A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL MODEL OF BORDERLINE PERSONALITY DISORDER (BPD): EXAMINING EVIDENCE FOR IMPAIRMENTS IN 'EXECUTIVE FUNCTION'

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Declaration

I declare that this thesis is my own account of my research and contains as its main content work which has not previously been submitted for a degree at any tertiary education institution.

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Abstract

Borderline Personality Disorder (BPD) is a serious psychiatric disorder characterised by turbulent interpersonal relationships, impaired self image, impulsivity, and a recurrent pattern of unstable affect which is usually evident by early adulthood. It has a community prevalence rate of two per cent, and approximately nine per cent of people diagnosed with BPD commit suicide. This suggests that BPD has one of the highest lethality rates of all psychiatric disorders. The course of the disorder shows a steady improvement over the course of early adulthood with the majority of cases remitting by middle age. This positive but incomplete long-term recovery is thought to be a naturalistic outcome that is independent of treatment effect.

The reported study sought to test selected components of a multidimensional developmental neuropsychological model of executive functioning in BPD. The model proposed that BPD is characterised by impairments to four neuropsychological executive functions. These include working memory, response inhibition, affective-attentional bias, and problem-solving. The model further proposed that impaired executive functioning in BPD occurs as a result of the failure of 'experience-dependent' maturation of orbitofrontal structures. These structures are closely associated with the development of the 'cognitive executive'.

The study incorporated a cross-sectional design to analyse data from a BPD group, a Depressed Control Group, and a Medical Control Group. The overall findings of the study returned limited support for the original hypotheses. There was no evidence of deficits in working memory, response-inhibition, or

problem-solving. In contrast, the BPD group returned some evidence of deficits in affective-attentional bias.

Therefore, the results suggest that executive functioning remains largely intact in BPD. This also suggests that people with BPD have the working memory resources necessary to facilitate abstract cognition, have the capacity to effectively plan and execute future-oriented acts, and are able to perform appropriate problem-solving functions. These problem-solving returns are also particularly significant because a number of the tasks utilised in the study are known to be associated with so-called 'frontal-executive' function. These unremarkable findings challenge the view that people with BPD might experience some form of subtle neurological impairment associated with frontal-lobe compromise.

The Stroop measure of affective-attentional bias provided the only supportive evidence for the proposed model, and these findings can be accounted for by at least two different explanations. The first suggests that BPD might be characterised by a hypervigilant attentional set. The specific cause of hypervigilance in BPD is unknown, but some candidate factors appear to be the often-reported abuse histories of borderlines, insecure attachment histories, and deficits in parental bonding. The second interpretation suggests that the Stroop findings reflect a form of 'response conflict' in which BPD participants experience difficulties overriding tasks that rely on the enunciation of automatic neural routines.

As a result of these findings, further research on the role of arousal, priming, hypervigilance, and response-conflict in BPD is required. It is likely that the Stroop findings reflect a basic, 'hard-wired' attentional mechanism that

consolidates by early adolescence at the latest. As a result, the Stroop findings have implications for both the prevention and treatment of BPD.

A number of prevention strategies could be developed to address the attentional issues identified in the present study. These include assisting children to more effectively regulate arousal and affect, and assisting parents to communicate affectively with children in order to enhance self-regulation. The treatment implications suggest that interventions directed at affective-attentional processes are required, and further suggest the need for new pharmacotherapies and psychological treatments to modify dysfunctional attentional process. Affective neuroscience will have an increasingly important role to play in the understanding of BPD, and the next quarter century is likely to witness exciting advances in understanding this most problematic of disorders.

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The capacity to show concern for another person whilst enduring one's own suffering is at the heart of the great faiths of the world. It also represents one aspect of the essence of our connection to each other as human beings. That Dr. Lloyd could show this attention to me under the most desperate of personal circumstances is testimony to her resilience, grace, and sense of self. It is this experience of humanness and care from the other that I believe is often lacking in the life experience of the person with BPD. To her, I dedicate this thesis.

It is not perfect, but it is finished.

TABLE OF CONTENTS

	PAGE
Abstract	iii
Acknowledgements	vi
Contents	x
List of Tables	xxi
List of Figures	xxvii
List of Appendices	xxviii

CONTENTS

SECTION I: THEORETICAL OVERVIEW

CHAPTER ONE: INTRODUCTION

		PAGE
1.1.	OVERVIEW	2
1.2.	HISTORICAL OVERVIEW	4
1.3.	AIMS OF THE PROJECT	17

CHAPTER TWO: BORDERLINE PERSONALITY DISORDER (BPD)

2.1.	OVERVIEW OF CHAPTER	22
2.2.	PSYCHOLOGICAL PERSPECTIVES ON BPD	22
2.2.1.	Psychoanalytic Perspectives	23
2.2.2.	BPD as an 'Affect Spectrum' Disorder	27
2.2.3.	BPD as an 'Impulse Spectrum Disorder'	31
2.2.4.	Disturbed Early Family Environment	41
2.2.5.	BPD as a 'Trauma Spectrum Disorder'	45
2.2.6.	Behavioural Perspectives	50
2.2.7.	BPD as a 'Neurobehavioural' or Psychobiological Disorder	57
2.3.	DIAGNOSTIC SYSTEMS IN BPD	68
2.3.1.	Kernberg's Borderline Personality Organization (BPO)	68
2.3.2.	Gunderson's BPD Criteria	71
2.3.3.	The International Classification of Diseases of the World Health Organisation (ICD)	72
2.3.4.	The Diagnostic and Statistical Manual of the American Psychiatric Association (DSM)	74
2.3.5.	Summary and Conclusions	83

PAGE

2.4. EPIDEMIOLOGY OF BPD 83

2.4.1. Prevalence of BPD 83

2.4.2. Incidence of BPD 93

2.4.3. Summary 94

2.5. THE COURSE OF BPD 95

2.5.1. Short-Term Outcome Studies of BPD 95

2.5.2. Long-Term Outcome Studies of BPD 106

2.6. THEORETICAL AND METHODOLOGICAL CRITIQUES OF BPD 112

2.6.1. Diagnostic Validity 112

2.6.2. Reliability 121

2.6.3. Assessment 122

2.6.4. Heterogeneity 126

2.7. SUMMARY AND CONCLUSIONS 131

CHAPTER THREE: A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL MODEL OF IMPAIRED EXECUTIVE FUNCTION IN BPD

3.1. OVERVIEW OF CHAPTER 134

3.2. FRONTAL LOBE FUNCTIONS AND 'FRONTAL' PATHOLOGY 137

3.2.1. Development of the Orbitofrontal Cortex 143

3.3. THE CONCEPT OF 'EXECUTIVE FUNCTION' (EF) 148

PAGE

3.4.	A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL THEORY OF EXECUTIVE DISORDER IN BPD	153
3.4.1.	A Multidimensional Risk Model for BPD	157
3.4.2.	Impairments to the 'Central Executive': Working Memory, Response Inhibition, Affective-Attentional Bias, and Problem-Solving	158
3.4.3.	Impaired Executive Function in BPD	160
3.5.	EXECUTIVE FUNCTION I: IMPAIRED WORKING MEMORY IN BPD	161
3.5.1.	Empirical Evidence for Impaired Working Memory in BPD	164
3.5.2.	Assessment of Working Memory in BPD	170
3.6.	EXECUTIVE FUNCTION II: IMPAIRED RESPONSE INHIBITION IN BPD	171
3.6.1.	Assessment of Response Inhibition in BPD: The Application of the 'Stop-Signal' Task	173
3.7.	EXECUTIVE FUNCTION III: IMPAIRED AFFECT REGULATION AND AFFECTIVE-ATTENTIONAL BIAS IN BPD	180
3.7.1.	Empirical Evidence for Impaired Affect Regulation in BPD	182
3.7.2.	Assessment of Affect Regulation in BPD: The Application of the 'Emotional Stroop' Method	193
3.7.3.	Assessment of Affective-Attentional Bias in BPD	198

PAGE

3.8. EXECUTIVE FUNCTION IV: IMPAIRED PROBLEM SOLVING IN BPD 199

3.8.1. Empirical Evidence for Impaired Problem-Solving in BPD 199

3.8.2. Assessment of Problem Solving in BPD 208

3.9. METHODOLOGICAL ISSUES ASSOCIATED WITH STUDIES EXAMINING EXECUTIVE FUNCTION IN BPD 209

3.9.1. Sampling Issues 210

3.9.2. Diagnostic Issues 219

3.9.3. Psychiatric Issues 224

3.9.4. Neurological/'Brain Impairment' Issues 229

3.10. HYPOTHESES AND DESIGN OF THE PROJECT 235

SECTION II: SELECTION OF DIAGNOSTIC INSTRUMENTS AND VALIDATION OF SCALE 'C' OF THE MCMI-III

CHAPTER FOUR: SELECTION OF DIAGNOSTIC INSTRUMENTS AND AN EXAMINATION OF THE VALIDITY OF SCALE 'C' (BORDERLINE) OF THE MCMI-III

4.1. OVERVIEW 238

4.2. DIAGNOSTIC INSTRUMENTS EMPLOYED IN THE PROJECT 238

4.3. CONFIRMATORY PSYCHOMETRIC RESEARCH ON THE MCMI-III 240

4.4. PARTICIPANTS 241

4.5. PROCEDURE 241

4.6. RESULTS 242

PAGE

4.7. SUMMARY AND CONCLUSIONS 244

SECTION III: DEVELOPMENT OF THE EMOTIONAL STROOP TASK AND DESCRIPTION OF THE STOP-SIGNAL PARADIGM

CHAPTER FIVE: METHOD: AFFECTIVE AND SEMANTIC REPRESENTATIONS IN BPD

5.1. OVERVIEW 247

5.1.1. Identification of Affect Categories for Inclusion in the Stroop Paradigm 248

5.2. DIAGNOSIS OF BPD 248

5.3. PARTICIPANTS 248

5.3.1. Situating the Sample 249

5.4. PROCEDURE 249

5.4.1. Screening of BPD Participants 250

5.5. RESEARCH INTERVIEWS 252

5.5.1. Interview Procedure 256

5.5.2. Data Transformation 257

5.5.3. Decision Rules for Inclusion of Affect Categories in the 'Emotional Stroop' Task 258

5.6. RESULTS 259

5.6.1. Theoretical and Clinical Implications of Informing Participants That They Meet Criterion for BPD 262

5.7. AFFECT CATEGORIES 266

5.7.1. Anger-Rage (Anger) 271

PAGE

5.7.2. Distress-Anguish (Sadness) 288

5.7.3. Shame-Humiliation (Shame) 293

5.7.4. Fear-Terror (Anxiety) 299

5.8. SUMMARY AND CONCLUSIONS 305

CHAPTER SIX: AFFECT CATEGORY JUDGEMENT TASK

6.1. OVERVIEW 306

6.2. DEVELOPMENT OF THE AFFECT CATEGORY JUDGEMENT TASK (ACJT) 307

6.3. PARTICIPANTS 310

6.4. PROCEDURE 311

6.5. RESULTS 312

CHAPTER SEVEN: CONSTRUCTION OF THE EMOTION WORD COLOUR-NAMING INTERFERENCE (EMOTIONAL STROOP) TASK AND DESCRIPTION OF THE STOP-SIGNAL PARADIGM

7.1. OVERVIEW 315

7.2. DEVELOPMENT OF THE STROOP TASK: EXPERIMENTAL HARDWARE 315

7.3. DEVELOPMENT OF THE STROOP TASK: EXPERIMENTAL SOFTWARE 316

7.4. PROCEDURE 320

7.5. DATA OUTPUT 324

7.6. STOP-SIGNAL PARADIGM 324

PAGE

7.6.1. Experimental Hardware 325

7.6.2. Experimental Software 325

7.6.3. Procedure 326

7.6.4. Data Output 328

SECTION IV: ASSESSMENT OF EXECUTIVE FUNCTION IN BPD

CHAPTER EIGHT: ASSESSMENT OF EXECUTIVE FUNCTION IN BPD

8.1. OVERVIEW 332

8.2. AIMS AND HYPOTHESES 332

8.3. PARTICIPANTS 334

8.3.1. Recruitment of BPD Participants 334

8.3.2. Recruitment of Depressed Control Participants 339

8.3.3. Recruitment of Medical Control Participants 340

8.4. PROCEDURE 342

8.4.1. Session One: Informed Consent, Screening, and Diagnostic Assessment 345

8.4.2. Session Two: Administration of the Emotional Stroop Task and the Stop-Signal Paradigm 349

8.4.3. Session Three: Administration of Screening Instruments, Working Memory and Problem-Solving Executive Tasks 350

8.5. RESULTS 352

8.5.1. Analysis of DIB-R and MCMI-III Data 353

8.5.2. Analyses of Hypotheses 374

PAGE

8.5.3. Analysis of Hypothesis One: Impaired Working Memory in BPD 377

8.5.4. Analysis of Hypothesis Two: Impaired Response Inhibition in BPD 379

8.5.5. Analysis of Hypothesis Three: Impaired Affective Attentional Bias in BPD 384

8.5.6. Analysis of Hypothesis Four: Impaired Problem Solving in BPD 392

8.5.7. Subsidiary Analyses 395

8.6. CONCLUSIONS 398

SECTION V: DISCUSSION

CHAPTER NINE: DISCUSSION

9.1. OVERVIEW 401

9.2. A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL THEORY OF EXECUTIVE DISORDER IN BPD 401

9.3. SUMMARY OF THE FINDINGS OF THE STUDIES 403

9.3.1. Study One: Validity Study of the MCMI-III 403

9.3.2. Study Two: Affective and Semantic Representations in BPD 404

9.3.3. Study Three: Affect Category Judgement Task 405

9.3.4. Study Four: Assessment of Executive Function in BPD 406

9.4. INTERPRETATION OF HYPOTHESES AND THE PRINCIPAL FINDINGS OF THE PROJECT 408

9.4.1. Impaired Working Memory in BPD 408

PAGE

9.4.2. Impaired Response Inhibition in BPD 408

9.4.3. Impaired Problem Solving In BPD 415

9.4.4. Impaired Affective Attentional Bias in BPD 415

9.4.5. Theoretical Accounts of Differences in Colour-Naming Response Latencies on the Stroop Task 427

9.4.6. A Modified Multidimensional Developmental Neuropsychological Model of Impaired Executive Function in BPD 434

9.5. CONCEPTUAL AND METHODOLOGICAL REVIEW OF THE PROJECT 436

9.5.1. The Categorical Approach to Studying BPD 436

9.5.2. General Methodological Issues Associated With the Project 438

9.5.3. Study One: Validity Study of the MCMI-III 439

9.5.4. Study Two: Affective and Semantic Representations in BPD 440

9.5.5. Study Three: Affect Category Judgement Task 443

9.5.6. Study Four: Assessment of Executive Function in BPD 446

9.6. IMPLICATIONS FOR FUTURE RESEARCH 466

9.6.1. Implications for Future Theoretical Research 467

9.6.2. Implications for Future Clinical Research 470

9.7. IMPLICATIONS FOR CLINICAL PRACTICE 473

9.7.1. Assessment 473

9.7.2. Information Provision and ‘Psychoeducation’ 474

9.7.3. Psychotherapy 476

PAGE

9.8. IMPLICATIONS FOR PUBLIC POLICY AND EARLY INTERVENTION TO REDUCE THE INCIDENCE OF BPD 485

9.9. CONCLUDING REMARKS 488

REFERENCES 491

LIST OF TABLES

PAGE

TABLE 1.1: HISTORICAL ORIGINS OF THE BPD DIAGNOSIS 14

TABLE 3.1: TESTS EMPLOYED IN THE STUDIES EXAMINING MEMORY IN BPD 166

TABLE 3.2: TESTS EMPLOYED IN THE STUDIES EXAMINING PROBLEM-SOLVING IN BPD 201

TABLE 3.3: SAMPLING/DESIGN ISSUES IN THE STUDIES EXAMINING EXECUTIVE FUNCTIONING IN BPD 211

TABLE 3.4: SAMPLE SIZES AND DEPENDENT VARIABLES TO CASE RATIOS FOR THE BPD EXECUTIVE FUNCTION STUDIES 218

TABLE 3.5: DIAGNOSTIC ISSUES IN STUDIES EXAMINING EXECUTIVE FUNCTIONING IN BPD 221

TABLE 3.6: PSYCHIATRIC ISSUES IN STUDIES EXAMINING EXECUTIVE FUNCTIONING IN BPD 225

TABLE 3.7: NEUROLOGICAL ISSUES IN STUDIES EXAMINING EXECUTIVE FUNCTIONING IN BPD 230

TABLE 4.1: DEMOGRAPHIC DATA FOR THE SAMPLE EXAMINING THE VALIDITY OF SCALE 'C' (BORDERLINE) OF THE MCMI-III 243

TABLE 4.2: CROSS-TABULATION ANALYSES FOR MCMI-III SCALE C BPD STATUS WITH DIB-R BPD STATUS 244

TABLE 5.1: TOMKINS AFFECT CATEGORIES 254

PAGE

TABLE 5.2: DEMOGRAPHIC DATA FOR THE BPD SAMPLE EXAMINING AFFECTIVE AND SEMANTIC REPRESENTATIONS IN BPD 260

TABLE 5.3: MEANS AND STANDARD DEVIATIONS FOR DIB-R SCALED SCORES, AND MCMI-III VALIDITY, CLINICAL PERSONALITY PATTERN, SEVERE PERSONALITY PATHOLOGY, AND CLINICAL SYNDROME SCALES FOR THE BPD SAMPLE TABLE 261

TABLE 6.1: AGE, GENDER, PROFESSIONAL DISCIPLINE, AND YEARS OF PROFESSIONAL EXPERIENCE FOR THE EXPERT JUDGE GROUP 311

TABLE 6.2: WORDS BY AFFECT CATEGORY ELIMINATED BY VALIDITY JUDGEMENT OF SPEECH PATHOLOGIST JUDGES 312

TABLE 6.3: AFFECT CATEGORY WORDS INCLUDED IN FINAL STROOP TASK 313

TABLE 6.4: WORD LENGTHS BY GROUP FOR WORDS IN STROOP TASK 314

TABLE 7.1: DATA OUTPUT PROVIDED BY THE STOP-SIGNAL PARADIGM 329

TABLE 8.1: SCREENING AND EXECUTIVE TASKS ADMINISTERED IN SESSION THREE 351

PAGE

TABLE 8.2: DEMOGRAPHIC DATA FOR THE IDD, PSYCHIATRY, AND NEWSPAPER BPD SUBGROUPS 354

TABLE 8.3: SAMPLE MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR DIB-R SCALED SCORES FOR BPD SUBGROUPS 357

TABLE 8.4: SAMPLE MEANS, STANDARD DEVIATIONS AND F STATISTICS FOR MCMI-III VALIDITY, CLINICAL PERSONALITY, AND SEVERE PERSONALITY PATHOLOGY SCALED SCORES 358

TABLE 8.5: SAMPLE MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR MCMI-III CLINICAL SYNDROMES SCALED SCORES FOR BPD SUBGROUPS 359

TABLE 8.6: DEMOGRAPHIC DATA FOR THE BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 366

TABLE 8.7: SAMPLE MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR DIB-R SCALED SCORES FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 369

PAGE

TABLE 8.8: SAMPLE MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR MCMI-III VALIDITY, CLINICAL PERSONALITY, AND SEVERE PERSONALITY PATHOLOGY SCALED SCORES FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 370

TABLE 8.9: SAMPLE MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR MCMI-III CLINICAL SYNDROME SCALED SCORES FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 371

TABLE 8.10: SAMPLE MEANS AND STANDARD DEVIATIONS FOR HYPOTHESISED CONFOUNDING VARIABLES FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 375

TABLE 8.11: SAMPLE MEANS, STANDARD DEVIATIONS AND F STATISTICS FOR WORKING MEMORY TASKS FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 378

PAGE

TABLE 8.12: STOP-SIGNAL PARADIGM MEAN REACTION TIME (MRT), STOP-SIGNAL RESPONSE TIMES (SSRT) AND NUMBER OF NON-RESPONSES AT 0, 100, 200, 300, 400, AND 500 MSECS DELAY MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 380

TABLE 8.13: HIGH BPD DIB-R IMPULSE SUBSCALE SUB-GROUP VS. LOW BPD DIB-R IMPULSE SUBSCALE SUB-GROUP ON MRT AND SSRT SCORES 382

TABLE 8.14: COMPARISON OF THE MEANS FOR MEAN REACTION TIME DATA (GO DATA) FOR THE CURRENT STUDY AND THE KUNERT ET AL. (2003) AND DINN ET AL. (2004) STUDIES 383

TABLE 8.15: MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR STROOP COLOUR-NAMING RESPONSE TIMES FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS AT SUPRALIMINAL (2000 MSEC), AND SUBLIMINAL (240 MSEC) PRESENTATION 387

PAGE

TABLE 8.16: STROOP INTERFERENCE SCORES MEANS, STANDARD DEVIATIONS, AND F STATISTICS FOR BPD, DEPRESSED AND MEDICAL CONTROLS FOR SUPRALIMINAL (2000 MSECS) AND SUBLIMINAL (240 MSEC) PRESENTATIONS 391

TABLE 8.17: SAMPLE MEANS, STANDARD DEVIATIONS AND F STATISTICS FOR PROBLEM SOLVING TASKS FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 393

TABLE 8.18: SAMPLE MEANS, STANDARD DEVIATIONS AND F STATISTICS FOR AUSTIN MAZE TRIALS ONE, FIVE, AND 10 FOR BPD, DEPRESSED, AND MEDICAL CONTROL GROUPS 394

TABLE 8.19: STUDY TWO AND STUDY FOUR DIB-R AND MCMI-III SCALED SCORE BPD ONEWAY ANOVA'S 396

LIST OF FIGURES

		PAGE
FIGURE 3.1:	MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL MODEL OF IMPAIRED EXECUTIVE FUNCTION IN BPD	156
FIGURE 3.2:	HYPOTHESISED IMPAIRMENTS TO THE CENTRAL EXECUTIVE IN BPD	159
FIGURE 3.3:	SEQUENCE OF STUDIES CONDUCTED IN THIS PROJECT	236
FIGURE 8.1:	RECRUITMENT PATHWAY FOR BPD CASES	338
FIGURE 8.2:	RECRUITMENT PATHWAY FOR DEPRESSED CONTROL CASES	340
FIGURE 8.3:	RECRUITMENT PATHWAY FOR MEDICAL CONTROL CASES	342
FIGURE 8.4:	ALGORITHM FOR ADMISSION OF PARTICIPANTS INTO STUDY FOUR	344
FIGURE 9.1:	MODIFIED MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL MODEL OF STROOP COLOUR-NAMING RESPONSE IN BPD	435

LIST OF APPENDICES

		PAGE
APPENDIX I:	ICD-10 CRITERIA FOR 'EMOTIONALLY UNSTABLE PERSONALITY DISORDER – BORDERLINE TYPE'	549
APPENDIX II:	DSM-IV AND DSM-IV-TR CRITERIA FOR BORDERLINE PERSONALITY DISORDER (BPD)	551
APPENDIX III:	DESCRIPTION OF MEASURES AND THE TESTS USED TO MEASURE HYPOTHESES	553
APPENDIX IV:	PERSONAL COMMUNICATION FROM MARY ZANARINI REGARDING THE DIB-R	567
APPENDIX V:	DIAGNOSTIC INTERVIEW FOR BORDERLINES – REVISED (DIB-R)	570
APPENDIX VI:	'OWNING' ONE'S PERSPECTIVE	589
APPENDIX VII:	DESCRIPTIONS OF EACH RESEARCH PARTICIPANT	595
APPENDIX VIII:	STUDY II INITIAL SCREENING INTERVIEWS	606
APPENDIX IX:	STUDY II INFORMATION SHEET	610
APPENDIX X:	STUDY II CONSENT FORM	615
APPENDIX XI:	SAMPLE ITEMS FROM THE AFFECT CATEGORY JUDGEMENT TASK	617

		PAGE
APPENDIX XII:	WORD LISTS FOR EACH AFFECT CATEGORY REVIEWED BY SPEECH PATHOLOGIST JUDGES	621
APPENDIX XIII:	NEWSPAPER ARTICLE IN SUNDAY TIMES 18 SEPTEMBER 1999	630
APPENDIX XIV:	TELEPHONE SCREENING INTERVIEW TO EXCLUDE POTENTIALLY CONFOUNDED CASES	633
APPENDIX XV:	TELEPHONE CONTACT TO RECRUIT BPD CASES THROUGH THE DEPARTMENT OF INFECTIOUS DISEASES	636
APPENDIX XVI:	TELEPHONE RECRUITMENT PROCEDURE FOR MEDICAL CONTROL PARTICIPANTS	639
APPENDIX XVII:	INFORMATION SHEET FOR STUDY FOUR	641
APPENDIX XVIII:	CONSENT FORM FOR STUDY FOUR	647
APPENDIX XIX:	SCREENING INSTRUMENT FOR STUDY FOUR	649
APPENDIX XX:	SUMMARY OF THE FINDINGS OF THE STUDY	653

SECTION I: THEORETICAL OVERVIEW

CHAPTER ONE: INTRODUCTION

1.1. OVERVIEW

Borderline Personality Disorder (BPD) is a serious psychiatric disorder characterised by turbulent interpersonal relationships, impaired self image, behavioural impulsivity, and a recurrent pattern of unstable affect which is usually evident by early adulthood (American Psychiatric Association, 1994, 2000). Although there is some evidence that BPD is detectable in childhood (Vela, Gottlieb, & Gottlieb, 1983), the majority of cases develop in adolescence (Bernstein, Cohen, Skodol, Bezirganian, & Brook, 1996) and persist into adulthood (Paris, 1999).

Prevalence rates for BPD vary widely. It is estimated to affect approximately two per cent of the general community (Paris, 1999), 11% of all psychiatric outpatients (Heard & Linehan, 1993), and up to 25% of all psychiatric inpatients (Baker, Silk, Westen, Nigg, & Lohr, 1992). BPD is also thought to be a disorder predominantly associated with young women (M. Swartz, Blazer, & Winfield, 1990). The course of the disorder shows a steady improvement over the course of early adulthood with some evidence suggesting that remission can occur in as little as six months (Gunderson et al., 2003), to five to seven years (Najavits & Gunderson, 1995). The bulk of the evidence however, suggests that cases diagnosed in adolescence or early adulthood remit by middle age, although approximately 20% of cases continue to meet BPD criteria in middle age and beyond (Stone, 1992). This relatively positive but incomplete long-term recovery is thought to be a naturalistic outcome that is independent of the effects of treatment (Paris, 1999, 2003a, 2003b). Whilst BPD

generally remits by middle age, many former BPD cases still meet criteria for other psychiatric disorders, most notably depression (McGlashan, 1986).

The clinical picture of BPD is also confused because there appears to be substantial co-morbidity with Axis I disorders (Widiger & Trull, 1993), and with other Axis II personality disorders (Paris, 1999, 2003a, 2003b; Zanarini et al., 1998). This often makes it difficult to determine the specific features associated with BPD in contrast to other Axis I or Axis II conditions.

BPD is also characterised by a number of key diagnostic features. These include unstable interpersonal relationships involving fluctuations between the extremes of idealization and devaluation of others, and the use of a variety of forms of self-defeating behaviour that often include dramatic efforts to avoid real or imagined abandonment. It is also characterised by 'identity disturbance', chronic feelings of emptiness and/or boredom, and recurrent suicidal threats and self-harm gestures. Most importantly, BPD is associated with 'affective instability' characterised by rapid fluctuations of mood including depression and anxiety, with co-occurring intense, inappropriate experiences of anger (American Psychiatric Association, 1994, 2000). It is estimated that up to nine per cent of people with BPD will commit suicide, suggesting that BPD has one of the highest lethality rates of all psychiatric disorders (Stone, 1992, 1999).

There is little consensus with regard to the causes of the disorder, and this represents a serious deficiency in the understanding of the condition (Paris, 1999, 2003a, 2003b). The available candidate factors include attachment disturbances (Barone, 2003; Patrick, Hobson, Castle, Howard, & Maughan, 1994), family environmental factors (Zanarini, 1997), biogenetic (Widiger & Trull, 1993), and neurobiological theories (Gunderson & Zanarini, 1989). In addition, there is

limited understanding of the psychological mechanisms that maintain the condition, and the number of empirically-supported psychological therapies available to manage the condition are limited to a handful of approaches with promising but as yet incomplete substantiation of their efficacy (Bateman & Fonagy, 1999, 2001, 2004; G. K. Brown, Newman, Charlesworth, Crits-Christoph, & Beck, 2004; Clarkin, Levy, Lenzenweger, & Kernberg, 2004; Clarkin, Yeomans, & Kernberg, 1999; Linehan, 1993; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991; Meares, Stevenson, & Comerford, 1999; Monsen, Odland, Faugli, Daae, & Eilertsen, 1995; Munroe-Blum & Marziali, 1995; Ryle, 2004; J. Stevenson & Meares, 1992, 1999).

1.2. HISTORICAL OVERVIEW

The origins of the diagnostic entity that is BPD probably extend back to the earliest known records of medical and psychiatric diagnosis (Millon & Davis, 1996). Greek scholars such as Aretaeus, Hippocrates, and Homer have all documented conditions characterised by impulsive anger, 'melancholia,' and manic states that follow an erratic course. Millon & Davis (1996) note that conditions characterised by impulsivity and mood lability disappeared from the medical literature during the course of the Mediaeval Period, but were re-reported in the 17th Century by Bonet who coined the term *'folie maniaco-melancholique'* to describe a syndrome consisting of impulsive and affectively labile symptoms (Millon, 1992). Subsequently, clinicians such as Schact and Herschel refined Bonet's observations and postulated that the affective instability followed a predictable and periodic pattern of elation and depression. At the time, the prevailing view suggested that a 'manic-depressive' fluctuation was the main feature of this pattern of unstable mood regulation.

Whilst 'Manic-Depressive' or Bipolar Affective Disorder (BPAD) (American Psychiatric Association, 1994, 2000) as it is now known has become a well documented clinical entity, Millon (1992) reports that eminent physicians of the time such as Baillarger and Jean-Pierre Falret reported that the 'manic-depressive' condition did not occur as frequently as had been assumed. The majority of cases displayed an erratic, inconsistent, and unpredictable course with an overlay of intense affective states that either co-occurred, or followed one another in rapid sequence. Baillarger and Falret also described a syndrome characterised by chronic depression with either attempted or completed acts of suicide. These cases often commenced with a prodroma punctuated by intermittent periods of irritability, rage, elation, and calm (Millon, 1992). Subsequently, physicians such as Hughes and Rosse applied terms such as 'borderland insanity' or 'borderline insanity' to describe a cohort whose mental state fluctuated between 'reason' on the one hand, and 'despair' on the other (Millon & Davis, 1996). Collectively, these early clinical descriptions appear to describe what is known today as the 'borderline syndrome' (Kernberg, 1975), and more recently as BPD.

During the same period, other clinicians such as Prichard described a condition which came to be identified as 'moral insanity.' Over the course of the 19th Century this condition became associated with antisocial personality. At that time, Kraepelin broadened the focus to include other severe forms of personality dysfunction (Akhtar, 1992).

Kraepelin regarded these conditions as also constituting 'borderline states'. These conditions were understood to lie between 'insanity' on the one hand, and the idiosyncrasies of normality on the other. Kraepelin identified three

forms of personality deviation of which one, characterised by an 'inability of will', appeared to resemble the modern borderline condition. These patients were characterised by an instability of self, self-centredness, and irritability. Despite this emergent trend, Kraepelin's later works collapsed earlier distinctions into a singular group termed the 'psychopathic personality'. As a result, the opportunity to develop a more sophisticated understanding of the severe personality disorders was temporarily lost (Akhtar, 1992).

Another impetus to reconsider BPD as a discrete diagnostic entity came from the work of Bleuler who had reformulated the understanding of psychotic states. Bleuler described two non-psychotic forms of the disorder known as 'simple' and 'latent' schizophrenia. This perspective originally considered BPD to be a variant of the schizophrenic spectrum disorders, and this had the effect of re-opening the area to further clinical investigation. Eventually the conditions originally described by Bleuler became subsumed under the Schizotypal Personality diagnosis (Akhtar, 1992).

Whilst the borderline diagnosis appears to have developed out of the convergence of a number of independent lines of conceptual refinement, the specific origins of the borderline concept first emerged in the context of an early psychoanalytic paradigm. This approach commenced with the works of A. Stern (1938) and Knight (1953), and continues to influence the area. The fundamental premise of this original psychoanalytic paradigm suggested that borderline conditions reflected varying levels of deficient intrapsychic organization (Millon, 1992). This viewpoint will be explored more fully in Section 2.2.1.

Although psychiatrists and physicians were studying a borderline-like condition prior to the 20th Century, it was not until the work of Kraepelin in the

early 20th Century that psychiatry experienced a revival of interest in the condition. Despite this, the earliest references to the borderline concept within descriptive psychiatry appear to have been reported by Zilboorg (1941) and Hoch & Polatin (1949).

Zilboorg (1941) described a condition known as 'ambulatory schizophrenia'. This category included antisocial individuals including psychopaths, 'perverts', murderers, as well as others who might otherwise be described as socially or psychologically 'impoverished'. The common characteristics of this group included shallow or deficient emotionality and impaired empathy, an inability to form and maintain relationships, and an incapacity to focus on maintaining a job or a life pursuit. These features were originally understood to be a schizophrenia variant rather than a frank personality disorder (Akhtar, 1992). Similarly, Hoch & Polatin (1949) described a condition known as 'pseudoneurotic schizophrenia'. The key features of this diagnosis included a so-called 'neurotic' adjustment which served to mask so-called core features of schizophrenia. These features included global anxiety, 'pansexuality', and a marked sensitivity to criticism with concomitant rageful outbursts.

Collectively, these early psychoanalytic and psychiatric reports do not appear to have stimulated significant research activity, and it was not until the publication of the seminal work of Grinker, Werble, & Drye (1968) that attention was redirected toward the study of borderline phenomena. This study appears to have been a stimulus to increase the research activity on BPD. Historically, it appears to be an important study and is briefly reported here.

Grinker et al. (1968) operationalised seven generic "ego-functions" in 51 borderline patients studied in an inpatient psychiatric unit. The seven ego-

functions included Relation to Reality; Regulation and Control of Drives; Object-Relations; Cognitive Functions; Defensive Functions; Autonomous Functions; and Synthetic Functions. Ratings of the ego-functions were made by nursing and allied health staff observing patients on the unit. Multivariate analysis revealed four clusters within the borderline grouping.

The first group was referred to as the 'psychotic border' group (Group I). They displayed clinically inappropriate and negative behaviours toward others, and were further defined as erratic, angry, and depressed. Two factors found within Group I accounted for their behaviour. The first factor involved negative behaviour directed towards the environment, and the second factor involved negative behaviour directed toward other persons.

The second group was referred to as the 'core borderline syndrome' group (Group II). Group II was characterised by a pervasive negative affect which was 'acted out' in a variety of ways. Two factors were found within Group II to account for their behaviour. The first factor involved negative behaviour directed toward others which often involved overt expressions of anger and/or depression. The second factor involved oppositional behaviour in relation to limit-setting and rule responsiveness, and this measure was inversely related to the level of depression the patient reported. Grinker et al. (1968) report that Group II participants were characterised by vacillating involvement with others, overt or acted-out expressions of anger, depression, and an absence of indications of consistent self-identity.

The third group was referred to as the adaptive, 'affectless', defended, 'as if" group (Group III). Group III was characterised by a combination of bland, adaptive behaviour with an absence of so-called negative behaviour, or positive

or negative affect. In addition, this group reported a poorly developed sense of self identity, and an incapacity to form or maintain relationships. Again, two factors characterised Group III patients. The first factor involved the accommodation to the demands of others. This finding suggested that these participants lacked a firm and stable sense of personal identity. The second factor also involved low levels of affective display, low spontaneity, and poor self-identity. Of all the groupings, Group III participants were the most likely to employ the defensive behaviours of withdrawal and intellectual isolation. Group III participants were characterised by adaptive and appropriate behaviour, complementary relationships, depleted affective response, and the use of the defences of withdrawal and intellectualisation.

The fourth group was referred to as the 'border with the neurosis' group (Group IV). Group IV was reported as a small group that was fundamentally different to Groups I, II, & III. The group was described as homogeneous, with characteristic "neurotic depressions." Two factors were found within Group IV to account for their behaviour. The first factor suggested that these patients were positive and co-operative in engaging in tasks in the ward setting, and were also able to form and maintain relationships with female patients. The second factor was associated with the development of effective relationships with staff and with male patients (in the case of female participants). Although the evidence suggested that they experienced anxiety and depression, the depression was not associated with anger or guilt. Group IV participants were characterised by a childlike 'clinging depression', anxiety, and a resemblance to 'neurotic characters.'

Grinker et al. (1968) summarise the findings of their study as follows:

1. The characteristic affect of the borderline was anger. This appeared to be the main affect that borderlines experience;
2. The borderline is characterised by a defect or poverty in affectional relationships. They were rarely capable of engaging in reciprocal relatedness;
3. The borderline is characterised by an absence of consistent self-identity;
4. The borderline is characterised by a form of depression involving a loneliness associated with the realisation of their inability to commit to involvement with others.

The study reported by Grinker et al. (1968) represents a watershed in the study of BPD, and formed the basis of a research tradition for studying borderline pathology which continues today. It is characterised by the transition from case-report methodologies to the empirical study of participants using a variety of operationalised measures. This approach includes studies which have sought to understand the temporal stability (Shea & Yen, 2003), long-term outcome (Paris, 1999, 2003a, 2003b; Paris & Zweig-Frank, 2001), co-morbidity (Paris, 1999), neuropsychological sequelae (O'Leary, 2000), and early family environment experiences (Gunderson & Zanarini, 1989) of BPD patients.

Historically, many of the developments in the understanding of BPD can be seen to have their origins in the efforts of Grinker et al. (1968). One aspect of this was the clarification of the relationship between BPD and schizophrenia on the one hand, and BPD and the affective disorders on the other.

In the mid 1970's, the attempt to understand BPD as a variant or subtype of schizophrenia (which had persisted from the work of Bleuler) was complemented by a renewed argument that borderline conditions shared a closer affinity to the affective spectrum disorders (Akiskal, 1981; D. F. Klein, 1975,

1977; Liebowitz & Klein, 1981). At this time, three reviews were published which had significant implications for the future course of the borderline diagnosis (Gunderson & Singer, 1975; Liebowitz, 1979; J. C. Perry & Klerman, 1978).

Gunderson & Singer's (1975) review listed six criteria considered diagnostic for 'BPD.' These included the presence of intense depressive or hostile affect, a history of impulsive behaviour, social adaptiveness, brief psychotic experiences, loose thought in unstructured situations, and interpersonal relationships that vacillated between superficiality and intense dependency. This review was also significant in that it appears to be the first use of the term 'Borderline Personality Disorder'. This foreshadowed its application as official nomenclature in the then forthcoming DSM-III (American Psychiatric Association, 1980). In addition, it appears to be the first attempt to codify diagnostic criteria for BPD. A detailed review of what has become known as the 'Gunderson Criteria' will be outlined in Section 2.3.2. This is significant also because the Gunderson criteria in part shape the diagnostic framework employed in this project.

J. C. Perry & Klerman (1978) analysed four sets of data and identified over 100 criteria pertaining to the borderline diagnosis. They concluded that there were a number of subtypes of BPD (a point first identified by Grinker et al. (1968) and subsequently by Andrulonis, Glueck, Stroebel, & Vogel (1982)), and argued that further research was required. Around the same time, Liebowitz (1979) identified four different usages for the diagnostic term 'borderline' which included a discrete behavioural syndrome, a schizophrenia variant, a cluster of atypical affective disorders, and a level of structural functioning underlying most

severe forms of character pathology. Liebowitz concluded that BPD was distinct from schizophrenia, but further research was necessary to delineate it from the affective disorders.

Akhtar (1992) argues that the delineation by Grinker et al. (1968) of a 'core borderline syndrome' and the development of the 'Gunderson Criteria' (Gunderson & Singer, 1975), provide the basis for discriminating borderlines from related conditions. This view was further supported by the work of Gunderson & Kolb (1978) and Spitzer, Endicott, & Gibbon (1979) who further delineated a set of borderline criterion.

The work of Spitzer et al. (1979) marks a turning point in the validation of the BPD diagnosis, as the findings of this study appear to directly form the basis of the DSM-III criterion introduced in 1980. Spitzer et al. (1979) sent a 17-item checklist based on the Gunderson & Kolb (1978) criteria to approximately 4,000 psychiatrists and requested them to judge the list's discriminating capacity. The responses indicated that the list would accurately discriminate borderline from non-borderline cases on approximately 90% of occasions.

In 1980, BPD was officially recognized when it was incorporated in the DSM-III (American Psychiatric Association, 1980). This development resulted in a significant increase in the volume of research conducted on the condition. Much of the research that emerged after the inclusion of BPD within the DSM focussed on addressing the issues of whether BPD is a 'subaffective' disorder, or whether BPD is an independent condition that coexists with affective disorders (Akhtar, 1992). Other areas of study have included the epidemiology of the disorder, the development of diagnostic methods and diagnostic instruments, and studies of the co-morbidity of the disorder. Table 1.1 provides a brief summary

of the significant events that shaped the development of the borderline construct. This includes the contribution of early diagnosticians ranging from the ancients such as Hippocrates and Aretaeus to 19th and early 20th Century contributors such as Schacht and Kraepelin. The table then identifies the importance of select descriptive psychiatrists such as Grinker et al. (1968), Gunderson & Singer (1975), Hoch & Polatin (1949), and Zilboorg (1941), and the contributions of the American Psychiatric Association (1980) in compiling the original formal diagnosis of BPD. Finally, the table also identifies parallel ideas developed by select psychoanalysts such as A. Stern (1938), Knight (1953), Kernberg (1967), Grotstein (1987), and Adler & Buie (1979), that also contributed to the development of the borderline concept.

Table 1.1: Historical Origins of the BPD Diagnosis

Hippocrates	
↓	
Aretaeus	
↓	
Schacht	
↓	
Kraepelin	
↔	
Descriptive Psychiatry	Psychoanalytic Perspective
↓	↓
	Stern (1938)
Zilboorg (1941)	
↓	↓
Hoch & Polatin (1949)	
	Knight (1953)
↓	↓
	Kernberg (1967)
Grinker et al. (1968)	
↓	
Gunderson & Singer (1975)	↓
↓	
Spitzer et al. (1979)	Adler & Buie (1979)
↓	↓
American Psychiatric Association (1980)	Grotstein (1987)

In summarising the conceptual development of the borderline diagnosis, Grotstein, Lang, & Solomon (1987) identify six frameworks employed to understand borderline conditions. They argue that the term 'borderline' has evolved to mean a level of personality functioning, a spectrum of related syndromes, a specific syndrome, a personality type, an attenuated form of a more severe condition, and a wastebasket category. This range of definitions highlights the difficulties inherent in the diagnosis, and foreshadows many of the diagnostic, theoretical and methodological issues that will be considered in more detail in Section 2.6. Despite this, Akhtar (1992) notes that whilst the early history of the borderline diagnosis associated the condition with schizophrenia and atypical affective disorders, it is now increasingly used to mean a specific personality disorder characterised by:

1. An unstable sense of self and/or identity disturbance;
2. A disturbed interpersonal life characterised by vacillating, intense relationships;
3. A superficial 'neurotic-like' picture associated transient psychotic episodes;
4. The experience of contradictory, intense affective states;
5. A sense of inner emptiness, and intolerance of aloneness;
6. Impulsivity;
7. Chronic rage and self-destructiveness;
8. Chaotic sexual life;
9. Inordinate sensitivity to rejection.

BPD appears to have become accepted as a legitimate diagnostic condition in relation to both the DSM (American Psychiatric Association, 1980, 1987, 1994, 2000) and also in the International Classification of Diseases (ICD)

system (A. W. Loranger, Janca, & Sartorius, 1997). This acceptance has resulted in a variety of different psychological studies which include the study of the cognitive-behavioural mechanisms involved in BPD (A. Beck, Freeman, & Associates, 1990; G. K. Brown et al., 2004; Linehan, 1993; Linehan et al., 1991; Young, 1990), a traumatogenic theory of BPD (Zanarini, 1997), and an integrationist perspective suggesting that BPD might be viewed as an 'impulse spectrum disorder' (Zanarini, 1993, 1997). Finally, a number of studies conducted since the early 1980's suggest that BPD is a 'neurobehavioural' disorder characterised by impaired neuropsychological executive functions (EF's). This is the thesis of the current research study, and the evidence for this perspective will be examined in detail in Chapter Three.

The proposal that impaired neuropsychological executive functions characterise BPD is further argued to provide the cognitive, behavioural, and affective basis for the clinical phenomenology of BPD. For example, one of the major systems for diagnosing BPD (DSM-IV-TR) (American Psychiatric Association, 2000) employs nine diagnostic criteria for diagnosing BPD. Of these, seven criteria imply the existence of impaired executive functions in BPD. These include efforts to avoid real or imagined abandonment (Criterion One), the alternating use of cognitive sets of idealization and devaluation (also known as 'splitting') (Criterion Two), identity disturbance (Criterion Three), impulsivity (Criterion Four), affective instability and inappropriate, intense anger (Criteria Six and Eight), and feelings of emptiness (Criterion Seven).

Impaired executive functions are argued to provide the cognitive basis for the 'classic' clinical signs of BPD: 'splitting' (the converting of mental representations of the world into 'all good' or 'all bad' properties), and

'projective identification' (imbuing objects with affective or motivational properties when there is either limited or no evidence to support such a view, or alternatively when the evidence specifically does not support this view) (Grotstein, 1987). In addition, one of the usual diagnostic phenomena regarded as pathognomonic for BPD - a sense of internal emptiness - can also be understood as reflecting impairment to the cognitive executive. In this sense, it is possible to reconfigure much of what has traditionally been regarded as the clinical phenomenology of BPD as the outcome of impaired executive functions.

1.3. AIMS OF THE PROJECT

The aim of the project is to examine selected components of a multidimensional developmental neuropsychobiological model of BPD functioning. The proposed model argues that the clinical phenomenology of BPD can be understood as the outcome of impairment to four neuropsychological executive functions. The specific executive functions include working memory, response inhibition, affective-attentional bias, and problem-solving. The model further argues that impaired executive functioning in BPD develops as a result of the failure of 'experience-dependent' maturation of orbitofrontal structures which are closely associated with the development of the cognitive executive (Cozolino, 2002; Grigsby & Stevens, 2000; Joseph, 1996; Schore, 1994, 2003a, 2003b). This viewpoint further suggests that a number of independent risk factors for BPD - including genetic, neurobiological, early family relations, parental bonding, and attachment - affect the development of orbitofrontal structures. It is speculated that these risk factors are central to the development both of BPD and also the executive disorders hypothesised to be implicated in BPD. In particular, an impaired 'attachment narrative' is thought to affect the development of a

sense of self in the nascent borderline (Patrick et al., 1994), and also compromises the development of neurological (Lyoo, Han, & Cho, 1998; Soloff, Meltzer, Greer, Constantine, & Kelly, 2000), and neuropsychological functioning in BPD (Bazanis et al., 2002; Dinn et al., 2004; Judd & Ruff, 1993; Mandes & Kellin, 1993; O'Leary, Brouwers, Gardner, & Cowdry, 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway, Gansler, White, & Bachman, 1993).

In order to evaluate selected aspects of the proposed model, the thesis is divided into five sections. The remainder of Section One describes the theoretical basis for the study, and consists of Chapters Two and Three. Chapter Two selectively reviews the relevant clinical and scientific literature on BPD. This includes a review of selected psychological perspectives on BPD, a description of the systems used to diagnose BPD, examination of the prevalence, incidence, and course of BPD, and a selective review of the theoretical and methodological controversies associated with BPD. Chapter Three outlines a multidimensional developmental neuropsychological model that proposes that four key executive functions (behavioural inhibition, working memory, affect regulation, and problem-solving) are impaired in BPD. The model outlines the developmental basis for BPD including the speculated psychobiological basis for the disorder.

Section Two reports on the selection of the diagnostic instruments employed throughout the project, and then describes a validity study conducted on the self-report instrument employed to make the preliminary diagnosis of BPD in each of the studies in the project. Chapter Four describes the diagnostic instruments employed throughout the project, and reports on a study designed to assess the construct validity of Scale C (Borderline) of the Millon Clinical

Multiaxial Inventory (3rd Edition) (MCMI-III) (Millon, Millon, & Davis, 1994). The MCMI-III was the first of the two diagnostic instruments employed in the studies. The reason for conducting this study arose because the MCMI-III was employed in this project as an 'instrument of first detection' for BPD, and as a 'gateway' instrument for further diagnostic assessment. For these reasons, it was considered necessary to ensure that the MCMI-III was a valid instrument for diagnosing BPD. The findings of the validity study supported the use of the MCMI-III as an 'instrument of first detection' for diagnosing BPD in the current project.

Section Three describes the development of the 'Emotional Stroop' Task, an instrument developed to examine whether BPD participants are attentionally biased to affectively valenced stimuli. Chapter Five (Affective and Semantic Representations in BPD), describes the interview methodology utilised to elicit categories of affective experience that were used to develop the 'Emotional Stroop' task. The chapter also reports the results of the study, and summarises the relevant affect (emotion) categories reported by BPD participants. Chapter Six reports on the development of the 'Affect Category Judgement Task' which was designed to select specific, affectively valenced words that represented the specific categories of affect identified in Chapter Five. The selected words were then included in the Stroop Task. Chapter Seven describes the hardware and procedural specifications of the Emotional Stroop task, and describes the procedural specifications of the Stop-Signal Paradigm.

Section Four reports the study designed to examine selected aspects of the multidimensional developmental neuropsychological model of BPD outlined

in Chapter Three. Chapter Eight describes the methodology and reports the results examining executive functioning in BPD.

Section Five argues for the significance of the data in understanding the theoretical, developmental, and clinical features of BPD. Chapter Nine examines the significance of the findings in terms of what is currently known about BPD, and identifies a number of implications for further research. The clinical implications of the findings of the study for the management and treatment of BPD are also briefly considered. A number of specific recommendations for the assessment and management of BPD are considered.

Finally, the data provide some suggestive directions for public policy. The first implication of the study is the need for a community-level education programme directed toward educating both professionals and the lay public about BPD. A second implication of the project calls for increased interventions to improve the quality of relationships between parents and young children. Early intervention services to date have emphasised the role of cognitive development in children. The findings of this study call for increased attention to be devoted to assisting parenting practices and for assistance to improve the quality of attachment between infants and parents. A related implication of the findings of the project calls for the development of specific educative programmes for children and adolescents in order to assist them to develop more effective methods for regulating affect. The thesis concludes by arguing that the future course of research into BPD should be directed toward increasing community awareness of BPD, developing programmes to reduce the incidence of BPD, and developing more effective and integrated treatment programmes to minimise the adverse effects of the disorder. Specific recommendations are offered for

additional clinical interventions to treat BPD. The thesis concludes by emphasising the importance of affective neuroscience for understanding the nature of BPD.

CHAPTER TWO: BORDERLINE PERSONALITY DISORDER (BPD)

2.1. OVERVIEW OF CHAPTER

This chapter selectively reviews the psychological literature and provides a conceptual framework for the major psychological perspectives on BPD. The review is organised into a number of sections. First, the major contemporary psychological perspectives on BPD will be reviewed. This will include psychoanalytic, affect-spectrum disorder, impulse-spectrum disorder, family environment, trauma, behavioural, and neurobehavioural perspectives on BPD. Second, the chapter reviews the four major diagnostic systems used in BPD. Third, a selective review of the epidemiological features of BPD is undertaken which includes an examination of the prevalence, incidence, and gender-prevalence of the disorder. Fourth, the natural history of BPD is reviewed by selectively examining the short and long-term outcome studies of BPD.

Finally, the chapter concludes by examining a number of theoretical and methodological issues that characterise BPD. These include problems associated with diagnostic validity, reliability, assessment, and heterogeneity of BPD. The review demonstrates that although difficulties remain in relation to the diagnosis of the condition, a consensus exists with regard to the salient features of the disorder. The chapter concludes by acknowledging that BPD appears to have consolidated itself as a legitimate psychiatric diagnosis, and provides the basis for describing a multidimensional developmental neuropsychological model of impaired executive function outlined in Chapter Three.

2.2. PSYCHOLOGICAL PERSPECTIVES ON BPD

There is an enormous psychological literature on BPD that includes psychoanalytic papers, case-report methodologies, descriptive essays, and

quantitative research studies (Akhtar, 1992). This includes an examination of the genetics, neuropsychiatry, and psychopharmacology of the disorder (Paris, 1999, 2003a, 2003b; Stone, 1993; van Reekum, Links, & Boiago, 1993). In addition, the psychodynamics (Grotstein, 1986, 1987), and developmental features of the disorder have been reported in detail (Clarkin & Kernberg, 1993), as have a number of contributions pertaining to the psychotherapy of BPD (Bateman & Fonagy, 2001, 2004; Clarkin et al., 2004; Clarkin et al., 1999; Karterud et al., 1992; Linehan, 1993; Linehan et al., 1991; Meares, Stevenson, & Comerford, 1999; Monsen et al., 1995; Munroe-Blum & Marziali, 1995; J. Stevenson & Meares, 1992, 1999). A comprehensive review of these literatures is beyond the scope of this thesis, and this chapter selectively reviews some of the salient psychological perspectives on BPD. For the sake of parsimony, selected psychoanalytic, affective, impulse, trauma, behavioural, and neurobehavioural perspectives on BPD will be considered.

2.2.1. Psychoanalytic Perspectives

Psychoanalytic theorists were the first to use the term ‘borderline’ with any regularity. The first reference to the term ‘borderline’ is attributed to A. Stern (1938) who analysed a number of patients who were thought to occupy a ‘borderline’ area between ‘neurosis’ and ‘psychosis’ who also did not respond satisfactorily to conventional psychoanalytic treatment.

In psychoanalytic theory, the use of the term ‘borderline’ came to signify three interrelated meanings (Gunderson, 1994). First, it described a quality or level of personality functioning that implied a more severe level of dysfunction than a ‘neurotic’ personality constellation on the one hand, but a less severe level of dysfunction than a ‘psychotic’ personality constellation on the other.

According to this view, the borderline suffered from a deficient personality structure. Second, the term 'borderline' was also used to distinguish frank cases of schizophrenia from less distinguishable variants. Third, the term 'borderline' appears to be employed to describe a specific personality disorder characterised by a pattern of disruptive affective states which were cyclical or intermittent in nature. Each of these perspectives is briefly reviewed.

The 'Structural Deficiency' Model

The structural deficiency model of borderline pathology suggests that the fundamental difficulty in BPD involves a deficit in self-cohesion or resilience. This view was first proposed by Knight (1953) who conceptualised borderline phenomena as involving 'structural deficiencies' which included 'ego weakness' and intermittent psychotic breaks with reality. Modell (1963) also noted a wide variety of 'symptom complexes', including depression, addictions, perversions, or eccentric and/or withdrawn behaviour, as characteristic of the borderline.

The structural deficiency model of borderline pathology is best represented through the work of Kernberg and his model of 'Borderline Personality Organisation' (BPO) (Clarkin et al., 2004; Clarkin et al., 1999; Kernberg, 1967, 1975, 1984, 1992; Yeomans, Clarkin, & Kernberg, 2002). His model has developed out of an integration of ego-analytic and object-relational thinking and the origins of this approach can be traced back to the works of A. Stern (1938), Knight (1953), and M. Klein (1957). Kernberg has developed a complex, multilevel model of borderline functioning in which the syndrome of identity diffusion represents the central feature of the condition. This theory has resulted in the development of an operationalised approach to treatment known as 'Transference Focussed Psychotherapy' (TFP) (Clarkin et al., 2004; Clarkin et

al., 1999; Yeomans et al., 2002). This 'structural deficiency' approach to understanding the psychodynamics of borderline conditions remains one of the major psychoanalytic perspectives for understanding BPD.

The Borderline as a 'Schizophrenia Variant'

This view developed as a result of the observation that many of the psychological mechanisms operating in BPD were also noted in schizophrenic patients (Gunderson, 1994). Terms such as 'ambulatory schizophrenia' (Zilboorg, 1941), and 'pseudoneurotic schizophrenia' (Hoch & Polatin, 1949), were used to imply a connection between borderline phenomena and psychotic (schizophrenic) processes. This tradition, whilst influential within psychoanalytic circles, was also an important perspective in early psychiatric approaches to understanding borderline pathology. Three panel discussions sponsored by the American Psychoanalytic Association in 1954, 1955, and 1959 explored the theme that the borderline construct might be a variant of schizophrenia (Akhtar, 1992), and contributed to the continued influence of this perspective.

This view was summarized by Frosch (1964; 1970), who argued that borderline cases are variants of 'psychotic' characters. The psychotic character was viewed as a stable structure that employed predictable modes of adaptation in response to stress. Frosch (1970) argued for a vulnerability model of borderline functioning in which the signs of the 'psychotic character' were reflected in symptoms such as decompensation, regressive behaviour, delusional states, and flawed reality testing under conditions of environmental challenge. This perspective appears to enjoy less influence in psychoanalytic circles, but has been influential in the development of the DSM-III, DSM-III-R, and DSM-IV Schizotypal Personality Disorder formulation (Akhtar, 1992).

The Borderline as a Specific Personality Disorder

This viewpoint proposed that the borderline represented a specific personality disorder that was characterised by 'structural vulnerability'. Whilst there are similarities with the structural deficiency perspective outlined above, this viewpoint argues that an increase in perceived stressful life events can provoke an array of compensatory actions and psychological features including self injuriousness, affect dysregulation, 'primary process' thinking, and the use of defences such as splitting and projective identification.. The origins of this 'diathesis-stress' vulnerability model is considered to be caused by a variety of developmental issues including incomplete separation-individuation from mother (Masterson, 1972, 1976), an incapacity to keep 'in mind' a sustaining memory or 'object representation' of mother (Adler & Buie, 1979), or an incapacity to engage in satisfactory 'self-soothing' in the context of the occurrence of external challenges and demands (D. N. Stern, 1985).

In the post World War II period, a number of psychoanalysts further elaborated this version of the borderline concept. Schmideberg (1959) described borderline patients as insightless and unempathic, incapable of tolerating routine, and chaotic in their organisation and lifestyle. Schmideberg is famous for her description of borderlines as 'stably unstable'. Wolberg (1952) also reported on a cyclical borderline process which was termed a 'vicious circle.' This process was also reported to occur in 'at risk' borderline children. It commences as ambivalence within the child which manifests itself as a wish to obey and love the parent on the one hand, and a defiance of the parent on the other. Wolberg argues that this results in an experience of anxiety and depression which in turn results in reassurance seeking, hypersensitivity to rejection, and an emergent

participative experience of failure, aloneness, and emptiness. This in turn 'fuels' projection and acting out against others who are perceived as responsible for the rejection, or alternately, to employ various forms of self injurious acts. Wolberg argues that the resultant guilt and shame fuel further episodes of anxiety and depression which in turn provoke a new episode of the vicious circle.

Conclusions

Whilst recent psychoanalytic integrations have begun to emphasise the role of self and affect dysregulation in BPD (Adler & Buie, 1979; Bollas, 1996; Grotstein, 1986, 1987, 1990, 1991; Volkan, 1976, 1987) and also defective 'executive function' (Grotstein, 1987; Searles, 1969, 1979, 1986), the dominant psychoanalytic perspectives regard borderline conditions to be associated with 'intermediate' levels of personality functioning in which the person operates between psychotic and so-called 'neurotic' states of functioning. Alternately, the borderline is seen in some quarters to be a stable variant of psychotically organized personality functioning (Frosch, 1964, 1970), and finally, as a vulnerable personality structure which is characterised by affective instability arising out of precipitative external events. Psychoanalytic contributions to the study of borderline conditions continue, but the literature appears to be of limited accessibility and is not integrated with other theoretical, therapeutic, and empirical literatures on BPD.

2.2.2. BPD as an 'Affect Spectrum' Disorder

Early formulations of BPD initially suggested that it was associated with schizophrenia (Akhtar, 1992). As the evidence mounted that there was no link between BPD and schizophrenia, other researchers suggested that BPD might share a closer affinity to the 'affective spectrum' disorders (Akiskal, 1981; D. F.

Klein, 1975, 1977; Liebowitz & Klein, 1981). This formulation suggested that BPD might represent one constellation of disturbances having common features with mood disorders such as depression, but also other affective disorders including Bipolar Affective Disorder (BPAD) (Akiskal, 1981). Two factors appear to account for this perspective. First, there appears to be a high degree of co-morbidity between the presence of mood disorders on the one hand, and BPD on the other (Jonas & Pope, 1992). Second, organic psychiatry has explored the interface between biological markers, drug therapy, and BPD over a number of years. The findings have yielded mixed results, but it appears that there is sufficient evidence to warrant consideration of a mood disorder-BPD interface as a valid perspective for understanding BPD (Gold & Silk, 1993).

The view that BPD might be an affective spectrum disorder originally emerged in the mid 1970's when D. F. Klein (1975; 1977) suggested that a subgroup of BPD patients with 'hysteroid dysphoria' were amenable to antidepressant therapy using monoamine oxidase (MAO) inhibitors. Klein speculated that these medications attenuated affective arousal, and had the secondary effect of reducing self-destructive, manipulative, and provocative behaviour. Klein regarded the fundamental difficulty of this group of patients as one of affective instability. Klein further speculated that the affective dysregulation in BPD might be associated with a poorly mediated releasing system of endogenous amphetamine-like substances, and that this regulatory problem could develop as a result of genetic factors, or as an 'acquired defect'.

In support of this view, Akiskal (1981) completed a study of 100 consecutively recruited DSM-III BPD patients over a two year period from a general clinic population. Akiskal noted a significant degree of co-morbid

pathology in the presentation of this borderline cohort that included substance abuse disorders, Schizotypal Personality Disorder, and various depressive-spectrum disorders. Akiskal argued that the reason for the high rates of co-morbidity in this population was that apart from the likelihood that a minority of the cohort experience 'primary characterological pathology', the majority of cases represented atypical, chronic and complicated forms of affective disorder with secondary personality dysfunction. In addition, Akiskal argued that the affective illness in BPD is usually masked, and more importantly, has an 'intermittent-chronic' course. In this regard, Akiskal links BPD with cyclical Axis I affective disorders such as cyclothymia, 'bipolar II' disorder, mixed bipolar disorder, and dysthymia which also display a life-long-intermittent course. BPD is also argued by Akiskal to be similar to, and therefore probably emanating out of, the same causal pathway as the affective disorders. Akiskal argued that BPD is a sub-syndromal form of affective disorder characterised by an intermittent life-long course which does not result in discrete episodes of affective illness. He argued that it is the intermittent nature of their affective illness that creates the impression of a personality disorder.

Akiskal's (1981) approach has a certain appeal, but ignores the crucial evidence of illness course (which in fairness to Akiskal was unavailable at the time of his paper). Akiskals argument in part turns on the proposition that 'subaffective' disorders have a lifetime course. Section 2.5 reviews the course of BPD, and the available long-term studies of the course of the disorder clearly suggest that of all of the personality disorders, BPD is the one most likely to remit in the long-term (Paris, 2003a, 2003b). This view is at variance with Akiskal (1981), and when combined with subsequent reviews (Gold & Silk,

1993), suggests that the link between BPD and affective illness is less direct than Akiskal (1981) might suggest.

In a review of the literature exploring the BPD – affective illness interface, Gold & Silk (1993) reviewed the evidence from the biological marker studies as well as the pharmacotherapy literature. They conclude that the evidence for a biologically based mood disorder account of BPD is mixed at best, and that many BPD patients differ significantly from those with affective disorder in terms of relevant neurophysiological factors. They emphasise however, that there is a subgroup of BPD's who share a common biological substrate with mood disordered patients. This suggests that there is a subtype of BPD might in fact be a variant of affective disorder. This interpretation of the data is also consistent with independent reviews (Gunderson, 1994; Korzekwa, Links, & Steiner, 1993) that note that biological marker studies generally do not support the view that BPD is related to the affective spectrum disorders.

Gold & Silk (1993) also note that the biological research on the relationship between mood disorder and BPD is methodologically compromised because many of the biological markers that have been studied (e.g., the dexamethosone suppression test) have comparatively poor sensitivity and specificity thresholds. In addition, Gold & Silk also argue that many of these studies fail to stratify depressed groups into different subgroups, and assume that the effects of medication act in a similar manner thus implying that the same underlying pharmacokinetic disturbance operates in both mood disorder and BPD. Gold and Silk further argue that claims suggesting that a similar pathophysiology underpin BPD and mood disorders must be treated cautiously, and the current status calls for prudence in assuming that the same causal

mechanisms underpin both disorders. Despite this, there remains the view in some quarters that BPD represents a variant of affective disorder. This view tends to be held more strongly within the more biologically-oriented psychiatric disciplines. The evidence for such a view however, appears to be equivocal.

2.2.3. BPD as an 'Impulse Spectrum Disorder'

A third perspective suggests that BPD might be viewed as an 'impulse spectrum disorder' (Zanarini, 1993). Millon & Davis (1996) note descriptions extending as far back as the Mediaeval Period of a borderline-like condition in which impulsivity was a major diagnostic feature. Similarly, Akhtar's (1992) historical review of the development of the BPD construct found that impulsivity was one of the major hallmarks of the condition. The suggestion that BPD is characterised in part as a disorder of impulsivity is also supported by all of the major diagnostic systems (American Psychiatric Association, 1980, 1987, 1994, 2000; Gunderson, Kolb, & Austin, 1981; Kernberg, 1984; A. W. Loranger et al., 1997; Zanarini, Gunderson, Frankenburg, & Chauncey, 1989).

The available data supporting the view that BPD is a form of 'impulse spectrum disorder' links a diverse range of studies including developmental research suggesting that impulsivity is a key defining feature of childhood BPD (Vela et al., 1983), that a link exists between attention deficit hyper-active disorder (ADHD) in childhood and the development of BPD in adulthood (Elia, Stoff, & Coccaro, 1992), neuropsychological evidence indicating pre-frontal planning and executive disorders in BPD (Andrulonis et al., 1982; Andrulonis et al., 1980; Bazanis et al., 2002; Gardner, Lucas, & Cowdry, 1987; Kimble, Oepen, Weinberg, Williams, & Zanarini, 1997; van Reekum, Conway et al., 1993), family studies linking BPD with Substance Abuse Disorders and

Antisocial Personality Disorder (ASPD) (Zanarini, 1993), and research that has attempted to assess operationalised aspects of impulsivity in BPD (Bazanis et al., 2002; Dahl, 1990; Dinn et al., 2004; Dougherty, Bjork, Huckabee, Moeller, & Swan, 1999; Hochhausen, Lorenz, & Newman, 2002; Kunert, Druecke, Sass, & Herpertz, 2003; Links, Heselgrave, & van Reekum, 1999). This literature is briefly reviewed.

Developmental Research Suggesting Impulsivity is Linked to BPD

There are two converging lines of evidence suggesting that impulsivity is linked to BPD. First, there are a number of studies of childhood BPD suggesting that impulsivity is a key diagnostic feature of the disorder (Biederman, Newcorn, & Sprich, 1991; Vela et al., 1983). Second, there is a developing literature which links impulsive/attentional disorders in childhood such as Attention Deficit Hyperactive Disorder (ADHD) with the development of BPD in both childhood and adulthood (Andrulonis et al., 1982; Elia et al., 1992).

Vela et al. (1983) identified six symptom clusters associated with the diagnosis of BPD in childhood. These included disturbed interpersonal relationships, impaired reality testing, anxiety, 'neurotic-like' symptoms, uneven or distorted development, and impulsive behaviour. Biederman et al. (1991) conducted a review examining evidence for co-morbidity of ADHD with other disorders. The review found evidence for the presence of ADHD in childhood and the presence of BPD in adulthood.

Andrulonis (1991) argues that there are aetiological differences in the development of BPD in men and women. Andrulonis claims that BPD in women is likely to be associated with affect spectrum disorders, whereas for men it is more likely to be associated with a history of developmental hyperactivity,

attentional difficulties, learning problems, poor impulse control, and conduct disorder. He argues that men with BPD are often over-represented within both the special education and juvenile justice system. In addition, borderline men not only meet the psychodynamic criterion for BPD, but also require specific medical and behavioural interventions to address the attentional, learning and impulse control difficulties that characterise BPD in men.

Cohen, Shaywitz, Young, & Shaywitz (1982) argue that there is a convergence between the diagnoses of ADHD and BPD. They argue that ADHD is of relevance to BPD for two reasons. First, it appears there might be similar neurochemical mechanisms mediating the development of ADHD and BPD, and that understanding the biological bases of inattention and impulsivity have implications for understanding facets of childhood borderline disorders, and by implication, adult BPD. Second, Cohen et al. argue that the psychobiological mechanisms underpinning ADHD are often observed in BPD suggesting that similar mechanisms might underpin the development of both disorders. They argue that both ADHD children and children with BPD experience difficulties in the modulation of arousal, focussing attention, and regulating affect.

Neuropsychological Evidence of Executive or 'Frontal' Deficits in BPD

There is also evidence from the neuropsychological literature suggesting that there are executive deficits in BPD (Bazanis et al., 2002; Burgess, 1990; Dinn et al., 2004; Judd & Ruff, 1993; O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993). The existence of executive/frontal disorders in BPD is thought to explain the disorders of impulsivity also observed in BPD (Andrulonis et al., 1982). This literature will not be reviewed here as it is further considered in Section 2.2.7 and

in great detail in Chapter Three. However, the relationship between frontal impairment and impulsivity is a well established phenomenon (Lezak, 1995), and it is hypothesised that this mechanism might also occur in BPD.

Family Studies Linking BPD with Substance Abuse and Antisocial Personality Disorder (ASPD)

Zanarini (1993) has proposed that BPD is a disorder characterised by a 'propensity to action'. In this regard, Zanarini argues that BPD is associated with a range of other so-called impulsive disorders which include substance use disorders, Antisocial Personality Disorder (ASPD), and to a lesser extent, eating disorders. According to this view, BPD is a specific form of personality disorder in which impulsivity is a central feature. Zanarini reviewed the literature on the association between BPD and other nominated disorders of impulse and suggests that there is a link between BPD and other impulse-related disorders. She found substantial evidence for a link between BPD and the presence of substance-use disorders including alcohol-use disorders and Antisocial Personality Disorder (ASPD).

Zanarini (1993) argues that this provides suggestive evidence for a relationship between BPD and impulse spectrum disorders. She speculates that there are a number of causal pathways for BPD suggesting either a heritable-impulsive component to BPD, or alternatively that impulsive behaviour operates as a self-soothing or self-regulatory mechanism in order to manage intense and painful affects characteristic of BPD.

Zanarini's (1993) proposal provokes some important questions. She proposes that BPD might be seen as a form of impulse-spectrum disorder occurring in the context of a broader affect regulatory disorder. Zanarini appears

to be focussing upon the impulsive features of some aspects of borderline phenomenology, and reifying these phenomena to the status of a fully developed clinical theory. In this sense, Zanarini appears to be drawing a distinction between a view of BPD as an impulse spectrum disorder on the one hand, and an affective dysregulation disorder on the other. This is an important theoretical distinction, and is deserving of further research attention. This issue will be considered further in Chapter Nine.

Studies That Have Attempted to Examine Operationalised Aspects of Impulsivity in BPD

In contrast to the previously cited studies that have examined a link between impulse disorders and BPD, the direct evidence linking BPD to impulse spectrum disorders is limited to a relatively small number of studies (Bazanis et al., 2002; Dahl, 1990; Dinn et al., 2004; Dougherty et al., 1999; Hochhausen et al., 2002; Hurt et al., 1990; Kunert et al., 2003; Leyton et al., 2001; Links et al., 1999).

Dahl (1990) studied 62 consecutively admitted inpatients with diagnoses of BPD, Schizotypal Personality Disorder, or a combination of both diagnoses. Dahl prefers the use of the term 'severe personality disorder' over the term 'borderline' (although most of the cohort met BPD criteria). Using a series of questionnaires, Dahl identified six criteria that delimit the 'core borderline syndrome'. These include identity disturbance, affective disturbance, impaired interpersonal relations, impaired social functioning, cognitive/perceptual disturbance, and impulse-action disturbance. These findings have some similarity to similar work by Hurt et al. (1990) who examined decision-making rules for diagnosing BPD. Their analysis suggests that there are three core dimensions to

BPD. These are: Identity Disturbance (ID), Affective Disturbance (AFF), and Impulse Disturbance (IMP). Hurt et al. argue that the presence of AFF and IMP markers are effective in detecting the presence of BPD.

Links et al. (1999) reported on a prospective, long-term follow-up study designed to address whether impulsivity in BPD is stable over a seven year follow-up period. They also sought to examine whether it is possible to predict remission versus persistence rates for BPD, and to determine if a measure of impulsivity is the most effective predictor of BPD at follow-up. 88 of an original sample of 130 participants who met Gunderson BPD criteria were followed up two and seven years post index admission. Scores on the Impulse Action Subscale of the DIB-R collected at index admission were used as a predictor variable at follow-up. At seven year follow-up, the original Impulse Action Subscale Score was correlated with the seven year follow-up score ($r=0.53$), and the Impulse Action Subscale Score best predicted borderline status at seven year follow-up. This finding provides some evidence suggesting that impulsivity is predictive of BPD, and is stable over time.

Three studies have employed various decision-making tasks to examine impulsivity in BPD (Bazanis et al., 2002; Dougherty et al., 1999; Hochhausen et al., 2002). These tasks have variously employed passive avoidance (Hochhausen et al., 2002), or 'delayed-gratification' (Bazanis et al., 2002; Dougherty et al., 1999) paradigms.

Hochhausen et al. (2002) compared 48 DIB-R female BPD inmates with 156 non-BPD female inmates on a passive-avoidance task. Participants were instructed to use trial-and-error learning to respond to an experimental task that would result in either monetary reward, or monetary loss. The experimental task

consisted of a list of 10 numbers of which half yielded monetary reward, and half yielded monetary loss. The relationship of the number to winning money or losing money was predetermined by the experimental protocol. At the commencement of the task, participants were provided with a number of 'chips' which served as reinforcers. Passive avoidance errors were defined as the number of times the participant responded to a losing number. The study found that the BPD sample committed more passive avoidance errors than controls, and reported higher rates of impulsivity on a self-report inventory of impulsivity. The authors conclude that these results provide evidence supporting the view that BPD is characterised by impulsivity.

Two studies examined 'delayed-gratification' type impulsive tasks. Dougherty et al. (1999) examined 14 BPD inpatients with 17 controls. They employed a delay of gratification task consisting of the presentation of 50 trials offering a choice between an immediate smaller monetary reward, and a delayed larger monetary reward. The selection of the short-delay responses was similar for both groups, but the BPD group avoided the delayed monetary reward condition. The authors suggest that these findings do not support the view of increased impulsivity in BPD. In contrast, Bazanis et al. (2002) compared 42 DSM-III-R BPD participants with 42 non-clinical controls on a 'decision-making' task. The task consisted of an array of computer-simulated red and blue boxes, the ratio of which varied between trials. The objective of the task involved the participant nominating whether a yellow token was placed inside a red or a blue box by nominating the colour on a response panel on the computer screen. After making a choice, the participant was required to increase their score by betting on whether their choice was correct. The available bets were made in

sequences defined as ‘ascending’ (the first bet was small, but successive bets were larger), and ‘descending’ (the first bet was large, but successive bets were smaller). The order of ascending and descending conditions was counterbalanced across both groups. The extent to which participants chose early bets in both the ascending and descending conditions was interpreted as evidence for impulsivity, and the findings of the study indicated that the BPD group responded significantly earlier in their choices of betting in both the ascending and descending condition. This result provides some supportive evidence for BPD as a disorder of impulsivity.

A related concept to impulsivity is that of response inhibition. The essential difference between these constructs is that inhibitory theories emphasise the capacity to stop or inhibit a prepotent response. This view emphasises the capacity to stop an action (Badcock, Michie, Johnson, & Combrinck, 2002), in contrast to the ‘propensity to action’ theories examining impulsivity (Zanarini, 1993). Inhibition paradigms have typically employed ‘go/no-go’ or ‘stop-signal’ type tasks to assess this phenomenon, and there have been mixed returns for the studies that have employed go/no-go tasks in the study of BPD (Dinn et al., 2004; Kunert et al., 2003; Leyton et al., 2001).

Leyton et al. (2001) examined the neurotransmission of serotonin (5-HT) in patients exhibiting impulsive behaviours. A 5-HT precursor analogue was studied through the use of Positron Emission Tomography (PET) scanning in 13 medication-free participants with BPD, and 11 comparison participants. Impulsivity was assessed by examining commission errors on a go/no-go task. Compared with controls, the BPD participants returned significantly lower 5-HT indices in corticostriatal sites, including the medial frontal gyrus, anterior

cingulate gyrus, superior temporal gyrus, and corpus striatum. In addition, BPD participants returned greater rates of commission error on the go/no-go task suggesting that they experienced difficulties in inhibiting the prepotent experimental task.

Kunert et al. (2003) examined a 'frontal deficit' hypothesis in BPD. One of the many tasks employed by Kunert et al. was a go/no-go task that assessed the capacity of BPD's to inhibit behaviour. Kunert et al. found no significant differences between their BPD cohort and a normal control group suggesting that inhibitory capacity was not compromised in BPD. This is an important finding as it provides empirical evidence which is at variance with the prevailing conventional wisdom that 'impulsivity' is a central feature of BPD.

Dinn et al. (2004) conducted two studies using a go/no-go task in BPD. Study One consisted of the examination of nine BPD inpatients and matched community controls, and Study Two compared 10 undergraduate recruited 'BPD' cases diagnosed solely by self-report with 129 comparison participants. Both studies employed the same go/no-go task which consisted of three conditions. Condition One required the participants to respond when a blue square appeared on the computer screen. Condition Two required the participants to respond when a blue square appeared but not when a blue cross appeared. Condition Three required the participants to respond when a blue cross appeared, but not when a blue square appeared. Collectively, the two studies returned significant but somewhat contradictory findings. Study One found that the BPD group committed more omission errors in Conditions One and Three but returned similar reaction times across all conditions when compared to controls. In contrast, 'undergraduate BPD' participants in Study Two were significantly

slower on Conditions One and Two, but returned similar results on measures of omission. Viewed collectively, the findings suggest that 'clinical' BPD cases demonstrate deficits in response inhibition as measured by omission error rates. These findings support the broader view that there are deficits in response inhibition in BPD.

In summary, there is mixed evidence suggesting that BPD and impulsivity are associated. One of the difficulties with this research is the absence of an operationalised concept of 'impulsivity', and a failure to measure impulsivity in a direct manner. It appears that many of the studies infer the existence of impulsivity on the basis of *post hoc* observations of various forms of behaviour. The use of *ex post facto* modes of explanation is, however, unsatisfactory and essentially unscientific.

Furthermore, it appears that the term 'impulsive' is used in relation to BPD with at least five interrelated meanings. First, the term 'impulsive' appears to be used to describe an inability to stop or inhibit a prepotent behavioural action or sequence. In this sense, impulsivity refers to a deficit in inhibitory capacity. Second, the term 'impulsive' appears to describe a class of behaviours observed in BPD that occur in a social or interpersonal context which have either a low probability of controlling or managing environmental variables on the one hand, or are not 'ecologically valid' on the other. Third, the term 'impulsive' appears to be used to describe various behaviours used to regulate emotional states when there is an absence of a more 'mature' mode of regulation available. Fourth, the term 'impulsive' appears to be used to suggest that there is some form of subtle brain impairment suggestive of 'frontal-lobe' compromise. Finally, the term 'impulsive' appears to be used to describe the employment of

so-called ‘mindless’ behaviour which is ‘irrational’ and not amenable to logical explanation. In relation to this project, the first identified meaning of the term impulsive (the inability to inhibit or stop a prepotent behaviour) will be directly examined, and consideration of various other meanings will be explored in detail in Section 9.4.2.

2.2.4. Disturbed Early Family Environment

A fourth causal perspective on BPD has examined the evidence for various forms of early disturbance or psychopathology in the family environment of BPD patients. ‘Family environment’ research typically includes studies examining evidence for early separation or loss, and/or psychopathology in family members (Links, 1992). In addition, it is also argued that there is considerable evidence for deficits in ‘parental bonding’ and/or attachment disorders in BPD, and this literature is also selectively reviewed here. Although sexual and physical abuse is regarded as a risk factor for BPD (Paris, 1998), the research associated with this area is extensive, and linked theoretically with so-called ‘trauma theory’ (B. D. Perry, Pollard, Blakley, Baker, & Vigilante, 1995; J. C. Perry & Herman, 1993). Therefore, independent review of this area is considered under the heading of trauma perspectives in Section 2.2.5.

Early Separation and Loss

A number of reviews have found support for the hypothesis that BPD patients report higher rates of early separations or losses of primary caretakers (Links, 1992; Paris, 1999; Zanarini, 1997). Many of these studies are quite dated, and are linked to what Zanarini (1997) has described as the ‘first generation’ of studies of the pathogenesis of BPD. Zanarini has argued that these studies were methodologically limited as a result of the failure to incorporate formal

diagnostic procedures, failure to confirm childhood experiences in a systematic manner, and the failure to collect information on diagnostic status and childhood experiences in such a way that each was blinded from the other. As a result, there appear to be some important qualifications to the view of early loss and BPD.

Paris (1999) notes that the majority of the studies examining the link between early loss and BPD have typically compared BPD patients to depressed controls, and Links (1992) has noted that a similar aetiological link has also been reported for depressive disorders. In this regard, Paris, Zweig-Frank, & Guzder (1994a; 1994b) compared BPD and non-BPD cases on measures of early separation and loss. They found that female BPD patients did not report higher rates of early separation and loss than an 'other-personality disorder' comparison group. The male group reported no differences between BPD and non-BPD groups before age five, but a significant difference in loss rates before age 16.

Paris (1999) argues that the fundamental problem with the early separation and loss perspective on BPD is that it fails to take into account the base-rate issue. Early separation and loss is not specific to any form of psychopathology, and occurs frequently in the community (Henderson, Byrne, & Duncan-Jones, 1981). Equally importantly, this perspective fails to incorporate the findings of studies on resilience in childhood that suggest that there are a number of buffers to adverse experience in childhood that act in protective ways to offset the effects of adversity (Rutter, 1989).

Family Psychopathology

A second group of family environment studies have examined the presence of psychiatric disorder in the parents or family members of borderlines. Paris (1999) notes that the parents of BPD's are more likely to manifest a variety

of different forms of psychopathology which include specific Axis I disorders, or personality traits and/or personality disorders that directly interfere with their capacity to parent effectively. For example, Links, Steiner, & Huxley (1988) found elevated rates of unipolar depression, alcoholism, BPD, and ASPD in the parents of a BPD cohort. Links (1992) has argued that parental psychopathology increases the risk of childhood loss or separation, sexual and physical abuse, and family breakdown.

Zanarini (1993) has also reported on a number of studies assessing psychiatric disorder in first degree BPD relatives. She concluded that there is limited evidence for a familial link between BPD and any of the Schizophrenic spectrum disorders. In contrast, affective disorders are common amongst first degree relatives of borderline probands. Zanarini also argued that the collective results of these studies suggested a strong familial link between BPD, Substance Use Disorders, and Antisocial Personality Disorder. Importantly, Zanarini also reported that BPD 'breeds true.' That is, BPD is significantly more common among first degree relatives of borderlines than amongst control participants.

Parental Bonding and Attachment Pathology

Another group of theories have suggested that a putative causal factor for BPD is impaired 'parental bonding' or that disturbed attachments exist between nascent BPD children and their parents. A number of retrospective studies of clinical BPD samples have examined the hypothesis of impaired parental bonding or attachment using various methods (Barone, 2003; Hooley & Hoffman, 1999; Nickell, Waudby, & Trull, 2002; Patrick et al., 1994; Torgersen & Alnaes, 1992; West, Keller, Links, & Patrick, 1993). Collectively, these studies have found that borderlines demonstrate a predominance of pathological

attachments with up to 50% characterised by 'unresolved trauma' (Barone, 2003), or they experience 'enmeshed' and 'unresolved' patterns of relating on the Adult Attachment Interview (Patrick et al., 1994). Other studies have found significant increases on measures of anxious or ambivalent attachment (Nickell et al., 2002), or measures of 'feared loss' (anxious attachment) (West et al., 1993). In addition, other studies have found evidence for high maternal overprotection (Patrick et al., 1994; Torgersen & Alnaes, 1992), emotional over-involvement (Hooley & Hoffman, 1999), and low maternal care (Patrick et al., 1994).

The major methodological flaw with all of these studies concerns their use of retrospective reporting methods based upon respondents' perceptions that they experienced abnormal bonding or attachment with their parents (Paris, 2003a, 2003b). In contrast, Bezirganian, Cohen, & Brook (1993) examined putative risk factors for the development of BPD in a prospective study of 776 adolescents. Mother-child and father-child interactions, maternal personality, and adolescent personality disorder diagnoses were measured on two occasions, two and a half years apart. The findings indicated that the combination of maternal inconsistency and maternal over-involvement in child-rearing predicted the emergence and maintenance of BPD, but not for any other personality disorder. This effect only occurred in the presence of both factors – the presence of either maternal inconsistency or maternal over-involvement in isolation failed to predict the development of BPD. Maternal personality was unrelated to the combined features of maternal inconsistency and maternal over-involvement. The findings of this study suggest that the causal factors associated with the development of BPD are involve a particular type of adverse mother-child

interactional style characterised by inconsistency and over-involvement and are not associated with specific features of maternal personality.

This study provides significant support for the proposition that early attachment or 'parental bonding' factors represent a risk for the development of BPD. However, these findings cannot be interpreted as suggesting that maternal inconsistency and over-involvement exclusively predict the development of BPD as they may also be predictive for other forms of psychopathology.

In summary, there appears to be evidence suggesting that early separation and loss, family psychopathology, and impaired parental bonding and attachment represent significant risk factors in BPD. They cannot however be considered exclusive risk factors for the development of BPD as many of these factors are known to be associated with the development of other disorders (Rutter, 1989). Therefore, these risk factors are not specific for BPD, but probably represent components of a larger multidimensional risk model for BPD (Paris, 1999, 2003a, 2003b).

2.2.5. BPD as a 'Trauma Spectrum Disorder'

A fifth perspective on BPD suggests that a history of childhood trauma is associated with the development of BPD in adulthood. A number of candidate factors including child physical abuse (CPA) and child sexual abuse (CSA) have been implicated in the development of BPD (Herman & van der Kolk, 1987; Murray, 1993; J. C. Perry & Herman, 1993). The large number of studies in this area suggest that BPD is also closely associated with the development of Post Traumatic Stress Disorder (PTSD) (American Psychiatric Association, 1994, 2000), and this perspective is used to explain the often-reported co-morbidity between BPD and PTSD .

There are considerable data suggesting a high incidence of child maltreatment, particularly for incest and sexual abuse, in the histories of BPD participants. This has led some authors to suggest that it is a causal factor for BPD (Bleiberg, 1994; G. R. Brown & Anderson, 1991; Bryer, Nelson, Miller, & Krol, 1987; Herman, Perry, & van der Kolk, 1989; Ogata et al., 1990; K. R. Silk, Lee, Hill, & Lohr, 1995). Individuals who report histories of child sexual abuse also report significant post-traumatic symptoms, and these can include sensory numbing, nightmares, 'flashback' experiences, impaired affect regulation, identity disturbance, dissociative experiences, sexual 'acting out', self-injuriousness, and substance misuse. Many of these diagnostic features (impaired affect regulation, identity disturbance, impulsive sexuality, self-injuriousness, and substance misuse) are also commonly reported by BPD patients (American Psychiatric Association, 1994, 2000; Grotstein, 1987; Linehan, 1993). The co-morbidity between a childhood sexual abuse history, post-traumatic symptomatology, and an adult diagnosis of BPD is a now well documented phenomenon (Landecker, 1992). It is the co-occurrence of these phenomena that has resulted in Zanarini (1997) arguing that BPD might be viewed as one group in a spectrum of traumatically induced psychopathologies.

One additional line of research which supports the view of BPD as representing a form of trauma spectrum pathology is associated with the literature on children who meet BPD criteria. Whilst the diagnosis of BPD in childhood remains a controversial issue (Bleiberg, 1994), there is an emerging literature which supports the view that the diagnosis of BPD can be made in childhood (Farrugia, 1992; Lewis, 1994; Vela et al., 1983).

Guzder, Paris, Zelkowitz, & Marchessault (1996), and Guzder, Paris, Zelkowitz, & Feldman (1999) examined risk factors associated with borderline pathology in latency aged groups of children. Guzder et al. (1996) studied 41 borderline and 57 non-borderline latency aged children using Global Assessment Scale scores and chart review in order to derive cumulative abuse, and cumulative parental dysfunction scores. The study found that the risk factors that discriminated the borderline from the non-borderline cohort were severe neglect, physical and sexual abuse, and parental substance abuse or criminality. In the Guzder et al. (1999) study, 41 borderline and 53 non-borderline school-aged children were compared on a range of behavioural measures. The findings of the study suggested that the borderline cohort experienced higher rates of neglect as well as physical and sexual abuse. In addition, the borderline group also experienced higher rates of family breakdown and parental criminality. The two factors predictive for BPD in childhood were parental criminality and sexual abuse.

Other studies of BPD in childhood and adolescence have also been conducted (Weaver & Clum, 1993; Westen, Ludolph, Misle, Ruffins, & Block, 1990). These studies also suggest that a history of childhood physical and sexual abuse discriminate child and adolescent BPD cases from non-borderline cases. Westen et al. (1990) argue that sexual abuse in particular discriminates for BPD since physical abuse is argued to occur across most psychiatric diagnoses. Weaver & Clum (1993) also reported that in a retrospective study of borderline and non-borderline depressed inpatients, the only predictor of BPD status was the experience of sexual abuse in childhood.

In contrast to the literature suggesting a link between childhood trauma and in particular child sexual abuse in the development of BPD, there is also a growing body of literature which challenges this viewpoint. This body of research has directly examined the presence of trauma in childhood and the development of BPD in adulthood (Fossati, Madeddu, & Maffei, 1999; Paris, 1998; Paris & Zweig-Frank, 1992).

Paris & Zweig-Frank (1992) acknowledge the high incidence of sexual abuse during childhood reported by persons diagnosed with BPD. Whilst noting that these findings are important, Paris & Zweig-Frank suggest that the presumed relationship between child sexual abuse and the development of BPD in adulthood is an oversimplification. They cite the literature on community-based studies of the long-term effects of CSA and note that both the parameters of abusive experience as well as family-of-origin environment factors are central to determining the long-term effects of CSA. They propose that a multifactorial model of BPD is necessary which includes biological predisposition, psychological factors, and social context, as well as their interactions with each other as a necessary mechanism for understanding the development of BPD.

In a more methodologically critical review, Paris (1998) noted the large body of empirical research suggesting that there is a high rate of traumatic events in the histories of people diagnosed with BPD, and as a result BPD, might be viewed as a chronic form of PTSD. Paris argued that the central methodological defect with these studies is that the data is correlational in nature, yet there remains a persistent attempt to argue for a causal relationship between CSA and BPD. Instead, Paris argues that ‘latent’ factors such as genetic vulnerability as well as coexisting environmental factors need to be included in any account of

BPD. In addition, Paris argues that associations between trauma and personality disorder suffer from the 'base rate' problem – there is evidence of a high rate of childhood trauma in the general population. From this perspective, Paris suggests that clinical samples (which form the bulk of the database of the CSA-BPD studies) do not include those cases of childhood trauma that achieve satisfactory outcomes and do not develop personality disorder. Paris concludes that the relationship between childhood trauma and the development of personality disorder is not a simple, linear one. Instead, Paris argues that the majority of children exposed to trauma are resilient, adaptive, and form secure attachments. Paris concludes by arguing that the more significant factor appears to be resilience, and that future efforts should be directed towards identifying children with low levels of resilience as it is this group that future cases of BPD are likely to emerge from.

In an attempt to resolve this issue, Fossati et al. (1999) conducted a meta-analysis on the association between CSA and BPD in 21 studies conducted between 1980 and 1995. The study yielded a total of 2,479 participants where the *r* coefficient was used as a measure of effect size. The findings indicated that a moderate pooled *r* (0.279) was returned for the association between CSA and BPD. The authors concluded that CSA is neither a major risk factor nor a causal factor in the development of BPD.

In summary, there are a large number of studies suggesting that a link has been established between early childhood trauma, particularly sexual abuse, and the development of BPD in adulthood (G. R. Brown & Anderson, 1991; Bryer et al., 1987; Herman et al., 1989; Mitton, Links, & Durocher, 1997; Ogata et al., 1990; Wagner & Linehan, 1997). In contrast, there is an emerging body of

evidence that acknowledges that whilst there might be elevated rates of reporting of childhood trauma and CSA in particular in BPD, the evidence that CSA causes BPD is yet to be established (Bernstein et al., 1996; Bezirganian et al., 1993; Fossati et al., 1999; Paris, 1998; Paris & Zweig-Frank, 1992). It appears that CSA cannot be considered a direct cause of BPD, but people with CSA histories are likely to be overrepresented in BPD cohorts (Zanarini, 1997). Therefore, trauma histories also appear to represent a risk-factor for the development of BPD.

2.2.6. Behavioural Perspectives

The application of behaviourally-based approaches to the understanding and treatment of BPD is a relatively recent phenomenon. This tradition appears to have developed exclusively within the context of clinical psychology, and the behavioural tradition has focussed almost exclusively upon the development of treatment models aimed at modifying the cognitive and behavioural disturbances associated with BPD.

There are at least five approaches to understanding and treating BPD that are informed by various behavioural perspectives. These include a social-learning perspective (Millon, 1981), a radical behavioural perspective (Koerner, Kohelenberg, & Parker, 1996; Nelson-Gray & Farmer, 1999), and four interrelated, behavioural or cognitive-behavioural approaches (Arntz, 1994; A. Beck et al., 1990; G. K. Brown et al., 2004; Linehan, 1993; Young, 1990). These are selectively reviewed.

Social-Learning Approaches

Millon (1981) conceptualises BPD from a social-learning vantage point, and argues that the principle difficulty emerges out of the absence of a consistent

sense of personal identity. This perspective appears to have much in common with the 'identity-diffusion' perspective of Kernberg and colleagues (Clarkin et al., 1999; Kernberg, 1984). According to Millon (1981), impaired identity is the result of biological, psychological, and sociological factors that interact with one another and in so doing compromise the development of a cohesive self-identity. As a result, the absence of coherent self-identity results in the employment of inconsistent goal directed behaviours and impulsive acts which in turn result in the failure to achieve satisfactory outcomes. Because of this fundamental identity difficulty and the attendant absence of coherent problem-solving strategies, Millon argues that emotional regulation and its consequential behavioural outcomes figure prominently in BPD. As a result, borderlines become dependent on others to provide reassurance and protection, yet at the same time experience intense conflicts over dependency and autonomy. This in turn provides the basis for the interpersonal dysregulation which is characteristic of BPD.

Cognitive-Behavioural Approaches

Beck et al. (1990) have developed a model for understanding and treating BPD based primarily on Beck's well-known view of psychopathology (A. T. Beck, Rush, Shaw, & Emery, 1979). This view emphasises the roles of 'appraisals' and cognitive processing in the development and maintenance of psychological disorder. A. Beck et al. (1990) suggest that three fundamental factors explain BPD pathology: a set of untested assumptions about the social world that the borderline endorses, the use of 'dichotomous' thinking, and a 'weak' or unstable sense of identity.

A. Beck et al. (1990) argue that three assumptions operate in BPD. These assumptions include the belief that the world is dangerous and malevolent, that

the borderline person is powerless and vulnerable, and that the borderline person is inherently unacceptable. Furthermore, Beck et al. also argue that BPD is characterised by the use of dichotomous thinking in which there is a tendency to evaluate experience in terms of one of two mutually exclusive categories. The usual candidate dichotomous thoughts include good/bad, success/failure, or trustworthy/untrustworthy. Beck et al. argue that the effect of this type of thinking results in the adoption of extreme positions or the endorsement of extreme interpretations of events that in turn result in adverse emotional reactions and behaviour. One further effect of the endorsement of dichotomous thinking is the tendency to oscillate between two extreme positions which can, in turn, result in abrupt shifts in mood.

Beck et al. (1990) concur with both Kernberg (1975; 1984) and Millon's (1981) view that a weak or unstable sense of identity is central to BPD. This represents the third factor of their model of BPD. They argue that confusion over the establishment of goals and priorities makes it difficult for borderlines to be effective and this in turn adversely affects their sense of self-efficacy. This feature also makes it difficult to pursue goals because there is often a significant degree of emotional turbulence in the patient. This in turn makes the task of accessing and challenging basic assumptions and dichotomous thinking processes difficult. As a result, Beck et al. emphasise the importance of addressing dichotomous thinking processes early in the therapy of BPD. Thereafter, the treatment goals include minimising non-compliance, increasing emotional regulation and impulse control, and strengthening a sense of personal identity.

Whilst Beck has an impressive pedigree in the development of cognitive-behavioural approaches for the treatment of depression (A. T. Beck et al., 1979), there has been little follow-up of his early work on personality disorder. To date, only one randomised control trial (RCT) of Beck et al's. (1990) CBT approach to the treatment of BPD has been reported (G. K. Brown et al., 2004). In this regard, the use of cognitive therapy for BPD appears to have been superseded by the findings of DBT (C. J. Robins & Chapman, 2004). This approach will be considered shortly.

Arntz (1994) has also developed a cognitive-behavioural model of treatment of BPD which represents a variation of the model originally proposed by Beck et al. (1990), in which a greater emphasis is placed upon the role of childhood abuse and trauma. Arntz proposes a treatment model consisting of five overlapping phases which include the construction of a working relationship, symptom management, correction of thinking errors, trauma processing and schema change, and termination. This model appears to be similar to the Beck et al. and Young (1990) models in its emphasis upon the requirement to modify pathological schemas and thinking errors, and similar to the work of Linehan (1993) in its emphasis upon acceptance and empathic connection with the patient. It represents an alternative approach however, in its emphasis upon the assumption that abuse and neglect is causal for BPD. This view appears to be at variance with the assumptions other behavioural researchers have made concerning the causal factors in BPD (A. Beck et al., 1990; Linehan, 1993; Millon, 1981; Young, 1990), and with the literature that has reviewed the role of trauma as a causal factor in BPD (Bernstein et al., 1996; Bezirganian et al., 1993; Fossati et al., 1999; Paris, 1998; Paris & Zweig-Frank, 1992). Again, there is

limited outcome data associated with this approach, and further research is required to confirm the utility of this approach.

Young (1990) has developed a cognitive-behavioural approach to the treatment of BPD which is termed 'Schema-Focused Therapy' (SFT). Young argues that BPD (like all other personality disorders) is characterised by three features: Rigidity, Avoidance, and Interpersonal Difficulties. According to this view, BPD is characterised by rigidity in thinking, avoidance or blocking of painful thoughts or feelings, and dysfunctional interpersonal relationships.

Young's (1990) model emphasises the role of 'Schema Theory' to describe the particular forms of primitive cognition central to the genesis and maintenance of personality disorders. Young does not provide a specific theory of schematic processing in BPD, but rather considers that a number of different schemas are activated in persons with BPD.

'Schemas' refer to enduring patterns of thinking that develop during the course of childhood. They are characterised as unconditional in nature, self-perpetuating, recurring, associated with high levels of affect, and appear to develop out of an interaction between temperamental factors with early childhood experience. Specific, 'Early Maladaptive Schemas' (A. Beck et al., 1990), are argued to operate in BPD. They include thematic issues associated with Abandonment/Loss, 'Unlovability', Dependence, Subjugation/Lack of Individuation, Mistrust, Inadequate Self-Discipline, Fear of Losing Emotional Control, Guilt/Punishment, and Emotional Deprivation. Schemas are argued to continue to operate in the case of BPD until an intervention is provided which specifically interferes with their activation and maintenance.

Dialectical Behaviour Therapy (DBT)

Linehan (1993) has developed a 'biosocial' model of treatment known as 'Dialectical Behaviour Therapy' (DBT). DBT emerged out of a combination of CBT techniques with Zen Buddhist practices that emphasise the importance of acceptance as a healing modality. DBT emphasises the role of emotional dysregulation as a fundamental characteristic of BPD. This arises as a result of an interaction between biological/temperamental features of the BPD individual with the experience of a 'negating' or 'disqualifying' early social environment.

Dysfunctional emotion regulation is believed to form the basis of the dramatic over-reaction to precipitative events in persons with BPD, and also is understood to lie behind much of the so-called impulsivity characteristic of the disorder. Linehan postulates that the early developmental experience of BPD patients is characterised by relationships with caregivers who negate or disavow subjective experience, and insist that the subject engage in a process of denial with regard to their 'true' feelings. The result of this process is that individuals who are already prone to emotional reactivity through a temperamental or biological predisposition compound their inability to regulate emotion, and at the same time develop a disavowing position to their own emotional states.

The combination of inadequate emotional regulation, intense emotional states, impulsive responses to emotional crises, and a disavowing position with regard to their own emotional states results in the use of parasuicidal acts as a signature for the disorder Linehan (1993). Linehan emphasises the importance of the use of validation as a central technique in the treatment of BPD. DBT emphasises a commitment to the use of 'mindfulness' practices couched within the philosophical principle of acceptance of the person.

The strength of this approach has been the commitment to the pursuit of an evidence-based model in which a number of randomised-controlled trials (RCT's) have been conducted to establish therapeutic efficacy (Linehan, 2000). DBT was initially conceived of as a treatment specifically for BPD, but is now being used in the treatment of a number of other disorders including anorexia nervosa (McCabe & Marcus, 2002), substance use problems in BPD (van den Bosch, Verheul, Schippers, & van den Brink, 2002), and partner abuse (Waltz, 2003).

Radical-Behavioural Approaches

Koerner et al. (1996) and Nelson-Gray & Farmer (1999) provide formulations of personality disorders from a 'radical behavioural' and 'functional analytical' framework respectively. Whilst these theorists do not address theoretical issues associated exclusively with BPD, many of their respective arguments do apply to BPD.

Nelson-Gray & Farmer (1999) note that some behavioural researchers reject the notions of personality and personality disorder because of a combination of their trait and mental illness connotations, whereas Koerner et al. (1996) argue that the important issue in 'diagnosis' is to understand the functional significance the behavioural aspects of the problem serve in maintaining the 'disorder'. They further argue that the important issue is the need to understand the role of specific behaviours in the context in which they occur. The respective models of Nelson-Gray & Farmer and Koerner et al. appear to focus exclusively upon understanding the measurable, behavioural aspects of so-called 'personality disorder behaviour'.

As a result of this behavioural reformulation, both Koerner et al. (1996) and Nelson-Gray & Farmer (1999) recast BPD related issues in terms of either their 'Clinically Relevant Behaviours' (CRB), or the applicability of the Stimuli-Organism-Response-Consequences (SORC) model of Goldfried & Sprafkin (1976). Both models employ the strategy of formulation of BPD markers into behavioural terms, and the selection of CRB's (Koerner et al., 1996) or 'target behaviours' (Nelson-Gray & Farmer, 1999) as the loci for intervention. Through an analysis of the factors that cause and/or maintain each specific CRB or target behaviour, each theorist is able to construct a multi-level intervention aimed at treating the behavioural referents of BPD.

Unfortunately, neither Koerner et al. (1996) nor Nelson-Gray & Farmer (1999) provide any empirical evidence confirming the efficacy of their respective approaches. Clearly, the behavioural tradition has been characterised by an emphasis on evidence-based practice, and it is surprising therefore that there are so few studies on BPD from an applied behavioural perspective.

In summary, the 1990's witnessed a burgeoning of interest by behaviourally oriented theorists to understanding BPD and developing treatments with demonstrated efficacy in managing the disorder. Currently, there appears to be some promise held for DBT, although Westen (2000) cautions against over-enthusiasm for DBT as there has been insufficient long-term follow-up of the RCT's for DBT. Despite this caution, behavioural approaches offer an important, evidence-based approach for understanding and treating BPD.

2.2.7. BPD as a 'Neurobehavioural' or Psychobiological Disorder

A final psychological perspective has developed out of the integration of the genetic, biological, neurological and neuropsychological research on BPD.

This perspective has been variously referred to as the 'neurobehavioural' or 'psychobiological' approach (Kimble et al., 1997; Marziali, 1992; Siever & Davis, 1991; Soloff & Millward, 1983; van Reekum, Conway et al., 1993). This perspective examines the role that psychobiological, neurological, and neuropsychological factors play in BPD (O'Leary, 2000; Siever & Davis, 1991; Stone, 1993). A brief review of these perspectives is provided below.

A 'Core Psychobiological Vulnerability'

Siever & Davis (1991) have argued that a variety of psychobiological deficits occur in personality disorders. They take issue with the categorical perspective employed in personality disorder diagnosis, and argue instead for a dimensional perspective. They argue that a 'core psychobiological predisposition' underpins the personality disorders, and that the psychobiological factors underpinning personality disorders include a 'cognitive/perceptual' dimension, an 'impulsivity/aggression' dimension, an 'affective instability' dimension, and an 'anxiety/inhibition' dimension. These dimensional predispositions are thought to underpin both the personality disorders, and their related Axis I disorders. From this perspective, the different categories of personality disorder reflect differential combinations of these 'core psychobiological predispositions'. Siever & Davis argue that BPD is characterised by impulsivity/aggression and affective instability, and that these clinical exaggerations might be associated with depleted serotonin activity and increased norepinephrine activity. This view is conceptual in nature, and empirical support for this perspective is limited.

The evidence that BPD is characterised by various forms of psychobiological impairment is, however, equivocal (Coccaro & Kavoussi, 1991;

Cowdry, 1992; Kimble et al., 1997; Korzekwa et al., 1993; Marziali, 1992; Schore, 1994, 2003a; Stone, 1993; van Reekum, Links et al., 1993), although a recent review argued that there is accruing evidence supporting the view that a biological substrate operates in BPD (Paris, 2000).

This core psychobiological predisposition is thought to influence the metabolic, neural and neuropsychological outcomes observed in BPD. This includes evidence concerning the genetics (Dahl, 1994; Jang, Livesley, Vernon, & Jackson, 1996; Torgersen, 2000; Torgersen et al., 2000), 'soft' neurological signs (Cornelius et al., 1989; Gardner et al., 1987; Kimble et al., 1997; Soloff & Millward, 1983), neurophysiological (EEG) activity (Cornelius et al., 1989; Kutcher, Blackwood, St Clair, Gaskell, & Muir, 1987), neuroradiological returns (Cowdry, Pickar, & Davies, 1985-1986; Donegan et al., 2003; Goyer et al., 1994; Goyer et al., 1992; Lucas, Gardner, Cowdry, & Pickar, 1989; Snyder, Pitts, & Gustin, 1983b; Soloff et al., 2000; Tebartz van Elst et al., 2003), and neuropsychological findings (Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary, 2000; O'Leary et al., 1991; Sprock, Rader, Kendall, & Yoder, 2000; Swirsky-Sacchetti et al., 1993) reported from studies examining BPD.

A Genetic Predisposition to the Development of BPD

There is limited data available concerning the genetics of BPD. Although there have been a number of family history studies conducted on BPD, these have been unable to distinguish between the influence of environmental and genetic factors (van Reekum, Links et al., 1993). Dahl's (1994) review of the family studies of BPD concluded that most studies failed to meet adequate

methodological rigour for family risk analysis and their findings could not be interpreted as supporting an increased risk for BPD among first-degree relatives. Torgersen (1994) also reported on a twin study aimed at examining the genetic factors associated with the development of Schizotypal Personality Disorder (SCZ) or BPD. Torgersen reported a MZ concordance of 0%, and a DZ concordance of 29% which was interpreted as not supporting a genetic account of BPD. The bulk of the literature on the genetics of BPD suggests that there is limited evidence to support a genetic basis for BPD, however a number of relatively recent studies challenge this assumption.

Jang et al. (1996) studied 236 MZ and 247 DZ twin-pairs with a self-report inventory designed to assess 18 factorially-based dimensions of personality problems. The questionnaire was designed to asses the hierarchy of trait dimensions that constitute the domain of personality disorder, and provided data on 69 'facet traits' of personality disorder. The results indicated that a heritable component approximating 40-50% occurred across all personality disorder dimensions. The shared environmental effects were reported to be negligible, and the non-shared environmental effects accounted for most of the variance.

In a more recent and empirically rigorous study, Torgersen et al. (2000) interviewed 92 MZ and 129 DZ twins with the SCID-II, and prevalence rates from a normal population of over 2,000 individuals were used in combination with the interview data to generate statistics assumed to be valid for a normal twin population. The best fitting models returned heritability coefficients for personality disorders in general at 0.60 and 0.69 for BPD. One surprising finding of the study found that for BPD there was a strong genetic influence and an

absence of effects for shared environment. The findings of this study lend considerable weight to the view that there is a heritable component to BPD. This view is also consistent with the review of Parker & Barrett (2000) who argue that as much as 50% of the variance in personality can be accounted for through the influence of genetic factors.

This possible genetic predisposition is probably related to the temperament organisations associated with impulsive-aggression and affective instability (Coccaro & Kavoussi, 1991; Siever & Davis, 1991). This presumed genetic predisposition provides the basis for the psychobiological vulnerability in BPD which elaborates itself in concert with adverse post-natal experience. The end result is the development of BPD in late adolescence or early adulthood.

In summary, the original studies examining the genetic basis for BPD found little support for a heritability component to the disorder. More recent evidence however, in the form of direct studies of heritability in BPD as well as other studies examining the heritability of personality and personality disorder traits have found some evidence for a heritability factor in the expression of BPD pathology. These latter findings provide some support for the psychobiological perspective of Siever & Davis (1991), and suggest that a genetic substrate might represent an independent risk factor for the development of BPD.

Neurological Deficits in BPD

There are a variety of sources of evidence of neurological deficits in BPD. These include 'neurodevelopmental' interview studies, neurophysiological (EEG) evidence, and neuroradiological data. This evidence is selectively reviewed.

Neurodevelopmental Interview Studies

There is mixed evidence that neurodevelopmental interview methods detect neurological deficits in BPD. Soloff & Millward (1983) and Cornelius et al. (1989) employed neurobehavioural checklists in each of their respective studies to elicit evidence of neurological deficits in BPD. Soloff & Millward's findings did not provide support for a neurobehavioural account of BPD, and Cornelius et al. reported no significant differences between groups on cumulative tallies of convulsions, head injury, premature birth, indices of delayed milestones, hyperkinesis, clumsiness, speech delay, or childhood developmental disorders.

In contrast, Kimble et al. (1997) reported pilot data from a prospective study of the longitudinal course of BPD. One component of the study included the collection of neurodevelopmental histories on 63 female patients. The results indicated that no individual neurological variable discriminated BPD from other personality disorders, however a composite variable assessing a 'vulnerable CNS substrate' was significantly more common amongst the borderline group than controls. Kimble et al. interpret this evidence to suggest that a non-specific CNS dysfunction operates in BPD. The causal basis remains unknown.

Neurophysiological Studies

There also appears to be mixed evidence for EEG abnormalities in BPD. A number of studies report evidence of abnormal EEG activity in BPD (Cowdry et al., 1985-1986; Kutcher et al., 1987; Snyder & Pitts, 1984), and other studies report no evidence of EEG abnormality (Cornelius et al., 1989; Ogiso et al., 1993).

Snyder & Pitts (1984) found evidence of greater slow-wave activity in BPD participants when compared with dysthymic controls. They account for this finding through a 'neural immaturity' hypothesis, and speculate that this finding might represent a neural lag indicator for BPD. Similarly, Cowdry et al. (1985-1986) compared BPD and unipolar depressed patients and found EEG profiles consistent with complex partial seizure or episodic dyscontrol phenomenon. Kutcher et al. (1987) also examined P300 and other long-latency EEG activity in BPD, other personality disordered, schizophrenic, depressed, and volunteer controls. Significant differences were found between BPD and other personality disordered controls on measures of P300 latency and amplitude, and long-latency event-related potentials were similar between the BPD and schizophrenic group. These were, in turn, different from the other controls. The findings were interpreted as supporting a hypothesis that, like schizophrenics, BPD is in part characterised as a disorder of auditory neurointegration.

Other studies report equivocal results for EEG activity in BPD. Cornelius et al. (1989) examined the prevalence of EEG dysrhythmias in BPD. No significant group differences were reported for either mild or severe EEG abnormalities. Similarly, Ogiso et al. (1993) examined EEG responses in a BPD and 'non-BPD' comparison group. No evidence of EEG abnormality was found.

In a review of the EEG literature on BPD, Boutros, Torello, & McGlashan (2003) identified 22 studies from which diagnostic criteria and data on co-morbidity and control groups could be identified. The majority of studies returned evidence of electrophysiological aberrations in BPD. However, Boutros et al. identified a number of methodological limitations with many of the studies including inadequate control groups and inadequate evaluation of co-morbidity.

They concluded that this research is at a preliminary stage, but that electrophysiological investigations are important in understanding the psychobiological basis of BPD.

Neuroimaging Studies

The neuroimaging research in BPD is characterised by a small number of studies that have returned a variety of results. A number of studies have examined brain volumetric indices (Driessen et al., 2000; Lucas et al., 1989; Lyoo et al., 1998; Snyder et al., 1983b; Tebartz van Elst et al., 2003), and others have used functional magnetic resonance imaging (fMRI) or positron emission tomography (PET) to examine impaired metabolic function (De la Fuente et al., 1997; Goyer et al., 1994; Soloff et al., 2000), in relation to fenfluramine challenge (Soloff et al., 2000), affective facial expressions (Donegan et al., 2003), or in response to go/no-go tasks designed to measure impulsivity (Leyton et al., 2001; Vollm et al., 2004).

Volumetric studies have generally returned mixed results, with early studies (Lucas et al., 1989; Snyder et al., 1983b) suggesting normal brain volume in BPD, whereas more recent studies (Driessen et al., 2000; Lyoo et al., 1998; Tebartz van Elst et al., 2003) have found reduced frontal lobe, hippocampal, amygdala, and anterior cingulate volumes in BPD. These findings appear equivocal, but earlier studies employed CT scan technology (Lucas et al., 1989; Snyder et al., 1983b), whereas more recent studies (Driessen et al., 2000; Lyoo et al., 1998; Tebartz van Elst et al., 2003) employed MRI technology. MRI is generally considered to be a superior method to CT technology because of its greater diagnostic sensitivity, and capacity to detect 'clinically silent' abnormalities (Kent, Haynor, Longstreth, & Larson, 1994).

The fMRI and PET studies have consistently demonstrated evidence consistent with impaired metabolic functioning in BPD. Goyer et al. (1994) found impaired regional cerebral glucose (rCMRG) levels in the prefrontal regions of their BPD cohort, and Soloff has also found evidence of a diminished serotonergic response in the prefrontal cortices of their BPD cohorts (Soloff et al., 2003; Soloff et al., 2000). In contrast, Donegan et al. (2003) reported that BPD participants returned significantly greater left amygdala activation in response to 'emotional faces' stimuli. Collectively, these studies suggest significant metabolic reactivity in BPD.

The two neuroimaging studies employing go/no-go tasks have also returned similar results. Völlm et al. (2004) employed fMRI whilst performing a go/no-go task. They reported that the BPD cohort displayed a bilateral pattern of activation involving the frontal gyri and anterior cingulate whereas activation in the control group was localised in the prefrontal regions. Leyton et al. (2001) employed PET scan technology to examine impulsivity and serotonin function. They found evidence of impaired serotonergic functioning in the medial frontal gyrus, anterior cingulate, temporal gyrus, and striatum.

In summary, there is evidence from the neurological literature suggesting a neurobiological underpinning to BPD. This evidence forms one component of the neurobehavioural perspective, and the supporting neuropsychological evidence will also be briefly reviewed to demonstrate legitimacy of this perspective.

Neuropsychological Deficits in BPD

A number of studies have examined neuropsychological functions in BPD. The findings of these studies have also returned mixed results. A number

of studies have examined whether intelligence is impaired in BPD, and these studies have returned equivocal results. Some studies suggest that BPD is characterised by normal IQ returns (Bazanis et al., 2002; Cornelius et al., 1989; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; Sprock et al., 2000) whereas other studies suggest that IQ returns are significantly lower in BPD (Swirsky-Sacchetti et al., 1993).

Similarly, a number of studies have examined a variety of memory functions, and these studies also return equivocal findings. Many studies have found intact mnestic returns including general memory function (Cornelius et al., 1989; Judd & Ruff, 1993), verbal memory (Driessen et al., 2000; Kunert et al., 2003; Sprock et al., 2000), visual recognition memory (Bazanis et al., 2002; Kunert et al., 2003), and visual working memory (Kunert et al., 2003). Other studies have suggested that BPD is characterised by various forms of memory impairment including immediate-recall verbal memory (Burgess, 1990; O'Leary et al., 1991), delayed-recall verbal memory (Burgess, 1990, 1991; Kurtz & Morey, 1999; O'Leary et al., 1991), delayed-recall visual memory (Judd & Ruff, 1993; Sprock et al., 2000) and 'evocative' memory (Richman & Sokolove, 1992).

Finally, a number of studies have examined 'problem-solving', 'executive', or 'frontal lobe' performance from a neuropsychological perspective. Again, the results have provided equivocal findings. A number of studies have found little evidence to support a problem-solving/executive deficit hypothesis in BPD. These studies have found intact performance on executive tasks including phonetic word retrieval (Judd & Ruff, 1993), attentional capacity (Kunert et al., 2003), 'set-shift' and 'decision-making' tasks (Bazanis et al.,

2002; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993), Tower of London and Tower of Hanoi planning tasks (Bazanis et al., 2002; Kunert et al., 2003), visuoconstructive problem-solving tasks including the Rey Figure (Cornelius et al., 1989; Sprock et al., 2000), the use of abstract conceptualisation in tasks such as Similarities (Burgess, 1990; O'Leary et al., 1991), maze-learning (Cornelius et al., 1989; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), 'response-conflict' tasks including the colour-conflict Stroop (Judd & Ruff, 1993; Kunert et al., 2003; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and the 'Emotional' Stroop task (Arntz, Appels, & Sieswerda, 2000; Sprock et al., 2000), behavioural inhibition as examined by the go/no-go task (Kunert et al., 2003), and general performance on the Luria-Nebraska Neuropsychological Battery (Rogalski, Val, Prasad, & Weiler, 1986).

Other studies have found support for various executive deficits including multidimensional dichotomous thinking (Veen & Arntz, 2000) and 'splitting' phenomenon (Leichsenring, 1999), visuoconstructive problem-solving tasks including the Rey Figure (Dinn et al., 2004; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993), the use of abstract conceptualisation (Burgess, 1991), measures of 'general cerebral efficiency' (Judd & Ruff, 1993), response inhibition as examined by the go/no-go task (Dinn et al., 2004; Leyton et al., 2001; Vollm et al., 2004), and response-conflict involving the 'colour conflict' Stroop (Swirsky-Sacchetti et al., 1993).

Despite the equivocal nature of these findings, this literature is reviewed in detail in Chapter Three. Therefore, it will not be considered in further detail at this point. There is however, sufficient evidence to warrant consideration of a 'neurobehavioural' perspective in BPD. This perspective considers BPD to be a

psychobiologically based disorder in characterised by impaired 'executive functions'. This perspective forms the basis of the multidimensional developmental neuropsychological model of BPD which will be described in Chapter Three. It is also argued that this perspective is the most capable perspective available to explain the multidimensional psychological disturbances noted in BPD. These include affect dysregulation, impulse dyscontrol/behavioural disinhibition, impaired attentional and mnestic capacity, and compromised problem-solving abilities.

2.3. DIAGNOSTIC SYSTEMS IN BPD

Four systems exist for diagnosing BPD. They include Kernberg's Structural Interview System for Borderline Personality Organization (Kernberg, 1967, 1984; Kernberg et al., 1981), the Gunderson criteria for diagnosing BPD (Gunderson, 1994; Gunderson et al., 1981), the International Classification of Diseases (ICD) of the World Health Organization (A. W. Loranger et al., 1997), and the Diagnostic and Statistical Manuals (DSM) of the American Psychiatric Association (American Psychiatric Association, 1980, 1987, 1994, 2000). This section will selectively review these systems, and report the diagnostic instruments aligned with each of the respective models of BPD.

2.3.1. Kernberg's Borderline Personality Organization (BPO)

Kernberg (1967; 1975; 1984) has developed a psychodynamically oriented system for diagnosing a borderline syndrome known as "Borderline Personality Organisation" (BPO). Kernberg does not conceptualise BPO as a primary diagnosis, but as a means of describing the severity of impairment of 'ego functioning' and of 'object relations'. Kernberg (1967; 1975; 1984) identifies the following symptoms as diagnostic for BPO:

1. Anxiety. BPO is characterised by chronic, diffuse, and free-floating anxiety.
2. 'Poly-symptomatic Neuroses'. This includes multiple 'simple' phobias, obsessive-compulsive, hypochondriacal and conversion symptoms, dissociative reactions, and 'paranoid' trends.
3. 'Polymorphous Perverse Sexual Trends'. Patients with BPO often report a clearly defined sexual deviancy.
4. 'Classical Prepsychotic Personality' Structures. These include the 'paranoid' personality, the 'schizoid' personality, and the 'hypomanic' personality.
5. 'Impulse Neuroses and Addictions'. BPO is often characterised by the use of impulsive gestures which are 'ego-dystonic' (unpleasant) when they are not being engaged in, but are 'ego-syntonic' (pleasurable) when actually engaged in. Kernberg includes such activities as alcoholism, drug addiction, eating, and 'kleptomania'.
6. 'Lower-level' Character Disorders. Included within this grouping are patients who evince signs of chaotic and 'impulse ridden' styles of behaviour. Kernberg includes within this grouping 'infantile', 'narcissistic', and 'antisocial' personalities.

Measurement of Kernberg's BPO Construct

Kernberg's BPO construct is assessed exclusively through an interview methodology. Kernberg et al. (1981) originally developed the 'Structural Interview' to examine symptoms, conflicts, and the manner in which the respondent manages these challenges in the interaction with the interviewer. The assumption underpinning the interview suggests that the focus upon conflict creates tension within the respondent that provokes their typical defensive organisation. This in turn enables a judgement to be made regarding the

respondent's level of personality organisation. This judgement is facilitated by assessing the degree of identity integration, type of defensive operations, and the capacity for reality testing observed in the interview. The interview combines a mental status examination with a psychoanalytically oriented interview which examines patient-therapist interaction, the response to confrontation, and the interpretation of identity conflicts, defensive operations, and reality distortions employed by the respondent. More recently, the Structural Interview has been updated as the Structural Interview for Personality Organisation (STIPO) (Clarkin, 2003), and Lenzenweger, Clarkin, Kernberg, & Foelsch (2001) have reported psychometric data on the STIPO suggesting that it has sound internal consistency, and good test-retest reliability.

Despite these findings, there is little available in the published literature on the STIPO. The most significant issue with Kernberg's methodology is, however, the relative absence of empirical support for the interview methodology. In comparison to other approaches, there remains insufficient reliability and validity data available on this measure for it to be regarded as a viable instrument for use in the current study. This is particularly the case when both the Structural Interview/STIPO are not aligned with any of the other major diagnostic systems. The Structural Interview/STIPO is problematic because it relies upon the interviewer having an intimate knowledge of an ego-psychological/object-relational psychoanalytic meta-psychology. Furthermore, the concept of BPO is a broader and more heterogeneous construct than the more parsimonious BPD (Gunderson, 1994). This results in an increased risk of Type I diagnostic error. These issues render the Structural Interview/STIPO redundant for the purposes of most empirical research into BPD. Accordingly, the

Structural Interview and it's newer cousin the STIPO, are rarely reported in clinical or experimental research (Gunderson, 1994).

2.3.2. Gunderson's BPD Criteria

Gunderson and colleagues (Gunderson, 1994; Gunderson & Kolb, 1978; Gunderson et al., 1981; Gunderson & Singer, 1975; Zanarini et al., 1989) have developed a diagnostic system for BPD which has its origins in psychoanalytic theory and descriptive psychiatry. The so-called Gunderson criteria appear to have been influenced by the work of Kernberg (1967) and Grinker et al. (1968). Gunderson criteria examine five areas of functioning thought to be both characteristic and discriminating for borderlines. These include: Social Adaptation, Impulse/Action Patterns, Affects, Psychosis, and Interpersonal Relations.

Measurement of Gunderson's BPD Construct

Gunderson's model of BPD is assessed exclusively through semi-structured interview. The original instrument - the Diagnostic Interview for Borderlines (DIB) (Gunderson et al., 1981) was subsequently revised as the Diagnostic Interview for Borderlines - Revised (DIB-R) (Zanarini et al., 1989). The DIB was superseded because it was unable to effectively discriminate BPD from other personality disorders (Reich, 1992).

The DIB-R is a semi-structured interview that is divided into four sections for diagnosing BPD. These sections are defined as the "Affect," "Cognition," "Impulse Action Patterns," and "Interpersonal Relationships." It enables the interviewer to rate 87 items concerning the way that the subject had felt, thought, and behaved during the past two years. The interview is further divided into 24 subsections that enable rating of important dimensions of the

disorder. These subsections yield a score which is scaled using an algorithm to determine a Scaled Section Score. The Scaled Section Scores are then added to yield a total DIB-R Score of 0-10. A cut-off score of eight (8) out of 10 is regarded as the probabilistically optimal level for diagnosing the condition. At a cut-off score of eight, Zanarini, et, al. (1989) report a Sensitivity of 0.82, a Specificity of 0.80, a Positive Predictive Power of 0.74, and a Negative Predictive Power of 0.87. The DIB-R is more effective in discriminating BPD from other personality disorders, and is designed for use with participants 18 years or older. It requires approximately 60 minutes to administer.

Kaye & Shea (2000) argue that although the DIB-R does not diagnose according to DSM-IV criteria, it provides more information than do the DSM-IV aligned protocols. In particular, it provides considerably more information concerning cognitive and dissociative features than do other, comparable instruments. The consensus suggests that it represents a sound instrument for the diagnosis of BPD.

2.3.3. The International Classification of Diseases of the World Health Organisation (ICD)

The International Classification of Diseases (ICD) is a classification system for all known illnesses including psychiatric disorders. The ICD is auspiced by the World Health Organisation (WHO), and is currently in its 10th revision (ICD-10). It incorporates personality disorder diagnoses within its psychiatric disorders classification, and the diagnosis of BPD has only recently been incorporated with the advent of the ICD-9. In the ICD-10, BPD is categorised as an independent diagnosis. It is included as a subcategory of

'Emotionally Unstable Personality Disorder – Borderline Type'. Appendix I describes the diagnostic criteria for this condition.

Measurement of the ICD BPD Construct

The ICD-10 assessment of BPD is assessed by the International Personality Disorders Examination (IPDE) (A. W. Loranger et al., 1997). The IPDE is a semi-structured, self-report interview designed for respondents over 18 years of age, although it can be used with respondents as young as 15 years (A. W. Loranger et al., 1997). The IPDE has also been designed to diagnose personality disorders according to DSM-III-R and DSM-IV criteria (American Psychiatric Association, 1987, 1994), but the schedule is included here as the IPDE was designed for the WHO International Pilot Study of Personality Disorders (IPSPD) (A. W. Loranger et al., 1997).

The IPDE consists of 67 questions organised under six headings that elicit information relevant to ICD-10 and DSM-IV diagnoses. These headings include Work, Self, Interpersonal Relationships, Affects, Reality Testing, and Impulse Control. The sections are introduced by the use of open-ended inquiries that develop a set for the questions that follow. Because the IPDE codes for both of the major diagnostic systems, common questions are employed to assess DSM/ICD criterion.

Psychometric data on the IPDE was not reported by Loranger et al. (1997), but the Personality Disorders Examination (A. Loranger, 1988) which was an earlier version of the IPDE was reported to have acceptable test-retest reliability ranging from 0.82 to 0.94 in various studies (Reich, 1992).

2.3.4. The Diagnostic and Statistical Manual of the American Psychiatric Association (DSM)

The Diagnostic and Statistical Manual (DSM) system was developed under the auspices of the American Psychiatric Association (APA), and is currently in its sixth revision (DSM-IV-TR) (American Psychiatric Association, 2000). The original DSM was published in 1952, and included no diagnostic information regarding BPD. Although the DSM-II included a section on the diagnosis of personality disorders, BPD was not included as an official diagnostic category (Novello, 1974). The precursor category for BPD in the DSM-II appears to be a category called 'Explosive Personality Disorder,' but this only referred to the angry/affective lability which came to be a feature of BPD in subsequent revisions. Elements of what has come to be known as BPD were probably also derived from the 'Asthenic' Personality Disorder diagnosis. This included anhedonia, vulnerability to stress, and lethargy and fatigability.

The formal use of the term 'Borderline Personality Disorder' (BPD) was not incorporated into the official nomenclature of the APA until the publication of DSM-III (American Psychiatric Association, 1980), when the DSM system first incorporated a 'multiaxial' mode of diagnosis (Akhtar, 1992). The multiaxial diagnostic system allocated an axis of diagnosis (Axis II) specifically to the diagnosis of personality disorder. 'State' or acute-episode disorders were concurrently allocated to Axis I of the DSM. BPD was included on Axis II of the DSM. The revisions of the DSM since the publication of DSM-III, the DSM-III-R (American Psychiatric Association, 1987), the DSM-IV (American Psychiatric Association, 1994) and the DSM-IV-TR (American Psychiatric Association, 2000) have maintained the multiaxial diagnostic system.

The diagnosis of BPD has undergone a number of revisions since the original DSM-III formulation. The revisions have resulted in additions to the inclusion criteria for the disorder. The DSM-IV/DSM-IV-TR diagnoses of BPD require that a patient meet five of nine listed criteria. The criteria for BPD operate on a 'polythetic' basis in which no specific criterion within the nine available criteria must be met. As a result, at least 256 different 'types' of borderline condition can be potentially classified on the DSM-IV depending upon the specific criteria met by a respondent (Burgmer, Jessen, & Freyberger, 2000). The DSM does not provide any conceptual distinction between different combinations of polythetic BPD criterion. Appendix II describes the diagnostic criteria for this condition.

Measurement of the DSM BPD Construct

Currently, there are two methods employed to assess DSM-IV or DSM-IV-TR personality disorders. These include self-report inventories and semi-structured interviews (Kaye & Shea, 2000). All of the available measures appear to have been designed for use with DSM-IV criteria and this discussion will concern itself with DSM-IV and DSM-IV-TR (American Psychiatric Association, 1994, 2000) categorisations, as they appear identical with one another.

SELF-REPORT INVENTORIES

Personality Diagnostic Questionnaire – 4th Edition (PDQ-4) (Hyler, 1994).

The Personality Diagnostic Questionnaire (PDQ-4) is a 100 item, self report inventory that provides estimates of the DSM-IV Axis II personality disorders. It has been designed as a screening measure for personality disorders,

and the most recent version includes a Clinical Significance Scale to control for Type I error (PDQ-4+). The PDQ-4 is an update of earlier versions, the Personality Diagnostic Questionnaire – Revised (PDQ-R) - a DSM-III-R aligned revision of the Personality Diagnostic Questionnaire (PDQ). The PDQ-4 is a 'forced choice' instrument that takes approximately 30 minutes to administer. The principal advantages of the PDQ-4 are that it enables rapid testing of subjects, and places little demand on clinician/interviewer time.

Some limitations have been reported for the PDQ-4. Fossati, et al. (1998) administered the PDQ-4+ along with the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (M. B. First, Gibbon, Spitzer, Williams, & Benjamin, 1997) to 300 consecutively admitted in-patients and outpatients. Low rates of agreement were returned for both dimensional and categorical modes of personality disorder diagnosis. Discriminatory capacity was only observed for the Dependent and Antisocial subscales of the PDQ-4. The authors concluded that the PDQ-4 was not an adequate instrument for detecting personality disorders. They recommended major modifications before the PDQ-4 could be considered as a screening instrument for DSM-IV personality disorders.

Wisconsin Personality Disorders Inventory – IV (WISPI-IV) (M. K. Klein, Benjamin, Rosenfeld, Greist, & Lohr, 1993)

The WISPI-IV was designed to assess DSM-IV personality disorders from both a categorical and dimensional perspective using Benjamin's (1993) Structural Analysis of Social Behaviour circumplex model. The WISPI-IV is a 214-item self-report inventory that provides indices on 10 DSM-IV personality disorder diagnoses as well as the 'negativistic' personality disorder from Appendix B of the DSM-IV. In addition, 10 items from the Marlowe-Crowne

social desirability scale are included to examine social desirability and response-set bias.

M.K. Klein, et, al. (1993) has reported both reliability and validity data on a DSM-III-R aligned version of the instrument. Two week test-retest reliabilities are reported as 0.88 and 0.84 for the instrument as a whole for two separate groups, and for the Borderline Scale, the retest kappa's are 0.88 and 0.84 respectively. Construct validity of the WISPI was examined by the administration of the WISPI, PDQ, and the MCMI to a clinical sample of 146 subjects. For the Borderline scale, the WISPI correlated significantly with the MCMI Borderline scale (r=0.57), and with the PDQ Borderline Scale (r=0.67). The reliability and validity data on the WISPI suggest that the internal consistency and reliability scales for specific personality disorders are very high, and that it correlates well with other paper and pencil instruments such as the MCMI and the PDQ (Kaye & Shea, 2000).

Kaye & Shea (2000) argue that the WISPI-IV appears to be a promising instrument for assessing personality disorder using a dimensional approach. The novel aspects of the instrument include its basis in an interpersonal circumplex model, and that items are written from the perspective of the respondent. However, there remain questions regarding its compatibility with other measures of personality disorder, but the available psychometric data suggest that it is an instrument of promise.

Millon Clinical Multiaxial Inventory - III (MCMI-III) (Millon et al., 1994).

The MCMI-III is a DSM-IV aligned, 175 question revision of the MCMI and the MCMI-II. It is a "true-false," forced choice, computer or hand scored,

DSM-IV aligned instrument which takes approximately 30 minutes to administer. It is designed for use with respondents aged 19 years or older. The MCMI-III utilizes a Base-Rate scoring system which yields scores on a range of orthogonal dimensions. Included within these are 'Clinical Personality Patterns', and 'Severe Personality Pathology's Scales which assess the presence or absence of personality disorder. The 'Severe Personality Pathology' section includes the 'Borderline' Scale. In addition, the MCMI-III includes four modifying indices that are used to adjust scale scores, and to assess the validity of the protocol.

The original MCMI was regarded as problematic for a number of reasons (Reich, 1987, 1992). These included whether the diagnoses were consistent with the then DSM-III, and whether the overlap in items diagnosing different disorders created an artificial overlap between the disorders (Widiger & Frances, 1989). Reich (1987) also suggested that the presence of state illness influenced the diagnosis of personality disorder status. Finally, the MCMI borderline diagnosis did not agree well with other measures of borderline diagnosis (Reich, 1992). Nevertheless, the MCMI generally received good reviews when compared to other non DSM-III self report inventories (Reich, 1992). The upgrade to the MCMI-II was an attempt to address the concerns outlined previously.

The MCMI borderline scale reported good test-retest reliability of 0.77 in one study of an inpatient and outpatient cohort (Millon, 1982), and 0.89 in an eight week test-retest of panic disorder patients (Reich, 1987). Reich (1992) concludes that the MCMI-II is a cost-effective method for diagnosing the degree of borderline traits.

Since that time, the Millon has been upgraded to Version Three which is aligned to DSM-IV. The MCMI-III's Borderline Scale Test-Retest reliability

over a 14 day period is reported to be 0.93, and the Internal Consistency Coefficient is reported as 0.85 (Millon et al., 1994). The principal advantages of the MCMI-III are that it enables rapid testing of subjects, places little demand on clinician/interviewer time, and is DSM-IV aligned.

Kaye & Shea (2000) note that whilst the MCMI-III has a long history when compared to other personality disorder measures, concerns have been expressed about item overlap amongst the scales which in turn result in high correlations between the scales. This in turn makes the task of profile interpretation problematic. Second, if the base rates for specific personality disorders are significantly different with the population under examination from the original standardisation sample, then it is likely that the diagnostic cut-offs will be invalid. This also has implications for profile interpretation as the MCMI-III manual provides little assistance with regard to profile interpretation. Despite these objections, the extensive use of the MCMI-III over a substantial period has resulted in the instrument being regarded as a clinically useful tool that is best used in conjunction with a semi-structured interview (Kaye & Shea, 2000).

SEMI-STRUCTURED INTERVIEWS

Structured Interview for DSM-IV Personality Disorders (SIDP-IV) (Pfohl, Blum, & Zimmerman, 1997).

The SIDP-IV is a semi-structured clinical interview that was designed to provide both dimensional and categorical assessments of both DSM-IV personality disorders. An ICD-10 version is currently being developed (Kaye & Shea, 2000). The DSM-IV version of the SIDP-IV is available in two formats – one in which items are organised thematically, and the other in which items are organised according to personality disorder category. The thematic version of the

SIDP-IV contains 101 questions organised according to the following themes: Interests and Activities, Work Style, Close Relationships, Social Relationships, Emotions, Observational Criteria, Self-Perception, Perception of Others, Stress and Anger, and Conformity.

Kaye & Shea (2000) note that the SIDP-IV is one of the more established Axis II semi-structured interviews. Little has been published with regard to the psychometric properties of the SIDP-IV, but its DSM-III-R aligned predecessor was reported to have an acceptable reliability of 0.85 for the BPD scale (Reich, 1992). A six month test-retest kappa of 0.70 has also been reported and J. Reich (1992) also reported that the SIDP-R was a useful instrument for diagnosing BPD.

Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II) (M. B. First et al., 1997; M. B. First, Spitzer, R.L., Williams, J.B.W., et al, 1997)

The SCID-II is a semi-structured interview designed to assess DSM-IV Axis II personality disorder. It is organised according to specific personality disorder categories, and the SCID-II has the capacity to categorically and/or dimensionally assess personality disorder.

A key design feature of the SCID II is a 119 item, pre-interview self-report instrument designed to indicate which personality disorders are likely to be present. The measure is designed to provide some false positives, but no false negatives. The principal underpinning the development of this aspect of the instrument was that if specific personality disorder symptoms are not present in

the screening instrument, there is little utility in examining them more completely in an interview.

If a respondent is unlikely to meet criteria for a particular personality disorder during the interview phase of the examination, there are 'skip outs' that allow the examiner to proceed to examine for the presence of the next personality disorder on the schedule. Reich (1992) reports that the SCID-II is the instrument of choice when a comprehensive screening for personality disorders is required. Reich reports that the use of the SCID II is not as widespread as would be anticipated and this is due to a lack of published reliability and validity data. It also appears that the SCID II over-diagnoses BPD resulting in an unacceptably high level of false positive diagnoses. Despite this, the SCID-II is used extensively in personality disorder research, and appears to be one of the principal diagnostic methods for diagnosing DSM-IV personality disorder.

Diagnostic Interview for Personality Disorders (DIPD) (Zanarini, Frankenburg, Sickel, & Yong, 1996).

The Diagnostic Interview for DSM-IV Personality Disorders is a semi-structured interview which assesses all DSM-IV Axis II disorders according to a categorical personality disorder model. The structural organisation and scoring system of the DIPD-IV is reminiscent of the DIB-R, which was also developed by Zanarini. Each Axis II disorder is assessed through the use of questions which examine the criterion pertaining to the relevant personality disorder. Each criterion is scored as zero for 'absent', one for 'present but of uncertain clinical significance', or two for 'present and clinically significant'. A confirmation of a specific personality disorder diagnosis is made when sufficient criteria with

scores of two are realised (i.e., five criteria for borderline personality disorder). The inter-rater reliability median kappa coefficients ranged from 0.58 to 1.0 for all Axis II personality disorders; the median kappa for borderline personality disorder was 0.68. The median kappa for 1-week test-retest reliability for all personality disorders was 0.69; the median test-retest kappa for borderline personality disorder was 0.69 (Zanarini et al., 2000).

Kaye & Shea (2000) note that the DIPD-IV appears to be a well constructed instrument, but there is limited data on reliability and validity. It appears that it is an instrument of promise that at this time has had insufficient 'field time' to suggest that it represents the most effective measure for detecting BPD.

Personality Disorders Interview – IV (PDI-IV) (Widiger, Mangine, Corbitt, Ellis, & Thomas, 1995).

The PDI–IV is a semi-structured interview designed to diagnose DSM-IV personality disorders in either a dimensional or categorical format. Two versions of the protocol have been developed – one in which items are grouped by personality disorder, and the other in which specific themes are explored. The thematic version includes 93 questions organised into the areas of Attitudes Toward Self, Attitudes Toward Others, Security of Comfort With Others, Friendships and Relationships, Conflicts and Disagreements, Work and Leisure, Social Norms, Mood and Appearance, and Perception. In addition, both versions include questions that examine negativistic and depressive personality disorders features.

Kaye & Shea (2000) note that one of the strengths of the PDI-IV is a detailed elaboration of each of the DSM-IV personality disorders and substantial

information regarding each diagnostic criterion. The primary weakness has been an absence of independent psychometric data on the PDI-IV, although the authors have reported acceptable reliability and validity indices. Further data is required on this instrument before it might be regarded as a more promising instrument for diagnosing BPD.

2.3.5. Summary and Conclusions

Although there are a number of competing systems for diagnosing BPD, most studies utilise DSM-III-R or DSM-IV formulations. This issue is also important for a second reason. There is a significant literature which criticises the DSM Axis II system on a number of theoretical and methodological grounds. These criticisms are generally applicable to the other diagnostic systems, and when this literature is reviewed in Section 2.6, it will be assumed that the criticism applies to all systems unless explicitly indicated otherwise.

2.4. EPIDEMIOLOGY OF BPD

There is limited data concerning the epidemiology of BPD. Widiger & Weissman (1991) argue that this is in part due to the technical requirements involved in making the diagnosis of BPD which include extensive training and clinical experience on the part of assessors, and the use of diagnostic instrumentation. As a result, there is a relative paucity of studies examining the prevalence and incidence of BPD.

2.4.1. Prevalence of BPD

Two sources of prevalence data are available on BPD. The first involves examining prevalence rates in non-clinical, community based samples. The second source examines prevalence rates in clinical populations. This evidence is selectively reviewed.

Prevalence of BPD in Non-Clinical Populations

There are at least six studies that examine prevalence rates for BPD in non-clinical populations. Five of these studies have derived their cohorts from large-scale epidemiological studies (Bernstein et al., 1993; Bezirganian et al., 1993; Reich, Yates, & Nduaguba, 1989; M. Swartz et al., 1990; Zimmerman & Coryell, 1989), and one study has examined BPD prevalence in a cohort of freshman students from one U.S. university population (Trull, 1995). With the exceptions of Bernstein et al. (1993), Bezirganian et al. (1993) and Trull (1995), these studies return consistent community prevalence rates. The apparent consistency of the community based prevalence rates for BPD can then in turn be used to interpret the usually significantly elevated rates of BPD in clinical samples.

One of the earliest prevalence studies employed a self-report methodology to estimate BPD prevalence. Reich et al. (1989) randomly sampled 401 members of a small, Midwestern U.S. community using the Personality Diagnostic Questionnaire (PDQ) (Hyler, Rieder, Spitzer, & Williams, 1983). A 1.3% prevalence rate for BPD was reported. One of the issues with this study concerned the possibility that self-report methods tend to overdiagnose personality disorder when compared to semi-structured interviews (Widiger & Trull, 1993). Subsequently, a number of studies have employed semi-structured interview methods to examine BPD prevalence.

Studies employing semi-structured interview methods have returned similar prevalence findings to that of Reich et al. (1989) (M. Swartz et al., 1990; Zimmerman & Coryell, 1989). Zimmerman & Coryell (1989) reported a rate of 1.6% using the Structured Interview for DSM-III Personality Disorders (SIDP)

(Stangl, Pfohl, Zimmerman, Bowers, & Corenthal, 1985) with 697 first-degree relatives of schizophrenic, psychotic and non-psychotic depressives, and normal controls participating in a family study of psychiatric disorders. Swartz et al. (1990) reported a prevalence of 1.8% for 1,541 respondents interviewed with the Diagnostic Interview Schedule (DIS) (L. N. Robins, Helzer, Croughan, & Ratcliff, 1981) as part of the Piedmont Health Survey (PHS) component of the NIMH Epidemiologic Catchment Area Program.

In contrast to the previous studies that have examined prevalence rates for BPD in adult populations, three studies have reported prevalence rates in child and adolescent cohorts. These findings have returned significantly higher prevalence rates for BPD than their adult counterpart studies (Bernstein et al., 1993; Bezirganian et al., 1993; Trull, 1995). Bernstein et al. (1993) randomly sampled 733 youths aged between nine and 19 years. The sample was followed over a two year period and structured interviews were conducted with the participants and their mothers. The findings suggested that personality disorders reached a peak at age 12 in males, and age 13 in females with a subsequent decline in prevalence over time for both groups. The mean BPD prevalence rate for all participants was 7.8%, with a 7.1% prevalence rate for males, and an 8.5% prevalence rate for females.

Bezirganian et al. (1993) studied a randomly sampled, prospectively followed group of 776 children over 10 years. Initial interviews were conducted when the children were approximately six years old (Time 1), and follow up interviews were conducted when the cohort was 13.7 (Time 2) and 16.4 years (Time 3). BPD prevalence data was collected at Time 2 and Time 3. The prevalence rate at Time 2 was 10.5%, and at Time 3 was reported to be 7.3%.

Trull (1995) sampled 1,697 undergraduate students from a large U.S public university. They were administered three self-report inventories used for diagnosing BPD – the MMPI BPD Scale (L. C. Morey, Waugh, & Blashfield, 1985), the Personality Disorders Questionnaire – Revised (Borderline Personality Disorder Scale) (PDQ-RBPD) (Hyler & Rieder, 1987), and the Personality Assessment Inventory – Borderline Features Scale (PAI-BOR) (L. C. Morey, 1991). The study returned BPD prevalence rates of 21.1% on the PDQR-BPD, 13.1% on the MMPI-BPD, and 14.8% on the PAI-BOR.

The prevalence rates of the latter three studies (Bernstein et al., 1993; Bezirganian et al., 1993; Trull, 1995) contrast with the prevalence findings in the comparable adult studies (Reich et al., 1989; M. Swartz et al., 1990; Zimmerman & Coryell, 1989). It appears that there is an approximate five-fold increase in prevalence of BPD in child and adolescent cohorts than is the case for adult samples. There are at least two explanations that might account for this discrepancy. First, Trull's (1995) study employed self-report methods, and these are known to overdiagnose BPD (Widiger & Trull, 1993). Therefore, the elevated prevalence rates in the Trull (1995) study could conceivably be a reporting artifact associated with self-report methodologies. Second, BPD is understood to be a disorder of youth (Paris, 1999), and it is possible that the findings of Bernstein et al. (1993) and Bezirganian et al. (1993) are consistent with this broader finding. Despite this, it appears that the prevalence of BPD in the general adult community is approximately two percent, and in the range of eight to 21% in child/adolescent populations. These findings suggest the possibility that BPD is a developmental-maturational disorder which attenuates over time. This issue will be further considered below.

Prevalence of BPD in Clinical Populations

In contrast to the previously reported prevalence studies in non-clinical BPD populations, the studies examining prevalence in clinical populations have returned highly variable results. The evidence for this is selectively reviewed.

Widiger & Trull (1993) identified 55 studies conducted between 1975 and 1988 that enabled prevalence to be estimated relative to a known, specified population. In addition, the included studies were also methodologically rigorous in the sense that they employed operationalised diagnostic criteria, used interview methods to diagnose BPD, employed specific inclusion criteria, and controlled for sampling bias. This analysis yielded a total of eight outpatient and 14 inpatient studies. The average BPD prevalence rate for the outpatient studies was estimated to be eight percent, and 15% for the inpatient studies.

Because the Widiger & Trull (1993) study carefully controlled for methodological issues, it is possible that their findings under-represent the prevalence of BPD in clinical populations. Other studies have reported prevalence rates of 35%, 36%, and 49% respectively using retrospective chart review methods (Koenigsberg, Kaplan, Gilmore, & Cooper, 1985; A. W. Loranger, 1990; A. W. Loranger, Oldham, & Tulis, 1982), 32% using clinician completed diagnostic ratings of psychotherapy patients (L. Morey, 1988), 80% using a LEAD (Longitudinal Expert Evaluation Using All Data) diagnostic method (Skodol, Rosnick, Kellman, Oldham, & Hyler, 1988), 35%, 56% and 55% respectively using semi-structured interviews in inpatient settings (Sansone, Gage, & Wiederman, 1998; Soderberg, 2001; M. S. Swartz et al., 1989), and 47% in primary care settings (Sansone, Whitecar, Meier, & Murry, 2001).

In contrast, other studies have reported much lower prevalence rates than reported by Widiger & Trull (1993). Shea, Glass, Pilkonis, Watkins, & Docherty (1987) studied participants involved in treatment in the NIMH 'Treatment of Depression Collaborative Program' (TDCP) and found that 35% met criteria for at least one personality disorder diagnosis, but only two percent of the sample met DSM-III criteria for BPD. Similarly, Oldham & Skodol (1991) compiled a clinical database for the New York State Hospital system for the calendar year 1988. They examined the prevalence of DSM-III personality disorders in state hospital patients. 11% of the population received a personality disorder diagnosis and 1.9% of the total patient population met BPD criteria.

Finally, a limited number of studies report the prevalence of BPD cross-culturally (Onchev & Ganev, 2000; Pinto, Dhavale, Nair, Patil, & Dewan, 2000). Onchev & Ganev (2000) examined patients from three psychiatric settings and reported period prevalence rates of 3.8% for a closed-door clinic, 5.2% for a 'day centre', and 1.4% for an outpatient service. The authors suggest that BPD is rare in Bulgaria. In contrast, Pinto et al. (2000) reported elevated rates of BPD in a consecutively recruited group of Indian suicide attempters. They speculate that BPD is an under-reported condition in India. Further research on international prevalence rates is required.

Widiger & Weissman (1991) argue that the high prevalence rates reported for BPD challenge the validity of the diagnosis. They note that the prevalence rate of BPD has increased exponentially in a short period of time from its absence on DSM-II, to being the most frequently diagnosed personality disorder on either the DSM-III, or the DSM-III-R. They suggest that one way of assessing the veracity of this claim would be to examine the incidence of the disorder, that

is, the rate of new cases diagnosed in the population during a specified time period. Unfortunately, there is little published data which examines this question. A small number of studies do, however, provide data which can in part address this issue. These are considered in Section 2.4.2.

Prevalence rates in clinical populations appear to be highly variable. It appears that prevalence of BPD is affected by a number of factors including the use of operationalised diagnostic measures, the method employed to diagnose BPD, the extent to which sampling bias affects recruitment, the nature and extent of co-morbidity in the sample, and whether explicit inclusion or exclusion criteria are used to delineate the sample.

Gender-Prevalence of BPD

The prevailing view suggests that BPD is diagnosed more frequently in women (Paris, 1999; M. Swartz et al., 1990). This has led some to argue that the DSM in general and the diagnosis of BPD in particular are sex-biased. Kaplan (1983) argues that 'masculine-biased' assumptions concerning health and pathology have been codified in diagnostic criteria, and these criteria then influence the diagnosis and treatment rates for each particular disorder. According to this perspective, psychopathology represents a gendered construction of clinical phenomena which has the effect of 'pathologising' women's experience.

At least two reviews of the gender-prevalence of BPD have been conducted (Akhtar, Byrne, & Doghramji, 1986; Widiger & Trull, 1993). Akhtar et al. (1986) reviewed 23 studies that provided data on gender. They reported that more women than men were found to meet BPD criteria, although they did not provide data concerning sex ratios, and whether these ratios met statistical

significance. Akhtar et al. reported that the averaged prevalence of female-diagnosed BPD was 77%, with some studies reporting a female-typed BPD rate of between 32% and 100% in various studies. Two of the studies reported that fewer than 50% of the sample were female, and 16 studies reported that a minimum of 70% of the sample were female. The authors concluded that the findings of a greater gender prevalence of BPD in women were likely to be associated with a combination of diagnostic bias and referral artifact. Unfortunately, Akhtar et al. only provided descriptive analyses of the data, and did not subject their data to any form of statistical analysis and/or meta-analytic examination, thus limiting the value of their findings.

Widiger & Trull (1993) identified 75 studies that provided unbiased estimates of the percentage of women diagnosed as BPD using either DSM-III, or DIB criteria. They found that on average 76% of the participants in each of the studies were women. Furthermore, they also found that those studies employing a semi-structured interview method obtained a significantly higher percentage of BPD women participants.

One of the possible interpretations of these findings suggests that clinicians might over-diagnose BPD in women. There is however, some evidence which contradicts this interpretation.

An early study that challenged the assumption of a gender-linked bias in BPD was reported by Kass, Spitzer, & Williams (1983). They examined data from two studies. The first study consisted of 2,712 patients (1,297 male, 1,415 female) reviewed as part of a DSM-III field trial, and the second study consisted of clinical evaluations of 531 outpatients (201 male, 330 female). In both studies, diagnosis was made by clinical interview with raters explicitly trained to rate

personality disorder according to DSM-III criteria. Assessors were blind to the research hypotheses, and the findings indicated that BPD was diagnosed equally frequently in men as in women.

Funtowicz & Widiger (1995) also examined the issue of gender bias in the diagnosis of personality disorders. They examined whether participants at the threshold for a 'female-typed' personality disorder display less dysfunction than participants at the threshold for a 'male-typed' personality disorder. No significant differences were reported between males and females on BPD measures as examined by the PDQ-R or the MCMI-III. Of most significance, the diagnostic thresholds for the personality disorders which are considered to occur more often in females were similar to the thresholds for personality disorders regarded as occurring more often in males. These results suggest that the presumption of a gender bias in the diagnosis of BPD may be unfounded.

These findings are also supported by the work of Morey & Ochoa (1989) who found that the gender of the evaluating clinician rather than the gender of the patient was the central feature that determined the over-diagnosis of personality disorders. Their findings indicated that female and inexperienced psychodynamically oriented clinicians were most likely to over-diagnose BPD. These findings suggest that the relationship between gender and BPD diagnosis is probably far more complex than a simple gender-linked phenomenon.

Widiger & Spitzer (1991) note that the view that personality disorders are gender-biased is an issue of considerable importance. They argue that attempts to resolve this matter empirically have been flawed by a combination of conceptual and methodological difficulties that have resulted in misinterpretations of data and the continued use of inappropriate research designs. They emphasise that

there are a number of different types of what they term ‘sex bias’. These include ‘social-cultural aetiologic sex bias’, ‘sampling sex bias’, and ‘diagnostic sex bias’. The bias that they identify as being of primary concern is that of ‘diagnostic sex bias’ which occurs when there is a differential prevalence of Type I or Type II error more often for one gender than the other. Two forms of this type of gender bias can be distinguished: bias that arises through the use of criteria for the personality disorder that are biased (criterion sex bias), or bias that arises through the instruments that are used to make the diagnosis (assessment sex bias).

Widiger & Spitzer (1991) argue that the critique of the DSM is often associated with ‘criterion sex bias’ or ‘assessment sex bias’. Importantly, most critiques fail to identify which form of bias they are concerned with. They argue that as a result, most critiques fail to distinguish between aetiologic, sampling, assessment, and criterion sex bias, and this prevents any conclusions regarding the presence of gender bias.

In contrast, a recent review by Skodol & Bender (2003) maintains the argument that BPD is diagnosed predominantly in females with an approximate 3:1 female to male gender ratio. They argue that the magnitude of this ratio is pronounced for a mental disorder. They question whether the higher rate of BPD in women is a result of a methodological or diagnostic bias, or a reflection of biological or socio-cultural factors. They argue that the differential gender prevalence of BPD in clinical settings is associated with sampling bias, and that the true prevalence by gender is unknown.

It appears that the issue of gender prevalence in BPD remains a controversial issue. It also appears that elevated rates of gender-prevalence for

women with BPD might be an artifact associated with sample bias, and this is a speculation offered by Paris (1999). An alternative perspective suggests that BPD is one part of a broader personality disorder that includes Antisocial Personality Disorder (ASPD). ASPD is known to be over-represented in males, and Paris (1999) suggests that BPD and ASPD might reflect different components of the same underlying pathology whereby women express a more 'affective' feature of the disorder, and men express a more 'impulsive' component. Clearly, further research on gender prevalence in BPD is indicated.

2.4.2. Incidence of BPD

Incidence data on BPD is rare (Widiger & Wiseman, 1991). Only two studies have been identified that examine incidence rates for BPD (Mors, 1988; Mors & Sorensen, 1994). Mors (1988) studied all first admission cases drawn from the Danish Psychiatric register for the period 1970 to 1985 inclusive. Mors employed the ICD-8 diagnostic codes of 301.83 and 295.59 in order to develop a cohort of borderline patients for that period. The results suggest that the incidence rates of borderline conditions increased for both males and females throughout the period 1970 to 1985 inclusive, and that the main increase in incidence was found in the age group 15 to 34 years of age. 80% of BPD diagnoses were made within this age range.

Mors and Sorensen (1994) subsequently studied 150 first admission psychiatric patients in the age group 18 to 49 years from a catchment area of 218,000. All participants were interviewed with the Present State Examination (10D – Danish Draft Version) (J. Wing, Cooper, & Sartorius, 1974; J. K. Wing et al., 1990), and the Personality Disorder Examination (A. Loranger, 1988). 23% received at least one DSM-III-R personality disorder diagnosis, and the majority

of diagnosed personality disorders were dependant and avoidant personality disorders. DSM-II-R BPD diagnoses were only made in three cases. The findings of these studies suggest a low incidence rate for BPD. Further research is required to clearly establish the incidence rate of BPD.

2.4.3. Summary

The findings from these studies suggest that BPD achieves a community prevalence rate of approximately two percent, with elevated prevalence rates in clinical samples. There appears to be significant variation in the prevalence rates for BPD in clinical populations depending upon the nature of the clinical population, the methods employed to diagnose BPD, the nature and extent of co-morbidity in the sample, and whether explicit inclusion/exclusion criteria were used to delineate the sample. There is equivocal evidence concerning whether women are over-represented in BPD diagnoses, and there is insufficient information concerning incidence rates in BPD to confidently claim the true incidence rate for BPD. This appears to be an area where further research is indicated.

Another significant implication emerging from the prevalence findings is a significant age-effect for BPD, with apparent higher rates of prevalence at younger ages. This suggests that BPD might be a 'developmental-maturational' disorder whose morbidity diminishes over time. This conclusion is also consistent with the findings from the studies of long-term outcome for BPD which conclude that a significant group of borderlines experience symptom remission over a long-term course (Paris, 1999; Stone, 1992). It is also consistent with the findings of Snyder, Pitts, & Gustin (1983a) who argued that it is uncommon to find patients over age 40 who meet criterion for BPD and

Stevenson, Meares, & Comerford's (2003) findings that impulsivity diminishes in severity over time. If these findings are replicable, then it would appear that BPD is a developmentally organised disorder which might best be viewed as a disorder of adolescence/early adulthood which generally remits by middle age.

2.5. THE COURSE OF BPD

Zanarini, Chauncey, Grady, & Gunderson (1991) reviewed outcome studies of BPD and categorised them into studies of short and long-term outcome. Short-term studies have followed up original cohorts for a period of up to five years after discharge from index contact. Long-term follow-up studies have typically followed up cohorts for a minimum of 15 years after discharge from index admission. Stone (1992) argues that chronic conditions such as personality disorders require long-term follow-up. As a result, he argues that short-term follow-up is of little value in identifying the 'life trajectory' of the participant. Despite this critique, this section will review both the short-term and long-term outcome studies of BPD.

2.5.1. Short-Term Outcome Studies of BPD

A number of studies have examined short-term outcomes in BPD. They include follow-up research on inpatient treatment studies (Clarke, Hafner, & Holme, 1995; Dolan, Warren, & Norton, 1997; Grinker et al., 1968; Modestin & Villiger, 1989; Senol, Dereboy, & Yuksel, 1997; Tucker, Bauer, Wagner, Harlam, & Sher, 1987; Werble, 1970; Zanarini, Frankenburg, Hennen, & Silk, 2003), various forms of outpatient treatment studies (Bateman & Fonagy, 2001; Karterud et al., 1992; Linehan, Heard, & Armstrong, 1993; Mehlum et al., 1991; Monsen et al., 1995; J. Stevenson & Meares, 1992; Wilberg et al., 1998), or other studies that have employed various methodologies including naturalistic

prospective-longitudinal outcome research (Barasch, Frances, Hurt, Clarkin, & Cohen, 1985; Gunderson et al., 2003; Links, Mitton, & Steiner, 1990; Mitton et al., 1997; Najavits & Gunderson, 1995).

Treatment Studies

Short-term follow-up treatment studies have generally returned inconsistent findings. A number of studies have reported outcomes indicating either deteriorated or unimproved outcomes (Clarke et al., 1995; Grinker et al., 1968; Karterud et al., 1992; Mehlum et al., 1991; Modestin & Villiger, 1989; Werble, 1970; Wilberg et al., 1998), whilst other studies suggest minimal to significant short-term improvement (Bateman & Fonagy, 2001; Dolan et al., 1997; Linehan et al., 1993; Meares, Stevenson, & Comerford, 1999; Monsen et al., 1995; Senol et al., 1997; J. Stevenson & Meares, 1992; Tucker et al., 1987; Zanarini et al., 2003).

Grinker et al., (1968) followed up 41 of 51 borderline patients from their original study three and a half years post discharge. Whilst 80% were resident in the community, 66% of the sample rated themselves as significantly worse off, unimproved, or only marginally improved since discharge. 30% of the original sample had required rehospitalisation during the follow-up period. Although no patient deaths had been recorded, social functioning was generally poor, with only 17% leading an 'active' social life. 50% of the group reported continuing poor quality relationships with their families. Subsequently, Werble (1970) followed-up 28 of the Grinker et al. (1968) cohort seven years after discharge. Most participants were resident in the community, but 50% of the cohort had been readmitted at least once. Most admissions tended to be short-term, and their social functioning was reported to have generally deteriorated.

Modestin & Villiger (1989) followed-up 18 DSM-III BPD and 17 Other Personality Disordered (OPD) participants 4.6 years after index admission. All participants were blind-reviewed through the use of a semi-structured interview. At review, 80% of the BPD cohort was single compared with 35% of controls. The groups did not differ significantly on measures of anxious or depressive morbidity, but the BPD group reported higher levels of rehospitalisation post index admission. The authors concluded that the overall psychosocial functioning of the BPD cohort was not significantly different from the OPD group.

Mehlum et al. (1991) studied 97 DSM-III-R diagnosed patients consecutively admitted to a day unit specialising in the management of personality disorders. The sample consisted of 28 men, and 69 women treated for an average length of 5.5 months. 73 of the original cohort were followed up on average 2.8 years after the completion of treatment by an interviewer blind to the patient's original diagnosis and subsequent information concerning their condition. The 24 cases that were not followed up included 12 participants who refused to participate, nine who had provided incomplete data sets, and three participants who had died (one via suicide). 29 of the original participants met DSM-III-R criteria for BPD at index admission. At follow-up, they displayed a moderate reduction in symptom level, a stable level of social adjustment, and a significantly higher rate of rehospitalisation than all other personality disorder groups with the exception of Schizotypal Personality Disorder which was re-hospitalised at approximately the same rate. Mehlum et al. compared the results of their borderline cohort with three other studies (Modestin & Villiger, 1989; J. C. Perry, 1985; Tucker et al., 1987) and found that the short-term outcomes for

borderlines were worst in their study. Despite this, the sample reported overall reduction in symptom level from admission to follow-up.

Karterud et al. (1992) studied 97 consecutively admitted patients to a day unit for the treatment of severe personality disorders. All patients were diagnosed according to DSM-III-R criteria, and 76% were found to have one or more Axis II disorders. BPD diagnoses constituted the most frequently occurring personality disorder, with 35% of the total sample meeting criteria for the disorder. The average length of stay (LOS) for all patients was 171 days. BPD patients demonstrated modest change across the course of day treatment, with change scores on the Health-Sickness Rating Scale suggesting that very few BPD cases achieved a level of functioning at discharge indicating that they did not require further treatment.

Clarke et al. (1995) followed up 47 Australian BPD patients diagnosed according to DSM-III-R criteria three years after their index admission. At follow-up, the authors reported that BPD remained the primary diagnosis for 89% of the cases available for analysis, and two participants (4%) had committed suicide. They also found that during the three year follow-up period, 74% of participants experienced at least one additional inpatient admission. The sole predictor of readmission frequency was the number of admissions prior to the index admission. The authors concluded that the study supports BPD as a valid diagnosis, and that treatment for this condition within the state psychiatric system was largely inconsistent and ineffective.

Senol et al. (1997) followed up 61 BPD inpatients up to four years post discharge. Two had committed suicide and 45 were included in the follow-up study. A semi-structured interview confirmed that 95% of the cohort continued to

meet BPD criterion, although other measures of psychiatric morbidity suggested that there was some improvement in functioning. Affective disorders were diagnosed in three-quarters of the cohort, and the lifetime prevalence for BPD was estimated to be 100%.

Wilberg et al. (1998) followed up 146 patients treated in a Day Unit specialising in the management of personality disorders. Of this original cohort, 48 participants met DSM-III-R criterion for BPD. Overall, the authors found that for all personality disorder configurations, there were four distinct courses of progression which were referred to as good, fair, late improvement, or poor. The authors note that those with the poorest prognosis were originally defined as meeting criteria for BPD.

In contrast, a number of studies report favourable short-term outcomes for BPD. Tucker et al. (1987) reported a two year follow-up of 40 patients admitted to a long-term, intensive inpatient treatment programme who met the criteria for 'Borderline Personality Organisation' (Kernberg, 1984). At one year follow-up, drug and alcohol use had reduced significantly, peer relationships had improved, and suicidal or self-destructive feelings had also significantly reduced. At two years follow-up, a significant number of patients reported improved social functioning as assessed by increases in the number of close relationships, and overall level of functioning was assessed to have improved significantly over time.

Stevenson & Meares (1992) and Meares et al. (1999) provided a specific form of psychoanalytically informed twice-weekly outpatient psychotherapy to 30 patients diagnosed as BPD according to DSM-III criteria. Treatment was provided for a period of 12 months by supervised trainee therapists, and outcome

measures included frequency of drug use, number of medical consultations, episodes of violence and self-harm, hospital admissions, loss of time from work, and number of DSM-III criteria fulfilled. The findings indicated that on every dependent variable, participants demonstrated a statistically significant improvement over the course of treatment and at 12 month follow-up. Most significantly however, 30% of participants were reported to no longer meet DSM-III BPD criteria at one year follow-up.

Linehan et al. (1993) reported a one year naturalistic follow-up on 39 female participants who met DSM-III-R criteria for BPD. This cohort had been involved in an RCT to evaluate the efficacy of Dialectical Behaviour Therapy (DBT) compared to a 'treatment as usual' (TAU) condition. The results indicated that during the year of treatment, the 'parasuicide repeat rate' and inpatient admission rate were significantly lower for participants completing DBT than for participants receiving TAU. At six months follow-up, participants receiving DBT reported fewer parasuicidal episodes, and fewer medical consultations. This latter finding was an artifact of the higher parasuicide rate amongst the TAU group. At 12 months follow-up, there was a difference in parasuicide rates between the DBT and the TAU group, and the DBT group reported significantly fewer inpatient admissions. At both six and 12 month follow-up participants in the DBT group reported significantly better employment performance and higher scores on global adjustment.

Monsen et al. (1995) conducted a five-year, prospective follow-up of 25 patients who received an average of 25.4 months of intensive psychotherapy from a specialist personality disorders unit. At treatment conclusion, 75% of the cohort that had originally met DSM-III criterion, no longer did so. At five-year

follow-up, all measures including DSM-III diagnostic status showed a significant reduction in morbidity.

Dolan et al. (1997) report on a one year follow-up of patients referred to a therapeutic community treatment programme for 'severe personality disorder' (SPD). Because of the high number of co-morbid personality diagnoses for the participants in the study, the authors did not analyse outcome data by personality disorder category. The data nevertheless suggested that 80% of participants receiving up to one year of inpatient milieu therapy met the criteria for DSM-III-R Borderline Personality Disorder (BPD) whereas 81% of control participants who did not receive treatment met DSM-III-R Borderline Personality Disorder (BPD) criteria. At one year follow-up, the data suggested that both the control and milieu treated group demonstrated improvement, but the admitted group demonstrated significantly greater improvement than controls. The data also suggested that treatment outcome was positively associated with length of stay (LOS) suggesting that treatment duration might have significant implications for the short-term course of the disorder.

Bateman & Fonagy (2001) followed up 44 patients who had received a psychoanalytically oriented, 'partial hospitalisation' programme for BPD. They were assessed quarterly after completion of the inpatient phase, with measures of suicide and readmission frequency, depression, anxiety, general distress, interpersonal functioning, and social adjustment. The results suggested that those who completed the programme maintained gains but also demonstrated continued improvement in comparison to a control group that received standard psychiatric treatment.

As part of the McLean Study of Adult Development, Zanarini et al. (2003) prospectively tracked 290 DSM-III-R BPD and 72 non-BPD DSM-III-R Axis II personality disordered inpatients from index admission over the following six years. 94% of the surviving cohort was reassessed at two, four and six years post index admission by interviewers blind to original diagnostic status. Approximately 35% of the BPD cohort no longer met BPD criteria at two-year follow-up, with nearly half (49.4%) no longer meeting BPD criteria at four-year follow-up. 68% of the index cohort no longer met criteria at the six year follow-up, and over the course of the entire study, just under three-quarters (73.5%) no longer met criterion for BPD. None of the other-personality disordered comparison group met BPD criteria during the follow-up period. Zanarini et al. (2003) noted that impulsive symptoms resolved most quickly whilst affective symptoms remained the most intractable feature of borderline pathology. They note that the prognosis for severely ill borderlines appears to be better than previously recognised.

Other Studies

Barasch et al. (1985) conducted a three year follow-up study of 10 patients diagnosed with a DSM-III Borderline Personality Disorder (BPD), and a reference group of 20 patients diagnosed with other personality disorders. At follow-up, 60% of the original BPD group maintained threshold for the disorder, and 30% just failed to meet criteria. Of the non-BPD reference group, 80% continued to report non-Borderline Personality Disorders, whereas 15% attained BPD status at the three year follow-up. An initial diagnosis of BPD predicted a follow-up diagnosis of BPD with a sensitivity of 60%, and a specificity of 85%, and the presence or absence of BPD was consistent over the three year period in

77% of cases. The findings of this study suggest that Borderline Personality Disorder (BPD) might not be a stable entity, or that it operates along a 'dormant-active' continuum.

Links et al. (1990) reported on the stability of BPD diagnoses and the variables that predict the continuation of borderline features in a sample of 65 participants followed up two years after index assessment. The authors report that at follow-up, 60% of participants retained a diagnosis of BPD (DIB Positive), whereas 40% no longer met the criterion for BPD (DIB Negative). During the follow-up period, the DIB positive group experienced more episodes of psychiatric disorder than did the DIB negative group, and there was a trend for the DIB positive group to abuse substances at a greater rate than the DIB negative group. The variables that predicted the stability of BPD included impulsiveness, a history of childhood trauma, younger age of onset, and positive family history of psychiatric disorder.

Najavits & Gunderson (1995) studied 37 Gunderson Criteria (DIB) BPD participants prospectively over a three year period. Participants were re-examined annually for three years after admission to the study. The authors report a 46% attrition rate across the three years of the study. The 54% of participants available for analysis at the completion of the study reported significant improvement in the areas of global functioning, borderline traits, social functioning, substance abuse, transient psychotic symptoms, somatisation, depression, impulsivity, and work functioning. The key issue with regard to the short-term improvement of participants reported in this study is the failure to report on the 46% of participants not investigated in this study. The authors also report that four of the original 37 participants reported poorer DIB scores at three

year follow-up than at baseline. This finding suggests variable outcomes for BPD, implying the possibility of multiple pathways for the course of the disorder. These include a pathway to improved functioning, and a pathway for a small but significant minority of BPD's to experience deteriorated functioning. This is a theme that has been addressed in detail by Stone (1992), and these findings are reported in Section 2.5.2.

Mitton et al. (1997) followed up 14 sexually abused (abused group) BPD participants from their original 1990 cohort with 14 matched non-sexually abused (non-abused group) BPD participants also from the original 1990 cohort. Reanalysis of the 1990 data suggested that the abused group was more impaired than the non-abused group at index assessment. Parallel improvement was observed between the abused and non-abused groups on measures associated with borderline symptomatology and functioning, assessment of psychiatric impairment, and measures of global functioning. Abused borderline participants were however, more likely to experience borderline symptoms associated with affective instability, dysphoria, intolerance of being alone, and suicidal attempts or gestures. The authors also report that the scores for the abused borderline group were similar to the non-abused group scores collected some seven years previously leading them to speculate that the borderline abused group lags behind the non-abused borderline group by a factor of about seven years. This is clearly a controversial finding and is deserving of further research attention. If this finding is confirmed by subsequent research, it will confirm that abused borderline patients require longer periods of time to consolidate their recovery.

Trull, Useda, Conforti, & Doan (1997) report stability coefficients for a number of BPD measures from a two-year follow-up study of non-clinical young

adults who, at study entry, exhibited a significant number of BPD features. At two year follow-up, individuals originally defined as BPD positive were more likely to experience academic difficulties, to meet lifetime criteria for mood disorder, and to report ongoing interpersonal dysfunction than their peers. The findings suggest that BPD features are associated with poorer outcome even within a non-clinical population.

Gunderson et al. (2003) have reported on a sub-sample of carefully diagnosed BPD patients for whom prospective follow-up data was collected as part of the Collaborative Longitudinal Personality Disorders Study (CLPS). From an index group of 160 BPD cases admitted to the study, a sub-sample of 18 cases were identified who reported fewer than two of nine DSM-IV criteria after the first six months of the study and maintained this reduced criterion rate six months later. Follow-up data was collected on this sub-sample after two years and only one of the original 18 had relapsed into BPD status after two years. Whilst one of the original cases was judged to have been misdiagnosed at index diagnosis, the most important determinants of remission were situational change and resolution of co-morbid Axis I disorders. This study raises the possibility that for a small but nevertheless significant proportion of BPD cases, the course of the disorder might be very brief. Alternately, this finding calls into question either their status as BPD cases, or alternately, calls into question the validity of the diagnosis itself. It raises a significant challenge to the current understanding of the course of the disorder (Paris, 1999), and requires further research to confirm the validity of this finding.

Zanarini et al. (1991) note that the generalisability of the findings from short-term studies of BPD outcome have been limited due to a range of

methodological shortcomings which include small sample sizes, absence of control groups, poor or non-existent BPD criteria, the use of unstructured methods for making BPD diagnoses, non-blinded assessment of outcome status, and little emphasis on the prediction of outcome. Despite these objections, Zanarini et al. suggest that three findings emerge from the literature on the short-term course of BPD. First, borderline participants continue to experience significant difficulties for periods of at least seven years post index assessment, although the studies of Najavits & Gunderson (1995) and Gunderson et al. (2003) contradict this conclusion. Second, their level of functioning is similar to that of schizophrenic and other personality disordered groups, and third, borderlines did not develop schizophrenia, but retained a core instability characteristic of the initial borderline diagnosis. Finally, the evidence from the limited number of studies of follow-up from psychotherapy suggest that those BPD patients receiving formal psychotherapy probably enjoy better outcomes in the short-term (Bateman & Fonagy, 2001; Linehan et al., 1991; Monsen et al., 1995; J. Stevenson & Meares, 1992), although the results of the Gunderson et al. (2003) contradict this conclusion. This general finding does, however, require further examination through the use of better defined and larger psychotherapy outcome studies.

2.5.2. Long-Term Outcome Studies of BPD

Five studies examining the long-term functioning in BPD have been reported. Stone (1992) refers to these studies as the Austen Riggs Study (Plakun, Burkhardt, & Muller, 1985), the Minneapolis Study (J. L. Kroll, Carey, & Sines, 1986), the Chestnut Lodge Study (Heinssen & McGlashan, 1988; McGlashan, 1986, 1992; McGlashan & Heinssen, 1988), the New York State Psychiatric

Institute (PI 500) Study (Stone, Hurt, & Stone, 1987), and the Jewish General Hospital Study (Paris, Brown, & Nowlis, 1987; Paris & Zweig-Frank, 2001). Because of the comparative recency of diagnostic criteria for BPD, most studies have retrospectively diagnosed BPD either from 'chart review' or by redesigning contemporary diagnostic instruments in order to diagnose participants retrospectively from DSM-II era clinical populations.

The Austen Riggs Study

Plakun et al. (1985) conducted a 15 year follow-up of 237 patients retrospectively diagnosed with either Borderline Personality Disorder (BPD), or Schizotypal Personality Disorder (SCZ) using DSM-III criteria. The findings suggested that BPD participants functioned better than schizophrenic participants at both baseline and follow-up, further supporting the view that BPD and schizophrenia are separate diagnostic entities. The data did not however, support the view that Major Affective Disorder (MAD) and BPD are separate diagnostic entities.

The Minneapolis Study

Kroll et al. (1986) followed-up 15 inpatient borderlines identified retrospectively by chart review on average 20 years post index admission. 87% of the sample were reviewed. Of the reviewed sample, 15% of participants had committed suicide, and 15% had died due to medical conditions not directly attributable to their borderline status. The remaining 70% were resident in the community. Follow-up review indicated that 22% of the sample had made a very poor long-term adjustment, 44% were rated as having made a fair adjustment, and 34% were functioning competently. Of note, 22% of the sample continued to

meet DSM-III criteria for Major Depressive Disorder some 20 years post index admission.

The Chestnut Lodge Study

McGlashan (1986; 1992) conducted a longitudinal study of BPD as part of a more comprehensive examination of the outcomes of schizophrenic (SCZ), affective disordered (AD), and borderline (BPD) patients. The study was retrospective, and employed operationally defined diagnostic criteria, demographic and predictor characterisation of samples, multidimensional measurement of outcomes, bias-testing of missing participant sub-samples, reliability testing of all measures, and independence of follow-up data collection from baseline predictor data collection.

Patients discharged from the Chestnut Lodge hospital between 1950 and 1975 and a smaller cohort of non-discharged patients from a comparable period were included in the study. Those participants without organic brain damage and aged between 16 and 55 years at admission who were treated for a minimum of 90 days formed the basis of the study. Outcome data was collected on average 15 years post discharge, with a range of two to 32 years. At follow-up, the BPD cohort was reported to be functioning comparatively well, with most living independently. Most BPD patients reported persisting psychopathology which was most often reflected in interpersonal conflict. Depression and substance abuse continued to characterise their ongoing difficulties.

The New York State Psychiatric Institute (PI 500) Study

Stone et al. (1987) followed up 464 of 550 consecutive admissions from the New York State Psychiatric Institute (referred to as the 'PI 500') for the years 1963 to 1976. Inclusion criteria required participants to have been an inpatient

for a minimum of three months, to be aged under 40 at index admission, and have an IQ of 90 or more. The study examined the long-term functioning of 251 of the 299 BPD participants re-diagnosed on DSM-III criteria or Kernberg's (1984) structural criteria. Stone reported that the average BPD patient functioned well at 16 years follow-up which contrasted with a schizophrenic comparison group. 42% of the BPD group returned scores suggesting that they had recovered from the disorder, 30% were rated as 'good,' 17% in the 'fair' range, and 11% in the 'incapacitated' range.

Stone et al. (1987) also report that over half of the borderline sample had worked in excess of 75% of the time since discharge, 17% had worked for approximately 50% of the time since discharge, and 17% had worked for less than 50% of the time, or not at all. In addition, 55% of the borderline sample were rated as working at complex jobs, 43% were rated as working at relatively uncomplicated jobs, and two per cent worked at menial and very simplified jobs. Fewer than half (47%) married, and less than a quarter (22%) had children. Although the long-term clinical picture for borderlines in this study appears generally positive, nine per cent of the sample had suicided at 16 year follow-up. This rate of morbidity was similar to the schizophrenic control sample.

The Jewish General Hospital Study

Paris et al. (1987) chart reviewed 322 patients diagnosed as borderline using a modified version of Gunderson's Diagnostic Interview for Borderlines (DIB) for the years 1958 through 1978. Of the originally identified cohort of 322 participants, Paris et al. (1987) formally reviewed 100 participants on average 15 years after discharge. The results of the study suggested that active borderline pathology diminished over the course of time. The evidence suggested that at

long-term follow-up, the majority of borderline participants no longer met criteria for BPD, and experienced significantly reduced rates of impulsive, affective, and psychotic symptoms. In addition, the data suggested that the interpersonal relationships of the cohort were less chaotic, although this is offset to some degree by their apparently reduced rates of social involvement. This latter finding is consistent with the findings of McGlashan (1986) and Stone et al. (1987). The findings of the study generally suggest significant improvement in borderline symptomatology over the course of time, although there was a significant suicide rate of 8.5%.

Paris & Zweig-Frank (2001) provided 27-year follow-up data on the original Paris et al. (1987) cohort. 64 of the original 100 participants were followed up by telephone and administered a series of measures including the DIB-R and the DSM-III-R SCID-II. The majority of BPD participants no longer met BPD criteria with only five (5) of the 64 continuing to meet BPD criteria. Approximately 22% of the cohort continued to meet DSM-III-R criterion for Dysthymia, and approximately 10% of the original cohort had committed suicide. This finding suggests that patients with BPD diagnosed in adolescence or early adulthood continue to improve well into middle age.

Stone (1992) summarises the results of five long-term outcome studies on BPD reported in the 1980's. He notes that the global functioning of BPD participants at 10 to 25 year follow up across all five studies was remarkably consistent where approximately two thirds of participants were functioning 'fair' to 'well.' A significant number do, however, continue to meet criterion for Major Depressive Disorder. This finding lends weight to Akiskal's (1981) view of a link between BPD and the Affect spectrum disorders. Despite this, the prospect

for most borderlines suggests that BPD caseness and symptom severity are likely to remit by the onset of middle age, although Kroll (1993) suggests that improvements in functioning can be observed as early as the late 20's or early 30's. This finding provides considerable hope and calls for optimism in what is otherwise regarded as a difficult clinical area.

In a summarising the long-term outcome of BPD, Paris (1999; 2003b) argues that long-term improvement is most likely to be a naturalistic outcome rather than a specific treatment effect. Paris argues that whilst there is evidence of short-term improvement, BPD usually remits by middle age and he attributes this to a 'burnout' phenomenon which is speculated to be associated with either neurological maturation or social-learning. In contrast, Links & Heselgrave (2000) refer to an earlier prospective study (Links et al., 1999) that illuminates potential mechanisms of change in BPD. They suggest that impulsivity is a core factor in BPD and that it is the interaction between the severity of impulsivity with exposure to a 'healing relationship' that determines the course of BPD.

This interpretation of the long-term outcome literature makes considerable sense. It assists in explaining why some studies report short-term improvement (Gunderson et al., 2003; Najavits & Gunderson, 1995) whereas other studies have found that it requires more than a decade to elicit improvement (Paris et al., 1987). This perspective also assists in understanding why therapeutic approaches that emphasise the development of a relationship with the patient have enjoyed good outcomes in the short-term (Bateman & Fonagy, 2001; Linehan et al., 1991; Monsen et al., 1995; J. Stevenson & Meares, 1992).

In summary, the research on the course of BPD yields the following findings. First, short-term studies of outcome report highly varied outcomes. Second, long-term studies tend to lead to more favourable outcomes although a small proportion of cases continue to meet criterion at long-term follow-up. Long-term improvement appears to be dependent upon a number of factors which include preventing suicide (Linehan et al., 1991; Linehan et al., 1993), managing co-morbid mood and substance use disorder (Paris, 1999), and regulation of impulsivity and exposure to a healing relationship (Links & Heselgrave, 2000). In view of these factors, Paris (Paris, 2003a) considers that BPD is the most likely of all of the personality disorders to remit in the long term.

2.6. THEORETICAL AND METHODOLOGICAL CRITIQUES OF BPD

Although BPD is one of the most common psychiatric diagnoses and the most studied of the personality disorders (Widiger & Frances, 1989), it nevertheless remains a controversial entity (Tyrer, 1999). The controversies involve concerns about the diagnostic validity, reliability, assessment and heterogeneity of BPD. These issues are selectively reviewed.

2.6.1. Diagnostic Validity

There are at least four issues associated with the diagnostic validity of BPD. These include the employment of a trait formulation of personality, the continued use of a categorical rather than a dimensional system of diagnosis, the selection of optimal diagnostic criteria, and whether co-morbid psychotic symptomatology should be employed in the diagnosis of BPD (Widiger, Miele, & Tilly, 1992). Each of these issues is briefly reviewed.

Trait Formulations of Personality

A major issue related to the validity of BPD is associated with the model of personality on which the theory of personality disorders is based – a 'trait' conception of personality. Whilst the trait based conception of personality can be traced back to Hippocrates, modern trait theory is usually associated with the work of Allport (1931). The trait-based conception sees personality as based on core themes that influence behaviour in a particular domain (S. C. Cloninger, 1996). At the same time however, trait based conceptions consider personality to operate independently of environmental context. This perspective has been challenged by Mischel (1968) who has argued that tight control and reliable measurement of environmental variables is a better predictor of behaviour than is the measurement of the trait itself.

A second criticism of the trait-based conception of personality is that behaviour is usually understood to be caused by a number of co-occurring factors (S. C. Cloninger, 1996). This is an important issue that has also been identified by Koerner et al. (1996). They observed that there are significant problems with the current organisation of Axis I and Axis II on the DSM. Koerner et al. argue that a DSM style trait-based formulation of behaviour does not permit functional relationships between variables to be determined. Trait-based formulations do not allow controlling variables to be identified, and do not permit variability to be explored. More importantly, they argue that a trait-based approach can potentially interfere with the reliable assessment of functional behaviour. Koerner et al. argue that the DSM-IV (as one form of trait-based diagnosis) is ineffective as a predictor of behaviour, as trait conceptions do not assist in the functional analysis of behaviour or in treatment planning.

Whilst there is some limited empirical support for applied behavioural analytic approaches in the treatment of BPD (see Section 2.2.6) it would appear that this approach is somewhat overstated. Other behaviourally oriented therapists such as Linehan (Heard & Linehan, 1993; Linehan et al., 1991) and Arntz (1994) appear to successfully apply behavioural methodologies to the management of BPD within a DSM/trait based formulation. Nevertheless, the current conception of BPD is based upon a trait-based formulation, and this approach does have inherent difficulties. The essential problem with the trait-based approach is that it is argued by some to be a crude approach to understanding behaviour and fails to appreciate the subtle yet complex person-environment interactions (Mischel, 1968). This conceptualisation represents a serious challenge to the diagnostic validity of BPD. This conflict is rarely alluded to in the literature on BPD, but has important implications for a more sophisticated understanding of personality-based difficulties, and has significant implications for the prediction of behaviour.

Categorical Versus Dimensional Diagnosis

A second issue raised by a number of authors concerns the merits of a categorical versus dimensional diagnostic system for BPD (Livesley, 1998; Siever & Davis, 1991; Widiger, 2000; Widiger et al., 1992). Many of the issues associated with the categorical diagnosis of BPD cannot be understood without considering the personality disorders more generally. For example, some have argued that the current personality disorder groupings have been established on the basis of arbitrary criteria (Widiger et al., 1992). According to this view, a reconfiguration of the personality disorder categories is required in order to improve parsimony and to create personality disorder categories that reflect

diagnostic configurations suggested by methods such as cluster analysis research (Livesley, 1998).

A related issue concerns the demarcation of the 'borders' of BPD in relation to Axis I and other Axis II disorders. Tyrer (1994) argues that the borders of BPD are so flexible that it renders the diagnosis of BPD invalid. This issue is considered more fully in Section 2.6.4 when co-morbidity of BPD is examined.

The issue of categorical versus dimensional diagnosis of BPD probably represents the single most important validity issue. All contemporary diagnostic systems (Kernbergs BPO system, the Gunderson-DIB Group, ICD, and the DSM) diagnose BPD categorically. The fundamental diagnostic question with each of these systems involves determining whether the participant meets criterion for the disorder.

The main problem with the categorical approach is that personality dimensions are known to be continuous rather than discontinuous variables (S. C. Cloninger, 1996; Widiger, 2000). Because of this, Widiger et al. (1992) and Livesley (1998) have argued that the categorical approach includes a number of significant disadvantages. These include:

1. Diagnostic criteria for BPD are continuous variables, and this would not be expected in the case of categorical data. Rather, some evidence suggests that personality disorder diagnoses are effectively represented by normative models of personality which in turn suggest that personality disorder diagnoses represent extremes of 'normal' personality variation (Costa & McCrae, 1992; Widiger, 2000).

2. The inability to easily differentiate BPD from other co-morbid disorders, and from 'normal' conditions (Overholser, 1994).
3. Poor inter-rater reliability which in turn appears to be the result of arbitrary criterion cut-offs for personality disorder. For example, patients who meet four of the nine DSM-IV/DSM-IV-TR BPD criteria are technically not borderline (the requirement is five of nine criteria), but clinically they might demonstrate equivalent severity and/or morbidity as patients who meet a minimum of five BPD criteria. For example, Widiger, Sanderson, & Warner (1986) found that participants meeting four DSM-III-R criteria for BPD were similar to participants who met criteria for BPD (five criteria) in contrast to a group of controls who did not meet any criterion for the disorder.
4. The categorical approach to diagnosis also results in the loss of significant amounts of data. There are significant variations in the expression of borderline pathology between patients, and this is reflected in the original decision in the development of the DSM to establish multiple and optional decision making systems referred to as 'polythetic' criteria. Widiger et al. (1992) note that whilst there are many ways to be borderline, only one diagnosis is provided and no coding or referencing system is provided to describe the variation in symptomatology.
5. The lack of a theoretical rationale for the design of the BPD category. Of all the personality disorder categories, BPD is probably the category which has been most influenced by an amalgam of theoretical positions (Livesley, 1998). This issue was examined in detail in Sections 1.2, and 2.2 respectively.
6. Multivariate analyses suggest that the DSM-IV categories of personality disorder are not supported by the available evidence (Livesley, 1998).

7. Diagnostic overlap between BPD and other personality disorder categories is extensive (Livesley, 1998; Tyrer, 1994).
8. A number of studies have directly examined the categorical and dimensional approaches to diagnosing BPD. These studies have generally yielded results suggesting that dimensional ratings have increased the reliability and validity of the data (Livesley, 1998).

In response to these criticisms, Widiger (2000) has called for the adoption of a dimensional model of personality disorder classification that recognises the artificial demarcation between normal and abnormal personality. In particular, Widiger (2000) calls for the adoption of the Five Factor Model (FFM) (Costa & McCrae, 1990, 1992) using specific cut-offs to diagnose personality disorder. BPD would still be able to be diagnosed using this system by comparing the individual respondent's FFM traits with the 'prototypic profile' for a particular disorder.

Although the arguments in favour of the dimensional approach to personality disorders appear theoretically and methodologically compelling, recent commentators have argued against the adoption of a dimensional classification system. In a challenge to the categorical-dimensional dichotomy, Oldham & Skodol (2000) argue that whilst a categorical system might imply discontinuity, clinicians using 'categorical' diagnoses do not formulate in such dichotomous terms. They argue that thresholds defining disease entities are somewhat arbitrary, and that the polythetic criteria sets for the DSM-IV/DSM-IV-TR personality disorders contain a degree of dimensionality. For example, a specific case might just meet criterion for BPD or alternately, might meet all nine DSM-IV criteria. Those cases where all criteria are met would represent a more

severe case of BPD. They argue that the inherent dimensionality in what is recognised as a categorical system could be operationalised by stratifying BPD into subcategories of 'absent', 'trait', 'sub-threshold', 'threshold', 'moderate', and 'extreme' depending on the number of criteria met.

Oldham & Skodol (2000) further suggest that replacing the current categorical system with a dimensional system is inappropriate because it is too discrepant from the traditional medical and clinical tradition. Instead, they argue that the categorical system should be retained in principle, but that only two Axis II diagnoses be made. If more than two Axis II diagnoses are available, then a supra-modal Axis II category 'Extensive Personality Disorder' should be applied. They argue that under this structure, the ratings for all extant personality disorders should be made and graphed in a manner akin to that used within the MMPI system (Dahlstrom, Welsh, & Dahlstrom, 1972). Oldham & Skodol argue that this approach would allow an integration of categorical and dimensional features with the important development that the dimensional traits would be 'pathology defined' because they represent the presence of some of the criteria for personality disorders.

Diagnostic Efficiency

Diagnostic efficiency requires the utilisation of a parsimonious set of criteria that facilitate accurate diagnosis. As diagnostic systems increase in complexity (such as the increased number of diagnostic criteria involved in the transition from BPD on the DSM-III-R to BPD on the DSM-IV/DSM-IV-TR), diagnostic efficiency becomes a critical issue. Widiger et al. (1992) reported on a number of studies that were concerned with the optimal criteria for making the diagnosis of BPD. They found that self-injurious behaviour (SIB), unstable and

intense relationships, and impulsivity were pathognomonic for BPD, and the absence of affective instability and impulsivity is optimal for excluding the presence of BPD.

Widiger et al. (1992) note however that diagnostic efficiency is also an artifact of the context and the alternative differential diagnoses under consideration. As an example they note that SIB might be more relevant to the diagnosis of BPD in outpatient rather than inpatient settings because SIB is much more common in non-borderline participants in inpatient settings, and more specific to borderlines in outpatient settings. This argument is similar to the critiques of the BPD diagnosis mounted by Koerner et al. (1996) who argue from a radical behavioural perspective for the primacy of control of environmental variables in any study of personality disorder.

On the basis of the data reported by Widiger et al. (1992) it appears that a number of the diagnostic criteria for BPD are redundant. This would suggest that diagnostic criteria such as attempts to avoid real or imagined abandonment or the experience of transient psychotic episodes do not assist the positive predictive power (PPP) of the BPD diagnosis. This is an important issue particularly for the diagnostic criterion of transient psychotic episodes, and this is further considered below.

Inclusion of Psychotic Symptomatology

Another issue highlighted by Widiger et al. (1992) concerns the issue of whether psychotic symptomatology should be included in the diagnosis of BPD. The difficulties involve a variety of theoretical and empirical issues, and these are considered below.

Widiger et al. (1992) argue that at a theoretical level, it is difficult to establish whether psychotic symptoms evident in some cases of BPD actually represent a diagnostic feature of BPD, or whether they might be a co-morbid feature more associated with the individual psychopathology of specific cases of persons concomitantly diagnosed with BPD. Widiger et al. argue that psychotic symptoms might be more appropriately considered as part of Axis I in such cases, and as a result might blur the distinction between a personality trait (BPD) and an Axis I disorder (acute psychotic state). They note that the distinction between Axis I and Axis II is at times illusory, and the inclusion of psychotic phenomena can serve to confuse the diagnostic picture even further. The 'dimensional' relationship between Axis I and Axis II is an issue that has also been independently identified by other commentators (Oldham & Skodol, 2000; Siever & Davis, 1991)

At an empirical level, Widiger et al. (1992) reviewed a number of studies that sought to determine which diagnostic features were optimal in making the diagnosis of BPD. They found that self harm, unstable relationships, and impulsivity were most predictive of a BPD diagnosis, and absence of impulsivity was most predictive for excluding a diagnosis of BPD. These findings suggest that it is more likely that psychotic features are correlational, and not causal for the diagnosis of BPD. This finding is also consistent with the literature reviewed in Section 1.1 that suggested that BPD was unrelated to the process psychotic illnesses such as schizophrenia. In addition, Widiger et al. cite evidence suggesting that the psychotic features observed in BPD might be associated with co-morbid mood, substance abuse, or factitious disorder. In addition, the early characterisation of BPD included many cases of what is now diagnosed as

Schizotypal Personality Disorder (Akhtar, 1992), and one of the diagnostic features of this disorder is that it is characterised by the presence of psychotic and quasi-psychotic features (American Psychiatric Association, 1994, 2000). It appears that the justification for inclusion of psychotic symptoms might have been driven in part by consensus rather than by empirical evidence overwhelmingly supporting the inclusion of psychotic symptomatology as diagnostic for BPD. Further research is indicated.

2.6.2. Reliability

Overholser (1994) identifies two related issues which affect the reliability of BPD. The first is the issue of inter-rater reliability and the second is that of the 'temporal stability' of the BPD diagnosis.

Overholser (1994) notes that personality disorder diagnoses are usually made on the basis of interview data collected by trained interviewers and then rated by trained raters. Many studies have reported disappointingly low diagnostic agreement between raters. Overholser notes that this low inter-rater reliability is an artifact of a number of factors including the subjective nature of the diagnostic criteria, symptom overlap, and difficulties in discriminating state from trait factors. In addition, the diagnosis of personality disorder is known to be affected by the presence of temporary mood (state factors), and this can result in an inflated estimate of the presence or severity of the personality disorder (Paris, 1999).

A related issue concerns the temporal stability of BPD. One of the major distinctions between Axis I and Axis II in the DSM system is the issue of the duration of the disorder. In all of the diagnostic systems, personality disorder is implicitly regarded as a trait condition. Therefore, one of the assumptions

inherent in personality disorder research is the presupposition that personality disorders remain stable over time. Skodol & Oldham (1991) argue that BPD should be stable over a period of two to five years, but Overholser (1994) has noted that many studies have examined the test-retest reliability of personality disorders using brief time intervals of approximately two months. This is clearly an insufficient time-frame to examine the temporal stability of a condition such as BPD and is at variance with the recommendations of Oldham & Skodol (1991).

Although research has documented the stability of personality functioning over time, these findings may not apply to personality disorders (Overholser, 1994). In fact, many of the studies of both the brief and long-term course of BPD referred to in Section 2.5 indicated that BPD improves over time, and that many cases of BPD either 'grow out' or 'burnout' over time (Paris, 2003a, 2003b; Paris & Zweig-Frank, 2001; Stone, 1992). In particular, the recent study by Gunderson et al. (2003) suggested that for a significant sub-sample of the BPD cohort in the CLPS study, BPD is characterised by a marked level of temporal instability. The prospect that BPD might not be temporally stable has significant implications for the reliability of BPD. The finding that BPD might not be as temporally stable a construct as originally thought raises questions with regard to the reliability of the diagnosis. It also raises questions about the very essence of what constitutes a 'personality disorder'. Whilst this is an important issue, it is beyond the scope of the thesis, and will not be considered further.

2.6.3. Assessment

There are two major issues associated with the assessment of BPD. These include the issue of whether BPD should be located on Axis I or remain on Axis

II of the DSM, and secondly, the difficulties associated with the current methods for assessing BPD. These are also briefly considered here.

The 'Location' of BPD

The bulk of the literature that is concerned with the 'location' of BPD has arisen as a result of the 'multiaxial' nature of the DSM, and the provision of a specific axis (Axis II) for personality disorders. This issue does not arise in the other diagnostic systems where a multiaxial system is not employed. Despite this, the bulk of the literature on BPD assumes the DSM-IV/DSM-IV-TR BPD convention is being employed and therefore this issue remains salient.

The critical objection that has been made in relation to the location of BPD refers to the question of whether BPD should be located on Axis I or Axis II of the DSM (Oldham & Skodol, 2000). Pfohl (1999) has argued that there is a lack of clear differentiation between Axis I and Axis II, and Livesley (1998) has also argued that the relationship between various diagnostic groups is not empirically based. As a result, the relationship between Axis I and Axis II appears empirically deficient. Siever & Davis (1991) have noted the tendency to view biological factors as the key determinants to Axis I, and psychosocial-developmental factors as the principal determinants of Axis II. Section 2.2.7 demonstrated numerous 'biological' factors associated with BPD which could be employed to argue that BPD should be relocated to Axis I. Siever & Davis (1991) further argue that whilst there might be clinical sense in the demarcation between Axis I and Axis II descriptors, the emergence of a range of genetic studies suggests that the pathophysiology of psychiatric disorders might involve spectrum linkages between Axis I and Axis II states. For these reasons, some

authors argue that there is a case for many of the Axis II personality disorders to be relocated to Axis I.

In an interesting act of conciliation, a number of authors who otherwise find themselves in adversarial positions (such as on the issue of categorical or dimensional diagnosis) are in accord in recommending that Axis II should continue to be used (Oldham & Skodol, 2000; Widiger, 2000). Widiger (2000) advocates the continuation of Axis II because its removal will not resolve the diagnostic boundary issues, and it forces clinicians to consider the presence of personality disorder. At this time, the issue of the location of BPD remains equivocal and is likely to only be resolved as a result of further evidence or a significant re-conceptualisation of the disorder.

Problems With Current Methods for Diagnosing BPD

The current methods for diagnosing BPD have also been criticised for a number of reasons. These include the combination of a number of disparate theoretical positions, and the use of polythetic diagnostic criteria allows a number of different pathways to reach the same diagnosis (Paris, 1999). Most importantly however, there remains an absence of a 'gold standard' diagnostic test (Kaye & Shea, 2000), and as a result, a series of differing methods for diagnosing the disorder have been developed. The diagnostic methods of choice include self-report and semi-structured interview methods, and there are two major difficulties associated with these approaches. First, they are thought to be poorly related to each other in relation to their measurement of similar theoretical constructs (J. C. Perry, 1992), and secondly, they return highly variable validity and reliability coefficients (Paris, 1999).

The inconsistency across diagnostic measures appears to be influenced by a number of factors and can lead to 'method variance' errors (Overholser, 1994). This can artificially inflate the correlation between measures as a result of item overlap, gender bias, and limited evaluation data. As a result, an inflated risk of Type II error occurs.

Skodol & Oldham (1991) concluded that a questionnaire diagnosis of BPD used in isolation has an unacceptably high false-positive rate. In addition, the absence of a diagnosis by self-report is rarely associated with a positive diagnosis by structured interview. They conclude that self-report instruments have a role to play as cost-effective screening instruments but no single instrument has demonstrated diagnostic superiority over other measures. Therefore, a patient diagnosed with BPD by more than one instrument is much more likely to be a valid 'hit' for BPD than is a patient where instruments disagree. In contrast, J. C. Perry (1992) reviewed all of the personality diagnostic methods available at the time. He reported that the average kappa statistic was 0.25, with interviews returning slightly improved outcomes. Because kappa can be considered to assess the degree of variance explained, a significant amount of the variance in personality disorder diagnosis is likely to be error variance. Although the kappa for BPD is higher than for a number of other personality disorders, the results are nevertheless poorer than for other areas of personality research (Westen, 1997).

In response, Westen (1997) has noted that the current approach for diagnosing Axis II disorders by systematic assessment has been based upon methods derived from the diagnosis of Axis I disorders. In contrast, Westen argues that the process for diagnosing personality disorder relies more on

listening to the 'feel' of the patient (i.e., understanding the represented internal psychological themes or psychodynamics), examining interpersonal reactions, and observing behaviour within the interview. Westen also argued that a number of personality disorders exist that are not articulated within the current DSM formulation. Westen suggests that the current methods for diagnosing BPD require further development and need to build in a variety of other components that involve measuring emotional and interpersonal processes occurring in the clinical interview.

2.6.4. Heterogeneity

There are two major implications that arise out of the use of a categorical system to diagnose BPD. These are compounded by the use of polythetic diagnostic criteria within a categorical diagnostic paradigm. First, BPD is a heterogeneous condition in which a number of differing clusters or 'types' of BPD can be identified (Andrulonis et al., 1982; Andrulonis & Vogel, 1984; Grinker et al., 1968; Rusch, Guastello, & Mason, 1992; Russ, Shearin, Clarkin, Harrison, & Hull, 1993). Secondly, because of the heterogeneity of the disorder, there is a significantly increased likelihood of diagnostic overlap or co-morbidity with other Axis I and Axis II disorders (Paris, 1999).

BPD 'Subtypes'

Because the DSM and ICD diagnostic systems involve polythetic diagnosis, the end result is that any five of the available nine diagnostic criteria can be employed in reaching criterion for the disorder within the DSM-IV-TR system. This results in over 256 different DSM-IV-TR combinations, and 416 different ICD-10 combinations of criteria by which BPD can be potentially

diagnosed (Burgmer et al., 2000). This situation has, in part, contributed to the significant heterogeneity observed in BPD.

One of the outcomes of this situation has been the emerging recognition that there are a number of different 'types' of BPD. These different types have not been well articulated, and there does not appear to any comprehensive consensus concerning what types of BPD might exist. Nonetheless, some consensus descriptors have emerged that include the multivariate four-cluster grouping including the Psychotic Border, Core Borderline, 'As If', and 'Border with Neurosis' BPD subgroups (Grinker et al., 1968), 'organic' and 'non-organic' BPD subtypes (Andrulonis et al., 1982), pain sensitive versus pain-insensitive self-injurious BPD subtypes (Russ et al., 1993), and an impulsive versus non-impulsive BPD subtype (Andrulonis et al., 1982; Links et al., 1999). Finally, a factor analytic study revealed four BPD subtypes consisting of an 'emotionally unstable' subtype characterised exclusively by emotional volatility; an 'identity impaired' subtype characterised by identity disturbance; a 'severely impaired' subtype characterised by emotional instability, self-destructiveness, and identity disturbance; and an 'undifferentiated' subtype characterised by self-destructive unpredictability (Rusch et al., 1992).

Subtype descriptors clearly represent an attempt to cope with the complexity of a heterogeneous/multidimensional construct by reducing the number of dimensions being examined at any one time. Whilst this represents a sensible approach to managing a complex clinical entity, it again raises questions regarding the diagnostic validity of BPD, and questions the ultimate utility of the diagnosis. More importantly however, it suggests that there is a need to identify the 'type' of BPD group more precisely when questions are raised concerning

the prevalence and course of the disorder, and the form of therapy that is best indicated for a particular borderline patient. There is no evidence currently available to assist with this discrimination task.

In an initial attempt at simplifying this complexity, Burgmer et al. (2000) has argued that the polythetic approach should be abandoned in favour of a hierarchical diagnostic model driven by the use of 'core criteria' for making the diagnosis. Whilst they acknowledge that no one criterion can be used to make the diagnosis of BPD, a number of authors have identified 'core borderline' criteria (Dahl, 1990). The implication of this approach suggests that a preferable method for diagnosing BPD would see the employment of diagnostic algorithms in which 'core criteria' need to be met followed by adjunctive subsidiary criteria. This method appears reminiscent of the 'prototype' approach (L. Morey & Ochoa, 1989), in which particular criteria are given a heavier weighting in diagnosis. The difficulty inherent in this approach is associated with the absence of consensus for prototype criteria with significant cross-Atlantic disputes regarding the optimal prototype criteria (Burgmer et al., 2000; Paris, 1999). Although this approach holds promise, its implementation awaits the development of a consensus position on optimal prototype criteria for BPD.

Co-Morbidity of BPD

The heterogeneity of BPD also results in extensive co-morbidity with both Axis I (Tyrer, 1999), and Axis II disorders (Zanarini et al., 1998). The literature demonstrating a significant degree of co-morbidity of BPD with both Axis I and Axis II disorders is huge and beyond the scope of this review. There is however, demonstrated evidence of co-morbidity between BPD and affective disorders (Akiskal, 1981; Sullivan, Joyce, & Mulder, 1994), post-traumatic stress

disorder (PTSD) (Herman & van der Kolk, 1987; Prodgers, 1984), eating disorders (Herzog, Keller, Lavori, Kenny, & Sacks, 1992; Sansone, Sansone, & Morris, 1996), and other personality disorders (Zanarini et al., 1998).

There are some important issues concerning co-morbidity of BPD with Axis I and other Axis II disorders. Widiger & Frances (1989) note that in relation to the co-morbidity between BPD and other personality disorders, it is common for BPD's to meet criteria for at least one other personality disorder. Therefore, it is probably appropriate to provide all personality disorder diagnoses rather than attempt to distinguish which is the more salient diagnosis. Widiger & Frances also argue that co-morbidity between an Axis I condition and BPD will result in a more difficult to treat Axis I condition. With regard to the presumed associations between affective disorders and BPD, and schizophrenic disorders and BPD, Gunderson's (1994) review concluded that no specific association exists between BPD and affective disorders on the one hand, and BPD and schizophrenia on the other. Finally, Widiger & Frances argue that BPD can be over diagnosed by self-report inventories, but the most critical issue is the potential for confusion between state and trait factors. This issue can be overcome by a systematic analysis of each diagnostic item, and this will result in a valid diagnosis of the disorder.

Tyrer (1994; 1999) takes issue with this view. He argues that co-morbidity implies the simultaneous presence of two or more independent disease entities. In relation to BPD, he argues that the term 'overlap' should be employed as the prevalence of the 'pure' form of the disorder (i.e.: without overlap with another disorder) is low. Part of the reason for this is that the diagnostic borders of BPD are highly flexible, and so the BPD construct does not represent a stable

entity. Tyrer further argues that the behavioural criteria for BPD are also common for other Axis II personality disorders and a number of Axis I disorders. The commonality of these criteria is argued by Tyrer to be the reason why patients with BPD have approximately twice as many mental state disorders and are four times as likely to have four or more other disorders than patients without BPD. Furthermore, the issue of co-morbidity becomes important because there is data available suggesting that when co-morbid Axis I disorders are resolved then the criterion for BPD are also no longer met (Gunderson et al., 2003). This latter point represents a highly important issue in its own right, and deserves a great deal more empirical scrutiny.

The overall conclusion that can be drawn from these findings suggests that BPD is a heterogeneous disorder with a range of probable subtypes which vary on the basis of the severity of identity diffusion, impulsivity, self-injuriousness, degree of interpersonal difficulty, and affect regulatory incapacity. These findings are probably an artifact of a categorical system of diagnosis. Developmentally, it would also appear that BPD is an expression of morbidity which is best viewed as a final common pathway arising out of the aggregation of a range of risk factors which include an 'at risk' genetic and neurobiological substrate, a predisposition toward co-morbidity with other psychiatric disorders, and a dysfunctional developmental/family history with 'parental bonding', trauma, and attachment disturbances in childhood.

Despite all of the issues outlined above, the use of a categorical diagnostic model of BPD continues. Whilst the seventh revision of the DSM (DSM-V) might result in conceptual and methodological changes to BPD, the prospect appears remote. The possible changes that have been proposed include

an incorporation of a dimensional system of classification (Widiger, 2000), relocation of some personality disorder categories (including BPD) into Axis I of the DSM, or significantly reducing the number of personality disorder diagnoses to achieve increased parsimony (Oldham & Skodol, 2000). At this time, it appears unlikely that a radical reformulation of BPD will be considered because of the strong association of the categorical form of BPD diagnosis with orthodox medical diagnosis (Oldham & Skodol, 2000). It appears that the continuance of a medical-diagnostic tradition steeped in practices of a 'sign-symptom-syndrome-illness' formulation of BPD remains central to the current understandings of the nature of BPD. This approach appears to have a certain desirability with some clinicians, in part because of the shorthand manner in which the term 'borderline' is sometimes used to communicate clinical information about the 'difficulty' of a patient and the extent of their 'pathology'. In addition, the maintenance of this tradition permits the continuance of a link with a well-established approach for thinking about the clinical phenomenology of 'borderline material'. In this sense, the continued allegiance to the current approach for understanding BPD has the appearance of adherence to a particular 'heuristic bias' (Tversky & Kahneman, 2002). As Tversky and Kahneman have demonstrated, heuristic biases are extremely resistant to change.

2.7. SUMMARY AND CONCLUSIONS

This chapter has considered some of the critical issues associated with BPD. First, the chapter considered a range of theoretical perspectives. These included the psychoanalytic, affect spectrum, impulse spectrum, trauma spectrum, behavioural, and neurobehavioural perspectives on BPD. The review then examined the four major diagnostic systems in BPD. These included a

consideration of Kernberg's (1984) concept of Borderline Personality Organisation (BPO), Gunderson's borderline criterion (Gunderson et al., 1981), the ICD (A. W. Loranger et al., 1997), and the DSM systems (American Psychiatric Association, 1994, 2000). This subsection also included a consideration of the major self-report and semi-structured interview assessment instruments available for each diagnostic system.

Thereafter, the review examined the prevalence and incidence data for BPD and demonstrated that it has a prevalence rate of approximately two percent in community studies with a significantly elevated rate in clinical populations. Incidence data is rare and requires further research. Gender prevalence is thought to over-represent women, and it has been argued that this is a selection artifact associated with the settings in which BPD cohorts have been recruited. The course of BPD was also reviewed, and the available evidence suggests mixed outcomes for the short-term course of BPD, but generally positive longer term outcomes suggesting that it remits by middle age.

Finally, a number of theoretical and methodological issues were identified. These included issues of diagnostic validity including diagnostic efficiency, whether psychotic symptomatology should be included as part of the diagnostic criteria for the disorder, and whether BPD should utilise a categorical or dimensional diagnostic system. A second group of critiques associated with the structural organisation of the DSM system, and the problems associated with the current methods for diagnosing BPD were considered. A third group of concerns that were identified involved identifying BPD as a heterogeneous rather than a homogeneous entity. This has resulted in the identification of a range of different types of BPD, and an extensive co-morbidity with both Axis I and Axis

II disorders. Finally, the temporal stability of the disorder has been identified as problematic which in turn raises questions regarding the ultimate validity of the disorder.

Despite these issues, the diagnosis remains and is likely to be included in the DSM-V (Oldham & Skodol, 2000). It seems likely that BPD will continue to be recognised as a personality disorder although its location in both the DSM and the ICD systems and how it is theoretically conceived will continue to receive significant attention. It also seems likely that BPD is an evolving diagnosis that will be modified over time as a result of the convergence of various lines of research. Despite these difficulties, BPD appears to have consolidated itself as a legitimate psychiatric diagnosis. The following chapter attempts to extend the understanding of this disorder by outlining a multidimensional developmental neuropsychological model of impaired executive function in BPD.

CHAPTER THREE: A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL MODEL OF IMPAIRED EXECUTIVE FUNCTION IN BPD

3.1. OVERVIEW OF CHAPTER

Chapter Three describes a multidimensional developmental neuropsychological model of impaired executive function in BPD. The model proposes that four key executive functions are impaired in BPD. These include working memory (WM), behavioural inhibition (BI), affect regulation (AR), and problem solving (PS). The proposed model argues that deficits in these executive functions occur as a result of the influence of a number of factors. These include a genetic and psychobiological predisposition to BPD, exposure to adverse early family environments including an insecure attachment history, stressful and/or traumatic experiences including child maltreatment and child sexual abuse, as well as a lack of empathic care in childhood and adolescence.

The proposed multidimensional developmental neuropsychological model of impaired executive function in BPD is informed by the perspectives of various authors including Grigsby & Stevens (2000), Meares, Stevenson, & Gordon (1999), Mega & Cummings (1994), Paris (1999), Schore (1994; 2003a; 2003b), Siegel (1999), Siever & Davis (1991), and Stone (1993). The model proposes that a genetic and psychobiological predisposition to BPD interacts with adverse developmental factors to produce various CNS impairments. The failure of appropriate neural 'sculpting' (Cozolino, 2002; Schore, 1994, 2003a, 2003b), results in the creation of aberrant neural pathways and dysfunctional neurotransmitter systems which in turn generate the interrelated series of symptom profiles that characterise BPD. As a result, the model proposes that

BPD phenomena are 'experience-dependent' (Joseph, 1996; Kandel, 1998, 1999; Siegel, 1999) and can be understood to be the outcome of a complex interaction of genetic, (Torgersen, 2000), psychobiological (Siever & Davis, 1991), early socio-developmental (Paris, 1999), and relational-attachment factors (Barone, 2003; Patrick et al., 1994).

Specifically, the model proposes that BPD is characterised by an 'experience-dependent' maturational failure of the development of a distributed regulatory system (Grigsby & Stevens, 2000) involving amygdala (Donegan et al., 2003; Driessen et al., 2000; Lyoo et al., 1998; Tebartz van Elst et al., 2003), anterior cingulate (Bazanis et al., 2002; Leyton et al., 2001; Tebartz van Elst et al., 2003), and orbital-prefrontal regions (Andrulonis et al., 1982; Andrulonis et al., 1980; Goyer et al., 1994; Kimble et al., 1997; Schore, 1994, 1996, 2003a, 2003b; Soloff et al., 2000; Vollm et al., 2004). The failure of this system to develop satisfactorily provides the neural basis for the development of BPD and the associated executive deficits that are hypothesised to characterise the disorder. There is evidence also that the orbital-prefrontal regions of the brain in particular are heavily 'experience-dependent' (Grigsby & Stevens, 2000; Joseph, 1996; Kandel, 1998, 1999; Siegel, 1999), and the failure of this system to develop satisfactorily also results in the self-regulatory deficits characteristic of the disorder. These self-regulatory and executive deficits are reflected in impairments to working memory, behavioural inhibition, affect regulation, and problem solving. The model predicts that deficits in working memory, behavioural inhibition, affect regulation, and problem-solving should be observable in adult cases of BPD.

In order to outline this model more adequately, Section 3.2 discusses the functions that the frontal lobes perform in human neuropsychology. This is an important first step, because knowledge of the function of the frontal lobes is an important backdrop for understanding the principles of executive function. Section 3.2 also describes the phenomenon of 'frontal lobe syndrome' and notes similarities between this syndrome and a number of the diagnostic features of BPD. The frontal lobes are examined in detail in this section because they are also known to be 'experience-dependent' (Grigsby & Stevens, 2000), and many of the experimental tasks employed in this project are known to be mediated by these brain regions (Lezak, 1995). Section 3.2.1 outlines the current understanding concerning the development of the orbitofrontal cortex, and demonstrates that the development of these regions is dependent upon appropriate, phase-attuned experiences particularly associated with attachment and mother-infant interaction. This section also describes different types of frontal syndromes, and links aspects of these to the phenomenon of BPD.

Section 3.3 describes a theory of executive function which is then linked to BPD. This is important because as Pennington & Ozonoff (1996) note, research on executive functions has been guided by the so-called 'frontal metaphor' derived from adult neuropsychology. This tradition has used the neuropsychological test returns of patients with documented frontal lobe lesions to understand the nature of the deficits associated with 'frontal' regions of the brain. The functions associated with the frontal lobe have also become known as 'executive functions' (Lezak, 1995). Therefore, there is a high degree of conceptual overlap between 'frontal functions' and 'executive functions'. They

are not, however, identical concepts although they often appear to be used interchangeably.

Section 3.4 describes a multidimensional developmental neuropsychological theory of executive disorder in BPD. It proposes that BPD involves a number of impaired executive functions which include working memory, behavioural inhibition, affect regulation, and problem-solving ability. Section 3.5 expands on the model by reviewing the evidence for impaired working memory in BPD, and Section 3.6 reviews the evidence for impaired behavioural inhibition in BPD. Section 3.7 reviews the evidence for impaired affect regulation in BPD, and Section 3.8 the evidence for impaired problem solving in BPD. Section 3.9 concludes the chapter and outlines the hypotheses which form the basis of the project.

3.2. FRONTAL LOBE FUNCTIONS AND 'FRONTAL' PATHOLOGY

The frontal lobes of the human brain are not a unitary structure, but consist of a number of functionally specific regions. There is debate within the literature regarding the number of regions, with some arguing that the frontal lobes consist of three regions (Joseph, 1996), and other arguing that the frontal lobe consists of as many as five subunits (Lichter & Cummings, 2001). The term 'frontal lobe' was first coined by Chaussier in 1807 (J. D. Russell & Roxanas, 1990), and this area has also been referred to as the 'organ of civilization' (G. A. Miller, Galanter, & Pribram, 1960).

Damage to the frontal regions evoke a variety of patterns of behaviour which are generically referred to as the 'frontal lobe syndrome' (Lezak, 1995). Although this term has been criticised for lacking specificity, it nevertheless retains clinical validity in describing various classes of aberrant behaviour

(Fuster, 1989). Frontal damage typically results in behavioural and personality change, altered social awareness, and changes in activity level, motivation, and mood (J. D. Russell & Roxanas, 1990).

Frontal lobe impairment is also often characterised by deficits in attention and memory (Joseph, 1996; Lezak, 1995). Typically, frontal dysfunction is reflected in difficulties in learning from experience, adapting to novel situations, and using context specific information to solve novel problems (Lezak, 1995; Walsh, 1978). In addition, frontal syndromes typically involve perseveration and an inability to alter strategy in order to manage novel situations (Fuster, 1989). Closely associated with this phenomenon is a difficulty in suppressing responses, and this phenomenon is typically associated with impulsive behaviour (Lezak, 1995). Dissociation can also occur between language as a self-correctional mechanism and ongoing behavioural responses such that there is an increased risk of perseveration (Barkley, 1997). Damage to frontal sites is also hypothesised to compromise the capacity to regulate a sense of time, and this phenomenon has major implications for the development of a continuous sense of self (Lezak, 1995). In addition, damage to specific regions of the prefrontal cortex lead to syndromes in which disorders of affectivity or empathy are noted (Fuster, 1989). Difficulties in learning from experience, adapting to novel situations, problem solving, perseveration, affect regulation, empathy, experiencing a continuous sense of self, and 'impulsivity' are often noted features of BPD (American Psychiatric Association, 1994, 2000; Grotstein, 1987; Gunderson, 1994).

The key issue is that a number of the behavioural features identified as synonymous with BPD (affect regulation, difficulties in learning from

experience, impulsivity, time orientation, adaptability, social awareness (reflective self function), and perseverative behaviour) also appear to be characteristic of impaired frontal lobe function. As a result, it raises the important question of whether BPD is 'localised' within the frontal regions as there is also some evidence available suggesting 'frontal' impairment in BPD (Lucas et al., 1989; Lyoo et al., 1998).

It also appears likely that a number of different frontal-subcortical circuits are implicated in BPD phenomenon, and this is likely to be mediated by the nature of the task demands confronting the BPD individual. Mega & Cummings (1994) have identified specific features associated with particular frontal-subcortical circuits, and each of these appear to play a role in BPD phenomenon. Mega and Cummings identify the following circuits:

1. A dorsolateral-prefrontal circuit that subserves 'executive function' including complex problem-solving, set-shift activity, the use of verbal behaviour to guide behaviour, and self-directedness;
2. A lateral-orbitofrontal circuit that subserves socially appropriate and empathic behaviour. Most importantly, rapid shifting of mood states – what clinicians refer to as 'affect lability' – are associated with the lateral orbitofrontal circuit;
3. An anterior-cingulate circuit that subserves a behavioural inhibitory function.

Because many of the features of these circuits appear consistent with BPD phenomena, it suggests that the network disturbances thought to occur in BPD are syndromal in nature rather than an artifact of a discrete, localised lesion as originally proposed by Andrulonis et al. (1980). Furthermore, the presumed diverse network circuitry underpinning BPD symptomatology again raises the

issue of the absence of diagnostic specificity for BPD. Nonetheless, there appear to be a series of neural referents underpinning BPD which can be seen to be associated with known frontal-subcortical deficits. The task now appears to be one of documenting the nature, context, and extent to which each of these circuits are specific for BPD. This task is however, beyond the scope of the present project.

Fuster (1989) and Schore (Schore, 1994, 2003a, 2003b) have synthesised the available research on lesion studies, human studies of pre-frontal neural trauma, neurophysiological data, and developmental psychopathology in order to develop comprehensive theories of frontal and pre-frontal functioning. Fuster's theory suggests that the pre-frontal cortex supports a number of cognitive functions of which at least three can be identified as specific for this particular region of the neocortex. These include short-term memory, preparatory set and interference control. Fuster (1989) argues that the primary function of the pre-frontal lobes is the formation of temporally structured behaviours which serve the purpose of structuring (and effecting) goal-directed, purposive behaviour.

Schore (1994; 1996; 2002; 2003a; 2003b) outlines a complex, multilevel theory of neural development which can be used to understand the development of a range of developmental psychopathologies including BPD. He emphasises the role of the orbitofrontal regions in human development. The close location of the orbitofrontal region to the limbic area has resulted in it being viewed as an 'association cortex' for the limbic forebrain. The limbic area has been well documented as the 'emotion centre' of the human brain (MacLean, 1954, 1958). This area of the frontal lobe is known to have extremely high levels of serotonin receptors. Serotonin is centrally important in the regulation of emotion. The

orbitofrontal regions project pathways to limbic areas in the temporal poles and amygdala, to subcortical centres in the hypothalamus, and to dopamine receptors in particular in the ventral tegmental limbic forebrain-midbrain circuit. These areas are well known regions for the initiation of emotional responses (Schore, 1994, 2003a, 2003b), and this area also integrates affective, motoric, verbal, and sensory CNS activity (Lezak, 1995).

The orbitofrontal area is expanded in the right cortex (Schore, 1994, 1997, 2002, 2003a), and this cortex is also known to be dominant for the processing of affectively based information and the storing of memories of emotional faces (Joseph, 1996). It is also known that this area is involved in appraisal processes, directed attention, and the processing of social information. The orbitofrontal region is centrally involved in the execution of social and emotional behaviours, and in the self-regulation of bodily and emotional states (Schore, 1994, 1997, 2003a, 2003b).

The orbitofrontal cortex also performs an appraisal function, and is responsible for the allocation of attentional resources to facilitate effective cognition (Barkley, 1997). It is also involved in the temporal organisation of behaviour and in the adjustment or correction of emotional responses (Fuster, 1989). The prefrontal region acts as a monitoring system for the regulation of affective states, and therefore enables the individual to recover from affective disruptions and integrate a sense of self across state experiences. This process enables the integration of a coherent sense of self which has a ‘seamless’ quality (Grigsby & Stevens, 2000).

Schore (1994; 2003a) proposes that affective transactions between the infant and the caregiver act as a growth promoting environment for the postnatal

development of a corticolimbic system in the prefrontal cortex. Schore (1996; 2003a) argues that the parasympathetic lateral tegmental forebrain-midbrain limbic circuit mediates regulatory, homeostatic, and attachment functions in the developing infant. The view that early environmental influences, particularly the quality of early maternal-infant interaction and attachment, predict neural development and optimal neural functioning is supported by a number of reviews (Bradley, 2000; Bremner, 1999; Davidson, 1994; Dawson & Ashman, 2000; Depue, Collins, & Luciana, 1996; Fox, Calkins, & Bell, 1994; B. D. Perry, 1997; Post & Weiss, 1997; Siegel, 1999; Trevarthen & Aitken, 1994).

Schore (1994; 2003a; 2003b) also argues that deprivation, trauma, or failures of interactive-affective experience act as growth-inhibiting environments for prefrontal structures, and in particular for the parasympathetic lateral tegmental forebrain-midbrain limbic system. The occurrence of non-regulated stressors operating within the infant-caregiver dyad, or stressors impinging from outside of the care-giving dyad generate the potential for the development of insecure attachments. There is now reasonable evidence that as a group, BPD is characterised by disordered attachment (Barone, 2003), and in particular are overrepresented in the disordered attachment in the 'E3' classification on the Adult Attachment Interview (AAI) (Patrick et al., 1994). The E3 AAI category is a highly specific subcategory associated with anxiously attached individuals who also are hypothesised to experience disorders of 'metacognitive functioning' (Fonagy et al., 1995; Main, 1991). This further predisposes the vulnerable individual to future psychopathology through the alteration of corticolimbic circuitry (Schore, 1996, 2002), or through disrupted self-regulation (Cicchetti & Tucker, 1994). These mechanisms are hypothesised to be responsible for the

regulatory failures underlying BPD. It is proposed that these mechanisms along with the establishment of the deficits in frontal-subcortical circuitry described by Mega & Cummings (1994) provide the neural basis for the development of BPD in adulthood.

3.2.1. Development of the Orbitofrontal Cortex

The frontal lobes of the human are not fully formed at birth, but develop over the course of childhood, adolescence, and early adulthood (Joseph, 1996). The development of the frontal lobes are particularly sensitive to phase-appropriate stimuli that are matched to 'sensitive periods' during which the neural system is primed for receipt of appropriate stimuli (Grigsby & Stevens, 2000; Kandel, 1998). These stimuli include appropriate sensory stimulation, and most importantly, sensitive and appropriate caregiver-child interactions (D. N. Stern, 1985, 1998). It is the caregiver-child interactions that facilitate the optimal growth and development of neural networks (Schore, 2003a, 2003b), provide the prototypical affective experiences for the infant (Trevarthen & Aitken, 1994), and provide the basis by which affect regulation ultimately develops (Bradley, 2000; Schore, 2003a, 2003b; Siegel, 1999).

Schore (1994; 2003a; 2003b) argues that infant-caregiver interactions generate intense positive affective states and provide the growth promoting environment for the development of the prefrontal cortex. The neural basis for this development is thought to involve the release of dopamine and endogenous opiates in the context of optimal infant-caregiver attunement (Schore, 2003a).

The prefrontal cortex undergoes a maturational change at the end of the first year of life (Grigsby & Stevens, 2000). This is characterised by the increasingly complex self-regulatory functions located primarily within the

orbital prefrontal cortex (Joseph, 1996). The development and elaboration of these functions is 'experience dependent' (Grigsby & Stevens, 2000), and take form post-natally as a direct result of the quality of the social environment of the child (Schore, 1994, 2003a). The pre-frontal cortex is argued to be central to the formation of behavioural patterns which respond to and manage complexity in the environment. The pre-frontal cortex also provides a synthetic function to human cognition and facilitates temporally structured units into hierarchical configurations (Fuster, 1989; Lezak, 1995). The task demands controlled by the pre-frontal cortex involve management of time, novelty and complexity (Joseph, 1996). Furthermore, the pre-frontal cortex permits the development of cognitive structures that facilitate the bridging of temporal discontinuities. This function permits the organism to engage in complex, novel and temporal regulation of behaviour in order to initiate goal directed behaviour (Barkley, 1997; Fuster, 1989; Schore, 1994, 2003a).

Fuster (1989) has categorised prefrontal lesions according to impairments in attention, perception, motility, and temporal integration. The implication of this suggests that the 'frontal metaphor' is a high sensitivity, low specificity concept in which various features are potentially reflected in a variety of disorders including Attention Deficit Hyperactive Disorder (ADHD), Conduct Disorder (CD), Autism, and Tourette's Syndrome (TS), Bipolar Affective Disorder (BPAD), Hypomania, Antisocial Personality Disorder (ASPD), and BPD. It would appear that the 'pseudopsychopathic' syndrome described by Fuster describes aspects of Bipolar Affective Disorder, Hypomania, and Antisocial Personality Disorder. The 'pseudodepressed' syndrome describes particular features of BPD such as degraded awareness and diminished initiative,

whereas the ‘euphoric’ syndrome describes other features of BPD, most notably a ‘labile-affective’ component. The overlap of BPD features across known frontal syndromes suggests that the disorder is probably not caused by specific, localised lesion sites, but is probably mediated by a number of ‘distributed’ frontal-subcortical/corticolimbic systems (Grigsby & Schneiders, 1991; Grigsby, Schneiders, & Kaye, 1991; Mega & Cummings, 1994). The known frontal-subcortical circuits have been described earlier in this chapter.

The four ‘frontal-executive’ functions hypothesised to be impaired in BPD (working memory, response inhibition, affect regulation, and problem solving) also appear to be associated with known frontal lobe impairment (Lezak, 1995). Some of the evidence for this is briefly reviewed below.

Fuster (1989) argues that effective working memory is a central feature of pre-frontal functioning because it enables behavioural structures to be linked across time. Working memory involves the use of a temporary storage system that retains information in order to formulate goal directed sequences or action plans (Baddeley, 1995). These sequences are then employed to execute effective behavioural performance. The capacity to delay responses (which is necessary for the development of temporally maintained behavioural structures) requires protection from interference factors which might degrade or disrupt planning processes (Barkley, 1997). The ability to hold events in mind in temporal sequence appears to be associated with a psychological sense of time (Fuster, 1989), and also to a psychological sense of self (Grotstein, 1987). Because goal directed sequences and action planning often appear to be defective in BPD (Judd & Ruff, 1993), the proposed model hypothesises that BPD involves impaired immediate-recall verbal working memory (Burgess, 1990; O'Leary et

al., 1991), delayed-recall verbal working memory (Burgess, 1990, 1991; Kurtz & Morey, 1999; O'Leary et al., 1991), and delayed-recall visual working memory (Judd & Ruff, 1993; Sprock et al., 2000). It is hypothesised that the phenomenon that underpins these collective findings is a generic impaired working memory system in BPD.

Disorders of the pre-frontal cortex (PFC) also provoke disturbances in the ability to inhibit behaviour (Lezak, 1995; Roberts & Pennington, 1996). Mega & Cummings (1994) argue that the lateral orbitofrontal-subcortical circuit within the prefrontal region probably mediates response inhibition and that failure in stop-signal or 'go/no go' tasks is mediated by medial-frontal damage. Clinically, this often presents as a variety of forms of perseverative response, but Lezak (1995) argues that the underlying deficit is generally one of impairment of response inhibition. Because impulsivity (or as examined in this project - response inhibition) is regarded as a central impairment in BPD (American Psychiatric Association, 1994, 2000; Links et al., 1999), it appears reasonable to speculate that this impairment might also be mediated by deficits in the lateral orbitofrontal-subcortical circuit within the prefrontal region.

Disorders of the pre-frontal cortex (PFC) also provoke disturbances in the regulation of affective states (Fuster, 1989; J. D. Russell & Roxanas, 1990). Distinct frontal syndromes are associated with lesions in specific sites within the PFC. Lesions to the medial structures of the PFC result in a syndrome of 'akinetic mutism,' whereby degraded production of speech and other forms of spontaneous behaviour is noted (Pennington & Ozonoff, 1996). A related set of syndromes involve lesions to the anterior convexity of the frontal poles and are characterised by degraded awareness and diminished initiative, reduced concern,

and blunted affective response. These syndromes are variously referred to as 'apathetic,' (Fuster, 1989), or 'pseudodepressed' (Stuss & Benson, 1986). A third set of syndromes occurs where orbital lesions are observed. These result in a hypomanic, puerile, disinhibited, antisocial, and non-empathic behavioural profile variously referred to as 'euphoric,' (Fuster, 1989), or 'pseudopsychopathic' (Stuss & Benson, 1986). Because the regulation of affective states is clearly impaired in BPD, it is possible that this impairment might also be mediated by deficits in these prefrontal regions.

It is important to note however, that affect regulation is difficult to examine directly. In contrast, attention is a phenomenon which is consistent with the cognitive-executive model described in this chapter, and the measurement of the allocation of attentional resources to affectively valenced stimuli is, in contrast, directly measurable. Therefore, it is proposed that within this project the measurement of affective-attentional resources through the measurement of attentional bias represents an analogue measure of affect regulation in BPD.

Finally, disorders of the frontal lobes and the pre-frontal cortex (PFC) also result in impaired problem solving and planning (Cozolino, 2002; Della Sala, Gray, Spinnler, & Trivelli, 1998; Walsh, 1978). Lezak (1995) notes that the prefrontal regions are central to the capacity for engaging in mental or behavioural shift-of-set, and the ability to think abstractly. These functions, along with the ability to hold material in mind in order to execute a problem solving initiative (working memory), inhibiting behaviour, and regulating affect are important in developing the capacity to problem-solve. Because these latter functions are reported to be impaired in BPD, it appears reasonable again to

speculate that problem-solving difficulties in BPD might also be mediated by prefrontal regions.

3.3. THE CONCEPT OF 'EXECUTIVE FUNCTION' (EF)

The concept of executive function emerged out of the neuropsychological literature examining impaired test performance amongst head injured patients where there was documented frontal lobe pathology (Lezak, 1995). There are a number of different perspectives on the constitution of executive functions, and it appears that the concept has been influenced by disparate literatures including cognitive psychology, developmental psychopathology, and neuropsychology.

Pennington & Ozonoff (1996) argue that the concept of executive function has been influenced by the 'frontal metaphor.' The frontal metaphor is derived from neuropsychology, where particular test profiles are returned by patients where there is independent, confirmatory evidence of frontal lesions. The use of the term 'executive function' also came to be employed in situations where structural neuropathology could not be confirmed, but respondents returned impaired test protocols. Evidence for structural pathology is also lacking in a number of studies of BPD where there is evidence of abnormal neuropsychological test returns. These findings include 'multidimensional dichotomous thinking' (Veen & Arntz, 2000), so-called 'splitting' phenomenon (Leichsenring, 1999), visuoconstructive problem-solving tasks including the Rey Figure (O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993), the use of abstract conceptualisation (Burgess, 1991), measures of 'general cerebral efficiency' (Digit Symbol) (Judd & Ruff, 1993), response-conflict tasks including a 'colour-conflict' Stroop Task (Judd & Ruff, 1993; Kunert et al., 2003; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and behavioural inhibition as examined by

the go/no-go task (Dinn et al., 2004; Kunert et al., 2003; Leyton et al., 2001; Vollm et al., 2004).

A significant assumption underpinning the application of the 'frontal metaphor' in the study of psychopathology is the central importance attributed to the role of the prefrontal cortices (PFC) in human cognition (Pennington & Ozonoff, 1996). Baddeley (1986) has also argued against the use of the term 'frontal' in describing prefrontal injury, and has instead suggested the preferred term of 'dysexecutive syndrome'. This perspective is consistent with an emerging neuroscience paradigm suggesting that cognitive impairments occur as a result of disturbances to heterarchically organised, distributed networks in contrast to specific, focal lesion sites (Grigsby & Stevens, 2000). Stuss (1992) also argues against a 'localisationist' approach because frontal lobe processes reflect psychological constructs as opposed to specific, anatomically localised functions.

Welsh & Pennington (1988) define executive function as the ability to utilise appropriate problem solving strategies in order to realise future goals. These problem-solving strategies can involve the inhibition or deferral of responses to more appropriate times, the development of a plan of strategic action sequences, and finally, the utilisation of mental representations of the task, including the relevant stimulus information encoded into memory in order to attain the desired future goal-state. This perspective on executive function emphasises the role of a limited-capacity, central processing system in which working memory is a central function (Baddeley, 1986, 1995; Shallice, 1982).

Lezak (1993) suggests that executive disorders associated with head trauma typically involve deficits in relation to self-determination, self-direction,

self-control and self-regulation. In this sense, the development of the concept of executive function has emerged out of the study of behavioural syndromes associated with what has generally been termed as the 'frontal lobe syndrome'. Therefore, whilst frontal lobe syndromes are highly diverse, they generally involve impaired memory systems (most importantly, to 'working memory'), impaired response inhibition, impaired ability to effectively regulate emotional states, and impaired organisation and planning of complex behavioural sequences. Grigsby & Stevens (2000) argue that executive functions enable the individual to 'engage with life', to respond flexibly to unfamiliar situations, to inhibit irrelevant or routine behaviour when it is functional to do so, and to learn from experience in real time.

Hanes, Andrewes, Smith, & Pantelis (1996) argue that executive control also involves abilities such as strategy formation and motor programming. These capacities are thought to be centrally involved in the organisation of complex behaviour. They argue that such abilities are amongst the most complex of intellectual functions and rely upon the integrity of neural structures, which emerge late in the course of ontogenetic development.

In recent times, the study of executive functioning has progressed from the application of anecdotal and qualitative descriptions of executive impairment, to the application of structured tests which are thought to assess salient aspects of executive function (Barkley, 1996, 1997; Denckla, 1996; Lezak, 1995; Pennington, 1997). Lezak (1995) describes executive disorders as consisting of:

1. Disorders of volition (the capacity for awareness of oneself, one's surroundings, and one's motivational state);

2. Impairments in planning capacity (the ability to conceptualise change and plan for the future, the capacity to psychologically distance oneself from an immediate situation in order to think abstractly, to conceptualise alternatives in decision making, and to sustain attention and to engage in planning);
3. Disorders of purposive action (the capacity to persist with specific action sequences, and to engage in activities which serve the purpose of regulating the self);
4. Disorders of performance perceptiveness (the capacity to examine the quality of one's behavioural actions in order to assess and, where necessary, engage in behaviour change in order to improve or otherwise adapt behavioural sequences such that they achieve an improved quality of behavioural outcome).

Executive function has the potential to be an extremely useful concept to both researchers and clinicians. The attractiveness is associated with a concept that has the capacity to explain the generation, maintenance, and generalisation of actions across place, space and time. Despite this, 'executive function' is a concept which has the capacity to become embroiled in theoretical and terminological confusion with related constructs such as 'self-regulation' and 'planning' (Borkowski & Burke, 1996). Barkley (1996) argues that executive function is difficult to operationally define, although Denckla (1996) and Stuss & Benson (1986) argue that planning and control are central features to the concept. Borkowski & Burke (1996) argue that inhibition, attention, and memory are also central features to the concept of executive function.

Part of the confusion in the development of the concept of executive function arises out of the association between executive function (which appears

to have originally been a term derived from cognitive psychology) with the observations from the neuropsychological literature where frontal lobe compromise affected executive function. As a result, 'frontal functions' are often considered to be synonymous with 'executive functions', although Denckla (1996) cautions against this formulation. In an attempt at rapprochement on this issue, Stuss (1992) proposed that terms such as 'executive function' (Stuss & Benson, 1986), 'supervisory system' (Shallice, 1982, 1994), or 'dysexecutive syndrome' (Baddeley & Wilson, 1988), relate to the psychological proposition of frontal-system function, and can be used irrespective of evidence for an underlying anatomical disturbance. Denckla (1996) argues that the concept of executive function has been confounded with the theories of 'prefrontal' function, and this should be resisted.

Many theories of executive function emphasise the information processing components of the task (Borkowski & Burke, 1996). In contrast, Denckla (1996) argues that executive functions are primarily control processes that involve inhibition and response delay, planning, working memory, and inhibition of emotional expression. Barkley (1996) argues that executive function incorporates a number of themes that distinguish it from the concept of attention. Firstly, it refers to functional rather than conditional relations. Executive functions involve 'chains' of behaviour in which environmental events set the context for appropriate action. Secondly, executive function acts as a response that serves the function of altering the probability of the subsequent response of the individual. Thirdly, the temporal proximity of the events within the behavioural chain is no longer a delimiting factor – links can be made between

environmental events, responses and consequences which are not temporally related.

The following sections draw upon these perspectives of executive functioning in order to articulate a multidimensional developmental neuropsychological theory of executive disorder in BPD. The model emphasises the role of control systems in executive functioning, and these are mediated by the capacities of the individual in relation to working memory, inhibition, attention, and problem-solving.

3.4. A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL THEORY OF EXECUTIVE DISORDER IN BPD

A multidimensional developmental neuropsychological model of BPD is proposed. This model suggests that BPD involves a number of impaired executive functions including working memory, behavioural inhibition, affective-attentional bias, and problem-solving ability. These impaired executive functions represent the cognitive manifestations of underlying deficits in a distributed corticolimbic regulatory system. These deficits occur as a result of the influence of number of independent risk factors that include a genetic and psychobiological predisposition to BPD, that are subsequently influenced by early loss and/or separation, parent and/or family psychopathology, impaired parental bonding and/or attachment pathology, and trauma usually in the form of child abuse and/or neglect. The interaction of these factors results in the failure of an 'experience-dependent' maturation of orbitofrontal-subcortical (limbic) networks that in turn result in the neuropsychological architecture of BPD and

the resultant impaired executive disorders hypothesised to characterise the disorder.

The proposed model argues that the executive functions of working memory, behavioural inhibition, affective-attentional bias (affect regulation), and problem solving share interdependent relationships with each other, and act in a 'co-operative' or 'seamless' fashion in order to effectively regulate the transactions between the person and the environment. Impairment in one domain of executive functioning has the potential to contribute to impairment in other domains of executive functioning. For example, the inability to effectively regulate affective states is likely to result in episodes of affect dysregulation which can in turn provoke behavioural dysregulation which can in turn provide the basis for 'impulsive' acting out. Similarly, failure to successfully execute a problem solving sequence can lead to affective dysregulation which in turn can lead to 'impulsive' behavioural enactments as a means of restabilizing a dysregulated affective-attentional system.

One advantage of the proposed model is that it does not assume one predominant causal pathway for BPD. Rather, BPD is viewed as a final common pathway for a number of independent risk factors. The proposed model is also consistent with the cognitive perspectives of Beck (A. Beck et al., 1990), Young (1990), as well as the biosocial-cognitive perspective of Linehan and colleagues (Heard & Linehan, 1993; Linehan, 1993; Linehan et al., 1991; Wagner & Linehan, 1997). The model is also consistent with the various evidence-based psychoanalytic theorists who emphasise identity diffusion (Clarkin et al., 2004; Clarkin et al., 1999), disturbed self-systems (Monsen et al., 1995; J. Stevenson &

Meares, 1992, 1999), or attachment-based difficulties (Bateman & Fonagy, 2001) in the genesis of BPD.

Figure 3.1 illustrates the factors associated with the developmental neuropsychological model of impaired executive function in BPD.

Figure 3.1: Multidimensional Developmental Neuropsychological Model of Impaired Executive Function in BPD

BPD RISK FACTORS						BPD CRITERIA		IMPAIRED EXECUTIVE FUNCTIONS
1. Genetic/ Psychobiological Predisposition		Maturational Failure				Affect Dysregulation	→	Affective Attentional Bias
2. Early Loss/ Separation		of a		Development		Impulsivity	→	Behavioural Disinhibition
3. Parental Psychopathology		CNS		of		Interpersonal Dysregulation		Impaired
4. Family Psychopathology	→	Distributed	→	BPD	→	Identity Disturbance		Working Memory
5. Impaired Parental Bonding		Regulatory				Fears of Abandonment		
6. Attachment Pathology		System				Social Maladaptation		Impaired Problem
7. Trauma: Child Abuse & Neglect						Transient Paranoid Ideation		Solving

3.4.1. A Multidimensional Risk Model for BPD

The multidimensional developmental neuropsychological model of executive disorder in BPD identifies a number of independent risk factors that are predictive for BPD. Many of these were identified in Sections 2.2.4, and 2.2.5. In brief, the risk factors for BPD include genetic and psychobiological factors, early loss and/or separation from caregivers, ongoing parent and/or family psychopathology, ongoing impaired parental bonding and/or attachment pathology, and trauma usually in the form of child abuse and/or neglect. It is proposed that various combinations of these factors predict the development of BPD. One co-related effect will be the development of poorly integrated orbitofrontal-subcortical regulatory systems in BPD participants.

Poor integration of orbitofrontal-subcortical regulatory systems is thought to occur at the following levels of analysis:

1. Reduced frontal lobe, hippocampal, amygdala, and anterior cingulate volumes (Driessen et al., 2000; Lyoo et al., 1998; Tebartz van Elst et al., 2003);
2. Impaired functioning of orbitofrontal-subcortical pathways that develop as a result of aberrations in the neurogenesis-pruning relationship which occurs in the first 12-18 months of life, and subsequent failure of the CNS to develop adequately (Schore, 2003a). This process is directly influenced by the quality of parent-child interaction across the course of the early developmental life-span (Schore, 2003a, 2003b; Trevarthen & Aitken, 1994);
3. Ineffective neurotransmitter systems which develop as a result of impaired metabolic functioning within orbitofrontal-subcortical systems in BPD

(Donegan et al., 2003; Goyer et al., 1994; Hansenne et al., 2002; Leyton et al., 2001; Vollm et al., 2004).

These effects are hypothesised to predict the development of the executive deficits of working memory, response inhibition, affective-attentional bias, and problem-solving in BPD.

3.4.2. Impairments to the 'Central Executive': Working Memory, Response Inhibition, Affective-Attentional Bias, and Problem-Solving

This aspect of the model proposes that the impaired regulatory systems in orbitofrontal-subcortical systems result in impairments to the 'central executive' (Shallice, 1982). For people diagnosed with BPD, the impairments in cognitive self-regulation result in deficits in working memory, the capacity to regulate impulsive behaviour, biased attention toward affectively laden stimuli, and deficits in problem-solving. Figure 3.2 details the nature of impairments to the central executive in BPD.

Figure 3.2: Hypothesised Impairments to the Central Executive in BPD

CENTRAL EXECUTIVE IN BPD

MEMORY SYSTEM

Procedural Memory

As a Result of Impaired Working Memory, Oversimplified Procedural Memory Systems Develop

Results in Defective Interpersonal Process Experiences, Attachment, And Primitive Modes of Processing

Working Memory

Impaired Ability to Hold In Mind Verbal and Non-Verbal Material

Impaired Limited Capacity Processor

Results in Poor Mnestic Returns

EMOTIONAL-ATTENTION SYSTEM

Identifies and Regulates Arousal and Affects

Allocates Too Many Attentional Resources to Affective, Novel, or Threatening Stimuli

INHIBITION SYSTEM

'Impulsivity' Reflected in Deficits in Response Inhibition

Results In:

Incapacity to Stop Ballistic Processes
Incapacity to Modify Ballistic Processes Ability Once Initiated

PROBLEM-SOLVING

Poor Strategy Selection for Regulating Emotion

Use of Oversimplified Cognitive Strategies

Deficits in 'Set-Shift'

Deficits in Sequential Learning

Deficits in Planning

Deficits in Hypothesis Testing Capability

Deficits in Concept Formation

3.4.3. Impaired Executive Function in BPD

There is no literature that has specifically examined executive impairment in BPD. A number of studies report evidence consistent with impaired executive functioning in BPD although this conception is not specifically referred to in these studies (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990; Dinn et al., 2004; Judd & Ruff, 1993; Swirsky-Sacchetti et al., 1993). These studies have generally employed different measures to assess functions which in this study are considered to be 'executive functions'. As a result, there is a lack of comparability between studies regarding the nature of executive deficits in BPD. Whilst all executive functions are important, it is often argued that 'working memory' (Baddeley, 1995) and behavioural inhibition (Barkley, 1996) are central to executive functioning (Lezak, 1995). It is imperative therefore, that these specific functions be considered in any systematic analysis of executive functioning in BPD.

It is argued here however, that there are four principle executive functions that underpin the hypothesised deficits in BPD. It is proposed that working memory, behavioural inhibition, affect regulation as examined by an affective-attentional bias paradigm, and problem-solving account for the impairments observed in BPD. It is further argued that these executive functions act in a 'seamless' manner to provide a sense of coherence and adaptability in non-BPD participants. The view of executive function presented here is at variance with the view that executive deficits in BPD are characterised by frank organicity (Andrulonis et al., 1982; Soloff & Millward, 1983). Instead, the proposed model suggests that BPD develops as a result of the failure of 'experience-dependent' maturation of the CNS (Grigsby & Stevens, 2000; Schore, 2003a, 2003b). The

failure of experience-dependent maturation of the CNS results in defective executive function. This perspective is elaborated below.

3.5. EXECUTIVE FUNCTION I: IMPAIRED WORKING MEMORY IN BPD

Pennington, Benetto, McAleer, & Roberts (1996) argue that developing a unified theoretical account of executive function has been difficult because of the apparent heterogeneity of these tasks. They argue however, that there are common features to all EF tasks. These include the capacity to plan, and the capacity to maintain these plans 'on-line' or 'in mind' in order to execute the task effectively and to exclude or inhibit irrelevant actions. The capacity to hold material on-line has been referred to as 'working memory', and a number of theorists have argued that working memory is a central executive function (Baddeley, 1995; Barkley, 1996; Pennington, 1997; Shallice, 1982, 1994).

Working memory has its origins in two independent areas of research. The first area, short-term memory research, sees working memory as a system that holds limited information for short periods of time. The second area, computational modelling of higher cognitive processes, sees working memory as both a retrieval system but also as a series of intermediate processes in the computation of higher-order processes. Various examples of this latter notion of working memory include the myriad of underpinning processes involved in the execution of activities such as language production and in problem-solving (Smith & Jonides, 1995).

There also appears to be some controversy concerning the duration of storage in working memory systems. Some see working memory as a limited capacity memory store lasting anywhere between 10 and 20 seconds (Grigsby &

Stevens, 2000), whereas others regard working memory as decaying after one to two seconds (Baddeley & Hitch, 1994). This represents a major theoretical issue with regard to the validity of the concept of working memory, and has significant implications for the nature of tasks designed to examine working memory.

Baddeley (1995) has argued that working memory refers to a temporary storage system for information that is necessary for the effective performance of a wide variety of skills and tasks including comprehension, learning, and reasoning (problem-solving). Baddeley also notes that the term 'working memory' has been used in different ways by different theorists. These include the capacity to retain information across trials within the same testing session, simulated computational models of memory using computer protocols, and studies examining different forms of short-term memory (STM).

Components of Working Memory

Working memory refers to a hypothesised temporary memory storage system required in order to perform a wide variety of tasks including comprehension, learning, and reasoning (Baddeley, 1995). This concept has evolved to distinguish a multicomponent memory system from the earlier notion of generic 'short-term' memory. Baddeley & Hitch (1994) note that working memory has at least three separate meanings: an unlimited computational capacity; a storage and processing system; and a fractionation system which divides memory into a series of subcomponents. This latter approach is closely associated with neuropsychological approaches to understanding working memory (Pennington et al., 1996).

This latter approach is best represented by Baddeley's (1995) model of working memory. According to this view, working memory consists of three

subcomponents: a ‘phonological loop’ which holds and manipulates speech based information, a ‘visuospatial sketchpad’ which holds and manipulates visual and spatial information, and a ‘central executive’ which is an attentional control system which is aided by the phonological loop, and the visuospatial sketchpad in order to execute command and control processes (Baddeley & Hitch, 1994).

Baddeley (1995) suggests that the central executive is a limited-capacity system that is responsible for linking the systems of the visuospatial sketchpad and the phonological loop with long-term memory (LTM). Furthermore, it is also responsible for planning and strategy selection and therefore emphasises the role of attentional control. Baddeley also argues that the two forms of working memory are dissociable from each other. One form, the visuospatial sketchpad stores visuospatial information. This system also appears to be dissociable into a subsystem that stores material associated with colour and shape on the one hand, and another subsystem that is concerned with spatial location. The phonological loop stores memory for sounds. This system also appears to be dissociable into a subsystem capable of holding phonological information for periods of between one and two seconds, and an articulatory control process that ‘refreshes’ phonological information through the use of sub-vocal articulation. This latter process is similar to the concepts of ‘private speech’ (Vygotsky, 1987), or ‘internalisation of language’ (Bronowski, 1977). The functional significance of the visuospatial sketchpad is associated with its involvement in planning and executing spatial tasks. Equally, the functional significance of the phonological loop appears associated with its involvement in language acquisition, language comprehension, and as one component of a verbally-mediated control system.

Pennington et al. (1996) also argue that working memory is important because it enables the temporary use of on-line constraints relevant to the immediate context such that effective adaptation to the environment is facilitated. This can include specific features of the immediate environment, the affective state of the subject, and interactive material drawn from long-term memory. These functions clearly subserve a range of capacities that impinge upon adaptability to the environment, and for these reasons appear central to any model of executive functioning in BPD.

Baddeley (1995) argues that this approach to understanding working memory can explain diverse phenomenon such as learning in animals, artificial intelligence, cognitive development, and language acquisition. Apart from the understanding that working memory probably underpins other executive functions, there is a body of evidence that suggests that deficits in learning from experience are characteristic of BPD (Grotstein, 1987; Grotstein et al., 1987). Therefore, it is possible that the difficulties attributed to borderlines with regard to learning from experience might be an artifact of deficits in working memory.

3.5.1. Empirical Evidence for Impaired Working Memory in BPD

A number of studies have examined memory function in BPD (Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993). However, only one study has explicitly reported examining working memory (Kunert et al., 2003), although other studies have employed tasks that could be interpreted within a working memory paradigm

(Bazanis et al., 2002; Cornelius et al., 1989; Judd & Ruff, 1993; Kurtz & Morey, 1999; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993).

A variety of memory functions have been examined in BPD. This has included complete Wechsler Memory Scale returns (WMS) (Wechsler, 1987) (Cornelius et al., 1989; O'Leary et al., 1991), or other studies that employed selected WMS subtests including Logical Memory (LM) (Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993)[1], Visual Reproduction (Cornelius et al., 1989; O'Leary et al., 1991), Paired Associates Learning (PAL) (Cornelius et al., 1989; Dinn et al., 2004; O'Leary et al., 1991; van Reekum, Conway et al., 1993)[2], Figural Memory (FM) (Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and Digit Span (DS) (Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[3]. These studies have returned mixed findings. Table 3.1 summarises the tests employed in the various studies examining memory in BPD.

[1] These Findings Refer to Dinn et al. (2004) (Study One)
[2] These Findings Refer to Dinn et al. (2004) (Study One)
[3] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

Table 3.1: Tests Employed in the Studies Examining Memory in BPD

	LOGICAL MEMORY	VISUAL REPRODUCTION	PAIRED ASSOCIATES	FIGURAL MEMORY	DIGIT SPAN	VERBAL LEARNING/ MEMORY TASKS
Bazanis et al. (2002)	N	N	N	N	N	N
Burgess (1990)	N	N	N	N	Y[b]	Y[a]
Burgess (1991)	N	N	N	N	Y[b]	N
Cornelius et al. (1989)	Y[b]	Y[b]	Y[b]	N	Y[b]	N
Dinn et al. (2004) (Study 1)	Y[a]	N	Y[b]	N	Y[b]	N
Dinn et al. (2004) (Study 2)	N	N	N	N	N	N
Driessen et al. (2000)	Y[b]	N	N	N	N	N
Judd & Ruff (1993)	N	N	N	N	Y[b]	Y[a]
Kunert et al. (2003)	N	N	N	N	N	Y[b]
Kurtz & Morey (1999)	N	N	N	N	N	Y[a]
O'Leary et al. (1991)	Y[a]	Y[b]	Y[b]	N	Y[a]	Y[b]
Sprock et al. (2000)	Y[b]	N	N	Y[b]	Y[b]	Y[b]
Swirsky-Sacchetti et al. (1993)	Y[b]	N	N	Y[a]	Y[b]	Y[b]
van Reekum et al. (1993)	N	N	Y[c]	N	Y[c]	N

Y = Yes; N = No; a = significant difference between groups; b = non- significant difference between groups; c = not interpretable

Table 3.1 (Continued): Tests Employed in the Studies Examining Memory in BPD

	STORY/ WORD RECALL	VISUAL RECOGNITION MEMORY	VERBAL WORD LISTS	FACIAL MEMORY TEST
Bazanis et al. (2002)	N	Y[b]	N	N
Burgess (1990)	N	N	Y[a]	N
Burgess (1991)	N	N	N	N
Cornelius et al. (1989)	N	N	N	N
Dinn et al. (2004) (Study 1)	N	N	N	N
Dinn et al. (2004) (Study 2)	N	N	N	N
Driessen et al. (2000)	N	N	N	N
Judd & Ruff (1993)	N	N	N	N
Kunert et al. (2003)	N	N	N	N
Kurtz & Morey (1999)	N	N	N	N
O'Leary et al. (1991)	N	N	N	Y[b]
Sprock et al. (2000)	Y[b]	N	N	N
Swirsky-Sacchetti et al. (1993)	N	N	N	N
van Reekum et al. (1993)	N	N	N	N

Y = Yes; N = No; a = significant difference between groups; b = non- significant difference between groups; c = not interpretable

WMS verbal memory functions have returned a variety of findings. Two studies (Dinn et al., 2004; O'Leary et al., 1991)[4], reported deficits on Logical Memory, whereas other studies have found no differences (Cornelius et al., 1989; Driessen et al., 2000; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). Paired Associates Learning (PAL) returns were not significant in the studies of Cornelius et al. (1989), Dinn et al. (2004) (Study One), and O'Leary et al. (1991), and were not able to be interpreted in van Reekum et al. (1993) because direct comparisons between the experimental and control group were not reported. Similarly, Digit Span (DS) returned significant differences in one study (O'Leary et al., 1991), and non-significant results in six others (Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993)[5]. A non-significant DS was also reported by Judd & Ruff (1993), but this task was drawn from the WAIS-R rather than the WMS. Again, the study by van Reekum et al. (1993) could not be interpreted.

WMS non-verbal memory functions have also returned a variety of findings. Two studies have reported findings on Visual Reproduction (VR), and both found that BPD is not characterised by deficits in this area (Cornelius et al., 1989; O'Leary et al., 1991). Two studies have examined Figural Memory, with Swirsky-Sacchetti et al. (1993) reporting deficits in BPD, and Sprock et al. (2000) reporting non-significant findings.

A series of verbal-learning paradigms have been reported, many of which appear to rely upon notions of working memory similar to that proposed by Baddeley (1995). These include Verbal Learning and Memory Tasks with various forms of affective and non-affective interference artifacts (Kunert et al.,

[4] These Findings Refer to Dinn et al. (2004) (Study One)
[5] These Findings Refer to Dinn et al. (2004) (Study One)

2003; Swirsky-Sacchetti et al., 1993), a Verbal Recall Memory Task (Kurtz & Morey, 1999), a Verbal Word-List Learning Task (Burgess, 1990), a Story Recall Task (Sprock et al., 2000), a word recall task with affective and neutral interference conditions (Sprock et al., 2000), Verbal Incidental Learning Test (O'Leary et al., 1991), and the Selective Reminding Test (Judd & Ruff, 1993; Kunert et al., 2003).

A number of these studies have found deficits in various verbal-learning paradigms including verbal recall (Kurtz & Morey, 1999) or word-list learning (Burgess, 1990), whereas other studies found no evidence of deficits in BPD for verbal learning and memory (Kunert et al., 2003; Swirsky-Sacchetti et al., 1993), story recall (Sprock et al., 2000), word recall (Sprock et al., 2000), or verbal-incidental learning (O'Leary et al., 1991). Mixed evidence has been reported for the Selective Reminding Test with Judd & Ruff (1993) reporting deficits in BPD, and Kunert et al. (2003) failing to detect differences.

Finally, visual learning paradigms including Visual Recognition Memory (Bazanis et al., 2002), and a Facial Memory Task (O'Leary et al., 1991) have also been examined. Neither study found deficits in BPD on these measures.

In summary, there is mixed evidence of deficits in a wide variety of memory functions in BPD. It is speculated that the equivocal nature of these findings is predominantly associated with a range of methodological issues inherent in the designs of the respective studies. Many of these methodological issues are common to other executive functions and will be considered in detail in Section 3.9.

There are however, two issues associated with characterising working memory that have implications for the current study. These include the duration

of storage involved in working memory systems, and the absence of available measures of working memory.

A major issue is associated with the question of the duration of storage in working memory systems. Some see working memory as a limited capacity memory system that stores information for periods between one and two seconds (Baddeley & Hitch, 1994), whereas others regard working memory a lasting anywhere between 10 and 20 seconds (Grigsby & Stevens, 2000). This has significant implications for the nature of the measurement of WM. Until consensus occurs with regard to the basic parameters of working memory, it will be difficult to make progress in understanding working memory in BPD.

As a result of this conceptual difficulty, there also appear to be a limited availability of appropriate measures of working memory. Although Pennington (Pennington, 1997; Pennington et al., 1996) have identified a number of measures of working memory, many of these are either experimental in nature or have been developed for use with paediatric populations. More importantly, there appears to be little evidence available to suggest that these tasks actually represent more effective measures of working memory than some of the tasks that comprise well-established memory tests for which adequate norms are available. This is a particularly important issue when combined with a consideration of the duration issues associated with working memory. This issue will continue to compromise research into WM until a consensus prevails regarding appropriate working memory measures.

3.5.2. Assessment of Working Memory in BPD

As a result of the absence of consensus described in the previous section, it was decided to employ a number of tasks selected from the Wechsler Memory

Scale – Revised (WMS-R) (Wechsler, 1987) and the Wechsler Adult Intelligence Scale – Revised (WAIS-R) (Wechsler, 1981) to examine working memory in the present study. The tasks that were selected were included because they come from authoritative tests with well established norms, and fit within the Grigsby & Stevens (2000) parameters of working memory. In addition, these tasks are commonly used, and thus allow the findings from this study to be compared with a wide variety of other studies. The tasks selected for the measurement of working memory included the following:

1. Logical Memory (LM) (Wechsler, 1987)
2. Visual Reproduction (VR) (Wechsler, 1987)
3. Paired Associates Learning (PAL) (Wechsler, 1987)
4. Digit Span (DSp) (Wechsler, 1981)
5. Visual Memory Span (VMS) (Wechsler, 1987)

A detailed description of these measures is provided in Appendix III.

3.6. EXECUTIVE FUNCTION II: IMPAIRED RESPONSE INHIBITION IN BPD

Impulsivity is considered to be a core feature of BPD, and the evidence for this was reviewed in Section 2.2.3. This view suggests that BPD is a specific personality disorder which shares common features with other disorders of impulse control such as substance use disorders, Antisocial Personality Disorder (ASPD), and to a lesser extent, eating disorders. The feature common to these disorders is a 'propensity to action' (Zanarini, 1993).

There are a number of conceptual problems associated with the available studies marshalled to support the view that impulsivity is a core problem in BPD. These include:

1. The meaning of 'impulsivity' is not well defined in the clinical or theoretical literature on BPD (Hochhausen et al., 2002). It is unclear whether the term 'impulsive' involves any of the definitions outlined in Section 2.2.3, or whether other meanings might be involved;
2. Few of the reported studies directly examine impulsivity in BPD. For example, the study reported by Links et al. (1999) relied upon a self-report measure of impulsivity (the 'Impulsivity' Scaled Score from the DIB). The problem with this approach is that this score is arrived at via self report, and this should not be regarded as an appropriate measure of impulsivity. Similarly, the decision-making task reported by Bazanis et al. (2002) which was regarded as a test of impulsivity, could equally be seen to be a measure of frustration-tolerance. Future studies need to be directed toward employing 'ecologically valid' methods of assessing impulsivity, and one resolution to this impasse would be to employ methods derived from cognitive or experimental psychology. Section 3.6.1 below describes an alternative methodology for assessing impulsivity which measures response inhibition rather than the more generic conception of 'impulsivity'.
3. A number of studies employ measures of go-no/go tasks in examining BPD (Dinn et al., 2004; Kunert et al., 2003; Leyton et al., 2001; Vollm et al., 2004)[6]. In the paradigm employed in this study, it is argued that the equivalent of the go/no-go task (the Stop-Signal paradigm) represents one method of measuring response inhibition. In the identified studies, go-no/go tasks have been employed as measures of response conflict. Whilst the concept of response inhibition is not identical to that of impulsivity, it is argued that it

[6] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

represents an operationalised derivative of the more global concept of 'impulsivity'. It is also argued response inhibition represents a more specific construct and is therefore more likely to meet acceptable scientific standards for measurability and reproducibility.

It is also important to recognise that the few studies that have directly examined 'impulsivity' in BPD also have a number of methodological limitations that result in difficulties in interpreting the findings. Again, many of these methodological issues are also common to the studies examining other executive functions in BPD. Therefore, these methodological issues will be addressed separately in Section 3.9.

3.6.1. Assessment of Response Inhibition in BPD: The Application of the 'Stop-Signal' Task

In a review of executive control of thought and action, Logan (1985) describes a cognitive model for examining successful task performance. He suggests that a number of executive functions are involved in successful task performance. These include:

1. The capacity to make choices about alternative strategies for processing environmental stimuli;
2. The capacity to develop a version of a chosen strategy in order to enable task performance;
3. The capacity to control and coordinate executive strategies during real time performance of the task;
4. The capacity to disable or disengage the strategy in response to changes in goals or changes in the task environment which render the current strategy inappropriate.

Point Four identifies the role of an executive function in terminating the production or continuance of a ballistic process in response to changes in the environment. This is the function that response inhibition performs in the control of behaviour, and it is argued that this represents the underlying process that is centrally important in understanding impulsivity in BPD.

Logan (1985) argues that cognitive strategies can be disengaged from motor systems in approximately 200 milliseconds, and this in part provides the basis for the capacity to inhibit actions in an efficient and timely manner. Furthermore, control structures for behavioural inhibition are thought to be hierarchical in nature. Therefore, functions such as speech and motor activity are controlled locally from second to second, by an 'executive process' that supervises production (Logan, 1985). This executive process appears to perform a monitoring function, with the capacity to intervene when necessary. This in turn suggests that thought and action are controlled in a linear fashion by an over-riding executive control mechanism which ensures that the task under consideration is performed effectively, and sustained in a highly adaptive manner. This further suggests that the capacity to inhibit action occurs almost immediately under normal circumstances, and can also be implemented almost instantaneously because control is both local and monitored on an ongoing basis.

One of the key features of an intact executive system is utilisation of appropriate control processes (Shallice, 1982). Logan & Cowan (1984) have described this aspect of executive function as consisting of a series of 'acts of control' that ensure coherent thought, action and appropriate task selection in order to facilitate successful goal-directed behaviour. The implication of this

conceptual framework suggests that one important feature of self-control is the ability to inhibit thought and action.

This aspect of executive functioning also assists in controlling the execution of novel behaviour that in turn enables the coordination of multiple behavioural responses. This can and does include the capacity to interrupt or modify behavioural sequences (Logan, 1994). In addition, in order to exert maximal control over the environment, it is also necessary to be able to inhibit inappropriate behaviours, or behavioural responses that are no longer contextually relevant. Accordingly, response inhibition is argued to be a measurable referent or analogue of the clinical phenomenon otherwise referred to as 'impulsivity'.

Logan (Logan, 1994; Logan, Cowan, & Davis, 1984) has developed an information-processing task known as the Stop–Signal paradigm which is a method for examining behavioural inhibition. The Stop–Signal paradigm employs a computer program that requires participants to inhibit responses (pressing the 0 or X keys on a computer keyboard) in response to specific task demands. The objective underpinning this task requires participants to respond when a specific environmental cue is produced, and inhibit responding when a paired cue signalling the requirement to stop the initial response is presented.

The goal of the current study was to examine the components underlying the hypothesised inhibitory deficit in BPD. This was facilitated by measuring the capacity of BPD participants to achieve inhibitory control across a range of conditions, and by measuring the speed (latency) of inhibitory control.

Response inhibition is understood to be mediated by the motor cortex during a response planning and preparation stage that occurs immediately prior

to the execution of the motor command (Badcock et al., 2002). Unlike other well documented neuropsychiatric conditions, BPD does not appear to be mediated by a specific cortical network. Nevertheless, there is sufficient neuropsychological, neuropsychiatric, and neuroimaging data available to suggest that 'impulsivity' in BPD is probably mediated by an orbitofrontal-subcortical network involving the amygdala, cingulate cortex, hippocampal, prefrontal, and orbitofrontal regions (Donegan et al., 2003; Driessen et al., 2000; Lyoo et al., 1998; Tebartz van Elst et al., 2003; Vollm et al., 2004). As a result, it is hypothesised that one of the end-stage processes involved in the execution of an 'impulsive' act in BPD involves the failure of the response planning and preparation stage in motor command execution to inhibit a prepotent response. This specific mechanism can be directly examined by the Stop-Signal Paradigm.

The 'Race' Model of Response Inhibition

The stop-signal paradigm is a computer generated inhibition task based on a cognitive-information processing model of the stopping process (Logan, 1994; Logan & Cowan, 1984). The paradigm involves examining the relationship between two temporally related tasks, a 'go-task' and a 'stop-task'. The protocol used in the present study employed a forced-choice reaction time task associated with the presentation of two visual stimuli. This was used as the dominant or 'prepotent' go-task response. The stop-task involved the presentation of an infrequent signal (a tone) that countermanded the 'go' signal. On 'stop-signal' trials (that subset of trials where the 'stop' signal was issued) the participant was required to inhibit their response for that trial. It is argued that this 'central act of control' (Badcock et al., 2002; Logan, 1994), is similar to the type of stopping mechanism involved in many routine activities in which behaviour must be re-

regulated in response to new task demands or situational requirements. The stopping task examines the capacity to inhibit a prepotent response. It is likely that it involves components of executive function similar to the Wisconsin Card Sorting Test, the Colour-Conflict Stroop, and various forms of the Antisaccade task. It differs from these tasks however because it involves the capacity to stop a behaviour which has already commenced (Badcock et al., 2002).

The theory of the stop-signal paradigm is based upon an underlying theory known as the 'race' model of inhibition (Badcock et al., 2002; Logan, 1994). This model predicts that the capacity to inhibit behavioural responses depends upon the outcome of a race between the initiation of, and the inhibition of, a response to a prepotent stimulus. The theory suggests that once the 'go-task' process has commenced, the 'stop' process must be initiated with sufficient temporal proximity to the 'go-task' process in order to be able to successfully inhibit the execution of the task. The stop-signal paradigm measures control in terms of the capacity to achieve control and in terms of measuring the latency of control. By implication, poor inhibitory control is associated with differing components of the 'go-stop' process. There are at least three factors which can affect inhibitory control. These include, fast responding to the prepotent (go) stimulus, slow responses to the 'stop' signal, or other difficulties in initiating the stop process.

The prepotent or 'go-task' process commences with the presentation of the 'go-task' stimulus. At this point, the participant is primed to initiate the 'go' task. The timing of the inhibitory response occurs at a point beyond this aspect of the process and is initiated with the onset of the 'stop-signal'. Thereafter, the relative finishing times of the go and the stop processes determine the outcome

of the race. If the go process is executed more quickly than the stopping process, then the response will occur. If the stop (inhibition) process is executed more quickly than the go process, then no discernible response will be observed – inhibition will be 'successful'. The finishing times of the stop and go processes are relative to each other, and are determined by the speed and variability of each process and by the likelihood of the inhibition process being executed. Furthermore, the latency between the onset of the go and the stop-signal also influences the outcome. If there is a short delay (i.e., up to approximately 200 mSecs) following the onset of the 'go' signal, it is highly likely that there will be inhibition of the response (Logan, 1994). If however, there is a longer delay following the onset of the go signal before the stop-signal is presented, there is a probabilistically higher likelihood that a response will occur. In other words, the go process under this condition will win the 'race', and inhibition of the response will be 'unsuccessful'. Therefore, the inhibition function describes the probability of inhibiting responses across a range of stop-signal delays. This can be expressed mathematically as the flatter the slope of the inhibition function, the less likely will be the capacity for successful response inhibition. Furthermore, if go task responses are significantly slower, then the probability of inhibition will be greater because the stopping process is the probabilistically more likely outcome (Badcock et al., 2002; Logan, 1994).

The anticipated individual variations associated with go processing speed are typically controlled for experimentally by presenting the stop-signal at different levels of delay prior to the individual's anticipated response. The stop-signal paradigm employed in this study calculated each of the individual's mean reaction times to trials with no stop-signal present. Inhibitory functions were then

assessed for stop-signals presented at various intervals prior to the individual's Mean Reaction Time (MRT).

The speed of response to the 'go' response can be measured directly from those trials in which no stop-signal is presented. In contrast, the response latency of the stop-signal to a response cannot be directly observed, but it is possible to estimate this (Logan, 1994). The presentation of the stop-signal was determined by the design of the experimental protocol (Badcock et al., 2002). The point at which the inhibition process concluded for each trial was calculated from the distribution of the go-task reaction times. The go-task response distribution was rank-ordered and the number of reaction times in the distribution was multiplied by the probability of responding at a given delay. This formula was subsequently used to estimate the point at which the stop process terminated relative to the onset of the go signal. The Stop Signal Reaction Time (SSRT) relative to the commencement of the stop-signal was then determined by subtracting the stop-signal delay from this value. This methodology was employed in the paradigm to calculate the SSRT at each level of delay. The mean score was examined to compare the latency of the stopping process between groups. Where a significantly longer stop-signal latency occurs, it is likely that this represents evidence of impaired inhibition. The SSRT represents the difference between the point in which the stop-signal was presented and the point at which the inhibitory process terminated (Badcock et al., 2002).

The 'race' model of stopping developed by Logan (1994) represents a sophisticated model of response inhibition. It is argued that this represents an effective means by which the issue of impulsivity in BPD can be examined in detail, and specific aspects of the stopping process understood more

comprehensively. This study will therefore employ the Stop-Signal Paradigm in the form reported by Badcock et al. (2002).

3.7. EXECUTIVE FUNCTION III: IMPAIRED AFFECT REGULATION AND AFFECTIVE-ATTENTIONAL BIAS IN BPD

The third executive function hypothesised to be impaired in BPD is that of affect regulation. There are a number of different aspects of affect regulation which might be potentially impaired in BPD. These include:

1. Poorly developed or poorly integrated affective states. These states are thought to arise out of chronic misattunements between the nascent borderline child-to-be and the caregiving environment. The failure to adequately develop affective states results in the person with BPD experiencing non-specific somatosensory states which are 'pre-symbolic', and these usually represent prototypical undeveloped affective states. Clinically, affective distress is often experienced as somatic in nature, and the respondent often reports difficulties in identifying phenomena as affective in nature (Krystal, 1988).
2. An impaired capacity to accurately identify affective states. This involves the relative inability of the borderline to be able to identify specific affective states. It is a subcomponent of a lack of developed affective knowledge of the self. This is also one component of broader impairments to 'reflective self functioning' (Fonagy et al., 1995). Evidence is provided in Chapter Five that supports the view that borderlines have difficulty in identifying affective states.
3. Poor affect modulation and/or regulatory capacity. This involves the relative inability of the borderline to be able to regulate or control the arousal associated with an emergent affective experience, or to regulate the gradient

(time frame) of the experience of an affective state. This results in the often noted 'explosiveness' or 'affective lability' of borderlines (Linehan, 1993; K. F. Stein, 1996). This aspect of an impaired affective-attentional system also represents an alternative means by which 'impulsivity' in BPD can be understood. Evidence is provided in Chapter Five that supports the view that borderlines have difficulty in regulating affect.

4. Poor affect tolerance. This involves the relative inability of borderlines to effectively tolerate and manage the emergence of affective experiences which are dysphoric in nature (Clarkin et al., 1999; Linehan, 1993). Evidence is provided in Chapter Five that supports the view that borderlines have difficulty in tolerating intense affective states.
5. Poor integration of 'affect-blends'. This deficit involves the relative inability of borderlines to manage affective experience which involves either the combination of two or more concurrently experienced affects, or when positively and negatively valenced affects co-occur (Grotstein, 1987; Linehan, 1993; K. F. Stein, 1996).

It is proposed that impaired affect regulation in BPD can be understood to operate when dormant pathological character routines are initiated (Grigsby & Hartlaub, 1994; Grigsby & Schneiders, 1991), usually by a provoking event in the interpersonal domain (Farrell & Shaw, 1994), although an internally mediated event can also precipitate such events. An 'emotion episode' occurs which leads to a temporary state of affective dysregulation (N. Stein, Trabasso, & Liwag, 1993). The 'emotion episode' can be brief (minutes), but often occurs over periods lasting from several hours to several days. Emotion episodes are typically provoked by an internal set of cognitions, or by an event or series of

events which occur in the interpersonal environment which is experienced as adverse in nature. This results in the experience of an affect or most typically, a 'blend' of affects which are usually 'negatively' or antagonistically valenced (Linehan & Heard, 1992). The affects predicted to operate during a dysregulated affective episode include anger, anxiety, sadness, or shame.

The outcome of the experience of an emotion episode is an attempt by the borderline subject to re-regulate their internal affective state utilising a variety of mechanisms of self-regulation (Linehan, 1993; Schore, 2003a, 2003b). A common method of affect regulation utilised by borderlines is hypothesised to be the use of behavioural enactments that are employed in order to re-regulate internal affective states or decrease arousal (Wagner & Linehan, 1997). These enactments can include the use of impulsive violence, self-injurious or self-medicating behaviour, so-called 'manipulative' behaviour, or behavioural avoidance. The persistent use of these maladaptive behavioural strategies serves the purpose of assisting affect regulation in order transform affective states, or to regulate the arousal associated with the adversely experienced affect. When the use of these strategies has been effective, a return to a re-regulated state is observed, and the 'emotion episode' has therefore resolved. The participant returns to a 'homeostatic balance' until the next event occurs which disrupts their affective regulatory capacities, and thus provokes a subsequent 'emotion episode.' This process of impaired affect regulatory executive function appears to be similar to Schmideberg's (1959) proposition of 'stable instability' in BPD.

3.7.1. Empirical Evidence for Impaired Affect Regulation in BPD

Although the prevailing clinical view asserts that patients with BPD experience significant difficulty regulating emotion, there is limited empirical

evidence to support this view. There is nevertheless a widespread view that impulsive self-destructive behaviour arises out of deficits in the capacity to recognise, organise, and process affectively valenced material (Linehan & Heard, 1992; Westen, 1991). The following sections selectively review the empirical evidence for impaired affect regulation in BPD.

Impaired Affect Regulation in Non-Clinical BPD Populations

Trull (1995) conducted two studies on an undergraduate cohort of non-clinical young adults who met self-report criteria for BPD. In phase one, 90 BPD participants and 54 control participants were recruited into a laboratory study. All participants completed a self-report battery of tests which included a semi-structured diagnostic interview, and a number of self-report mood measures. Interviewers were blind to the group membership of participants (B+ for the Borderline Personality Disorder (BPD) group and B- for the control sample). B+ participants returned significantly higher scores on a variety of measures of depression and negative affect and significantly lower scores on measures of positive affect. B+ participants also returned significantly higher scores on trait dimensions of neuroticism and significantly lower on the trait dimensions of extroversion, agreeableness and conscientiousness. B+ participants obtained significantly higher scores on all measures of general psychopathology.

In a second study, Trull (1995) screened 1800 introductory psychology students using the same methods as described in the first study. Participants were assigned to an above threshold (B+), or a below threshold group (B-) on the basis of their self-report borderline score. 34 B+ participants and 54 B- participants were recruited into a laboratory study which examined interpersonal difficulties

in BPD. The results confirmed higher rates of interpersonal problems and distress on the part of the B+ group.

The results of these 'non-clinical' studies suggest that mood, coping style, and interpersonal distress is impaired in BPD. In addition, higher prevalence rates for a number of lifetime Axis I conditions including anxiety and mood disorder were observed to be elevated in non-clinical borderlines. These findings provide some evidence for affect dysregulation in BPD. The BPD criteria most prevalent in B+ cohorts included inappropriate or intense anger, impulsiveness, and affective instability. Significantly, a number of BPD features typically considered pathognomonic in clinical samples such as parasuicidal gestures were relatively rare in this collegiate cohort.

This study provided two other significant findings. Firstly, the absence of significant levels of parasuicidal gestures/acts in this sample provides some suggestive evidence that BPD is a dimensional rather than a categorical disorder because parasuicidal acts might be associated with more severe forms of the disorder. In addition, intelligence might act as a 'buffering' or resiliency variable that protects individuals from the more extreme features of the disorder. Secondly, this study provides further evidence of high prevalence rates for BPD in adolescent/young adult populations and the findings are therefore consistent with other studies (Bernstein et al., 1996; Bezirganian et al., 1993). The self-report measures for BPD suggested that 13-21% of the total sample met criteria for BPD. These prevalence rates are significantly higher than those reported for the general community, although these results may be an artifact of the use of a self-report instrument. This finding again provides further confirmatory support for the proposition that BPD might be best viewed as a developmental disorder

with the highest prevalence rates in the late adolescent/early adulthood phase of life.

Impaired Affect Regulation in Clinical BPD Populations

A number of different approaches have been employed to examine affect regulation in clinical BPD populations. These include studies examining the processing of affective information, and studies examining the regulation of affect in BPD. These are briefly reviewed.

Recognition and Processing of Affective Information in BPD

There are a small number of studies that have examined affect recognition and affect processing in BPD. These have included examining the quality of affect in the early memories of borderlines (Arnow & Harrison, 1991) and studies examining the identification and interpersonal perception of affect in BPD populations (Levine, Marziali, & Hood, 1997; M. I. Stern, Herron, Primavera, & Kakuma, 1997; Wagner & Linehan, 1999).

Arnow & Harmon (1991) studied the affective quality in the early memories of 15 'neurotic' participants, 15 DSM-III paranoid schizophrenics, and 15 DSM-III BPD participants. The BPD group reported fewer positively toned memories than controls, and the evidence was interpreted as supporting the hypothesis that borderlines experience limited or impoverished internally sustaining images of self and other.

Levine et al. (1997) and Wagner & Linehan (1999) examined emotion recognition and processing in BPD. Levine et al. studied a cohort of 30 DSM-III-R BPD participants and a non-psychiatric comparison group with a number of different self-report instruments measuring emotional awareness, facial expression, and affect intensity. Wagner & Linehan examined a cohort of 21

sexually abused DSM-III-R BPD participants, 21 sexually abused non-BPD participants, and a non-clinical control group.

Levine et al. (1997) confirmed a significant difference between the groups for measures of emotional recognition and emotional processing involving emotion differentiation in self and others, emotional ambivalence, recognition of facial expression of emotion and affect intensity. The data suggested that BPD participants experienced difficulties in recognising, differentiating, and integrating emotional states. Furthermore, BPD's responses to negatively valenced emotions were more intense than controls. Borderlines demonstrated a limited capacity for processing emotional information related to self and others, and this appeared most pronounced for ambiguous and conflicted emotional states.

Wagner & Linehan (1999) found that their BPD cohort did not demonstrate deficits in the capacity to recognise basic emotions. Furthermore, they found limited support for the hypothesis for a heightened sensitivity to negative emotional cues. Interestingly, the BPD group was more accurate in assessing fearful facial expressions only. The absence of a heightened sensitivity to negative emotions in BPD is interpreted by the authors as suggesting that facial recognition of emotion is poorly associated with affect dysregulation in BPD.

M. I. Stern et al. (1997) examined the interpersonal perceptions of 55 depressed DSM-III-R BPD's and 22 Major Depressed, non personality disordered patients in order to assess whether distortions in interpersonal perceptions differentiate the two groups. The findings suggested that the depressed BPD group did not distort interpersonal perceptions more than the

non-borderline depressed-only group and that depressed-only patients who were not personality disordered inhibit the expression of anger when hostility is directed towards them. The evidence also suggested that the depressed BPD cohort tended to behave in a more hostile manner when they experienced personal attack and they viewed themselves as more hostile and emotionally labile than did the Major Depressed cohort. The Borderline group rated their own behaviour and that of both parents more negatively than was the case for the Major Depressed comparison group. When assessing current relationships, borderline patients assessed themselves but not their relatives as significantly more hostile than did the Major Depressed group.

Affective Instability and Regulation of Affect in BPD

There are a small number of studies that have directly examined affective instability and affect regulation in BPD. This has included examining the nature of the 'soothing tactics' employed by borderlines (Sansone, Fine, & Mulderig, 1991), studying patterns of affect lability in BPD (K. F. Stein, 1996), the use of behaviour as a regulator of emotion (Wagner & Linehan, 1997), examining specific features of affect regulation and it's relationship to borderline phenomenon (Yen, Zlotnick, & Costello, 2002), and investigation of arousal-based hypervigilance processes in BPD (Arntz et al., 2000; Herpertz et al., 2000; Koenigsberg et al., 2002).

Sansone et al. (1991) studied the use of 'self soothing' tactics in a cohort of 25 DSM-III-R BPD's and 43 college student controls. Participants completed an instrument termed the 'Soothing Questionnaire' (SQ). The SQ consisted of 14 items assessing how often specific objects were used to soothe the participant, 16 items measuring specific actions employed to self-soothe, 16 items measuring

maladaptive soothing behaviours such as smoking, drug taking, alcohol usage, and 10 items measuring psychological activities which assess the frequency with which abstract activities were employed as self-soothing mechanisms.

The results suggest that Borderline participants reported comparable usage of soothing objects, behaviours, and activities. The findings also demonstrated that borderlines employed more maladaptive soothing behaviours than controls but found that borderlines did not report different levels of usage of soothing objects and soothing behaviours as well as self-soothing psychological activities. The authors suggest that these unexpected findings were an artifact of the select nature of the borderline sample (a military and private hospital sample) and as a result might not reflect a more heterogeneous general population of borderline participants. Nonetheless, the authors suggest that the results are consistent with studies that report borderline participants employing prolonged and/or maladaptive use of 'transitional objects'. These findings lend further weight to the view that affect regulatory capacities, as assessed by the increased need for the use of transitional objects, are impaired in BPD participants.

K. F. Stein (1996) examined 15 DIB diagnosed BPD participants, four anorexia nervosa participants, and 10 asymptomatic controls. Participants were administered the self report Affect Circumplex Scale (R. J. Larsen, & Diener, E., 1987; R. J. Larsen, Diener, & Emmons, 1986) which was employed to measure stability of affect. In addition, participants were 'experience-sampled' in order to obtain multiple measures of affect during the course of everyday activity for a period of 10 days. Data was collected a total of 50 times for each participant over a 10 day period. The findings suggested that BPD is characterised by a unique pattern of affect dysregulation. The BPD participants reported higher levels of

unpleasant affects and greater fluctuations in unpleasant affect state than did asymptomatic adults. The evidence also suggested that borderline participants experience significant rapid fluctuations in affect gradients, and these fluctuations occurred over intervals of a few hours rather than more prolonged mood states occurring over the course of a number of days.

A curious finding of the study suggested that, although significant group differences were found between the BPD and asymptomatic participant groups with regard to level of unpleasant and activated 'negative' affects, the mean level of unpleasant affects reported by the BPD group was low. This result contradicts prevailing theoretical and clinical viewpoints suggesting that BPD participants are highly sensitive to emotional stimuli and that their affective reactions are notably intense with a slow return to baseline. Another unexpected finding suggested that borderline participants did not differ from the asymptomatic groups with regard to the persistence of unpleasant affects across time. This finding also offers a challenge to the hypothesis that BPD participants have difficulty restoring affect to baseline levels. Replication of this study is warranted.

These results can be explained in one of two ways. Firstly, the sample sizes on which the point series analyses were taken were notably small in number and raises the possibility that the findings reflect a Type I error. Alternatively, the data might provide greater support for an impulse dysregulation theory of BPD. The findings of intense, short latency affective experiences and the failure of the persistence of unpleasant affects across the course of time is interpretable within a theoretical paradigm consistent with poor impulse regulation. Nevertheless, the findings of this study are also generally interpretable within an

affective dysregulation paradigm, although the methodological limitations of the study restrict the generalisability of these findings.

Wagner & Linehan (1997) provide anecdotal evidence suggesting that borderlines use suicidal behaviour and parasuicidal acts as emotion regulators in order to manage painful and/or overwhelming negatively valenced emotions. They report that borderlines engage in parasuicidal acts as a means of avoiding or escaping experiences associated with anger, anxiety, and shame. Wagner & Linehan suggest that overdosing (self medication) is often employed as a mechanism to induce relaxation, and self-mutilation can serve tension reduction or for ending dissociative episodes.

Yen et al. (2002) examined affect regulation in 39 women exhibiting borderline features from the perspective of Linehan's (1993) biosocial theory of BPD. Participants completed the Affect Intensity Measure (R. J. Larsen, & Diener, E., 1987), and the Affect Control Scale (K. E. Williams, Chambless, & Ahrens, 1997). Hierarchical regression analysis revealed that the level of affect intensity and affect control were associated with the number of reported BPD traits even after the influence of mood was controlled for. The findings for affect control also persisted even after the influence of affect intensity was controlled for. The findings of this study indicate that difficulties with the intensity of affect experience and the control of affective experience remains a central difficulty in BPD.

Arntz et al. (2000) examined the hypothesis that BPD participants would be hypervigilant for negative emotional stimuli and whether this hypervigilance is related to specific emotional themes. They studied 15 BPD, 12 Cluster C personality disordered, and 15 non-patient controls with an Emotional Stroop

paradigm. In this task, participants were required to colour name emotional and non-emotional words presented in three colours via microcomputer technology. Three classes of negative, emotionally-laden words hypothesised to be related to BPD pathology (negative views of others, sexual abuse related words, negative self descriptors) were included as well as one class of negatively laden words which were hypothesized to be unrelated to BPD pathology. In addition, a group of neutrally valenced words were also included. The words were presented in both supraliminal and subliminal conditions that were individually calibrated according to participant response to an initial testing session in which a set of test words was presented to the participant in decreasing presentation latencies.

Significant results were returned for both the BPD and the Cluster C personality disorder groups in the supraliminal condition only, and no differences between these groups was found for specific classes of emotional stimuli. The subliminal Stroop did not return differences between any of the groups. The authors interpret the findings as indicating that a hypervigilance for emotionally negative stimuli is not specifically indicative for BPD, but is more likely to be a general feature of Axis II disorders.

Herpertz et al. (2000) studied 24 BPD, 23 Avoidant Personality Disordered (APD), and 27 normal control participants on a series of psychophysiological measures obtained from exposure to photographic slides depicting pleasant, neutral, and unpleasant stimuli. Physiological responses including heart rate, skin conductance, and startle reflex were recorded whilst participants viewed the slides. The findings did not support the view that BPD participants experience an increased affective hyperresponsivity compared to APD or control participants. Instead, the study found that there was a decreased

physiological responsiveness to affective stimuli in BPD. The authors speculate that this phenomenon is associated with a stimulus-bound activation which subsumes attentional and affective processing. They explain this phenomenon by suggesting that it represents a link between psychopathic and BPD personalities in the sense that psychopathic characters are believed to be characterised by hypo-arousal.

Koenigsberg et al. (2002) examined the features of affective instability among a group of 152 DSM-III-R personality disordered outpatients. Of these, 42 (27.6%) met BPD criteria. All participants completed self-report measures of affect intensity and affect lability. The study found that the affective instability thought to be characteristic of BPD did not involve all affects. When factors such as co-morbid mood and personality disorder were controlled for, BPD was associated with greater levels of affective instability than other personality disorders for anger, anxiety, and an oscillating affective state consisting of depression and anxiety. Curiously, affect intensity was similar to other personality disorders, suggesting that the experience of intense affective states does not discriminate for BPD.

Viewed collectively, these findings provide mixed evidence for affect regulatory difficulties in borderlines. The studies reporting impairment of affect regulation in BPD tend to be studies relying on self-report data, or alternatively use methods where the data was derived out of some form of interpersonal context. The studies where no differences were returned tended to be 'laboratory' studies where information processing and/or biological markers were employed to examine affective variables. These findings have raised the question whether information processing paradigms are the most appropriate paradigm for

assessing affective variables in BPD (Arntz et al., 2000). One implication of this viewpoint suggests that affect dysregulation in BPD might be interpersonally determined, or that the presumed affect dysregulation of BPD is an artifact of BPD participant self-report and self-perception. In other words, appraisal processes might be centrally important in understanding the role of affect regulation in BPD (Ortony & Turner, 1990). The findings also raise the possibility that affect regulation operates independently of any propensity towards impulsive behaviour. It also further suggests that the twin features of affect lability and impulsivity in borderlines might be dissociable from one another. If so, the study reported in Section Four might help to shed some light on this issue.

There are a number of methodological limitations with the reported studies of impaired affect regulation in BPD. These include the predominant use of cross-sectional designs to detect differences in emotion processing variables, the absence of process studies of emotion reactivity across time, and the absence of information processing studies of affect in BPD. Nonetheless, the absence of information processing studies of affective variables in BPD represents a significant gap in knowledge and this approach will be used in the current study. This is further discussed in Section 3.7.2 below.

3.7.2. Assessment Of Affect Regulation In BPD: The Application of the 'Emotional Stroop' Method

An alternative approach to examining affect regulation in BPD suggests viewing it as a form of 'priming' or 'attentional bias' associated with externally mediated, affectively valenced information. Priming refers to a process in which the occurrence of one stimulus inhibits subsequent responses because of a

semantic relationship between the stimulus and response. According to this view, BPD participants should be 'primed' to respond to particular classes of events which contain within them specific, affect laden components which are semantically consistent with their hypothesized mood-regulatory deficits. In this sense, measuring 'affective-attentional bias' in BPD is argued to operate as an analogue for affect regulation.

The study of priming has a long tradition in experimental psychology, and is often associated with the 'Stroop' method. The Stroop Method (Stroop, 1935) has been used to examine attentional bias under a variety of conditions. The original Stroop task required participants to name the colour which an item was printed in whilst concurrently attempting to ignore the item itself. The items might consist of stimuli such as symbols or benign stimuli such as X's, or words including those that are the actual names of colours. In the case of colour-word naming, a word such as 'blue' might appear in red ink, the word green in blue ink, and so on. A consistent finding from numerous studies has demonstrated that participants take longer to name colours when the base items are contradictory colour names than when they are benign stimuli. Finally, research has demonstrated that when the meaning of a word is semantically activated, colour naming interference is likely to occur (J. M. G. Williams, Mathews, & MacLeod, 1996).

The development of cognitive theories of psychopathology has resulted in the use of Stroop tasks to examine cognitive processing associated with emotional disorders. A range of studies have consistently measured the latency of colour-naming to in response to negatively-valenced affect words. A number of studies have examined different psychological disorders including depression

(Gotlib & McCann, 1984), anxiety (Mathews & MacLeod, 1985), spiderphobia (Watts, McKenna, Sharrock, & Trezise, 1986) suicidal ideation (J. M. G. Williams & Broadbent, 1986), eating disorders (Cooper, Anastasiades, & Fairburn, 1992), persecutory delusions (Bentnall & Kaney, 1989; Kinderman, 1994), and Post Traumatic Stress Disorder (PTSD) (J. G. Beck, Freeman, Shipherd, Hamblen, & Lackner, 2001). The methodology employing affectively laden stimulus words has come to be known as the 'Emotional Stroop' task (J. M. G. Williams et al., 1996).

The Parallel-Distributed Processing model of information processing (J. D. Cohen, Dunbar, & McClelland, 1990) is currently viewed as providing the most comprehensive account of Stroop function (J. M. G. Williams et al., 1996). This view suggests that the interference-effect associated with the Stroop method occurs because of an 'associational strength of processing variable'. The performance of any task which requires a specific processing pathway provokes a pattern of activation in the relevant sensory nodes in order to generate the relevant pattern of activation in the output modules. This model is consistent with the theory of BPD development proposed earlier. Significantly, J. D. Cohen et al. (1990) argue that the Stroop effect is observable in two distinct pathways, which are dissociable from one another. The first pathway involves colour naming, and the second involves word reading. The Stroop interference effect occurs when antagonistic patterns of activation intersect at a single point in the processing sequence after the stimuli have been presented.

J. M. G. Williams et al. (1996) further suggest that the Stroop interference effect occurs without reliance upon attentional allocation and is therefore consistent with the proposition that the bias is 'pre-attentive' or 'automatic'.

Despite this, J. D. Cohen et al. (1990) note that automatic processes are not completely independent of attentional control, and it is well known that patients with specific emotional disorders are known to ruminate on themes associated with the nature of their psychopathology (J. M. G. Williams et al., 1996). For example, Segal, Truchon, Horrowitz, Gemar, & Guirguis (1995) suggest that this rumination might result in a highly interconnected mode of processing for individuals with particular emotional disorders, which results in them becoming 'experts' in processing information related to their specific problem.

J. M. G. Williams et al. (1996) draw upon two sets of experiments using the Stroop method to examine this question. The first category is referred to as expertise studies, and the second group of studies involve the employment of the Emotional Stroop method as an outcome measure to assess the efficacy of therapy with a variety of clinical conditions. They note that only two expertise studies are reported in the literature, and that conflicting outcomes have been realised in these studies. They therefore conclude that there is insufficient data to assess whether an expert effect occurs in the Emotional Stroop method at this time. A significantly larger number of therapy studies have been reported, and the evidence from these studies suggests that the frequency of usage or inter-category association due to practice or expertise affects does not explain the Stroop interference process in emotional disorders.

These findings have clear implications for the examination of impaired affect regulation in BPD. The evidence overwhelmingly supports the application of the Emotional Stroop method as a measure of attentional bias related to the specific emotional disorders under investigation (J. M. G. Williams et al., 1996). The critical issue becomes one of determining the specific affect categories to be

included in the Stroop task that are relevant to the particular psychopathology under examination. Because of an absence of information in the clinical literature concerning the specific affect categories implicated in BPD, it appears necessary to pre-emptively determine the specific affect categories which might be built into the design of a Stroop method for examining BPD. This issue forms the basis of Section Three of this thesis.

Five studies have employed Stroop tasks in examining BPD (Arntz et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). These have included four studies employing colour-conflict Stroop tasks (Judd & Ruff, 1993; Kunert et al., 2003; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and two studies employing 'Emotional Stroop' tasks (Arntz et al., 2000; Sprock et al., 2000). Of these, four studies have employed card-form Stroop tasks (Judd & Ruff, 1993; Kunert et al., 2003; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and one has employed a computerised mode of word delivery (Arntz et al., 2000).

The studies employing a colour-conflict Stroop task have returned mixed results. Judd & Ruff (1993), Kunert et al. (2003), and Sprock et al. (2000) found no differences between BPD and controls with regard to colour-conflict Stroop performance, whereas Swirsky-Sacchetti et al. (1993) reported significant colour-naming response latencies between BPD and non-psychiatric, community controls. Similarly, the available 'Emotional Stroop' studies have also returned mixed results with Sprock et al. (2000) returning non-significant findings and Arntz et al. (2000) returning significant colour-naming response latencies between BPD and community controls, but not with Cluster C personality disorder controls for supraliminally delivered stimuli only. As a result, it is

difficult to offer an interpretation regarding the status of Stroop findings in BPD. Despite this, Arntz et al. (2000) suggest that in relation to the Emotional Stroop Task, a crude form of hypervigilance might operate in BPD.

There are also specific issues associated with the various Stroop studies that have been reported. The majority of studies employ colour-naming of lists of words on the card-form version of the Stroop (Judd & Ruff, 1993; Kunert et al., 2003; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). This method relies on the total-time taken to read word-lists, and it is therefore difficult to isolate the specific factors involved in colour-naming response latency. It is argued that computerised versions of the Stroop task are likely to yield more accurate measures of colour-naming response latency, and to date only one study has employed this methodology (Arntz et al., 2000).

The equivocal nature of these findings is thought to be associated with a range of methodological issues inherent in the designs of the respective studies. Because many of these methodological issues are also common to the studies examining other executive functions in BPD, these methodological issues are considered in Section 3.9.

3.7.3. Assessment of Affective-Attentional Bias in BPD

Affect regulation in BPD will be examined through an affective-attentional paradigm employing an 'Emotional Stroop' task. Section Three will describe the construction of this task in detail. The reader is referred to this section for elaboration of the Stroop task design.

3.8. EXECUTIVE FUNCTION IV: IMPAIRED PROBLEM SOLVING IN BPD

Lezak (1995) argues that executive functions include volition, planning, purposive action, and effective performance. This view of executive function appears to represent a more global level of conceptualisation of executive function than the framework considered in this thesis. In contrast, the view proposed here incorporates the four components of executive function outlined by Lezak as representing one of four executive functions that are proposed in this study – problem-solving ability. Therefore, the model of executive function proposed here is considered to be a more comprehensive model of executive function that is specifically related to BPD.

There is a long-standing view that BPD is characterised by a number of features suggestive of impaired problem-solving capacity. These include the inability to learn from experience (Grotstein, 1987), the use of the cognitive organisers known as 'splitting' and 'projective identification' (Kernberg, 1975, 1984, 1992), and 'impulsive' acts (Zanarini, 1993), to manage the environment.

It nevertheless remains difficult to measure problem-solving in an 'ecologically valid' manner (Cripe, 1996). One way in which the issue of 'problem-solving' in BPD can be assessed is through the use of neuropsychological tests which involve problem-solving components. This approach is considered below.

3.8.1. Empirical Evidence for Impaired Problem-Solving in BPD

A number of studies have examined problem-solving tasks in BPD (Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et

al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[7], and two recent reviews have concluded that BPD is associated with impaired cognitive processing (O'Leary, 2000; Rogers, 2003). These reviews appear to be somewhat overstated because whilst a number of studies have found that BPD's experience significant deficits in neuropsychological functioning (Bazanis et al., 2002; Burgess, 1990, 1991; Dinn et al., 2004; Judd & Ruff, 1993; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[8], other studies have not found evidence of deficits in neuropsychological functioning in BPD (Cornelius et al., 1989; Kunert et al., 2003; Sprock et al., 2000). In addition, many of the studies reporting neuropsychological deficits in BPD also have a number of methodological flaws which limit the veracity of their findings. For the sake of parsimony, problem-solving tasks relevant to this project are defined as involving the functions of hypothesis-testing, shift-of-set, planning, and conceptual ideation. Where appropriate, the link between the reported studies and the specific problem-solving function will be identified. Table 3.2 summarises the tests employed in the various studies examining problem-solving in BPD.

[7] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

[8] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

Table 3.2: Tests Employed in the Studies Examining Problem-Solving in BPD

	COMPLEX FIGURE OF REY	TOWER TESTS	DECISION-MAKING OBJECT ALT TASK	WISCONSIN/ OTHER CARD SORT	TRAIL MAKING TEST
Bazanis et al. (2002)	N	Y[a]	Y[a]	N	N
Burgess (1990)	N	N	N	N	N
Burgess (1991)	N	N	N	N	N
Cornelius et al. (1989)	N	N	N	N	N
Dinn et al. (2004) (Study 1)	Y[a]	N	Y	N	Y[a]
Dinn et al. (2004) (Study 2)	N	N	N	N	Y[a]
Driessen et al. (2000)	Y[b]	N	N	N	N
Judd & Ruff (1993)	Y[a]	N	N	Y[b]	N
Kunert et al. (2003)	N	Y[b]	N	N	N
O'Leary et al. (1991)	Y[b]	N	N	Y[b]	N
Sprock et al. (2000)	Y[b]	N	N	N	Y[b]
Stein, Hollander et al. (1993)	Y[c]	N	N	Y[c]	Y[c]
Swirsky-Sacchetti et al. (1993)	Y[a]	N	N	Y[b]	Y[b]
van Reekum et al. (1993)	Y[c]	N	N	Y[c]	Y[c]

Y = Yes; N = No; a = significant difference between groups; b = non- significant difference between groups; c = not interpretable

Table 3.2 (Continued): Tests Employed in the Studies Examining Problem-Solving in BPD

	SYMBOL DIGIT/DIGIT SYMBOL	COWAT	PROVERBS/ SIMILARITIES	CROSSES TEST	BLOCK DESIGN	OBJECT ALTERNATE TASK
Bazanis et al. (2002)	N	N	N	N	N	N
Burgess (1990)	N	N	Y[b]	N	N	N
Burgess (1991)	N	N	Y[a]	N	N	N
Cornelius et al. (1989)	Y[b]	N	Y[b]	Y[b]	Y[b]	N
Dinn et al. (2004) (Study 1)	N	Y[b]	N	N	N	Y[a]
Dinn et al. (2004) (Study 2)	Y[a]	Y[b]	N	N	N	Y[a]
Driessen et al. (2000)	Y[b]	N	Y[b]	N	Y[b]	N
Judd & Ruff (1993)	Y[a]	Y[b]	N	N	Y[b]	N
Kunert et al. (2003)	N	N	Y[b]	N	Y[b]	N
Kurtz & Morey (1999)	N	N	N	N	N	N
O'Leary et al. (1991) N	Y[a]	N	Y[b]	N	Y[b]	N
Sprock et al. (2000)	N	N	N	N	Y[b]	N
Stein, Hollander et al. (1993)	N	N	N	N	N	N
Swirsky-Sacchetti et al. (1993)	Y[b]	Y[b]	Y[b]	N	Y[b]	N
van Reekum et al. (1993)	N	N	Y[c]	N	N	N

Y = Yes; N = No; a = significant difference between groups; b = non- significant difference between groups; c = not interpretable

The tasks identified as meeting these problem-solving criteria include visuoconstructive tasks such as the Complex Figure of Rey (Rey Figure) (Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[9]; Towers of London/Hanoi (Bazanis et al., 2002; Kunert et al., 2003); Decision-Making Task/Object Alteration Task (Bazanis et al., 2002; Dinn et al., 2004)[10], Card Sorting Tasks including the Wisconsin Card Sorting Test (Judd & Ruff, 1993; O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993), Trail-Making Test (Dinn et al., 2004; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[11], Symbol Digit Modalities Test (Swirsky-Sacchetti et al., 1993) and the Digit Symbol Test (Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993),[12] Controlled Oral Word Association Test (COWAT) (FAS) (Dinn et al., 2004; Judd & Ruff, 1993; Swirsky-Sacchetti et al., 1993)[13], Proverb Interpretation/Similarities (Burgess, 1990, 1991; Cornelius et al., 1989; Driessen et al., 2000; Kunert et al., 2003; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993); the Crosses Test (Cornelius et al., 1989); and Block Design from the WAIS-R (Wechsler, 1981) (Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). The findings from these studies are selectively reviewed.

[9] These Findings Refer to Dinn et al. (2004) (Study One)
[10] These Findings Refer to Dinn et al. (2004) (Studies One and Two)
[11] These Findings Refer to Dinn et al. (2004) (Studies One and Two)
[12] These Findings Refer to Dinn et al. (2004) (Study Two)

The results of visuoconstructive studies have yielded mixed results in studies of BPD. A number of studies have found mixed support for visuoconstructive deficits in BPD. Some studies report non-significant findings on both the Crosses Test and the Complex Figure of Rey (Rey Figure) (Cornelius et al., 1989; Driessen et al., 2000; O'Leary et al., 1991; Sprock et al., 2000), whereas other studies have found deficits on the Complex Figure of Rey (Rey Figure) in BPD (Dinn et al., 2004; Judd & Ruff, 1993; Swirsky-Sacchetti et al., 1993)[14]. The studies conducted by D. J. Stein et al. (1993) and van Reekum et al. (1993) correlated Rey Figure returns with measures of organicity, but did not report their data in a manner that allowed direct comparisons with control data.

The studies examining Block Design have generally returned consistent, non-significant findings in relation to normal controls (Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). Of note, Sprock et al. (2000) also found that their BPD cohort returned superior results to a comparison group of depressed controls. This is an anomalous finding, and requires further investigation.

Tower tasks are thought to measure 'central executive' processes associated with the executive function theories of Shallice (1982) and Baddeley & Hitch (1994). Two tower task studies have been reported (Bazanis et al., 2002; Kunert et al., 2003). Bazanis et al. (2002) found that BPD participants required a greater number of overall attempts, and a greater number of attempts per trial to arrive at the correct solution compared with controls on the Tower of London task. In contrast, Kunert et al. (2003) reported that no differences were

[13] These Findings Refer to Dinn et al. (2004) (Studies One and Two)
[14] These Findings Refer to Dinn et al. (2004) (Study One)

found between BPD's and controls on their performance on the Tower of Hanoi task. An insufficient number of these types of studies have been conducted to draw meaningful conclusions concerning tower task functioning in BPD.

Card sorting tasks are thought to measure 'shift-of-set' functions (Lezak, 1995), and probably also examine hypothesis testing capabilities. Five studies of card-sorting tasks including an unspecified card task (Judd & Ruff, 1993), three studies of the 128 card presentation Wisconsin Card Sorting Test (WCST) (O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993), and one study employing the 64 card WCST (van Reekum, Conway et al., 1993), have been reported. Non-significant findings were reported where direct comparisons between BPD and control groups were made (Judd & Ruff, 1993; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993). Again, the studies of D. J. Stein et al. (1993), and van Reekum, Conway et al. (1993) did not report their data in a manner that allowed direct comparisons with control group data. The results of these studies probably represent one of the most unequivocal findings suggesting that shift-of-set functions are not impaired in BPD. Despite this, there are methodological difficulties associated with the reported studies that suggest that further research on this task is required. This will be considered in detail in Section 3.9.

Eight studies examined proverb interpretation or include the Similarities subtest of the WAIS-R (Burgess, 1990, 1991; Cornelius et al., 1989; Driessen et al., 2000; Kunert et al., 2003; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993). Six studies returned non-significant findings (Burgess, 1990; Cornelius et al., 1989; Driessen et al., 2000; Kunert et

al., 2003; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993), and Burgess (1991) only returned differences between the BPD and a schizophrenic comparison group. The study reported by van Reekum, Conway et al. (1993) could not be interpreted as it provided correlational data only. Overall, these findings again appear consistent, but they too are compromised by other methodological issues such as small sample sizes which raise the risk that low statistical power might explain the findings in at least some of the studies (Burgess, 1990; O'Leary et al., 1991).

Digit Symbol (DS) has often been regarded as a measure of 'general cerebral efficiency' (Lezak, 1995), or as a measure of visual discrimination (O'Leary et al., 1991). Six studies have examined Digit Symbol (DS) returns from the WAIS-R (Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993)[15]. Cornelius et al. (1989), Driessen et al. (2000), and Swirsky-Sacchetti et al. (1993) found no differences between BPD participants and non-patient volunteers or 'historical values', and Swirsky-Sacchetti et al. (1993) also reported non-significant findings on the Symbol Digit Modalities Test. In contrast, Dinn et al. (2004) (Study Two), Judd & Ruff (1993), and O'Leary et al. (1991) reported significant deficits when BPD's were compared with matched controls. These findings again appear equivocal but there are methodological issues such as small sample size (Swirsky-Sacchetti et al., 1993), failure to employ appropriate controls (Cornelius et al., 1989), and a failure to control for substance use histories in the BPD cohort (Judd & Ruff, 1993) which challenge the validity of these findings.

[15] These Findings Refer to Dinn et al. (2004) (Study Two)

Six studies have examined the Trail-Making Test in BPD (Dinn et al., 2004; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[16]. Two studies returned non-significant findings (Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and Dinn et al. (2004) (Studies One & Two) reported significant differences for a clinical BPD study (Study One), and a 'student-BPD' study (Study Two). The studies of D. J. Stein et al. (1993) and van Reekum et al. (1993) were again not interpretable because of the reporting of correlation data only.

The Decision-Making Task (Bazanis et al., 2002), represents an example of a specific problem-solving task and has been described in detail in Section 2.2.3. BPD participants demonstrated unequivocal evidence of impairment on this task as a result of taking significantly longer to decide which box held the target token, and chose earlier bets than controls on both the ascending and descending conditions. Bazanis et al. argue that the findings of this study suggest that borderlines demonstrate slower and suboptimal decision-making combined with impulsivity in the choices they make. In contrast, Dinn et al. (2004) (Studies One & Two) reported that borderlines returned similar scores to controls on an 'Object Alternation Task' which was similar in many respects to the Decision-Making Task of Bazanis et al. (2002).

Finally, four studies have found no differences on the Controlled Oral Word Association Test (COWAT) (FAS) (Dinn et al., 2004; Judd & Ruff, 1993; Swirsky-Sacchetti et al., 1993)[17]. These findings, like those reported for the

[16] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

[17] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

Wisconsin Card Sorting Test, probably represent one of the most unequivocal findings concerning the neuropsychology of BPD.

In summary, there is mixed evidence for deficits in problem-solving in BPD. The nature of these equivocal findings is most likely to be related to a range of methodological issues inherent in the designs of the respective studies. Because many of these methodological issues are also common to the studies examining other executive functions in BPD, these methodological issues are outlined in Section 3.9.

3.8.2. Assessment of Problem Solving in BPD

A number of tasks have been selected in order to examine problem-solving in BPD. Most of the tasks selected have been used previously, and they have been selected because there has been some evidence from previous work of salience in the use of the particular task. The exceptions to this are the COWAT (Lezak, 1995), Wisconsin Card Sorting Test (WCST) (Heaton, Chelune, Talley, Kay, & Curtiss, 1993), and the Austin Maze Task (Walsh, 1978). The COWAT and the WCST were included because despite the evidence of non-significant returns in the extant studies, there were sufficient methodological concerns inherent in these designs to warrant inclusion in the present study. In addition, the clinical utility of these tasks in other areas of neuropsychological research was sufficiently robust to warrant their inclusion in the present study. The Austin Maze was included as there is emerging evidence that it examines important features of executive function (Bowden & Smith, 1994). Therefore, the following tasks have been selected for use in the proposed study:

1. The Tower of London (Shallice, 1982)
2. The Tower of Hanoi (Simon, 1975)

3. The Austin Maze Task (Walsh, 1978)
4. The Wisconsin Card Sorting Test (Heaton et al., 1993)
5. The Rey-Osterreith Complex Figure (Lezak, 1995)
6. The Similarities subtest from the Wechsler Adult Intelligence Scale - Revised (WAIS-R) (Wechsler, 1981).
7. The Controlled Oral Word Association Test (Lezak, 1995)

A detailed description of these measures and the specific executive functions they are being employed to examine are described in greater detail in Appendix III.

3.9. METHODOLOGICAL ISSUES ASSOCIATED WITH STUDIES EXAMINING EXECUTIVE FUNCTION IN BPD

There are a range of different methodological issues which compromise the findings of many of the neuropsychological studies examining executive function in BPD. Many of these issues relate to all modes of executive functioning including working memory, response inhibition, affective-attentional bias, and problem-solving. Other methodological issues appear to be related to specific executive functions and have been addressed elsewhere in the review. For these reasons, general methodological issues common to all executive functions will be reviewed collectively in Section 3.9.1. Section 3.9.1 identifies a number of sampling issues associated with the respective studies, and Section 3.9.2 considers diagnostic issues that compromise these studies. Section 3.9.3 examines a variety of psychiatric issues which delimit the findings of the studies, and Section 3.9.4 considers a number of neurological factors which again compromise the integrity of a number of studies.

3.9.1. Sampling Issues

A number of different sampling issues have been identified. These include problems associated with whether a control group, or additional control groups were included in the study design, the use of inpatient, outpatient, or mixed inpatient/outpatient samples, or whether the study controlled for the effects of IQ Other sampling issues include concerns regarding the respective sample sizes of a number of the studies, and the gender ratios of the respective samples. Table 3.3 identifies the relevant sampling issues associated with each of the studies examining executive functioning in BPD.

Table 3.3: Sampling/Design the Issues in Studies Examining Executive Functioning in BPD

	EMPLOYED 'NORMAL' CONTROL GROUP	EMPLOYED ADDITIONAL MOOD DISORDER/ PERSONALITY DISORDER CONTROL GP	INPATIENT ONLY DESIGN	OUTPATIENT ONLY DESIGN	MIXED INPATIENT/ OUTPATIENT DESIGN
Arntz et al. (2000)	Y	Y	N	Y	N
Bazanis et al. (2002)	Y	N	N	N	Y
Burgess (1990)	Y	N	NR	NR	NR
Burgess (1991)	N	Y	NR	NR	NR
Cornelius et al. (1989)	N	N	Y	N	N
Dinn et al. (2004) (Study One)	Y	N	Y	N	N
Dinn et al. (2004) (Study Two)	Y	N	N	Y	N
Driessen et al. (2000)	Y	N	N	N	Y
Judd & Ruff (1993)	N	N	N	Y	N
Kunert et al. (2003)	Y	N	Y	N	N
Kurtz & Morey (1999)	Y	N	N	Y	N
O'Leary et al. (1991)	Y	N	N	Y	N
Sprock et al. (2000)	Y	Y	N	N	Y
Stein, Hollander et al. (1993)	Y	N	N	N	Y
Swirsky-Sacchetti et al. (1993)	Y	N	N	Y	N
van Reekum et al. (1993)	Y	N	Y	N	N

Y = Yes; N = No; NR = Not Reported

Table 3.3 (Continued): Sampling/Design Issues in Studies Examining Executive Functioning in BPD

	MEASURED/ CONTROLLED I.Q	ADEQUATE SAMPLE SIZE	FEMALE ONLY DESIGN	MALE ONLY DESIGN	MIXED GENDER DESIGN
Arntz et al. (2000)	Y	N	Y	N	N
Bazanis et al. (2002)	Y	Y	N	N	Y
Burgess (1990)	NR	N	N	N	Y
Burgess (1991)	NR	N	NR	NR	NR
Cornelius et al. (1989)	Y	N	N	N	Y
Dinn et al. (2004) (Study One)	N	N	Y	N	N
Dinn et al. (2004) (Study Two)	N	Y	N	N	Y
Driessen et al. (2000)	Y	N	Y	N	N
Judd & Ruff (1993)	Y	N	N	N	Y
Kunert et al. (2003)	Y	N	N	N	Y
Kurtz & Morey (1999)	NR	Y	N	N	Y
O'Leary et al. (1991)	Y	N	N	N	Y
Sprock et al. (2000)	Y	N	Y	N	N
Stein, Hollander et al. (1993)	NR	N	N	N	Y
Swirsky-Sacchetti et al. (1993)	Y	N	Y	N	N
van Reekum et al. (1993)	NR	N	NR	NR	NR

Y = Yes; N = No; NR = Not Reported

Utilisation of a Control Group or Additional Control Groups

A number of studies have failed to recruit control groups in a manner that would accord with acceptable scientific practice. Although the majority of studies employed at least one non-psychiatric or 'normal' control sample (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[18], a small number of studies failed to specifically recruit a non-psychiatric or 'normal' control sample (Burgess, 1991; Cornelius et al., 1989; Judd & Ruff, 1993).

Three studies did not include a non-psychiatric or 'normal' control sample in their study design, and relied on 'historical values' (Cornelius et al., 1989), 'archival controls' (Judd & Ruff, 1993), or simply failed to include this type of control condition (Burgess, 1991). The studies of Cornelius et al. (1989) and Judd & Ruff (1993) drew their comparison data from extant databases that do not appear to have been explicitly recruited for these particular studies. Cornelius et al. (1989) made comparisons between their BPD cohort and 'historical values' because they used a number of well-normed instruments such as the WAIS and the WMS for which adequate norms were available. In contrast, Judd & Ruff (1993) matched their BPD cohort with volunteer 'archival controls' on age, gender, and educational variables. Importantly, it appears that these controls were recruited prior to the recruitment of the BPD cohort, and it also appears that they were not recruited from the same hospital catchment area as

[18] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

were their respective BPD groups. Burgess (1991) did not compare his BPD cohort with a 'normal' control group, but did so with Major Depressed, and Schizophrenic controls.

The majority of studies failed to include control groups in addition to non-psychiatric or 'normal' controls in order to control for the effects of confounds such as the presence of Axis I mood disorder, or other forms of Axis II personality disorder (Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[19]. However, two studies included depressed control groups in addition to a non-psychiatric or 'normal' control samples (Kurtz & Morey, 1999; Sprock et al., 2000), and Arntz et al. (2000) also employed a non-BPD 'Cluster C' personality disordered control group. The comparative recency of these studies suggests that this might represent a methodological trend that will improve the future quality of research in this area.

Ambulatory Status

The ambulatory status of participants in the reported studies is also highly variable, and this also renders comparability between the studies problematic. Four studies recruited their BPD cohorts exclusively from inpatient sources (Cornelius et al., 1989; Dinn et al., 2004; Kunert et al., 2003; van Reekum, Conway et al., 1993)[20], and six studies recruited their cohort exclusively from outpatient sources (Arntz et al., 2000; Dinn et al., 2004; Judd & Ruff, 1993;

[19] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

[20] These Findings Refer to Dinn et al. (2004) (Study One)

Kurtz & Morey, 1999; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993)[21]. In contrast, four studies included both inpatients and outpatients in their cohorts (Bazanis et al., 2002; Driessen et al., 2000; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993), and two studies failed to report the ambulatory status of their samples (Burgess, 1990, 1991).

The highly diverse nature of ambulatory status in these studies makes it difficult to draw conclusions regarding the effect that this variable plays in the neuropsychological sequelae of BPD. This lack of comparability between studies represents a methodological difficulty which limits the comparability between studies.

IQ Status

Nine studies reported measuring IQ status in their BPD cohorts (Arntz et al., 2000; Bazanis et al., 2002; Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993), and seven studies either failed to examine or did not report measuring IQ (Burgess, 1990, 1991; Dinn et al., 2004; Kurtz & Morey, 1999; D. J. Stein, Hollander et al., 1993; van Reekum, Conway et al., 1993)[22]. Of the nine studies that measured IQ status, only two study partially controlled for the influence of IQ on neuropsychological performance by excluding participants who returned an IQ under 70 (Cornelius et al., 1989), or 80 (Arntz et al., 2000). None of the studies controlled for the influence of IQ on test returns by employing IQ scores as a covariate and controlling for the effects of IQ statistically.

[21] These Findings Refer to Dinn et al. (2004) (Study Two)

[22] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

Female-Only Versus Mixed Gender Designs

A third sampling issue concerns the gender constitution of the respective samples. Four studies report female-only samples (Arntz et al., 2000; Dinn et al., 2004; Driessen et al., 2000; Sprock et al., 2000)[23], two studies do not report the gender-composition in their studies (Burgess, 1991; van Reekum, Conway et al., 1993), and none of the studies examine a male-only cohort. In contrast, the majority of studies report mixed-gender designs, (Bazanis et al., 2002; Burgess, 1990; Cornelius et al., 1989; Dinn et al., 2004; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993)[24]. Importantly, these studies have not stratified their data by gender in order to ascertain if gender affects executive performance.

The comparatively small number of female-only studies is somewhat surprising considering the literature on the gender-ratio of BPD, and it remains unclear whether gender influences neuropsychological function in BPD. For this reason, further research is required that controls for the effects of gender in the design of studies on BPD.

Inadequate Sample Sizes/ Type I Error Issues

A fourth issue concerns the sample sizes of a number of studies. This is a significant issue because of the risk of low statistical power in a number of studies with the attendant risk that there will be an increase of Type I error. Table

[23] These Findings Refer to Dinn et al. (2004) (Study One)

[24] These Findings Refer to Dinn et al. (2004) (Study Two)

3.4 reports the samples sizes and the ratio of dependent variables to study sample size for the respective studies.

Table 3.4: Sample Sizes and Dependent Variables to Case Ratios for the BPD Executive Function Studies

	BPD	NON—PSYCHIATRIC, VOLUNTEER , OR COMMUNITY CONTROL	DEPRESSED/ PERSONALITY DISORDER CONTROL	NUMBERS OF DEPENDENT VARIABLES REPORTED	APPROX VBLES: CASES RATIO
Arntz et al. (2000)	16	12	15	10	1:4
Bazanis et al. (2002)	42	42	Not Included	14	1:6
Burgess (1990)	18	14	Not Included	11	1:3
Burgess (1991)	27	Not Included	17	20	1:2
Cornelius et al. (1989)	24	Not Included	Not Included	28	1:1
Dinn et al. (2004) (Study One)	9	9	Not Included	21	1:1
Dinn et al. (2004) (Study Two)	10	129	Not Included	17	1:8
Driessen et al. (2000)	21	21	Not Included	15	1:3
Judd & Ruff (1993)	25	25	Not Included	15	1:3
Kunert et al. (2003)	23	23	Not Included	58	1:1
Kurtz & Morey (1999)	20	20	20	8	1:7
O'Leary et al. (1991)	16	16	Not Included	49	1.5:1
Sprock et al. (2000)	18	16	17	31	1:3
Stein, Hollander et al. (1993)	28	28	Not Included	NR	NA
Swirsky-Sacchetti et al. (1993)	10	10	Not Included	38	2:1
van Reekum et al. (1993)	10	10	Not Included	NR	NA

NR = Not Reported; NA = Not Available

The key feature identified in this analysis involves the relatively small sample sizes with large numbers of dependent variables in many of the studies. This finding suggests that many of the studies have a relatively high risk for Type I error. Whilst small samples are often typical in research with clinical populations, the major concern involves the dependent variables to cases ratio. A significant proportion of the studies report unacceptable dependent variables to cases ratios (Arntz et al., 2000; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[25]. The exceptions to this are Bazanis et al. (2002), Dinn et al. (2004) (Study Two), and Kurtz & Morey (1999) who report dependent variable to cases ratios varying between 1:6 and 1:8 respectively.

The implication of this finding is that many of the studies probably suffer from excessively low power with the attendant likelihood that they would be unable to realise statistical significance if in fact differences existed. In addition, it remains likely that those studies reporting significant results might be capitalising on Type I error and claiming effects which might not in reality exist. This represents a systematic flaw involving the majority of studies in this area. It appears that meta-analytic research is now required.

3.9.2. Diagnostic Issues

Two diagnostic issues have been identified which raise questions concerning the methodological rigour of the reported studies. The first issue is associated with the nature of the diagnosis of BPD cohorts, and the second issue

[25] These Findings Refer to Dinn et al. (2004) (Study One)

concerns whether the testers or the interpreters of the neuropsychological tests were blind to the diagnostic status of the participants at the time of testing or interpretation. Table 3.5 identifies the relevant diagnostic issues associated with each of the studies examining executive functioning in BPD.

Table 3.5: Diagnostic Issues in Studies Examining Executive Functioning in BPD

	DIAGNOSIS BY SELF-REPORT ONLY	DIAGNOSIS BY SEMI-STRUCTURED INTERVIEW	DIAGNOSIS BY CLINICAL JUDGEMENT/ CONCENSUS	WAS TESTER/ INTERPRETER BLIND TO DIAGNOSTIC STATUS?
Arntz et al. (2000)	N	Y	N	NR
Bazanis et al. (2002)	N	Y	N	NR
Burgess (1990)	N	N	Y	Y
Burgess (1991)	N	N	Y	NR
Cornelius et al. (1989)	N	Y	N	NR
Dinn et al. (2004) (Study One)	N	N	Y	NR
Dinn et al. (2004) (Study Two)	Y	N	N	NR
Driessen et al. (2000)	N	Y	N	NR
Judd & Ruff (1993)	N	Y	Y	NR
Kunert et al. (2003)	N	Y	Y	NR
Kurtz & Morey (1999)	N	Y	N	NR
O'Leary et al. (1991)	N	Y	N	NR
Sprock et al. (2000)	N	Y	N	Y
Stein, Hollander et al. (1993)	N	Y	N	NR
Swirsky-Sacchetti et al. (1993)	N	Y	N	NR
van Reekum et al. (1993)	N	N	Y	NR

Y = Yes; N = No; NR = Not Reported

Method of Diagnosis

Two diagnostic-methodological issues are identified. These include the nature of the methodology employed for making a BPD diagnosis, and which diagnostic system was used to diagnose participants.

One study reported the exclusive use of a self-report instrument as the primary diagnostic methodology for the diagnosis of BPD (Dinn et al., 2004) (Study Two), and Kurtz & Morey (1999) also used a self-report instrument in conjunction with a semi-structured interview. Three studies reported making BPD diagnoses exclusively by clinical judgement (Burgess, 1990, 1991; Dinn et al., 2004)[26], and van Reekum et al. (1993) employed a 'retrospective DIB' in order to diagnose their BPD cohort. The remaining studies employed at least one of a number of well-known semi-structured interviews to confirm BPD diagnoses (Arntz et al., 2000; Bazanis et al., 2002; Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993). In addition, Judd & Ruff (1993) and Kunert et al. (2003) also supplemented the use of a semi-structured interview with confirmation by the use of clinical judgement in order to confirm a DSM-III diagnosis in the case of Judd & Ruff (1993), and a DSM-IV diagnosis in the case of Kunert et al. (2003).

A second diagnostic issue concerns the diagnostic alignment of the respective studies. Two studies diagnosed according to DIB criteria alone (Cornelius et al., 1989; van Reekum, Conway et al., 1993), one according to DIB/DSM-III criteria (Judd & Ruff, 1993), nine according to DSM-III-R criteria

[26] These Findings Refer to Dinn et al. (2004) (Study One)

(Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990, 1991; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993), and four according to DSM-IV criteria (Dinn et al., 2004; Driessen et al., 2000; Kunert et al., 2003)[27]. The fundamental issue raised by this analysis concerns itself with the issue of diagnostic heterogeneity. Section 2.6.4 identified the heterogeneous nature of BPD as an ongoing matter of concern. The usage of a variety of diagnostic systems challenges the assumption that the studies are examining the same phenomenon. This, in turn, challenges the comparability of the findings across studies.

The utilisation across the studies of either clinical judgement alone, or alternately, a number of different semi-structured interviews underscores the fact that in the diagnosis of BPD there remains an absence of a 'gold standard' diagnostic measure. The instruments employed across the studies are not necessarily commensurate with one another, and the inconsistent selection of diagnostic instruments results in a potentially low level of diagnostic comparability between studies. This represents a systemic-methodological flaw which can only be remedied by the adoption of a standardised protocol for making the diagnosis of BPD.

Tester/Interpreter Blind to Diagnostic Status

The majority of studies did not indicate if the tester and/or the test interpreter was blind to the diagnostic status of the participant or the participants test protocol (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; D. J. Stein, Hollander et

[27] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[28]. This is an important consideration because knowledge of diagnostic status might have influenced tester behaviour, or decision-making by the test interpreter. In contrast, only two studies reported that the tester/interpreter was blind to diagnostic status (Burgess, 1990; Sprock et al., 2000), and Dinn et al. (2004) (Study One) had only the Rey-Figure returns blind-scored in order to partially control for diagnostic bias. The failure in the majority of studies to report controlling for tester/test interpreter knowledge of diagnostic status represents a significant methodological flaw which requires modification in future studies of executive function in BPD. Future studies will need to ensure control of this factor in order to improve the integrity of study designs. It is a potential confounding factor that is partially controlled for in the current project.

3.9.3. Psychiatric Issues

A number of psychiatric factors are also known to affect performance on psychological tests. These include the effects of co-morbid mood and/or psychotic disorders, the effects of ECT, and the influence of sedating medication at the time of testing. Table 3.6 identifies the relevant psychiatric issues associated with each of the studies examining executive functioning in BPD.

[28] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

Table 3.6: Psychiatric Issues in Studies Examining Executive Functioning in BPD

	CONTROLLED FOR:			
	CO-MORBID MOOD DISORDER	CO-MORBID PSYCHOTIC STATES	EFFECTS OF ECT	EFFECTS OF SEDATING MEDICATION
Arntz et al. (2000)	NR	Y	NR	NR
Bazanis et al. (2002)	Y	Y	NR	N
Burgess (1990)	Y	Y	NR	NR
Burgess (1991)	Y	Y	NR	NR
Cornelius et al. (1989)	Y	Y	NR	NR
Dinn et al. (2004) (Study One)	Y	N	NR	N
Dinn et al. (2004) (Study Two)	NR	NR	NR	NR
Driessen et al. (2000)	Y	Y	NR	Y
Judd & Ruff (1993)	Y	Y	NR	Y
Kunert et al. (2003)	Y	Y	NR	N
Kurtz & Morey (1999)	Y	Y	NR	N
O'Leary et al. (1991)	Y	NR	NR	Y
Sprock et al. (2000)	Y	Y	NR	N
Stein, Hollander et al. (1993)	NR	NR	NR	NR
Swirsky-Sacchetti et al. (1993)	Y	Y	Y	N
van Reekum et al. (1993)	NR	Y	NR	NR

Y = Yes; N = No; NR = Not Reported

Co-Morbid Mood Disorder

Section 2.2.5 identified the high rates of co-morbidity between Axis I mood disorder and BPD. In addition, co-morbid mood disorder is known to adversely affect performance on psychological tests (Lezak, 1995). For these reasons, it is important to control for the influence of co-morbid mood disorder affecting test performance in BPD.

Three approaches are identified as appropriate methods for controlling for co-morbid mood disorder. These include exclusion of mood disordered participants, incorporating a mood-disordered, non-BPD control group into the study design, or covarying the effects of mood in statistical analyses when examining neuropsychological returns.

Four studies failed to report if mood disorder data was collected (Arntz et al., 2000; Dinn et al., 2004; D. J. Stein, Hollander et al., 1993; van Reekum, Conway et al., 1993)[29]. Six studies reported collecting mood disorder data by psychiatric interview (Burgess, 1990; Cornelius et al., 1989; Dinn et al., 2004; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991)[30], and six studies reported collecting mood disorder data by the use of psychometric assessment (Bazanis et al., 2002; Burgess, 1991; Driessen et al., 2000; Kurtz & Morey, 1999; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). Finally, three studies also included a mood-disordered, non-BPD control group for comparison purposes (Burgess, 1991; Kurtz & Morey, 1999; Sprock et al., 2000). In total, 12 studies collected mood-disorder data of which three also employed a mood-disorder, non-BPD control group.

[29] These Findings Refer to Dinn et al. (2004) (Study Two)

[30] These Findings Refer to Dinn et al. (2004) (Study One)

Two studies excluded participants with mood disorder (Burgess, 1990; Judd & Ruff, 1993), and one study employed depression scores as a covariate (Driessen et al., 2000). Seven failed to utilise mood data either to exclude participants or to use as a co-variate (Bazanis et al., 2002; Cornelius et al., 1989; Dinn et al., 2004; Kunert et al., 2003; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993)[31].

In summary, co-morbid mood disorder was poorly controlled for in a significant number of studies. This is unfortunate given the adverse effects that mood has on neuropsychological test performance (Lezak, 1995). Future studies will need to ensure control of this factor in order to improve the integrity of study designs. It is a confounding factor that is specifically controlled for in the current project through the use of a non-BPD, depressed control group.

Co-Morbid Psychotic Disorder

Psychotic states are known to adversely affect performance on executive tasks (Hutton et al., 1998; Murphy et al., 1999). The majority of studies excluded participants with a history of psychotic illness (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993), but four studies failed to explicitly report controlling for psychotic states in their cohorts (Dinn et al., 2004; O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993)[32]. In the case of Dinn et al. (2004) (Study One), over half of the BPD sample also met criterion for schizoaffective disorder. In the majority of studies, this confounding

[31] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

[32] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

factor appears to have been satisfactorily controlled for. It is a confounding factor that is specifically controlled for in the current project.

The Effects of ECT

There is a well established relationship between the administration of ECT and memory impairment (Fink, 2001; Reisner, 2003). In turn, this is likely to have implications for memory, response inhibition, Stroop, and problem-solving performance because all of these tasks rely to various degrees upon working memory resources for effective task execution. The majority of studies failed to report whether ECT was controlled for in their designs (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; van Reekum, Conway et al., 1993)[33]. Only one study reported excluding participants with a history of ECT (Swirsky-Sacchetti et al., 1993).

The failure of the majority of studies to control for ECT again represents a significant methodological limitation in a number of studies. Future studies will need to ensure control of this factor in order to improve the integrity of study design. It is a confounding factor that is specifically controlled for in the current project.

Effects of Sedating Medication

The use of sedating psychotropic medication again represents an important factor that can adversely affect performance on psychological tests (Lezak, 1995). Three approaches are identified as methods for controlling for the influence of psychotropic medication. These include exclusion of participants on

[33] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

medication, withdrawal of participants from medication for the duration of the study, or controlling for the effects of medication in statistical analyses.

None of the studies appeared to exclude participants on the basis that they were in receipt of sedating psychotropic medication. Three studies controlled for the effects of medication by withdrawing participants from medication regimes for a period of at least one week prior to testing (Driessen et al., 2000; Judd & Ruff, 1993; O'Leary et al., 1991). Six studies provided information concerning medication usage in their cohorts, but subsequently failed to employ co-variate analysis to control for this (Bazanis et al., 2002; Dinn et al., 2004; Kunert et al., 2003; Kurtz & Morey, 1999; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993)[34].

In summary, the majority of studies failed to adequately control for the effects of sedating medication. Future studies will need to ensure control of this factor in order to improve the integrity of study design.

3.9.4. Neurological/'Brain Impairment' Issues

Five neurological and/or brain impairment issues are also identified as having significance in examining the executive function studies in BPD. These include whether the studies controlled for the effects of head trauma, for various neurological/medical conditions known to compromise cognitive function, handedness, substance use at the time of testing, and the effects of cumulative, lifetime substance abuse. Table 3.7 identifies the relevant neurological issues associated with each of the studies examining executive functioning in BPD.

[34] These Findings Refer to Dinn et al. (2004) (Study One)

Table 3.7: Neurological Issues in Studies Examining Executive Functioning in BPD

	CONTROLLED FOR:				
	HEAD INJURY	NEUROLOGICAL CONDITIONS/ ILLNESS	HANDEDNESS	SUBSTANCE USE AT TIME OF TESTING	CUMULATIVE LIFETIME SUBSTANCE USE
Arntz et al. (2000)	NR	NR	NR	Y	NR
Bazanis et al. (2002)	Y	Y	Y	Y	NR
Burgess (1990)	Y	Y	NR	Y	NR
Burgess (1991)	NR	N	NR	Y	NR
Cornelius et al. (1989)	NR	Y	NR	Y	NR
Dinn et al. (2004) (Study One)	NR	NR	Y	NR	NR
Dinn et al. (2004) (Study Two)	Y	NR	Y	NR	NR
Driessen et al. (2000)	Y	Y	Y	Y	NR
Judd & Ruff (1993)	Y	Y	NR	Y	NR
Kunert et al. (2003)	Y	Y	NR	Y	NR
Kurtz & Morey (1999)	Y	Y	NR	NR	NR
O'Leary et al. (1991)	NR	Y	NR	Y	N
Sprock et al. (2000)	Y	Y	NR	Y	NR
Stein, Hollander et al. (1993)	Y	Y	NR	N	NR
Swirsky-Sacchetti et al. (1993)	Y	Y	Y	Y	N
van Reekum et al. (1993)	NR	NR	NR	NR	NR

Y = Yes; N = No; NR = Not Reported

Head Injury / Head Trauma

A history of head trauma is a well-documented factor that is known to affect test performance (Lezak, 1995). The majority of studies report excluding participants with a history of head injury or evidence of some form of 'organic' profile (Bazanis et al., 2002; Burgess, 1990; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993)[35]. Five studies failed to report if participants with a history of head injury were excluded from their cohorts (Arntz et al., 2000; Burgess, 1991; Dinn et al., 2004; O'Leary et al., 1991; van Reekum, Conway et al., 1993)[36]. Cornelius et al. (1989) specifically excluded participants with 'overt organicity', but did not nominate a history of head trauma within this criterion. Future studies will need to ensure control of this factor in order to improve the integrity of study designs. It is a confounding factor that is specifically controlled for in the current project.

Neurological Conditions/Illnesses

A history of neurological compromise is also known to affect test performance (Lezak, 1995). The majority of studies reported excluding participants with neurological conditions, illnesses, or other metabolic disorders likely to affect test performance (Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993). Four studies failed to report controlling for the effects of neurological conditions (Arntz et al.,

[35] These Findings Refer to Dinn et al. (2004) (Study Two)

[36] These Findings Refer to Dinn et al. (2004) (Study One)

2000; Dinn et al., 2004; van Reekum, Conway et al., 1993)[37]. Future studies will need to ensure control of this factor in order to maintain the integrity of study designs. It is a confounding factor that is specifically controlled for in the current project.

Handedness

The identification of handedness is an important consideration in the neuropsychological/executive examinations because there is a substantial body of evidence suggesting that lateralisation of hemispheric function is related to handedness (Lezak, 1995). This relationship has implications for the pattern of executive deficits associated with so-called 'frontal functions', and for this reason handedness data is routinely collected in comprehensive neuropsychological examinations (Walsh, 1978). This factor was poorly controlled for in the majority of studies with only five studies attempting to control for handedness (Bazanis et al., 2002; Dinn et al., 2004; Driessen et al., 2000; Swirsky-Sacchetti et al., 1993)[38]. Of these five studies, two studies appear to have matched participants for handedness (Bazanis et al., 2002; Driessen et al., 2000), and Swirsky-Sacchetti et al. (1993) appears to have employed a 'screening' questionnaire.

Swirsky-Sacchetti et al. (1993) reported a significantly higher number of left handed BPD participants in their study, and Dinn et al. (2004) (Studies One & Two) reported the ratios of right versus left handed participants in their studies. In these studies, handedness does not appear to have been controlled for either by eliminating these subgroups from their studies, comparing test

[37] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

[38] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

performances between right and left-handed BPD participants, analysing their data separately, or utilising handedness as a covariate in subsequent analyses.

The remainder of the studies failed to report if handedness was controlled for (Arntz et al., 2000; Burgess, 1990, 1991; Cornelius et al., 1989; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; van Reekum, Conway et al., 1993). This represents a significant methodological failure that compromises the integrity of a number of studies. Future studies will need to ensure control of this factor in order to improve the integrity of study designs. It is a confounding factor that is specifically controlled for in the current project.

Substance Use at the Time of Testing

The use of illicit substances is also known to impair test performance (Lezak, 1995). The majority of studies report controlling for this factor in their designs by excluding participants who had taken drugs or consumed alcohol (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). The exceptions to this were Dinn et al. (2004) (Studies One & Two), Kurtz & Morey (1999), and van Reekum et al. (1993), who failed to report if these factors were controlled for. D. J. Stein, Hollander et al. (1993) specifically included participants with a history of drug and alcohol use because of the noted co-morbidity of these factors with the presence of BPD. Future studies will need to ensure control of this factor in order to maintain the integrity of study designs. It is a confounding factor that is specifically controlled for in the current project.

Effects of Cumulative Lifetime Substance/Alcohol Use

Another methodological limitation involves the failure to control for the influence of the cumulative effects of lifetime alcohol and substance use in study designs. There is abundant evidence indicating that a chronic history of alcohol and substance use is associated with adverse frontal-executive function, and this in turn is likely to affect the performance on various tasks reported in these studies (Lezak, 1995). The failure of most, if not all studies to control for the influence of cumulative dosage represents a significant issue that urgently requires attention. It is acknowledged however, that this represents a formidable methodological challenge because of the difficulties inherent in realising an accurate, retrospective estimate of drug and/or alcohol utilisation. It is nevertheless important because the nature of executive deficits in BPD is likely to be subtle, and the capacity to discriminate poor executive performance which is related to BPD functioning from brain damage associated with drug misuse will represent an important development in understanding the nature of cognitive impairment in BPD.

The majority of studies failed to control for this confounding factor (Arntz et al., 2000; Bazanis et al., 2002; Burgess, 1990, 1991; Dinn et al., 2004; Driessen et al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993)[39]. Only Cornelius et al. (1989) excluded participants on the basis of alcohol related deficits, although it does not appear that they controlled for cumulative lifetime drug usage. However, two studies appear to have implicitly acknowledged this

[39] These Findings Refer to Dinn et al. (2004) (Studies One and Two)

issue by excluding participants with an admitted history of using alcohol or substance dependence in the two years prior to testing (O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993). A methodology to control for the cumulative lifetime effects of alcohol and substance use in study designs remains elusive but necessary.

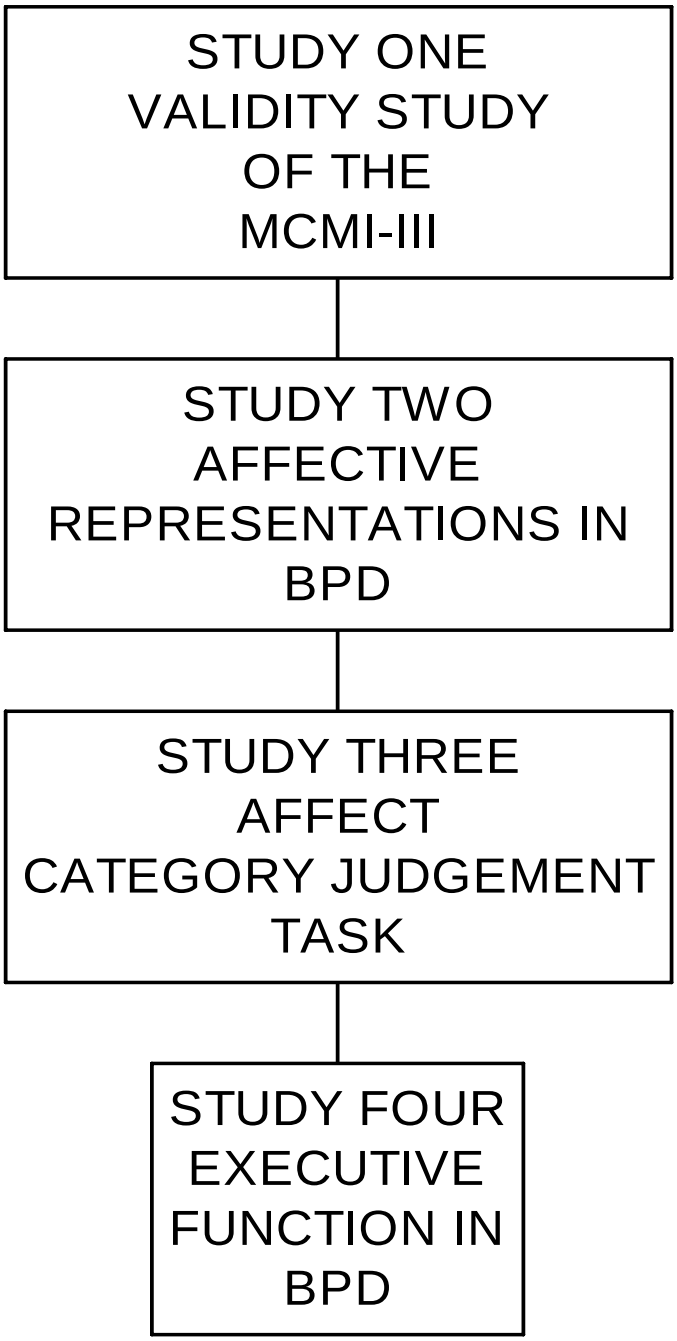
3.10. HYPOTHESES AND DESIGN OF THE PROJECT

The following hypotheses will be examined in the current study:

1. BPD participants will demonstrate impairments to working memory when compared with mood impaired and normal controls;
2. BPD participants will demonstrate impaired impulse control as assessed by their performance on a measure of behavioural disinhibition when compared with mood impaired and normal controls;
3. BPD participants will demonstrate impaired attentional bias (affect regulation) as assessed by an Emotional Stroop Task when compared with mood impaired and normal controls;
4. BPD participants will demonstrate impaired problem solving capacity when compared with mood impaired and normal controls.

In order to test these hypotheses, three preliminary studies will be conducted in order to validate the BPD scale of one of the diagnostic instruments (Study One), and to develop the Stroop task (Studies Two and Three). Study Four will formally test the hypotheses outlined above. Figure 3.3 outlines the sequence of studies conducted in the project.

Figure 3.3: Sequence of Studies Conducted in this Project



SECTION II: SELECTION OF DIAGNOSTIC INSTRUMENTS

AND VALIDATION OF SCALE ‘C’ OF THE MCMI-III

CHAPTER FOUR: SELECTION OF DIAGNOSTIC INSTRUMENTS AND AN EXAMINATION OF THE VALIDITY OF SCALE 'C' (BORDERLINE) OF THE MCMI-III

4.1. OVERVIEW

Section 2.6 outlined various theoretical and methodological critiques of BPD. As a result of the diversity of theoretical approaches to the study of BPD, a number of instruments have been developed to diagnose BPD. This has, in turn, increased the level of diagnostic confusion often associated with the construct. This chapter describes the diagnostic instruments selected for use in the present series of studies, and reports the findings of a validity study conducted on Scale C (Borderline) of the Millon Clinical Multiaxial Inventory (3rd Edition) (MCMI-III) (Millon et al., 1994). The validity study reported in this section commenced in January 1997 and concluded in June 1997, and occurred prior to the execution of the other studies reported in this project.

4.2. DIAGNOSTIC INSTRUMENTS EMPLOYED IN THE PROJECT

Whilst there are a number of approaches available for diagnosing BPD, all studies reported in this project have employed the combined use of questionnaire and structured interview for diagnosing BPD in accordance with the views of Skodol & Oldham (1991). Both Skodol and Oldham and Kaye & Shea (2000) argue that the use of a self-report inventory coupled with a semi-structured interview is a method most likely to return an efficient, valid, and reliable diagnosis. It was therefore decided that if a self-report inventory was augmented with the use of a semi-structured interview, and the participant was positive for BPD on both instruments then it would be highly likely that the participant would represent a 'true' case of BPD.

Whilst there is no 'gold standard' for diagnosing BPD, there is some evidence that an 'aggregation' approach increases the likelihood of making a valid and reliable diagnosis (Kaye & Shea, 2000; Overholser, 1994). As a result, the combined use of two reliable, well utilized instruments was selected as the diagnostic strategy to be employed in all studies. In order to enhance efficiency, the utilization of a self-report method followed by a semi-structured interview accorded the best combination of adherence to methodological rigour with the use of a time efficient approach to initial screening for the presence of BPD.

The instruments of choice included the Millon Clinical Multiaxial Inventory (3rd Edition) (MCMI-III) (Millon et al., 1994), and the Diagnostic Interview for Borderlines (DIB-R) (Gunderson et al., 1981; Zanarini et al., 1989). These instruments were selected because each demonstrated sound psychometrics, ease of administration, scoring and interpretation, and had a history of consistent and successful use as defined by a significant number of references employing each particular instrument (Kaye & Shea, 2000). These instruments were described in detail in Sections 2.3.2 and 2.3.4 respectively, and the reader is referred to these sections for details regarding each instrument.

The MCMI-III (Millon et al., 1994) was chosen as an initial screening instrument for detecting the presence of BPD because:

1. It is the most commonly employed instrument for this purpose;
2. It was designed to be DSM-IV aligned;
3. The reported psychometrics on this instrument appeared to be sound;
4. It is easily administered, with little evidence of patient resistance to responding.

5. The author has extensive clinical experience in both the administration and interpretation of the instrument.

The DIB-R was selected as the semi-structured interview of choice because:

1. The reported psychometrics of the instrument were sound;
2. It is a well known, commonly used instrument for diagnosing BPD;
3. It did not require extensive training for its use, and did not require calibration training or extensive reliability coding (Zanarini – Personal Communication, 1998) (See Appendix IV)

Because the MCMI-III is a copyrighted test it was not possible to include as an Appendix. However, the DIB-R is in the public domain (Kaye & Shea, 2000), and is included as Appendix V.

4.3. CONFIRMATORY PSYCHOMETRIC RESEARCH ON THE MCMI-III

Although the reported psychometric data for both the MCMI-III and the DIB-R were reported to be sound, there have been concerns that the exclusive use of questionnaire methodologies for diagnosing personality disorder results in an unacceptable false-positive rate. A small number of studies have reported unacceptable false-positive rates with earlier versions of the MCMI (Reich, 1992). Whilst no specific evidence is available which confirms this difficulty in relation to the MCMI-III, it was decided to conduct a validity study on the Borderline Scale (Scale C) of the MCMI-III. This was regarded as important because of the centrality of this instrument as the initial 'instrument of first detection' with study participants. Secondly, no Australian data was available for the MCMI-III. As a result, a study examining the validity of the Borderline scale (Scale C) of the MCMI-III was initiated prior to the commencement of any other

studies designed to test the hypotheses reported in Section 3.10. Section 4.4 describes the participant sample, Section 4.5 describes the study procedure, and Section 4.6 reports the results of the study.

4.4. PARTICIPANTS

The participants included in this study were recruited from referrals to the author for outpatient psychological assessment and therapy through an outpatient clinical psychology clinic at Fremantle Hospital in Western Australia. The author is a 'Specialist Clinical Psychologist' employed at Fremantle Hospital, and participants were recruited into the study during the period 1 January to 30 June 1997. Referrals to this clinic came from a number of separate units within Fremantle Hospital including the Mental Health Directorate (Psychiatry), the Departments of General Medicine, Genitourinary Medicine, and Gastroenterology. Participants referred to this clinic were placed on a waiting list, and reviewed by the author as outpatient appointments became available. All participants were assessed as outpatients, and those participants who were referred from inpatient psychiatric referring teams were not reviewed until they had been discharged from hospital for a minimum period of 14 days. Participants were also informed that refusal to participate in the study would not jeopardise ongoing treatment.

4.5. PROCEDURE

The initial outpatient clinic appointment consisted of three parts. Firstly, the patient was interviewed to assess the nature of their difficulties, and to assess the need for psychological intervention. Secondly, each participant completed the Millon Clinical Multiaxial Inventory (3rd Edition) (MCMI-III) (Millon et al., 1994) as part of their clinical assessment and as part of the research objectives of

the study. Upon completion of the MCMI-III, each participant was administered the Diagnostic Interview for Borderlines - Revised (DIB-R) (Zanarini et al., 1989). Participant performance on the MCMI-III was unknown at the time of the administration of the DIB-R. The scoring and interpretation of both instruments occurred at the completion of the interview. Therefore, the administration of the DIB-R was conducted blind to participants' MCMI-III Scale C status.

4.6. RESULTS

19 participants participated in the study. The demographic features of the sample including the ages, gender, occupational status, marital status, and educational status of the sample is reported in Table 4.1.

Table 4.1: Demographic Data for the Sample Examining the Validity of Scale 'C' (Borderline) of the MCMI-III

PARTICIPANT NUMBER	AGE	GENDER STATUS	OCCUPATIONAL STATUS	MARITAL STATUS	EDUCATION STATUS	MEETS MCMI-III BPD STATUS	MEETS DIB-R BPD STATUS
1.	43	1	7	1	1	2	2
2.	48	1	5	2	1	2	2
3.	38	2	1	3	2	2	2
4.	27	2	9	2	1	2	2
5.	36	1	6	3	1	1	1
6.	23	2	9	4	1	1	1
7.	59	2	9	6	1	2	2
8.	20	2	10	4	1	1	1
9.	40	1	6	4	2	2	2
10.	25	2	5	4	3	1	1
11.	39	1	6	4	1	1	1
12.	31	1	10	5	3	1	1
13.	24	2	8	4	2	1	1
14.	40	1	6	1	2	2	2
15.	43	2	4	3	3	2	2
16.	35	1	6	3	2	2	1
17.	52	1	7	3	1	2	2
18.	22	2	10	4	1	1	1
19.	34	2	8	4	3	2	2
		1 = Male 2 = Female	1 = Professional 2 = Managerial 3 = Technical 4 = Clerical/Sales 5 = Skilled Labour 6 = Semi Skilled Labour 7 = Unskilled Labour 8 = Student 9 = Home Duties 10= Unemployed	1= Married 2= Divorced 3= De Facto 4= Single 5= Separated 6= Widowed	1 = Completed Yr 10 2 = Completed Yr 12 3 = Completed Degree 4 = Completed Postgraduate Degree	1 = Yes 2 = No	1 = Yes 2 = No

Data was coded on a Presence/Absence for BPD on both the MCMI-III and the DIB-R. Presence of BPD on the MCMI-III was defined as occurring when participants returned a Scaled Score of 85 or more on Scale C of the MCMI-III. Presence of BPD on the DIB-R was defined as occurring when participants returned a Scaled Score of eight (8) or more on the DIB-R. A 2x2 Pearson Chi-Square returned significant results (χ^2= 15.354, df=1) ($p<0.0005$) suggesting significant differences between groups. Table 4.2 reports the frequencies and percentages by BPD status for the sample.

Table 4.2: Cross-Tabulation Analyses for MCMI-III Scale C BPD Status With DIB-R BPD Status

		DIB-R BPD STATUS	
		YES	NO
MCMI-III SCALE C STATUS	YES	8 (42.1%)	0 (0%)
	NO	1 (5.3%)	10 (52.6%)

4.7. SUMMARY AND CONCLUSIONS

The evidence from this study suggests that Scale C of the MCMI-III is a valid scale for the detection of BPD. The findings of the current study suggested a satisfactory convergence in classification between Scale C of the MCMI-III, and the DIB-R. The data suggested that Scale C of the MCMI-III and the DIB-R jointly identified cases of BPD on 89% of occasions, and jointly discriminated against non-BPD cases 100% of the time. These findings suggest that Scale C of the MCMI-III enjoys low Type I and Type II error rates. The findings of this

study provide the empirical justification necessary for the use of the MCMI-III as a diagnostic instrument for use in subsequent studies.

SECTION III: DEVELOPMENT OF THE EMOTIONAL STROOP TASK

AND

DESCRIPTION OF THE STOP-SIGNAL PARADIGM

CHAPTER FIVE: METHOD: AFFECTIVE AND SEMANTIC REPRESENTATIONS IN BPD

5.1. OVERVIEW

The studies included in Section Three were designed to construct an information-processing paradigm referred to as the 'Emotional Stroop'. The 'Emotional Stroop' paradigm was one component of the overall study designed to examine executive dysfunction in BPD. The other components of the study have been described earlier.

In order to develop this paradigm for use in the present study, a number of preliminary design tasks were required:

1. The first stage of building a Stroop task called for the identification of the categories of affect to be included in the paradigm. These categories were determined through the use of interviews with diagnosed BPD participants in order to ascertain the relevant affect categories to include in the Stroop paradigm. This procedure is reported in the current chapter.
2. Once the categories of affect to be included in the Stroop paradigm had been identified, a word list dictionary was employed in order to select specific words to include in each Stroop affect category. Once this had been developed, it was necessary to employ a judgement team to identify specific words selected from the emotion-word dictionary for inclusion in the Stroop paradigm. This procedure is described in Chapter Six.
3. The technical platform for the delivery of the Stroop paradigm had to be designed and tested. The description of hardware, operational platform, and instructional procedures is described in Chapter Seven.

5.1.1. Identification of Affect Categories for Inclusion in the Stroop Paradigm

The first component of the design of the Stroop paradigm required identification of the specific affect categories to include in the task. This aspect of the design of the task was resolved through the use of interviews with borderline participants in order to identify specific affect categories. This approach required the use of qualitative interview methodology. Elliot, Fischer, & Rennie (1999) have developed a set of guidelines for the use of qualitative research. These guidelines have been developed in order to ensure that an adequate level of scientific rigour operates in the conduct of qualitative research. The evolving guidelines suggest that good qualitative research requires the researcher to 'own one's perspective', 'situate' the sample to be studied, ground conclusions derived in the study in demonstrable examples, provide credibility checks, and ensure coherence. This information is contained in Appendix VI.

5.2. DIAGNOSIS OF BPD

The diagnosis of BPD was undertaken through the administration of the Millon Clinical Multiaxial Inventory (3rd Edition) (MCMI-III) (Millon et al., 1994), and the Diagnostic Interview For Borderlines - Revised (DIB-R) (Zanarini et al., 1989). The rationale for the selection of these instruments has been discussed in Chapter Four, and the reader is referred to this section for reiteration if required.

5.3. PARTICIPANTS

The participants included in this study were recruited from referrals made for psychological assessment and psychotherapy through an outpatient clinical psychology service at Fremantle Hospital during the period 1 January 1998 to 30 June 1998. Referrals to this clinic came from a number of units within Fremantle

Hospital. These sources included the Departments of Psychiatry, General Medicine, Genitourinary Medicine, Paediatrics, and Gastroenterology. Participants referred to this clinic were placed on a waiting list, and reviewed as outpatient appointments became available. All participants were assessed as outpatients, and those participants who were referred from inpatient psychiatric referring teams were not reviewed until they had been discharged from hospital for a minimum period of 14 days.

5.3.1. Situating the Sample

The study reports data from 33 transcripted interviews conducted on 11 participants. The demographics of the sample are included in Section 5.6. Elliot et al. (1999) recommend that the research participants should be described along with their life circumstances in order to articulate the range of persons and situations for whom the findings might be relevant. In order to explicate this, a description of each research participant is provided in Appendix VII.

5.4. PROCEDURE

The clinic procedure employed a routine screening assessment for all participants. This involved the administration, scoring, and interpretation of the Millon Clinical Multiaxial Inventory (3rd Edition) (MCMI-III) (Millon et al., 1994) at the initial clinical interview. In the course of the first clinical interview, those participants achieving a minimum base rate score of 85 on Scale C (Borderline) on the MCMI-III were administered the Diagnostic Interview for Borderlines - Revised (DIB-R) (Zanarini et al., 1989). Those participants who subsequently scored a minimum of eight (8) or more on the DIB-R R were identified as meeting research criterion for BPD and were eligible for inclusion in the study.

As part of the assessment and formulation of the case, participants were also informed that they met criterion for BPD. They were provided with information regarding this diagnosis, and advised that a research study was being conducted into this condition. Each patient was requested to consent to involvement in the study. All patients agreed to participate, and they were then requested to undertake a second screening interview. At this point, a total of 16 participants met MCMI-III and DIB-R criterion for BPD.

5.4.1. Screening of BPD Participants

16 participants undertook a second screening interview (Appendix VIII). The screening interview was administered verbally by the researcher. The screening criteria employed in Study One were identical to the screening criteria for Study Three. The same criteria were employed in order to ensure that there was similarity in the BPD samples employed across the two studies. The screening criteria for Study One eliminated participants who:

1. Reported evidence of neurological illness, neurological trauma, or head injury with loss of consciousness for a period in excess of five minutes at any time in their life.
2. Reported a history of receiving Electroconvulsive Therapy (ECT) during the previous 90 days.
3. Reported a history of psychotic illness. Psychotic illness was defined as a medical diagnosis consisting of Schizophrenia, Bipolar Affective Disorder, Schizoaffective Disorder, Organic Psychosis, or Psychosis Not Otherwise Specified.
4. Did not report a history of exclusive right handedness.

5. Did not accurately discriminate between the colours of Red, Blue, Green, and Yellow on a colour discrimination task.
6. Did not employ English as their primary written and spoken language.
7. Admitted to a current use of illicit drugs.

Of the original 16 participants, five (5) were eliminated at this phase of the study for the following reasons:

1. Three participants admitted to concurrent, regular (at least twice weekly) narcotics usage;
2. One participant admitted to a co-morbid diagnosis of Bipolar Affective Disorder (BPAD).
3. One participant was facing criminal proceedings, and was subsequently incarcerated

As a result, 11 participants were identified as meeting criteria for admission to the study. All 11 participants agreed to participate in the study. Each participant was provided with an Information Sheet (Appendix IX), and a Consent Form (Appendix X). Participants were required to read the Information Sheet prior to signing the Consent Form. After signing the Consent Form, participants were formally admitted into the study. Each participant was then provided with four (4) appointments to complete the interview series.

All eleven participants attended for the research interviews, and nine participants completed all four interviews. The two participants who did not complete four interviews each completed three interviews. One terminated the interview series prematurely as a result of a job offer that required relocation and therefore precluded further involvement in the study. The second participant terminated further interviews because the task was experienced as ‘boring’.

Those participants who did not meet criterion on either the MCMI-III or the DIB-R (persons referred to the clinic during the recruitment period), remained eligible to receive psychological services where required. A statistical analysis of differences between the diagnostic returns of those participants who completed four, and those who completed less than four interviews was not undertaken because of inadequate sample sizes and an attendant lack of power to detect differences.

5.5. RESEARCH INTERVIEWS

The objective of this study was to identify categories of emotion salient to the experience of participants diagnosed with BPD. The identification of salient categories of affect was informed by Tomkin's (1962; 1963; 1991; 1992) affect theory. Tomkins developed a sophisticated theory of affective development which has significant implications for understanding the development of psychopathology (Vaillant, 1997). Tomkins sees the evolutionary significance of affect as constituting a basic motivational system. He proposed that there are three components to the emotion system. These are referred to as Pain, Drives, and Affects. Pain operates as a signalling system in order to communicate the presence of danger. This aspect of Tomkins' model is attentional in nature, and is similar to the affective-attentional component proposed in the multidimensional developmental neuropsychological model of BPD proposed in Section 3.4. Drives represent predominantly biologically organised, innate programs designed to maintain physical integrity. The drives in this conception include breathing, eating, defecation and urination, sexual arousal, and rudimentary forms of social response. Affects represent biologically based mechanisms designed to amplify or inhibit drive functions. These affects are categorical in nature, and represent

an irreducible set of categories. Each category of affect is organised along a continuum ranging in intensity from 'mild' to 'strong'.

The term 'affect' is used here in contradistinction to the term emotion to describe subjective experience. The notion of 'emotion' in this model is used as a global category to describe internal bodily experience, whereas 'affect' describes subtypes or categories of emotion. The term 'feeling' refers to a more broadly based experience associated with the constancy of affective states which are present irrespective of whether there is conscious awareness of their existence or not. Therefore, 'affect' refers to a more precise set of categorical constructs.

Vaillant (1997) argues that one of the important features of Tomkins' model lies in its capacity to explain the notion of 'affective association'. Affective associations refer to the process by which a variety of phenomena become imbued with affectively laden value. For example, a cloth teddy bear does not possess affective value in and of itself. It becomes affectively laden *in the mind of the child/person who possesses it.* These associations are learned, and it is possible therefore to conceive that any object or phenomenon can become affectively valenced. This perspective has significant implications for understanding emotional development and the development and maintenance of psychopathology.

Tomkins identified nine categories of affect. They are characterised by specific patterns and densities of neural firing in response to internal or external stimuli, a relatively specific set of internal psychophysiological sensations, and a specific facial configuration. Table 5.1 identifies the basic affects in the Tomkins system.

Table 5.1: Tomkins Affect Categories

INTEREST-EXCITEMENT
ENJOYMENT-JOY
SURPRISE-STARTLE
FEAR-TERROR
ANGER-RAGE
DISTRESS-ANGUISH (Grief/Sadness)
SHAME-HUMILIATION
CONTEMPT-DISGUST
DISMELL

Tomkins identifies two positive affects (Interest-Excitement, Enjoyment-Joy), one neutral affect (Surprise-Startle), and six negative affects (Fear-Terror; Anger-Rage; Distress-Anguish; Shame-Humiliation; Contempt-Disgust; and Dismell). Tomkins argues that each of these categories is not a unitary state, but rather a constellation or family of related states reflecting a specific theme. For example, the grouping of affects represented by the continuum distress-anguish includes the affective experiences denoted by terms such as sorrow, sadness, grief, and despair. In contrast, the affect category defined as anger-rage is characterised by affect nominations such as irritation, annoyance, and fury (Vaillant, 1997). This in turn has implications for the variety of experiences of affective tone, and the seemingly infinite ways in which both human experience in general and psychopathology in particular can be both expressed and experienced.

Of greater theoretical significance is Tomkins' account of the relationship between three systems of emotion, and the manner of their interaction. Tomkins argues that infants are born with neuro-biologically organised predispositions for affective development. These capacities are not fully formed at birth, but mature in an 'experience dependent' fashion (Grigsby & Stevens, 2000). Tomkins (1962; 1963), argued that the affective system constitutes the primary motivational system of the human being. Affects serve the function of providing a sense of 'colour' and tone to experience, which in turn leads to the execution of preference, which can in turn be understood as reflecting a sense of underlying motivation.

Tomkins' affect theory was explicitly used when reading the text of the interview transcript in order to identify and classify affective codes reported by participants. Specific affect states were endorsed when the following criteria were met:

1. There was specific identification of the affect either by the participant or the interviewer as reported in the text. In the case of initial identification by the interviewer, it was then necessary for the participant confirm that the construct/affect category was correct. This process, including confirmation by the participant, must be represented within the text.
2. For a category to be included, it was also necessary that all participants report or confirm the experience of the category. In other words, all 11 participants were required to confirm the existence of the affective experience for the particular category to be employed in the Stroop task.

5.5.1. Interview Procedure

All interviews were conducted in the same office at Fremantle Hospital. The office was routinely used to conduct psychological assessments and psychotherapy with patients of Fremantle Hospital. The data was sampled from a maximum of four, 45 minute unstructured interviews with the author. The format of the interviews was non-directive, and open-ended. The hypotheses were not identified with participants, and no explicit attempts were made to shape or direct the nature of the material that participants elected to introduce and/or explore in each interview.

The interviews were conducted in the same manner as all other clinical interviews conducted by the author. The author has received extensive training and supervision in a specific model of psychotherapy (Contemporary Self and Object-Relational Psychoanalytic Psychotherapy) and this model routinely employs audio taping of material for the purposes of supervision and quality control. Therefore, the use of audio taping was usual practice.

Each initial interview commenced with a standard probe inquiry:

> 'By now, you have become acquainted with the broad objectives of this study. Could we begin by you telling me a little about yourself, about your life, and something about the difficulties you currently encounter. What brings you to see me?'

The material, which ensued from this probe, was unstructured and reflected the different experiences of each borderline participant. The material was managed through the use of an 'empathic-introspective' mode of clinical interviewing (Kohut, 1977, 1984). The interview process was not based upon the

use of predetermined interview questions. Subsequent interviews commenced with the interviewer inquiring: ‘How shall we begin today’?

The objective of the study was to sample the affective experience of each borderline participant. Once the interview series with each participant was concluded, participants were thanked and advised that their research involvement concluded. The nine participants who had not withdrawn from the study were advised that they were free to continue to receive psychological services, and all nine participants continued to do so.

5.5.2. Data Transformation

The data from the interviews was audiotaped using a Dictaphone Desktop Voice Processor (Model 2714). The unit had a detachable, hand-held remote control unit which could be used to control the operation of the unit, but also included a multi-directional microphone. The hand held remote control unit was housed on a seating mechanism attached to the Dictaphone machine during all interviews, and was operated from the main console located on the machine itself. Recording of each interview session was initiated prior to the participant entering the room by pressing the conference recording button (CONF). The procedure of switching the recording system on prior to the entrance of the participant was undertaken to ensure that all participant discourse was recorded. Interview sessions were recorded on BASF Ferro Extra I 45 minute audiotapes. 45 minute audiotapes were employed in this study as they recorded the entire interview session without the interruption of the Dictaphone machine switching off during the interview.

Interviews were transcribed using the same Dictaphone Desktop Voice Processor (Model 2714) using a detachable foot pedal. All interviews were

transcribed according to the principles established by Mergenthaler & Stinson (1992) for psychotherapy transcripts. Transcripts were typed into a Microsoft Notepad text editor. The first three interviews for each of the 11 participants were transcribed, resulting in a total of 33 interviews for the study. Transcription of the interviews occurred after the conclusion of all interviews in the series. Therefore, second and subsequent interviews for each participant were not influenced by the reading of transcripts of interview prior to the commencement of the subsequent interview.

Transcripts of the interview data were then introduced, coded, and analysed using the Non-numerical Unstructured Data Indexing Searching and Theorizing (NUD*IST) (Version 4.0) (Richards, 1998) software. NUD*IST is a qualitative data software programme designed to manage non-quantitative data. It has the capacity to organise data in a flexible manner, and to index data under an infinite number of categories. The programme also allows categories to be adjusted as the analysis of data develops.

5.5.3. Decision Rules for Inclusion of Affect Categories in the 'Emotional Stroop' Task

The identification of affect categories to be included in the 'Emotional Stroop' paradigm was determined by the following decision-making criteria:

1. Each transcript was initially read in its entirety
2. The transcript was then re-read, with the specific aim of identifying those sections of the transcript where there was clear reference to affectively valenced material.

3. The specific affectively valenced experience of the participant was identified either by the interviewer or the participant and a category for the particular affect was created.
4. The specific discourse-related material for a particular category could only be included if the participant confirmed that they were experiencing the specific affect. The transcript was required to include confirmatory discourse initiated by the participant indicating that they experienced the affect nominated by the interviewer. Alternatively, the participant must independently nominate the affect category without prompting from the interviewer.
5. For an affect category to be included in the 'Emotional Stroop' paradigm, all participants in the study must endorse the experiencing of the category of affect. If any one participant failed to endorse a specific affect category, the category was not included in the Stroop paradigm.

5.6. RESULTS

The demographic features of the sample including the age, gender, occupational status, marital status, and educational status is reported in Table 5.2, and Table 5.3 reports the means and standard deviations for DIB-R Scaled Scores, and MCMI-III Validity, Clinical Personality Pattern, Severe Personality Pathology, and Clinical Syndrome Scales for the BPD Sample.

Table 5.2: Demographic Data for the BPD Sample Examining Affective and Semantic Representations in BPD

PARTICIPANT NUMBER	AGE	GENDER	OCCUPATIONAL STATUS	MARITAL STATUS	EDUCATION STATUS
1.	45	2	7	2	1
2.	41	2	9	3	1
3.	29	2	9	5	1
4.	30	2	2	5	2
5.	24	1	7	5	2
6.	33	2	9	1	1
7.	31	1	7	3	1
8.	52	2	9	1	1
9.	41	2	9	4	1
10.	42	2	9	2	1
11.	32	2	8	5	3

1 = Male 2 = Female	1= Professional 2= Managerial 3= Technical 4= Clerical/Sales 5= Skilled Labour 6= Semi Skilled Labour 7= Unskilled Labour 8= Student 9= Home Duties 10= Unemployed	1 = Married 2 = Divorced 3 = De Facto 4 = Separated 5 = Single 6 = Widowed	1 = Completed Yr 10 2 = Completed Yr 12 3 = Completed Degree 4 = Completed Postgraduate Degree

Table 5.3: Means and Standard Deviations for DIB-R Scaled Scores, and MCMI-III Validity, Clinical Personality Pattern, Severe Personality Pathology, and Clinical Syndrome Scales for the BPD Sample

	MEAN	SD
DIB-R TOTAL SCALED SCORE	9.45	0.52
DIB-R AFFECT SCALED SCORE	2.00	0.00
DIB-R COGNITION SCALED SCORE	1.73	0.47
DIB-R IMPULSE SCALED SCORE	2.91	0.30
DIB-R INTERPERSONAL RELATIONSHIPS SCALED SCORE	2.82	0.40
MCMI-III VALIDITY SCALES		
SCALE X (DISCLSURE)	85.09	7.40
SCALE Y (DESIRABILITY)	41.82	15.72
SCALE Z (DEBASEMENT)	83.09	6.38
MCMI-III CLINICAL PERSONALITY PATTERN		
SCALE 1 (SCHIZOID)	69.09	16.62
SCALE 2A (AVOIDANT)	73.82	19.51
SCALE 2B (DEPRESSIVE)	84.73	13.96
SCALE 3 (DEPENDENT)	79.09	8.14
SCALE 4 (HISTRIONIC)	28.91	26.79
SCALE 5 (NARCISSISTIC)	35.36	20.70
SCALE 6A (ANTISOCIAL)	71.09	12.37
SCALE 6B (SADISTIC)	65.36	12.41
SCALE 7 (COMPULSIVE)	31.82	14.65
SCALE 8A (PASSIVE- AGGRESSIVE)	78.09	11.03
SCALE 8B (SELF-DEFEATING)	78.09	6.63
MCMI-III SEVERE PERSONALITY PATHOLOGY		
SCALE S (SCHIZOTYPAL)	72.73	16.33
SCALE C (BORDERLINE)	89.73	3.17
SCALE P (PARANOID)	68.36	14.71
MCMI-III CLINICAL SYNDROME		
SCALE A (ANXIETY)	93.36	9.56
SCALE H (SOMATOFORM)	71.82	17.81
SCALE N (BIPOLAR)	71.18	14.01
SCALE D (DYSTHYMIA)	82.00	14.56
SCALE B (ALCOHOL DEPENDENCE)	70.09	9.12
SCALE T (DRUG DEPENDENCE)	71.09	17.13
SCALE R (PTSD)	79.18	16.90
SCALE SS (THOUGHT DISORDER)	75.55	9.86
SCALE CC (MAJOR DEPRESSION)	82.64	15.40
SCALE PP (DELUSIONAL DISORDER)	63.55	9.76

5.6.1. Theoretical and Clinical Implications of Informing Participants That They Meet Criterion for BPD

One of the more controversial issues in the diagnosis of BPD is the issue of informing patients that they meet criterion for this disorder. Unfortunately, no protocols exist to guide decision making with regard to this issue, and limited empirical evidence is available to assist with the decision to advise people of this diagnosis (S. G. Miller, 1994). One of the concerns inherent in notifying people of this diagnosis has been the issues associated with co-morbidity, reliability of the diagnosis, and concerns that despite the provision of the diagnosis, a cost effective and easy to administer treatment is lacking. Another concern is that there appears to be a stigmatising process associated with the use of personality disorder diagnoses, and that the application of such a diagnosis is potentially experienced by the recipient in an adverse manner. There is however, little evidence available to support the view that the provision of a personality disorder diagnosis is experienced as a stigmatising phenomenon, and no data is currently available to guide decision-making regarding when and how to advise patients of this diagnosis.

Despite this, one of the ethical requirements involved in the execution of this study was the provision of information concerning the diagnosis for study participants, and information with regard to the key features of the disorder. Clearly, one of the concerns inherent in this approach was the possible adverse effect for study participants in learning that they met criterion for BPD.

This concern appeared unfounded, as the participants reported experiences of relief and feelings of a greater level of understanding with regard to the difficulties they confronted in their lives. It seemed that information

concerning the diagnosis and phenomenology of BPD provided participants with a greater degree of understanding, and seemed to assist then in making greater sense of their lives. The following vignette provides an example of the positive benefit the provision of diagnostic information made for the study participants. In this vignette, the participant reports the content of a final consultation with her psychiatrist prior to relocating to Western Australia. In her account of the consultation, the diagnosis of BPD was confirmed in the following manner.

Participant: Well.....I don't know. I never knew what was wrong with me until just before I left.

Interviewer: How did you learn about what was wrong with you?

Participant: I can't remember who I was pumping. Oh, he'd (psychiatrist) spoken to my mother or something and on top of it I came in and I said what is 'What are you treating me for'? 'What do I have'? He said 'Oh you've got a personality disorder L.' And I said 'A personality disorder'? He said, 'Yes'. I said 'What type'? And he denies this, but I know he said it, 'Oh' he said, 'You're a smorgasbord' and laughed. It was just as I was going out the door. So I went to the library and I hadn't got any books out before, nothing on child abuse, nothing at all and so I started searching and started reading and everything that I read nothing really…. There was a bit of me in all of them but there was nothing that I would say was me and there was a book that had a lot of pages missing and I asked the library would they get another one in. 'Listening to Prozac'.. No I read that one, I can't recall which one it was now and it took three months for it to come from State Library and when it came it was the section on Borderline Personality Disorder. I couldn't find borderline in any of the books, there was nothing written on borderline just everything else but there's nothing on borderline, and when I read it I couldn't believe it it was like..it was my life. I thought this is me to a tee. This is absolutely me. It's like I'd written it myself.

Interviewer: Can you remember what the book's name was?

Participant: No. Nup. I can't. Like I said I waited 12 weeks for it to come from State Library and Dr M was away overseas and when I saw Dr T, I came in and I put the book down and I said 'Dr T. I know what I have.' He said 'What's that?' 'I've been reading and reading and I said nothing fitted, but this is me to a tee,' I said. 'I can't believe there's another person in the world can think and react and feel like I do, but there it is, it's called Borderline Personality.' He said, 'That's right.' And he said 'Have you ever seen that movie Breakfast at Tiffany's?' I said 'Yes.' He said 'What did you think of it?' I said I couldn't follow it very much. He said 'Well we use that as a training film. That's all about borderline.' 'We use that in psychiatry.' I said 'Oh shit. No wonder I couldn't follow her she was all about like I am, same type of life.' Umm and then Dr M before I left...Dr M went away for six or seven weeks he went overseas and I'd only seen him twice before I came back over here so I never got round to discussing it with him, but he would never ever diagnose it...he would never ever say what was wrong with me. Every time I said 'Well what's wrong with me', he'd never tell me, it was just infuriating.

Interviewer: Can I ask you this question because I think this is an important issue for people with this, this problem. I don't know the book you read that you saw your life in the pages so to speak....

Participant: Yes.

Interviewer: But it sounds like that was more of a revelation than anything?

Participant: No. It was more..can't remember actually. I can remember reading it and thinking, shit, that's me.

Interviewer: But to have something that you now know about yourself like, my impression is that you've spent years saying 'What's wrong with me'?

Participant: I have.

Interviewer: And there you read it basically in what, four or five pages?

Participant: Yes, four pages. Four or five pages.

Interviewer: What was that experience like to actually see it in print and to now know that it's known to professionals?

Participant: How did it feel?

Interviewer: Yes.

Participant: I felt absolutely furious.

Interviewer: Furious?

Participant: Absolutely furious that I had gone to psychiatrists...not the money that I've paid..it had nothing to do with, you know, the money. It was the fact that I knew inside that I was drowning. I was drowning as a mother, I was trying to bring up three kids and I carted a useless husband along with me, that I'd gone for help and I got Valium, Serapax thrown at me which I had the good sense to throw the prescriptions in the bin and I felt like ringing the AMA as soon as I walked out the door and putting the doctors in, because not one of them had said to me 'Why are you crying all the time L'? I'd just go in and say 'I'm crying all the time. I just can't stop crying'. Not to mention the itches and the crawling for years, like bugs all over your face and your eyelids all the time.

Interviewer: So when you found out the name of this disorder, you felt furious because you hadn't been told before?

Participant: I'd been diagnosed with PMT. The doctor, I think his name's C. He works out of (Hospital).

Interviewer: Uh Huh.

Participant: He wanted to flip my ovaries four years ago before I left to go to (Another State). He reckoned the PMT was that bad, they would flip the ovaries.

Interviewer: Okay. Can I just back this up, because I think this is an important issue. It sounds like you're saying you're angry because nobody told you about this disorder?

Participant: I was diagnosed with PMT all the way through.

Interviewer: So you're angry at the misdiagnosis, or the fact that maybe people knew what the diagnosis was but didn't tell you.

Participant: Well Dr M certainly didn't tell me, which I...I'm pretty pissed off at him for.

Interviewer: Do you think it's better to know or not know?

Participant: Well...you try to commit suicide because there's no hope, there's no hope at all, life is hopeless, but at least if they diagnose something you've got the chance to fight it. You don't know what you're fighting. It's like I said to him I couldn't care if he said I had AIDS or cancer at least if you know what you've got you know what you're fighting

against. If you don't tell people then here we are, we're chaotic, we're disruptive, we embarrass ourselves.

Interviewer: So would you say that as a matter of principle it's better that people know?

Participant: People should know. Because most of them commit suicide anyway, but then the others that really have the willpower to live will fight it. Yes, withholding it from people is absolutely disgraceful.

Interviewer: What about the idea that telling people that they have this problem sets in greater despair?

Participant: You can't. There's no such thing as a greater sense of despair that you could already have - it's impossible.

Interviewer: So when you learned of what this disorder was, this psychiatric condition, did that give you a sense of relief?

Participant: Sure. Absolutely. For the first time I thought so that's what it is, that's all that it is. All it is is a personality disorder and, yes, dysfunctional thinking. I can turn that around, but I didn't know what was going on, well when they talk psychology they talk about the brain and I was wondering, you know, I believe we should be told. Everybody should be told as soon as they're diagnosed.

Whilst this material has no direct bearing on the development of the Stroop paradigm, this finding has implications for the clinical management of BPD and will be considered in greater detail in Section 9.7.1.

5.7. AFFECT CATEGORIES

Section 5.5.3 described the decision rules by which discourse material on affective states in BPD would be included in the study. Four so-called 'negative' affect categories were consistently reported by all participants in the study: Anger-Rage, Distress-Anguish, (Grief/Sadness), Shame-Humiliation, and Fear-Terror. The following sections will outline the parameters of the experience of negative affective states consistently reported by the BPD cohort.

Affect Identification and Affect Regulation

All participants readily identified difficulties in relation to both the identification and regulation of affect states. In the first vignette, the participant outlines both levels of difficulty. The discourse commences with the opening discussion of the first interview in the series conducted with this participant. The participant inquires about commencing the interview, and thereafter produces the following material:

Interviewer: By now, you have become acquainted with the broad objectives of this study. Could we begin by you telling me a little about yourself, about your life, and something about the difficulties you currently encounter?

Participant: Where do I start?

Interviewer: Perhaps the best thing that you could start with is for you to just tell me a little bit about yourself and what your experience of life is like for you.

Participant: Alright. What sort of person I am. I'm basically, when I'm out in the public I put on my mask and I'm usually very quiet and cooperative and do as I'm told. But when I'm in my own space I'm completely different I.. tend to be umm, umm, take my mask off and I then I become more sometimes I can be aggressive I can be loving and kind or umm I'm just different. Umm when umm.. when I'd have the rages I was telling you about (on phone when initial contact was made) I'll swear and say dirty words and things like that which normally even in my own home environment and I wouldn't say I don't even like I don't agree with it in fact I'd probably find umm some of the things that I say if somebody did to me I'd find them terribly offensive, but when I really just go off it just all comes out, everything, the lot like an old fishwife. I scream, I rant, I rave but I'm not like that all the time, that's when I have these episodes.

Interviewer: How often would you have these episodes?

Participant: How often? Probably not as often now as I used to...umm but, I still have them. But I need very little or no provocation. It just, they just it just happens. But I find even which I don't have an episode that I'm not I don't know how to control my own feelings. I, if

I'm under any sort of stress, if for instance I'm having a something, one of my children…. I've got a daughter 16, 17 and a son 19, and just recently in particular with the daughter I'm having lots of problems and I'm not able to talk to her. I don't know how to talk to her. Umm as soon as we start talking I find myself getting all churned up and I end up in an argument.

Interviewer: So when you say churned up, [do] you mean annoyed?

Participant: Umm no, not even annoyed.

Interviewer: A kind of a distressed feeling?

Participant: Nervous, nervous

Interviewer: Like anxious?

Participant S: Frightened and anxious.

Interviewer: Frightened and anxious? I see. And then what happens after that, you talk about this experience of feeling churned up and anxious?

Participant: Well then of course my daughter will react to how I am and we end up screaming at each other and with no result.

Interviewer: So the sense of feeling anxious just transforms into feeling angry. Is that what you're saying?

Participant: There's anger at the end of it, but it's more ummm...I guess I'm frightened to say things to her maybe even, I can't really quite understand...I really don't know what the feeling is. Ummm and it happens with my son as well that and other people as well ummm I'm chopping and changing and I was at work and even if I had a discussion with somebody and we disagreed I could never ever speak how I felt it would everything would just be an absolute turmoil and I would have to back off from what the discussion was I couldn't proceed with it or just have a normal a normal discussion if there was a disagreement in what we were talking about umm...because part of my heart would start beating quickly and I wasn't able to, to ummm, express myself or say what I wanted to say and then I'd be all shaking I would shake yes.

This vignette highlights one of the major difficulties of the borderline – accurately identifying affective material. Participants often demonstrated

difficulties in identifying affective states, or alternately demonstrated deficits in relation to the use of a linguistic syntax to identify affective states. These situations called upon the interviewer to inquire about the presumed affective experience. This was usually done by asking the participant if they were experiencing a particular affective experience. In situations where the interviewer was unsure what the participant's affective experience might be, the participant was asked to elaborate their experience.

This phenomenon also has implications for the analysis of Stroop data. 'Interference' analyses typically rely upon subtracting the response latencies of 'neutrally' valenced words from affectively laden ones in order to obtain measures of 'pure' interference. This methodology has the potential to be seriously compromised if the participant experiences difficulties in identifying affective states. This is an issue that will be considered in detail in Chapter Nine.

A second difficulty that was often observed was the concurrent reported experience of binary or multiple co-occurring affective states. These are referred to as 'affect blends'. From the perspective of developing the Stroop paradigm, the following participant was able to identify the experience of a number of different concurrently experienced, affective states.

Interviewer: What I'd like to do to begin with is to examine some issues in a little bit of detail which I think are important for me to know about. Now, the things I'm particularly interested in are issues to do with emotion. Could we begin by talking about your emotions? One of the things that I'm particularly interested in understanding is about how people with BPD organise their emotional lives and how they, what's called regulate, their emotions. Can you tell me a little bit about how you organise your emotional life?

Participant: **Anger. Anger** is really the main one.

Interviewer: What other emotions are you aware of that you experience?

Participant: **Sadness**. That's about it. About the only other time, **grief**.

Interviewer: Anything else that you're aware of?

Participant: When you're **happy**.

Interviewer: So happy, grief, sadness, anger. Are there any other emotions that you are aware of?

Participant: No.

Interviewer: Now the way that you control those emotions, does it differ between them?

Participant: No.

Interviewer: You do the same thing to manage them?

Participant: Oh, no, probably not **happiness** I wouldn't sort of..., no I wouldn't leave the room for that. All the rest I do.

Interviewer: So can you tell me what you do when you're feeling one of these feelings?

Participant: I just go in my room.....or leave the house.

Interviewer: And what do you do?

Participant: I just brood.

Interviewer: Let's just go back to what you know that you experience emotionally. The emotions that you are aware of are anger, grief, depression and joy I think it was. Are there any other feelings that you know you experience?

Participant: No.

Interviewer: Fear?

Participant: Oh yeah. I forgot about that one.

Interviewer: Anything else?

Participant: I just can't put my finger on it, I just...I don't know, it's like **being empty** or just **nothing**.

Interviewer: Can you describe this sense of emptiness for me please?

Participant: Well you don't know where to put yourself or what to do or...there doesn't seem to be a way out.

Interviewer: Sounds like a sort of feeling of being out of place?

Participant: Yeah.

In the example reported, the participant was able to independently identify the affective states of anger, sadness, and joy. With some clarification from the interviewer, the participant was able to also identify the experience of fear or anxiety. Thereafter, the participant began to report the experience of a less specific internal experience which was understood as an affective state but was less able to be described. It was associated with a state of emptiness, nothingness, or being trapped. This experience, often referred to as a state of 'existential aloneness' (Adler & Buie, 1979), or of the 'black hole' (Grotstein, 1990, 1991) is frequently reported in the clinical literature on borderlines but does not appear to link directly with a specific affective state. Despite this, the vignette provides a sense of the nature of the salient affective experience of the borderline. It also provides some evidence for the major affect categories likely to be involved in a Stroop paradigm – anger, sadness, joy, and fear.

The following sections provide evidence supporting the identification of specific affect categories that were employed in the Stroop paradigm. Each reported vignette will be introduced by providing some context for its inclusion, and where possible, transcripted material will be included under sub-categories which will illustrate features of the affect category such as how the affect is experienced, and the means by which the affect is regulated.

5.7.1. Anger-Rage (Anger)

The most commonly reported affective experience was that of angry, rageful states. Specifically, participants reported experiencing anger episodes more frequently than other affective states. The origins of each participant's experience of episodic anger varied significantly, but the main dynamic which drove this appeared to be an experience of feeling misused, or treated in a

manner which disavowed their personal experience. These experiences are consistent with the biosocial theory of Linehan (1993) who has argued that BPD is in part provoked as a result of the childhood experience of living in a 'negating' environment. The experiences in question could either be current experiences, or thematic-episodic issues remembered from the past. This section will report a variety of themes related to each participants experience and reporting of anger. They will include the participant's reports of the experience of anger, and the experience of regulating anger states.

Experiences of Anger

A number of participants described the experience of angry rageful states that were based on a long-standing sense of interpersonal injustice. One participant offered a particularly salient example. The participant recalled memories associated with the manner in which the participants' father responded to her as an adolescent. In recalling a series of episodes involving a family celebration and another occasion where she and her father were travelling in a vehicle. The transcript material commences at a point where memories from the participant's courtship period were used to describe an experience which enhanced her self esteem. In the episode in question, the participant commences by speaking with a sense of pride about how she, rather than other women, was the object of her husband's affections. The participant then produced the following discourse material:

Interviewer: So that was a real boost for your sense of self? (The understanding that her husband wanted a relationship with the participant and not other women)

Participant: Oh God, yes. You know, to make sure that it was me that he wanted you know, but that only comes from the fact that when I was growing up I was the oldest of three girls

and one brother and umm how old was I.. 13.. I must have been 13 it was my thirteenth birthday party my father called me a slut in front of all of my friends who were at that party and all I was doing was playing spin the bottle and I just happened to spin the bottle on one guy that I had liked for so long at school and he never paid me the time of day ever but I got to spin the bottle and I got him and just as I was about to kiss him in front of all of my friends and my dad was standing up on the steps and he just turned around and said 'You're a slut'.....and it's like anything I have to do with guys, anything at all, you know, always trying to prove that I'm what they need, I'm important in their lives. You know what I mean? Umm and it's that that comes from my dad.

Interviewer: I can see even now that the memory of that time at 13 is still very painful for you.

Participant: Oh God, yes. It still really **annoys** me that, you know, I couldn't even look at a guy. And I got a bit... My dad always sat me in the back of the car when we'd be driving. Always behind him so that he could look in the rear vision mirror and look at me so that while he was driving he could check the road and check me to see where I was looking. And mum would be sitting in the front seat. And if I just happened to see...I was just looking on the road looking at the scenery but if there happened to be some guys walking along there my dad would turn around to my mother and say 'She looks at anything in pants. You're going to have to watch your daughter, you know, she's going to end up a slut.' And I think he was in fear because all the girls in our street everyone where I came from. Umm all the girls, before they were 13 were pregnant,, you know, so he didn't want me to get pregnant before I was married. And then low and behold at the age of 29 I find myself pregnant with L, my daughter, now but before that when I was 21 I was pregnant to D as well and he made me, oh he insisted that I get an abortion.

In the above example, the participant independently nominates the affective descriptor of 'annoys' in response to the preceding material under discussion as well as the interviewer's empathic linkage with the participant's experience by acknowledging that she was experiencing a painful affective state.

The use of the term painful by the interviewer appears to assist the participant to more explicitly categorise her internal state as one of 'annoyance.'

A different phenomenon was also observed when another participant remembered the manner in which one of her parents tutored her in mathematics. In the process of recall, the participant's response included the production of a concurrent experience of anger even though the event which was under discussion occurred many decades earlier. The participant had been recalling the manner in which her parent had humiliated her as her tutoring proceeded by implying that she was 'stupid'.

Interviewer : That's the feeling you had conveyed to you?

Participant: Always. You know umm learning maths [was] the most difficult participant I had at school. Maths. I could not comprehend, I just didn't get it, you know, and my father's very smart with maths, he's a very umm he's a well spoken quiet man. Umm...he's ..he when he punishes he punishes you, you know, looking at a leather strap with a gold buckle like a snakeskin blue strap with a gold buckle on it and he only had to say it once and if you didn't do what he said the first time you got that strap across the legs you just copped it and you nothing was going to get you out of it you know what I mean? Umm oh I've lost my track...everything's making me **mad** now.

Interviewer: Remembering this provokes anger in you?

Participant: Uh Huh.

In this example, there appears to be evidence of an interdiction process whereby the memory of events that were physically abusive led to a loss of the narrative account of the event because of the interference effect of strong emotion – in this case anger.

Similarly, another participant described the experience of a mode of interpersonal misuse as a basis for ongoing experiences of anger and rage. In this instance, the source of the misuse appears associated with memories of parents requiring more from the participant than was developmentally reasonable. In this instance the demand included caring for her adolescent sister when the participant was aged 21. This sense of inappropriate use was then compounded by the experience of an absence of empathic understanding from her husband with regard to the significance of the event for the participant.

Participant: Absolutely. It's not...my dad,..my mum and dad when I was 21 and going through a divorce, my mum and dad knocked on my door at home with my fifteen year old sister K who now lives in M, they had her bed and her clothes in the back of the car, didn't ask me anything, knocked on my door and said we can't control her, she doesn't want to stay with us you look after her. So at 21 I've got a 15 year old to look after, I'm working a day job and I'm working in hotels at night to try and sort of get me by and I have to look after my 15 year old sister who is working in hotels under age or she's coming with me to hotels because I'm old enough to go but she's not, so I've got my 15 year old sister to look after and they don't want the responsibility so they dump that on to me. Do you know what I mean? I didn't need that I was still trying to get me organised, you know, getting married oh I was so **angry** and D (husband) keeps on saying to me you have to forgive your parents, they don't owe you anything but as far as I'm concerned they owe me a lot. Okay fair enough they did the best that they could, everyone who becomes a parent they don't have a book of rules no one's taught them how to become a parent and you do the best you can, but as far as I'm concerned my parents didn't. You can't tell me that parents who've got an 11 year old child, right, no, I must have been about 11 first year high school, over the school holidays they got me a job in a factory and I was working for (Well known Australian Company) in M (City), my Aunty was the foreman or forelady whatever you want to call them, in the factory and I got a job in there and every week my mother would take my money off me. She'd give me enough

money for bus fare and give me lunch and the she'd take the money telling me that the money was being put in the bank so that whatever came first my 21st or my wedding I would have money for the wedding or the 21st right? And because I wanted to do modelling, I wanted to do finishing school I needed to do four years of that and I wanted to learn to sing so all the part time jobs that I got mum took all the money, I started work when I was 15 full time, she took the money right, and then when the time came that I was 21 nope I got married first, there was no money because all of a sudden it was well you paid for your piano singing lessons and you paid for the four years of finishing school and it's like I'm paying for everything? So when the time came for a wedding there was no money so I had to pay for that and then when my 21st came up there was no money for that and I had to pay for that too. First up is **anger**, towards both mum and dad and D (husband). I feel like I've been controlled, I have, I have been controlled for so long that the real me's never been able to come out umm you've always I've always been a people pleaser, umm, make everybody happy and forget about me.

Another participant described a process of repetitive, chronic rage directed toward her partner which did not resolve. This experience lead to a sense of feeling trapped, and the manner in which this resolved itself through an episode of attempted suicide. The affective intensity of this experience remained present at the time of the interview, and appeared to result in a disorganized level of discourse associated with the reporting of the event.

Interviewer: What were you feeling at that time in your life?

Participant: **Anger** and **hate**. Just **anger** and **hate** towards him. I can't be with him anymore. I can't stand the fact that he watches over me he ....he's always so critical about everything you know like I can have my kitchen in a mess and it doesn't bother me I'll fix it later but it's like does this kitchen bench have to be so cluttered, does the sink have to be so full you know, when're you going to do the laundry and over years and years and years of doing this and then for him to turn around after February now he feels

really bad. Do you know that since February all he can see is me excuse me and not being able to revive me and trying to get me to hospital and stuff therefore realising the stage that I'd gotten to and wanting to make everything better. It's too late to do that and he's not letting me go, do you know? And it's like I feel so...

Interviewer: So you were angry and depressed?

Participant: Yes. Really, really and I was just like tears are streaming down my face I'm so **angry** thinking what can I do you know... and then I took a few deep breaths and started to walk around I looked and I just happened to catch a glimpse in you know the fridges the reflection and I stood there and I looked at myself and I thought nup don't want to be here I can't do this anymore I don't want to argue anymore all I want to do is be happy and I'm not going to be happy here. So I thought fine so I did my shopping got in the car drove home thought okay umm and he knew something was wrong when I walked home because he wanted to talk and I wouldn't talk wouldn't talk I was crying and crying and telling him to leave me alone there was nothing that he could say that could make me feel any better and so I just said leave me alone and got myself up into the bathroom and locked the door in the bathroom I just sat on the step for a while the spa step just sat there and thought about everything nup can't do this I just can't be here as much as I love my daughters they're going to be better off with their dad they're better off without me. So I took all the D (sedative) got in the shower and started to get quite relaxed and by the time I had a shower so yes anyway by the time I got out of the shower I was quite relaxed and ummm put on a nice dress you know summery casual dress you know no make up nothing went into my beauty room I love my beauty room I love it it's peaceful and I pulled out this beautiful book and just started to write in the book and that's all I remember. That's basically it I mean I don't even remember getting dressed. I remember taking the dress out ready to wear I don't remember putting the dress on I can't remember walking to the beauty room but I do remember getting the book because it's got a fairy on it and I love fairies and that's basically it I don't know remember (husband) slapping me in the face to wake me up I don't remember the ride to the hospital and then the next thing I remember I was at some hospital I was either I think it was M I was in some room and they were giving me charcoal to drink and there

> was some priest in there and Dr S came in the psychiatrist and someone asked me if I wanted to say there or go somewhere else. You can stay here or go to K (hospital) I said I'll go to K and in the ambulance ride and I was sick and I remember saying to the ambulance driver people pay money to be in umm in this fund the ambulance fund this is the crappiest ride I've ever had it's just horrible it's comfortable at all cos they bash you in here and in there you know and yeah that's it that was that and the next thing I'm in hospital and (husband) was there every day you know he'd ring me up to see how I was and I just wanted to be away from him you know it was like I'm back to reality and you're still here you know but that was really bad that was my when L (daughter) when ummm I sat down with my oldest daughter and told her this was quite a while after because she didn't quite understand what was going on and I was sick and tired of lying trying to make up stories about why D (husband) and I weren't getting on and what's going on with the family and she's a mature enough girl she's 17 so I sat down and I talked to her and I told her what had happened and she just looked at me and started crying and said how could you leave me how could you ever think of leaving me. You know and that pretty much made me realise that I can't leave my girls.

This section has provided some evidence of the identification and experience of anger as a commonly occurring affective state in borderlines. The next section identifies some of the mechanisms employed to manage overwhelming experiences of anger.

Anger Regulation

Affect categories were also reported by participants when describing difficulties in managing intense states of anger. Regulation of intense anger states is a diagnostic feature of all BPD diagnostic systems, and participants readily identified difficulties in managing this affective state. One participant was able to articulate her difficulties with regard to regulation of anger or rageful states. This

also included the use of suicidal and self harm gestures as a means of regulating an intense state of anger (Wagner & Linehan, 1997).

Another participant also reported the use of self-harm gestures as a means of regulating overwhelming affective states by using self-injurious behaviour as a means of initiating an alternative, less overwhelming affective state.

Interviewer: So, sorry, C (Name), are you saying that you used it (cutting wrists) as a way of stopping yourself from doing more? Is that...

Participant: Stopping myself from being **angry**. It's like it……..

Interviewer: Uh Huh, yeah sure. I just wanted to clarify. It's a way that you stop yourself from getting worse, or just stopping yourself from feeling?

Participant: Getting worse probably.

Interviewer: Uh Huh. So you kind of stop the feeling by ..

Participant: Hurting my self.

Interviewer: By using a sensory experience? That stops you from feeling angry?

Participant: I used to do it **scare** myself. If I **scared** myself enough, I would stop and I would be left feeling **embarrassed** and **ashamed** and this and that. And so the days that I used to have to bandage myself up because the bleeding wouldn't stop straight away 'cause I was such an active sports person I, I used to have sweat bands on me instead or I'd have um a sprained wristband or something.

Another mechanism for the regulation of states of anger generated by interpersonal conflict was reported by yet another participant. In this instance, the participant reports the use of two interrelated mechanisms. These include the use of disavowal - a cognitive strategy which eliminates the valency of an event or person. In this case disavowal was used to lessen or minimise the psychological valency of members of the family *in the participant's mind.* This

is exemplified by the use of the phrase 'I don't give two shits'. Second, the participant employs a method of behavioural elimination of problematic others - those who provoke an experience of interpersonal conflict are rendered impotent of their power by literally disconnecting from them - they are no longer part of the participant's relational network. Third, the participant reports the experience of confusion concerning the contents of the mind of others. This is exemplified by the statement: 'I mean, I don't know whether to believe her or not'.

Interviewer: Uh huh. Sounds very confusing? I wonder if what happens also is that people take sides but those sides always seem to change?

Participant: I don't care about them anymore. I don't I don't give two shits... I care about my brother because he's looking after my dad... and I love my dad... before I never used to love him because only when my mum was alive... I couldn't give a shit about them and they don't give a shit about me .... now it's a different story now that she's gone... my older sister she just causes trouble.. I mean, I don't know whether to believe her or not.

Interviewer: What do you feel about that?

Participant: **Angry**.. she's caused so much. trouble in the family it's not funny....

Interviewer: So the way to manage these people is to cut them out of your life, have nothing to do with them?

Participant: Just move as far away as possible.

Identification of Anger States

A number of participants also reported varying abilities to identify affective experiences. This difficulty was particularly marked in relation to the recognition of emergent anger states. The first example describes the experience of one participant who reports an apparent lack of awareness of the experience of anger in relation to a sense of injury perpetrated by her father. In response, the

participant acts in a retaliatory manner toward her intimate partner. The material commences with the participant discussing a sense of personal emptiness, and thereafter the material moves towards a discussion of the difficulties associated with identifying anger states.

Interviewer: What about the emptiness?

Participant: Oh, well the **emptiness** would go with the pills, that's why you take pills. That's why you take a whole heap of pills and put yourself in hospital so for five days you don't need to deal with it, and hopefully you're as crook as a dog when you wake up, because even though you're not a hypochondriac if you're body isn't feeling a hundred percent you don't really feel like concentrating on some **emptiness** when you're really thinking about, oh shit, I really have no potassium in my body and umm I've still got double vision, I wonder whether that will clear up or my kidneys are really stuffed this time. So it gives you a bit of time to think about things other than total boredom.

Interviewer: Sounds like this feeling, kind of prickly, angry this morning (This was inferred from the tone, facial gestures, and posture of the participant).

Participant: Prickly **anger**?

Interviewer: Mmmm.

Participant: Oh, Dr M (former treating psychiatrist) says I'm a very, very **angry** woman.

Interviewer: Do you think that he was correct?

Participant: **Nup. I don't feel angry**, that's the thing that got me. **I don't feel angry** but I don't have any **anger** vented at somebody else, I couldn't go and kick somebody else's dog, I couldn't go and swear at anybody else.

Interviewer: So it sounds like at least one person persistently says you're angry, but **it sounds like you don't feel aware of being angry**.

Participant: **No. I don't know when I'm angry**.

Interviewer: So do you mean it's possible that you are, it's just that you don't know that you're angry?

Participant: Oh, yeah. I would say after what he proved, yeah, I am very **angry** and I don't know how I got **angry** and I don't know why I'm **angry**.

Interviewer: What could possibly have happened that would make you so angry?

Participant: My dad upset me badly a week ago and I turned around and I kicked S (current partner), who really is a very nice guy and I really hurt him badly.

Interviewer: S is the man that you've been having a relationship with?

Participant: Yes. And I hurt him. I hurt him really badly. When I sabotage a relationship I sabotage it in a big way. There's so much water under the bridge that you can't go back.

Another participant described an experience of anger that was associated with residual memories of family life.

Interviewer: If you were to have a life you'd be like other people? How would it be different to how it is now?

Participant: Heaps so. I'd do what I want to do... or what I wanted to do..... sometimes I blame my family for what I've become..... I get **angry** all the time.....

Interviewer: So, I get the impression that what it feels necessary to do is to kind of back off from those feelings because if you don't kind of distract yourself from those feelings they're going to get out of control and it's going to be quite turbulent.

Participant: I'm always making them happen. Always making..always make, provoking an argument and then I get **angry**.... I need to get **angry** and then the other person gets **angry** and then either hits me or says something that's hurtful so I'm putting myself down all the time.... that's probably how I want to be.... because I've never known how it's been to be happy.... never been happy.

Interviewer: I see.

In this vignette, the participant describes a phenomenon reported by Bollas (1996) in which the borderline patient seeks linkage with the 'object of desire'. This refers to an engagement with another person who serves the

function of maintaining some form of turbulent relationship in which the participant is able to maintain a characteristic and volatile mode of relating – a form of connection which engenders the re-experiencing of familiar patterns of relatedness. In the vignette reported above, the participant describes one such scenario.

The following vignette also illustrates the difficulty some participants have in identifying anger states within themselves, but also serves to demonstrate the operation of 'anger episodes'.

Interviewer: You've used this metaphor today a couple of times and it's quote "ripping me apart," which I'm not arguing with this, just it's a very dramatic metaphor. Can you say a bit more about feeling ripped apart?

Participant: Well before the, before the um before I ended up here (in hospital), this guy moved into the house, and prior to him moving into the house S (ex-partner) and I had a few background differences over what is, I don't know, what is courteous or respectful or whatever. May be social etiquette. We had a few differences there, but she was fine in me letting her know, just because she had never, it had never been brought to her attention before. So we worked out a lot of our relationship things about me that she didn't particularly like or was annoyed about. We, you know, we talked over a lot of stuff. Then she went down south, this guy moved in with me, and we had this three way relationship going. And as soon as he moved back in um I was having a,, it was at the stage where I was having a lot of hassles at work, um, which didn't help but um when S (ex-partner) did arrive it was like everything that we worked on went out the window. And it would be things like well, you know, she's been out on a work do with this guy down south, it's really bad weather, it's pissing down with rain, I'm on my own in Perth, and she said to me "Oh well, I will call you" at whatever time. And it's like "o.k." And she wouldn't ring and then I find out they've been to the work do, um in which they would have both been drinking at, in a country town, driving on pretty bad roads, getting home and her having enough consciousness to um go and get herself a coffee or

whatever, walk past the telephone probably about five times, sit herself down on the lounge, get the television on, have a glass of wine - whatever, and um pass out. So I'm still you know, by this stage it's like it's ten thirty, eleven o'clock, I'm thinking "shit, have they crashed," I mean she's been pulled up by the cops so many times like 'what the hells happened? 'And so by the time I get to the phone I'm like really anxious, I'm hoping that they're o.k., but at the same time I'm **furious**. By the time I get through it's like "Yep, yep, yeah she's here. She's asleep on the lounge." It's like why didn't you ring? " Oh I fell asleep, I was tired, I was this and I was that." "it's like "Yeah, but haven't you already done da, da, da." "Yeah, I did all that when I got home." It's like "Why couldn't you just give me a call just to let me know that you're o.k." And then I could get on with my life.

Interviewer: You can get pretty angry?

Participant: Yeah.

Interviewer: How angry is angry?

Participant: Well, they call me **volatile**.

Interviewer: Who calls you volatile?

Participant: A (Name), the guy. **He thinks I've got a raging fury**.

Interviewer: That's what he thinks. Do you think you do?

Participant: Um. I would say that I'm, **I've got a pretty bad temper.** But it doesn't um, it's not exposed very often at all.

Anger Episodes

Other participants were also able to describe discrete episodes of anger experience that were difficult to manage. The reporting of these experiences was important both theoretically and clinically because unlike earlier responses that were essentially historical, the reported vignette illustrates the contemporaneous nature of difficulties with anger regulation. It further illustrates the episodic nature of anger experiences and illustrates how anger episodes can be precipitated by specific events and thereafter maintained by other co-existing

factors. In the reported vignette, the participant describes a situation occurring a few days prior to the interview where she experienced a rapidly deteriorating mood state initially described as a 'rut'.

Participant : Oh just ah .. it's noth..., it's nothing bizarre, it's nothing that I wouldn't handle any other day. You know, things like computers broke down. Photocopier broke down. Um, I had disappointment and there was nobody there to help me. Um, and a lot of my day, well that's an exaggeration, couple of hours of my day was lost because of a technician who came in who gave me a new computer and um by the time he left my time was getting really pushed. But when he left, what he had installed didn't work. And it's a major part of my job. There's ah something that I really have to have done every single day because it affects the entire centre. And I couldn't get that done in time to be here. And that kind of just started it off.

Interviewer: What were you feeling at that point?

Participant: **I was really pissed off**.

Interviewer: You felt angry?

Participant: **Yeah**. I was getting really, well I was **anxious** I suppose because of the appointment. Um, I was also supposed to go out with a friend that night who hadn't contacted me all through the week and I had contacted her at work, left a message and that was in the morning and I hadn't heard back from her. And I think I was getting, by this stage, after being **annoyed** with the computer and then the photocopier, I still hadn't heard back from from this friend by four o'clock in the afternoon. And that started to **annoy** me. **Um, so I guess yeah, by the time I got home I was just a ..... I was just so, I was just ang, angry** would probably describe it best.

Interviewer: Can you say a little bit more about getting ... look my sense is that this is what happened. Events transpired, the computer, the photocopier whatever. You became ah, there was developing sense of being burdened I suspect.

Participant: Um, pushed for time.

Interviewer: O.K. Pushed for time. Then after that you became angry?

Participant: **I was getting angry at work**. As I was trying to resolve these little problems.

Interviewer: Right. O.K. Then you got home and you and you so you then left the office and on the way home you were feeling furious and then started to be distressed. That is, you started to cry. And then you went back to being angry?

Participant: Yeah.

Interviewer: Uh Huh. At what point did you start to feel angry again?

Participant: Um, probably it was the presence of this guy at home.

Interviewer: Oh, so you got angry when you got home, not on the way home.

Participant: **Well I was angry from the time I left the office** (I: Yes) um **and ..... I was still angry when I got home** but it was it was just like, it was chop and change. It was like I was ... **stopping myself from being in tears because I was just so full of rage** on one side, and then when I took a few minutes to sit down and see some, whatever I don't know. **I was just trying to take a few minutes out um, I was angry again.** You know, **and then I went from being angry** to to being just, yeah distressed or upset again. And I just rocked back and forth that entire afternoon. **(note evidence of affect lability)**

Interviewer: And when you got home, you got **angry** again?

Participant: Yeah.

Interviewer: Sounds like, my guess is that something provoked that. Would that be correct?

Participant: **I was already angry but I think it, ah, fuel to the fire was this chap being at home**. Um, it, I think it could have been anything, it just happened to be him. It could have been ... you know someone kicking a cat on the street It, it wouldn't have mattered.

Interviewer : What was it exactly though that happened?

Participant: Um, .. with the, with the guy at home? Well he didn't do anything towards me at all. Um, I just knew that he's married to one of my best friends and he's fifty years old and is treating her like shit. And he's an alcoholic and he's not helping himself with it and he's been, he's on school holidays so he'd been drinking and being really irresponsible and I knew that from the moment I walked in the door. **And that annoyed me**. But it could've, like I said even if he wasn't home and I happened to see something **that would nor.. normally just, you know, kind of piss me off a bit, um that would've sparked me off.** I knew it would have because I was a bit, I was feeling so vulnerable to that, to anything um ..... it was ... I don't know, it was just so overwhelming I I don't

know what happened. You know I woke up the next morning I was fine. But it was like my whole se.. sort of day from like three o'clock that afternoon till seven ... about seven o'clock or six o'clock ..

Interviewer: And if you think about that four hour period, how would you characterise it?

Participant: The period or, or how I felt?

Interviewer: Well, how you felt.

Participant: **I was furious.** I was, ... **I didn't know if I'd be able to contain my, my anger**. It was .. **I was seething**. It was just so .....

Interviewer: What got you out of it?

Participant: This guy running into to, well, the stupid thing was that he came into my bedroom and said "How are you?" And I said "I'm having a really bad day." And he said "You think you're having a bad day. Someone's just run into my car." And when I looked at it, um, I mean as soon as I walked out of the room it was like, there was something that's just diverting my attention.

Interviewer: So it was a distraction type of thing?

Participant: Yeah. And it kind of calmed me down. I, I was ... I was still **annoyed** because in my assessment of the damage, I'm not a professional by any means but um I couldn't see how any other car could have done what he had shown me and what he had told me. I believe that he had actually driven it off into a corner of the wall himself. And that, Yeah, I guess after that I started to calm down a bit ... but um I was still, by his stage I guess I was feeling a bit **anxious**. I was like, I just started, um the things I was, the thoughts that I was thinking um was kind of like rev, reverting back to how **scared** I was about wh, where my life's going, what I'm doing and this, that and everything else. You know, a few months back and just for that short period of time in the house that's all I can think, that's all I could think of. I was back into this hole that I was in before. I didn't know how to get myself out of it. But my attention was diverted by this guy's um accident.

Interviewer: What you're describing almost is a sense of falling into an emotional hole and not being able to get yourself out.

Participant: Yeah.

Of significance, the participant reports a phenomenon often noted in the clinical literature which is associated with a sense of internal 'psychological collapse' (Kernberg, 1984; Linehan, 1993). It is thought to be associated with the inability to regulate overwhelming affective states. In the reported case the affective state is that of anger. It appears that when such an episode occurs, the participant utilises a series of psychological and behavioural responses that appear to serve the purpose of altering or modifying the uncontrollable nature of the original anger episode. This appears to be a common phenomenon in BPD.

This section has outlined a range of experiences across the participant sample confirming the common, problematic experience of anger and the difficulties associated with its management. There appears to be sufficient and wide-ranging evidence of the experience of anger as a discrete affect to justify this as one category for inclusion in an 'Emotional Stroop' paradigm.

5.7.2. Distress-Anguish (Sadness)

A second commonly reported affective experience was that of distress-anguish which is typically referred to as 'sadness'. The experience of sadness was similar to the experience of anger in that the origins of each reported episode of sadness varied significantly. They included reports associated with a sense of hopelessness or futility in life, or unresolved issues from earlier periods of life. This section reports a variety of themes related to participants' experience of sadness, and the experience and manner by which participants regulated states of sadness/depression.

Identification of Experiences of Sadness

As a discrete affect, some participants were able to independently identify and articulate sadness experiences. One example of this is as follows:

Interviewer: Can I just ask you some broader questions just for a moment? Just so that I can get some things clear in my mind? You came to see me because there were difficulties with how life was going, that's fair enough. If I were to ask you to think about your emotional life, what feelings do you have that you find difficult to cope with, that are difficult to manage? What emotions would they be?

Participant: Anger. Angry at myself and I just keep going. **Sadness** and sometimes I cry, you know.

This vignette again confirms the participant's experience of anger but also independently confirms the experience of sadness. Sadness was identified in this vignette as an independent affective experience that was nominated by the participant without prompting by the interviewer.

A number of participants reported sadness experiences associated with dysfunctional intimate relationships. As an example, one participant described the experience of anger, sadness, and despair in relation to her husband. The participant reported that she experienced her relationship with her husband as one where she felt that they were chronically empathically misattuned. In response to a specific relational misattunement, the following interchange occurred in which the participant described the processing of a recent suicide attempt:

Interviewer: What led up to you deciding to kill yourself?

Participant: We (husband and the research participant) were arguing umm...we were always arguing and disagreeing but it was the 1st of February and the kids were going back to school. Now he (husband) knew that I had to get the oldest one (oldest daughter - L) to her friends place M (friend of daughter) to get both of them to the high school by what time was it, by quarter by eight o'clock. They had to be in there by eight o'clock which meant that I would then get E (youngest daughter) to school just after 8 o'clock and get her into class by about quarter past eight to see her friends and stuff like that. He (husband) was awake because D (husband) never gets out of bed before ten or eleven o'clock. He works at night but even on the nights - he's a DJ - but even on the nights he doesn't work like Monday night, Tuesday night, Wednesday night, Thursday night he will still stay up drink his Scotch, drink his port, drink his beer, have his chocolate, have his cheese, watch videos, and sleep. Like go to bed at two o'clock in the morning and sleep and I have always said that if you work and you want to sleep in that's fine because you've got to look after your voice and look after your health but when you're not working you don't have to stay up that late and you can get up and help me with the kids and he never did, not until after February. That's when it all started when he wanted to be so helpful but he would umm, he knew that E (youngest daughter) was going to school and he promised E (daughter) that he would be there for her first day. Anyway it was five past eight and he still wasn't out of bed and I thought 'I'm late. I've got to get L (oldest daughter) to M's' (L's friend). So we quickly ran out got in the car and drove off to school. I've got M (Oldest daughter L's friend) got L (oldest daughter) organised, took E (youngest daughter) to primary school and was sitting there and E (youngest daughter) says can I ring daddy and I said 'Yep fine.' So she rings up and he goes can I talk to your mother - 'Yes. Why didn't you get me up. You knew I was awake. Why didn't you just come and get me?' And I'm thinking 'What for?' If you were awake and you promised your daughter that you were going to go to the school get up, get organised, and let's go. I'm busy I've got things to do...so it was on, on the phone.....So next thing he drives down we both go in and get E (youngest daughter) settled. As we're coming out the argument's on again. 'Why didn't you get me out of

bed. You knew that I wanted to be with E (youngest daughter)' da, da, da. 'Are you in control of your own life? Do you need me to get you out of bed? Do you need me to organise you when I've got two kids to organise and myself and you want me to organise you?' And I thought 'I just I'm not going home.' I thought 'I'm not going home. I'm going shopping.' And I thought I'd go food shopping because I thought if I go home it'll be on for the next two or three hours and it's just full on about how I'm not thinking right or I'm irrational ummm, I'm abusive, umm I'm disruptive. I'm, God there are so many words that he used. Well anyway, so I took off and I went shopping and I was getting stuff in the trolley and I was just **depressed**, like really **pissed off** thinking what am I going to do?

Interviewer: So you were angry and **depressed**?

Participant: Yes.

A second example of the independent reporting of sadness was elicited from another participant who described the recent break-up of a long-term intimate relationship.

Interviewer: I take it that the decision to do that was not your decision. It was hers. So, does that mean that you were an unwilling ...

Participant: No, I agreed to it. **You know it's just sad**. There's no guarantees either.

Interviewer: Is that what you're feeling today?

Participant: Um, sort of been feeling like that (sad) for most of the week. Trying to focus on getting my life together. That keeps my mind a bit more occupied.

Sadness Regulation

A number of participants also reported a variety of difficulties with regard to the regulation of sad or depressive states. As an example, one participant described the combined difficulty of the identification of depressive states, and the use of 'behavioural enactments' to regulate internal states of

depressive pain. In this vignette, the participant had already independently identified the experiences of both anger and sadness. At this point in the transcript, the interviewer directed the participant's attention to the experience of sadness as it has already been independently identified by the participant.

Interviewer: Okay. What about when you're **sad**, do you know when you're feeling **sad**?

Participant: **I just feel, feel down, feel depressed**... I lose my appetite you know. And I do silly things.

Interviewer: Silly things?

Participant: Yeah.

Interviewer: What sort of things would they be?

Participant: Always being close to someone, following them around all the time.

Interviewer: Can you say some more about that?

Participant: It's a bit of a trust sort of thing as well.

Interviewer: Trust?

Participant: Yeah. It's the way life's going to be with someone I like all the time.

Interviewer: That's just when you're feeling sad?

Participant: Yeah.

Interviewer: What makes you sad?

Participant: When I think about mum and I don't get what I want. Just to into a spiral in fact I just go downhill.

Another participant also identified a cyclical pattern to her experience of sadness. This is significant because it provides support for the often reported phenomenon of 'rapid cycling' of affect and provides narrative support for the proposition that affect regulation is a central deficit in BPD.

Interviewer: Fluctuation? That sounds like it's a pretty constant experience in your life.

Participant: Yeah. I don't think ah ... a week goes by without a significant low part. But I don't know if that's normal or not. So I mean I'm sure everyone has their downs. **It's just that my downs just seem that it's the end of the world**.

Interviewer: Yes, and that happens quite a lot.

Participant: Yeah.

This section has outlined a range of experiences across the participant sample confirming the common, problematic experience of sadness and the difficulties associated with its management. There appears to be sufficient and wide-ranging evidence of the experience of sadness as a discrete affect to justify this as one category for inclusion in an 'Emotional Stroop' paradigm.

5.7.3. Shame-Humiliation (Shame)

A third affective experience reported by all participants was the experience of Shame-Humiliation. Unlike the experiences of anger or sadness which often appeared to be linked to participants' reports of the actions of others, the experience of shame-humiliation appeared related to adverse judgements with regard to their own behaviour. Alternately, shame-humiliation experiences were related to a more fundamental state of 'existential badness.' Participants' reporting of shame-based material was more difficult to elicit, and typically required the use of a greater number of probe questions in order to elicit recognition of shame experiences.

Experiences of Shame

In the first two examples, the participant's experience of shame-based material is elicited in relation to reports of episodes of suicidal ideation. In the

first example this was precipitated through an unwanted pregnancy and the conflict associated with terminating the pregnancy. In the second example, it was initiated through the termination of an intimate relationship.

First Example

Interviewer: Is that the first time in your life you've ever thought about doing it? (Attempting suicide).

Participant: No.

Interviewer: You've thought about it before?

Participant: Yep. Yeah I have but that was the first time that I actually did it.

Interviewer: How old were you the first time that you thought about taking your own life?

Participant: Umm..just after I'd met D (husband). I was umm 21 going on 22. And when I was pregnant, I got pregnant to D (husband) and he insisted that I get the abortion. That was really, that shook me. I'm still, I still think about that because I didn't want to do that. That's, that's not my way you know. And the doctor that we saw at the time said to him 'If you make her do this she'll never forgive you and she'll never forget it'. You know and I don't. Every June I remember, you know...though I do I resent him he keeps on telling me I have to forgive him for all this stuff. I just can't, you know, I can't. I'm living a lie. I'm living a lie umm....

Interviewer: I get the sense that you feel some regret but I also get the sense that you **feel bad** about having had the abortion as well?

Participant: Yes I do.

Interviewer: **Do you feel ashamed about it?**

Participant: Oh God, yes. You know, my mother. I kept it from my mum and dad. I mean if you're brought up in a family and they keep on saying to you 'don't ever get pregnant before you're married'. And my dad said 'if you ever get pregnant before you get married you are not welcome in this house'. And then you've got your mother saying to you that if you get pregnant I'll kick your father out and I'll look after you. It's like 'where am I? Am I welcome in this house? You know?

Second Example

Participant: Um, ... I remember having the thought that I didn't want to be around any more. Because I'm having to face what I did, which is, having survived it it's a pretty **embarrassing** thing to ever have to explain to anybody if the situation has happened. Um, or hoping the hell that some people just don't find out about it. It's like a **dark part** of my, my past now I don't, I don't like. So um, ..

Interviewer: Do you mean like feeling **ashamed** of it?

Participant: Do I?

Interviewer: Feel ashamed about it?

Participant: **I feel ashamed because I'm still here to talk about it**.

Interviewer: So you feel ashamed that it, that it was a, a gesture or an attempt that failed? Or were you ashamed that you tried in the first place?

Participant: No, um, I'm **ashamed** that it got as much attention as it did. Which makes me look pretty stupid because when you .... um I haven't had to, except for maybe once since it happened, actually had to turn around to somebody and say "Well this is what happened to me." You know, you feel, it's it sounds so stupid because you're still here. You obviously didn't do it well enough and you hadn't, it was like did you just want attention or something? And it's like I fucked it up. You know, if I had been a bit more gutsy about it and if I didn't feel like such a bloody wimp, I wouldn't have to put up with the rest of it. So the thought that's that did come into my head some time ago was "do the same thing again but jump into my car and drive to the Nullabor or some place like that." Where I wouldn't have, leave my phone, leave all communication things away, I wouldn't have that option. And if I decided not to, no-one would know. It would just be my, my test. No-one else has to know about it. If I succ, If I succeeded well then good on me, if I didn't then it's it's my private um deal. Nobody else has to be a part of that. That's what I feel, pissed off about it. I said that to ... hospital doctor that I'm probably going to have to deal now with more shit now than I've ever had to deal with before because I stuffed up. But I'm not in that frame of mind right now. So .. I'm not um angry or anything, I'm just maybe like you said a bit small....

In the following vignette, the participant had been describing her concerns regarding the compulsive use of alcohol, food, and tobacco. Her concerns up until this point in the transcript had implied that she had felt that she had transgressed a personal rule regarding the use of these substances. The implied nature of the participant's 'moral code' prompted the question posed by the interviewer.

Interviewer: Would you describe them as bad things you're doing?

Participant: It's over, it's over the top. Way too much um smoking, way too much eating, and way too much, I mean, as you can see, I mean, you know, what I mean and it's all way over the top.

Interviewer: So it gets fed by you being bad? (The use of the term 'it' was an adjective first introduced by the participant. This referred to an experience of a primitive affective state that was characterised by an insatiable form of 'hunger').

Participant: I suppose, yeah that's what happens.

Interviewer: Is it not, I don't want to put words into your mouth nor your mind. Does that seem correct?

Participant: It's just out of control at the moment because I'm out of control completely. And it's sort of like when I'm out of control it demands more and it becomes it's like being on a rollercoaster ride. When I used to think about it when I was into drugs as well cos, because I used to drop a lot of pills. And that some drink, and it was such a rollercoaster. And it was like being on a rollercoaster and you get spat out the other end, but the binge would last for a week or whatever. And you get spat out the other end and be told 'Get your shit together' and have a couple of days where you're just like getting your shit together and you feel calm and then you get this call and I mean start all over again. And you're back on the rollercoaster and it's really like that it's like that and a whole lot it's like being like that with everything you know, it's sort of like not just with drugs and alcohol. And, I mean, I used to be a compulsive, umm masturbator, I used to masturbate all the time. Umm, I'm starting to do that again, umm, and that worries me because I

don't - the reason for me to be doing that it gives me a great deal of comfort though. And I don't understand.

Interviewer: Is that what it's about though?

Participant: No. I've always I've always been a masturbator even when I was little. I think it was because it gave me, I could concentrate on one part of my body and enjoy it, ..it used to give me a great, it gives me a great deal of **guilt** as well. I mean it's from one extreme to the other you know you have this moment of pleasure and it's lovely and then you have this, this kind of **guilt** and shame and the whole trip you know.....and.....it's like that like that saying sleeping dogs lie let sleeping dogs lie..it's like that..you can see it all and don't touch it or anything and all of a sudden it just overruns me and I want to do something to appease it and so I do. And then **it makes me feel guilty** and **I feel ashamed** and then the whole thing starts again but it's the same it just goes round and round and round.

Interviewer: So it sounds to me like you're talking about a series of compulsive behaviours. What you've described as compulsively hopeless, which you feel in fact are bad things to do but then doing these bad things gives you pleasure even briefly or temporarily. So it sounds like you feel very caught. These things give pleasure but they also make **you feel guilty and ashamed afterwards**.

Participant: **Yeah. Yeah they do** and you don't know how to stop. It's like you don't know what to do to stop and I'm afraid that if I stop everything..like stopping eating and drinking and smoking and all this I don't know what's going to happen.

In the following vignette, the participant has been describing the conflict, which occurs between herself and her family. She reports the experience of engaging in an internal review of conflict between herself and her family, and this results in a process of self critique, which ultimately results in the experience of shame.

Participant: I've no idea really. Umm...there's nothing that I've tried to think of why these sorts of things happen and there's no real explanation that I can come up with that I can find

as to why it's like that. But what I find is difficult is the fact that I can't communicate with my own family even, I've either just got to let them do what they want to do as soon as I try to be assertive it just turns into a firing match and then you know I don't sleep good at night and I lay there and I'll all the things just come bashing into my mind and they're always really negative things there's never any positive things there's always all the things I've done and what I've done to hurt people and and all the mistakes I've made all this comes rushing into my...

Interviewer: So you become very critical of yourself?

Participant: Very critical, yes and I mean there's often times that I'll just be crying and I wake my husband up oh I try not to wake him but you know I just because of all this it's just moot **despair** I suppose because when I have discussions with my children for instance that don't go right ummm..and I get angry..umm..I then sort of think back on that and think well look I could've handled it better and I could've done this and I could've done that I mean I just I just seem to to I can only pinpoint all the negative things in my own behaviour I never can find anything of that's positive.

Interviewer: So you feel very critical of yourself?

Participant: Mmmmm.

Interviewer: I get a sense that there's a lot of shame within you.

Participant**: Shame and guilt. Guilt**.

Interviewer: About your own behaviour?

Participant: Hmmmm.

Interviewer: Is that an old feeling - one that you've had for a long time?

Participant: Yes.

This material illustrates the relationship between the employment of compulsive behaviour to serve an experienced need which in turn results in the experience of shame because of the use of the behaviour in question.

This section has outlined a range of experiences across the participant sample confirming the common, problematic experience of shame and the

difficulties associated with its management. There appears to be sufficient and wide-ranging evidence of the experience of shame-humiliation as a discrete affect to justify this as one category for inclusion in an 'Emotional Stroop' paradigm.

5.7.4. Fear-Terror (Anxiety)

A fourth affective experience reported by all participants was that of Fear-Terror or anxiety. The experience of anxiety often appeared to be associated with either the anticipated loss of a relationship or alternatively, the experience of some form of traumatic and abusive episode perpetrated upon the participant by another person. In summary, anxiety experiences were reported as artifacts of the loss of important relationships, or the experience of being in an abusive relationship, or of being in a relationship where some form of misuse of the participant occurred.

The Experience of Anxiety

The following vignettes report examples of both types of experience reported previously. In the first example, the respondent links anxious experience with somatic or bodily representations. The material leading up to the report outlined below saw the respondent describe various interpersonal experiences as representing a form of being 'ripped apart'. Upon further inquiry, the respondent ultimately reported the experience of anxiety. In the second example, the participant reports an episodic memory of sexual abuse committed by an older cousin. What is salient about this episode, is the combination of partial amnesia for the event with a clear recognition of the experience of fear in relation to another person.

Vignette One

Interviewer: You've used this metaphor today a couple of times and it's quote 'ripping me apart', which I'm not arguing with this, just it's a very dramatic metaphor. Can you say a bit more about feeling 'ripped apart'?

Participant: Well before the, before the um before I ended up here (in hospital), this guy moved into the house and prior to him moving into the house S (ex-partner) and I had a few background differences over what is, I don't know, what is courteous or respectful or whatever. Maybe it's social etiquette. We had a few differences there, but she was fine in me letting her know, just because she had never, it had never been brought to her attention before. So we worked out a lot our relationship things about me that she didn't particularly like or was annoyed about. We, you know, we talked over a lot of stuff then she went down south, this guy moved in with me, and we had this three way relationship going. And as soon as he moved back in um I was having a, it was at the stage where I was having a lot of hassles at work, um, which didn't help but um when S(ex-partner) did arrive it was like everything that we worked on went out the window. And it would be things like well you know she's been out on a work do with this guy down south, it's really bad weather, it's pissing down with rain, I'm on my own in Perth, and she said to me "Oh well, I will call you" at whatever time. And it's like "o.k." And she wouldn't ring and then I find out they've been to the work do, um in which they would have both been drinking at, in a country town, driving on pretty bad roads, getting home and her having enough consciousness to um go and get herself a coffee or whatever, walk past the telephone probably about five times, sit herself down on the lounge, get the television on, have a glass of wine - whatever, and um pass out. So I'm still you know, by this stage it's like it's ten thirty, eleven o'clock, I'm thinking "shit, have they crashed," I mean she's been pulled up by the cops so many times like what the hells happened? And so by the time I get to the phone I'm like really **anxious**, I'm hoping that they're o.k., but at the same time I'm furious. By the time I get through it's like "Yep, yep, yeah she's here. She's asleep on the lounge." It's like why didn't you ring? "Oh I fell asleep, I was tired, I was this and I was that." "it's like "Yeah, but haven't you already done da, da, da." "Yeah, I did all that when I got home." It's like "Why couldn't

you just give me a call just to let me know that you're o.k." And then I could get on with my life.

Vignette Two

Interviewer: O.K. Well we'll see what we can do. Now, which of these do you want to go into first?

Participant: I don't mind. They're all valid to me.

Interviewer: What would your preference be?

Participant: I guess for me would be the (city in Australia) event which happened with the cousin.

Interviewer: Can you tell me a little about it?

Participant: What I can remember of it. Um, I was in his care um and I think he was still a teenager himself.

Interviewer: You would have been how old?

Participant: Eleven. I don't really know how old he is, but he seemed really old. Um from an eleven year olds point of view I guess he looked, he could've been anywhere from sixteen or, I don't know he just you know he seemed fully grown and mature. Um, I just remember going, him asking me to a room um and it was a big room with two single beds. Um, I can't remember if it was a room I used to sleep in or not. Um, but I watched, I remember watching my cousin who's the same age as me have sex on the bed, on the other single bed. Like the day before with ... the guy next door. And she was the same age as me. And he was the same age as my cousin. Um, and that, I didn't really know what was happening there. But I was glad it wasn't me. Um, and then the next day I was to, he, he, I was in his care and if it is this guy um and he asked me to lie down in bed with him which normally I wouldn't have a problem with because I'm an only child and any sort of family closeness I was apa, about. I didn't ah you know, I like enjoyed being close to family. So I didn't see that that as an issue at first. Um, I just remember him making me lie next to him and "Oh yeah, o.k. fine," and then oh ... oh I don't know if he made me kiss him or not. I, I don't remember anything like that. But then um undoing his pants and making me put my hand down his pants and play with him and I didn't want to do it and he kept, he kept my arm in a lock. I wouldn't do anything. I jumped

out of the bed and ran to the door. Um, then he, I got to the door, he got there pretty much the same time I did, locked it and had his hand above me. So he would've been a foot taller or something I guess. So I just saw his arm there and um he said I wasn't allowed to leave. I had to go back and do whatever he said. And, but when I think about it, I look up and there's a face but it's blacked out. And that's where it ends. **But I know something happened, 'cause I know I was scared shitless each time I went** 'cause we used to go to (City in Australia) quite often. Like every few years or so. I knew each time I met him I was, **I was scared of him**.

Quality and Intensity of Anxious Experience

Another participant reported the experience of primitive terror-like states associated with what has been referred to elsewhere as 'nameless dread' (Ogden, 1989) or the experience of the 'black hole' (Grotstein, 1990). In the following vignette, the participant described the difficulty she experienced in identifying affective states within herself.

Participant: A lot of the time I don't feel anything.

Interviewer: Uh Huh.

Participant: I don't feel anything.

Interviewer: Do you mean you feel nothing, or you don't know what you are feeling and the possibility exists you are feeling something but don't know it.

Participant: Well both I suppose. I, I sometimes I think 'How do you (I) feel right now'? And I think 'I don't know'. A lot of the time I feel empty. When, **when I'm scared**, **when I was scared the other day** when I was on the boat, that's horrible. That's a horrible feeling. It's like um, like that black thing you know, the hole and....

Interviewer: The black hole?

Participant: Mmm, it's horrible. Makes me feel sick. I want to throw up. It's really um, I, I'm paranoid, I feel paranoid. I want to run away. Um, the other day, a couple of days ago I felt like that. I couldn't understand why I was feeling like that. I just felt like I wanted to

scream or something. I felt like I was wound up inside. And I wanted something to make it snap and I couldn't understand why I was feeling like that. And I said to G (Partner) 'I'm feeling really wound up'. He said to me 'Why'? And I said 'I don't know'. But I really did. I felt like, I feel like I do now. I feel really sort of tense inside. Really wound up and .... I'm not angry, **I'm just scared. I just feel scared**. **Really paranoid**. I feel really ...., and I don't know why. I don't know why I feel like that. I've been feeling like this since the weekend.

In the following example, the participant describes the use of a behavioural enactment (cutting) in order to manipulate an affective state. The participant describes the use of the self harm gesture as a means by which she was able to shift from one affective state to another.

Participant: ..... But it wasn't until ..... I remember doing it (cutting) and thinking it was just to really snap me out of it.

Interviewer: Can you tell me about that please?

Participant: It was kind of like to nudge me back into reality as to what I was really doing. And if it hurt, then I'd stop. Or if I saw .. then one morning I woke up and my sheets were covered in blood. And I was living at home still, **and that really scared me**. And I realised what I had done. It kind of like ..

Interviewer: So, what you're saying is that you would use other emotions to stop particular emotions.

Participant: Oh yeah. (Spoken in a 'contemptuous' tone which implied that the interpretation of the interviewer was so obvious that it was superfluous to state it).

Interviewer: So you would use cutting as a way of invoking fear as a way of stopping yourself from feeling increasingly angry.

Participant: Yeah. It was a diversion.

In the following vignette, the participant describes a sense of developing anxiety associated with an addictive state. The participant reports that the only way that this state can be sated is to 'feed it' alcohol and food.

Interviewer: You've just mentioned, that feeling. Can you say what that feeling was like?

Participant: It's a real driving...force it's like umm...

Interviewer: Is it the feeling to do with the compulsion? (The participant had previously described this compulsion)

Participant: Yeah, it's like a.., you know how you know how heroin addicts have a monkey on their back well it's sort of like that. It, it, it, you know, becomes out of control but the trouble is that if you feed it, it gets bigger and bigger and bigger, do you know what I mean? Like it's out of control and so it's really hard to find a umm.., you must think I'm insane This sounds so insane talking about this umm it's like umm.....,you've got to constantly be on top of watching what's happening all the time you've got to be in control of this, this thing whatever it is.

Interviewer: Can you describe this 'thing'? I mean I'm not doubting you, it's really important I think to get as much detail in the description of this 'thing' as possible.

Participant: I don't know.., it's umm.....as long as I keep feeding it alcohol at the moment and food and smoking it's appeasing it....but when I stop...I get this feeling of ummm...overwhelming urge to do something.., to **fear**.

Interviewer: Like what?

Participant: It doesn't matter what um it's like. I don't know, like a real umm sometimes it has a voice.....it's strange it's just this real urge to want it, it overwhelms me sometimes and it's hungry and it wants to be.. well that's about the only way I can explain it.., it's hungry, it wants to be fed and it wants to be fed and it wants to be fed now. **So then I get really anxious and upset about it** and try and figure out someway of appeasing it so that it doesn't come out and destroy my life because that's how I feel.

This section has outlined a range of experiences across the participant sample confirming the common, problematic experience of anxiety and the difficulties associated with its management. There appears to be sufficient and wide-ranging evidence of the experience of anxiety as a discrete affect to justify this as one category for inclusion in an 'Emotional Stroop' paradigm.

5.8. SUMMARY AND CONCLUSIONS

The outcome of this phase of the research was that a total of four affect constructs were reliably reported by all 11 participants. These affects were: Anger-Rage, Distress-Anguish (Sadness), Shame-Humiliation, and Fear-Terror. These constructs were then employed in a subsidiary study in order to elicit specific word representations of each of these generic constructs.

One of the significant findings from this study was the identification of 'affect blends.' This phenomenon refers to experiences reported by a number of participants of multiple, co-occurring negative affects linked to specific experiences. This finding suggests that borderlines might experience a general affect regulatory impairment in contrast to difficulties regulating specific, discrete affects. If this is the case, so-called affect-regulatory deficits in BPD might be more associated with arousal based phenomenon that represent overlearned responses to the social environment, a response to affective phenomenon which signal threat or adversity in the interpersonal domain, or finally, convey significant information with regard to self-referential negativity.

CHAPTER SIX: AFFECT CATEGORY JUDGEMENT TASK

6.1. OVERVIEW

The objective of the current study was to develop word lists for each of the previously nominated affect categories for inclusion in an 'Emotional Stroop' paradigm. Chapter Five identified the categories of Anger-Rage, Distress-Anguish (Sadness), Fear-Terror, and Shame-Humiliation, as the salient negative affective states reported by borderline participants.

In addition to the previously reported affect categories, it was decided to include affect categories reflecting Neutral and Joyful affective experiences. Neutral words were included as a result of the recommendations of Williams & Broadbent (1986) who argue that it is also necessary to analyse 'interference effects' in Stroop tasks by subtracting the value of neutrally valenced words from affectively laden words in order to calculate the amount of interference experienced in the affective conditions of the task. A Joy-word category was also included as a result of K. F. Stein's (1996) study in which she observed 'rapid cycling' of affective experience in borderlines from highly dysphoric to positively toned experience which occurred in relatively brief time periods.

In addition, if BPD is associated with affect dysregulation, then the inclusion of neutral or positive categories of affect should more adequately test this hypothesis. It was hypothesised that borderline participants would return delayed Stroop responses for so-called negative affective states (Anger, Sadness, Anxiety, & Shame), but positive and neutrally valenced affective words would yield comparatively faster rates of response relative to negative words. Two reasons are posited for slower rates of response on the Stroop for negatively valenced words. First, some cognitive theories of BPD (i.e., DBT) emphasize the

importance of dysphoric affect (Linehan, 1993), and this should be reflected in delayed colour-naming response latencies on negatively-valenced word stimuli. Second, some clinical studies have returned speeded colour-naming response latencies for neutral stimuli relative to negatively-laden word stimuli (J. M. G. Williams & Broadbent, 1986). For these reasons, it was decided to incorporate word lists that sampled a range of negative, neutral, and positive affects.

An instrument known as the 'Affect Category Judgement Task' (ACJT) was developed in order to specify and create word lists which reflected the affect categories of Anger, Sadness, Anxiety, Shame, Neutral, and Joy. The development of this instrument is outlined in Section 6.2.

6.2. DEVELOPMENT OF THE AFFECT CATEGORY JUDGEMENT TASK (ACJT)

The ACJT was derived from the Dictionary of Affect in Language (DAL) (Sweeney & Whissell, 1984; Whissell, 1989). The dictionary was developed as a result of the compilation of words employed by various experimenters (Conte & Plutchik, 1981; J. A. Russell, 1980; Whissell, 1981) and common English language words with known or acknowledged affectively laden content. The DAL contains over 4,000 English language words that have been rated for affectivity according to two orthogonal dimensions. These are referred to as 'Evaluation' (Pleasantness) and 'Activation' (Arousal). Both the Evaluation and the Activation scales rated each word on a Likert-type rating scale ranging from zero to seven, with a mean of four and a standard deviation of one. Because of the complexity of this task, words were rated according to their Evaluation score only. Therefore, a word rated with an Evaluation score of 1.000 would have been judged to have a pleasantness rating significantly lower than a word rated with an

Evaluation score of 6.000. All of the words included in the DAL were utilised in the development of the ACJT.

The ACJT consisted of a booklet which contained all of the words in the DAL. Listed next to each word was an array of six boxes which corresponded to six categories of affect to be employed in the Stroop study. A sample of the task is contained in Appendix XI. The shortened terms of Anger, Sadness, Fear, Shame, Neutral, and Joy were employed rather than Tomkins' hyphenated affect terms. This was done in order to simplify the task for judges. Participants were instructed to allocate each word to one or more of the previously identified categories. This was done by endorsing one or more of the corresponding boxes linked to a specific word. A guide to rating was included in the introduction to this task. This guide is included as part of Appendix XI.

DAL Evaluation ratings were then examined for each endorsed word in the categories of Anger, Sadness, Shame, Anxiety, Joy, and Neutral. Evaluation ratings were employed in contradistinction to Arousal ratings for two principal reasons:

1. Whilst there is evidence suggesting that the arousal component of affective experience is an important feature, contemporary theories of BPD emphasize the dysphoric component of affect (Linehan, 1993). The dysphoria associated with affect dysregulation in BPD appears to be more closely associated with the Evaluation (Pleasantness) dimension as opposed to Arousal ratings of affect. The nature of the affect regulatory difficulties outlined in Chapter Two and confirmed in Chapter Five suggests that borderlines have difficulty with the identification and articulation of affective experience. Linehan (1993)

emphasizes the importance of deficits in the identification of dysphoric affect in the genesis and maintenance of BPD.

2. The DAL Evaluation ratings appeared to be more broadly distributed than were Arousal ratings. The words selected for each category appeared to return Arousal ratings closer to the theoretical mean (4.0) for the DAL. This suggested the possibility of greater difficulty for participants in discrimination judgements of Arousal. Because it appeared possible to select an array of words with more extreme Pleasantness ratings, these ratings were adopted.

The words allocated to each of the 'negative' affect categories (Anger, Sadness, Shame, Anxiety) with Evaluation ratings from the DAL of a minimum of two standard deviations below the mean were sampled for inclusion as stimulus words. This meant that words were considered for inclusion in each affect category if they achieved an Evaluation rating of between One and Two. In the case of the affect category of Joy, it was decided that words with Evaluation ratings a minimum of two standard deviations above the mean would be sampled for inclusion. This meant that words were considered for inclusion in the affect category of Joy if they achieved an Evaluation rating of between Six and Seven. The selection of words for inclusion in the Neutral category was completed by listing all words with Evaluation ratings between 3.9 and 4.1. This list was then reduced by the selection of words receiving ratings as close as possible to 4.000 as possible.

It was also further determined that if two words of a similar root were included in a category, then the word with the more extreme Evaluation rating would be selected for inclusion. This decision was made in order to satisfy the

requirement that all words included within a category would differ from one another.

At the completion of this aspect of the task, word lists were generated for the affect categories of Anger, Sadness, Shame, Anxiety, Neutral, and Joy. For the categories of Shame, Anger, Anxiety, and Sadness, only words with arousal ratings of less than two were selected for inclusion in the task. For the Joy category, words with arousal ratings greater than six were selected for inclusion in the task. Words in the Neutral category had Evaluation ratings between 3.9 and 4.1.

When lists of words specific to each affect category had been selected, two Speech Pathologists reviewed each of the word lists in order to provide a face validity check of word by affect categories (Appendix XII). Those words not achieving a 100% consensus rate by these expert judges were eliminated from further analysis. The included lists of words for each category achieved a 100% endorsement by the Speech Pathologist judges. The 10 words within each list which returned the most extreme ratings in the case of the 'non-neutral' categories, and the words with ratings closest to 4.0 in the case of the Neutral category were then included in the Stroop task.

6.3. PARTICIPANTS

The participants included in this study consisted of five health professionals who served as expert judges. These participants were all clinical professionals who were practicing in counselling/psychotherapy roles at the time of the study. Their ages, professional discipline, and years of experience are included in Table 6.1.

Table 6.1: Age, Gender, Professional Discipline, and Years of Professional Experience for the Expert Judge Group

JUDGE	AGE	PROFESSIONAL DISCIPLINE	YEARS OF EXPERIENCE
1.	48	Nursing	29
2.	26	Clinical Psychologist	1
3.	43	Clinical Psychologist	1
4.	51	Social Work	25
5.	45	Nursing	25

6.4. PROCEDURE

Allocation of Words to Specific Affect Categories

The task for the judges was to allocate each word to at least one of the six affect categories. Each judge was instructed to rate each word by endorsing each affect category box the word corresponded to. Thus, each word could potentially be associated with a minimum of one category, or a maximum of six categories of affect. At the completion of the rating task by the judges, each word was assessed according to two rules:

1. The words to be considered for inclusion in the Stroop Task could only be allocated to one category. Those words endorsed in two or more categories were immediately eliminated from further analysis, and;
2. For a word to be included in a particular category, it had to achieve 100% endorsement from the judging group. In other words, all judges must have

allocated the word to the same affect category. If the particular word did not achieve 100% consensus, it was eliminated from further analysis.

6.5. RESULTS

The task described in Chapter Six yielded a list of words for each affect category for which there was 100% endorsement. These lists were not included as they were too extensive to be included. Table 6.2 lists words considered for inclusion from this original list that were eliminated by the Speech Pathologist Judges.

Table 6.2: Words by Affect Category Eliminated By Validity Judgement of Speech Pathologist Judges

NEUTRAL	ANGER	SAD
COPE	TREACHEROUS	EVILS
FOCUS	ALIENATES	EVILLY
CRACKED	TORMENTING	SUFFERS
CONQUER		BURNED
MALIGNANCIES		SUFFER
CUNNINGLY		BLEEDING
BEAMING		
DEVOUTLY		
TRANSFORMATIONS		
REPUTATIONS		
ANXIETY	**SHAME**	**JOY**
CHAOS		EXCELLENT
ARMAMENT		KISS
DEATHLY		KISSED
		CHARM

Table 6.3 lists the words included in the Stroop Task as rated by the Speech Pathologist Judges.

Table 6.3: Affect Category Words Included in Final Stroop Task

NEUTRAL	ANGER	SAD
NUMBERS	HOSTILE	LONELINESS
MESSAGES	BASTARD	WRETCHEDLY
COURSE	ATTACKS	DESPAIR
DIPPED	BITTERNESS	LETDOWN
COMPARING	IRRITATES	DEPRESSING
MEDICINE	QUARREL	BEREAVES
MONOPOLY	FIGHTING	DROWNED
ATTENDING	HATEFUL	FUNERAL
MACHINE	ANGERING	GRIMNESS
JOYSTICK	ANNOYING	GRIEVING
ANXIETY	**SHAME**	**JOY**
TERRIFIES	BELITTLES	DELIGHTED
STRESS	GUILTY	HAPPINESS
PHOBIA	DISGUSTING	INSPIRE
SCARED	WICKEDNESS	ENJOYMENT
CHAOTIC	PUNISHES	FRIEND
ANXIETY	DISGRACING	JOYFUL
BEWILDERED	FORBIDDING	PLAYING
EERINESS	SHOPLIFT	ROMANTIC
FRIGHTS	REMORSEFUL	GOODWILL
ALARMED	ASHAMED	EXCITING

Stroop Word List Frequency Analysis

Once the final word lists for each Stroop affect category had been determined, an analysis of word-lengths was undertaken in order to ensure that words in each category were of similar length. This analysis was undertaken in order to control for a possible confounding effect in the Stroop task whereby response times might be affected differential word lengths.

The length of each word was defined as the number of letters in each word. A one-way ANOVA on affect category word frequencies was then conducted. This analysis revealed no significant differences between the groups

(categories of affect-words) (F = 1.81, *d.f.* 5, 54, p = 0.13). Table 6.4 reports the means and standard deviations for word lengths by affect category for the final word lists included in final Stroop task.

Table 6.4: Word Lengths by Group for Words in Stroop Task

	ANGER	SAD	SHAME
Mean	7.8	8.2	8.8
Standard Deviation	1.03	1.32	1.48
	ANXIETY	JOY	NEUTRAL
Mean	7.3	7.7	7.6
Standard Deviation	1.34	1.16	1.07

The results of this analysis suggest that there are no differential word lengths between the words contained in each of the six affect categories. This result suggests that if differences emerge in the Stroop task, it is unlikely to occur as a result of differences in word length between specific words.

CHAPTER SEVEN: CONSTRUCTION OF THE EMOTION WORD COLOUR-NAMING INTERFERENCE (EMOTIONAL STROOP) TASK AND DESCRIPTION OF THE STOP-SIGNAL PARADIGM

7.1. OVERVIEW

Chapter Seven describes the development of the Stroop Task, and reports on the methodology of the Stop-Signal paradigm. Sections 7.2 to 7.5 inclusive describe the methodology employed to develop the Stroop Task, and also includes a description of the technical platform and design specifications of the Stroop task. Section 7.6 describes the methodology and procedural use of the Stop-Signal Paradigm.

7.2. DEVELOPMENT OF THE STROOP TASK: EXPERIMENTAL HARDWARE

An Archimedes 4000 microcomputer with a high-resolution monitor was used to present the emotional Stroop task. A voice-activated junction box with microphone headset was attached to the Archimedes microcomputer via the mouse port. A headset microphone was employed to record participant responses. The headset microphone was preferred to a desk mounted unit as it was anticipated that the proximal location of the microphone to the participant's mouth would increase the probability of accurate and reliable detection of the participant's colour-naming responses. The 'arm' of the microphone was adjustable, and enabled the microphone to be placed in close proximity, immediately adjacent to the participant's mouth. The voice activated junction box also had a feedback system consisting of a white light mounted in the console which flashed when a response was recorded by the computer. This also signified that the volume of the participant's utterances had been detected by the

computer. In this way, participants were able to receive immediate feedback confirming that their colour-naming responses had been recorded.

7.3. DEVELOPMENT OF THE STROOP TASK: EXPERIMENTAL SOFTWARE

Three separate programmes were developed for the Stroop task. These included a practice programme (Practice), an experimental programme (Stroop), and a conversion programme (Convert) used to convert output from the Stroop programme into text based files readable in a Windows format. Each programme was initiated by activating the relevant programme icon located on the main task window of the computer VDU by the attached mouse. Each of these programmes is described below.

1. Practice Programme (Practice)

The practice programme was designed to familiarise the participant with the requirements of the Stroop task. The characteristics of the practice phase of the task were identical to those of the experimental (Stroop) programme thus ensuring consistency in the task between practice and experimental phases of the task.

The practice trials consisted of five neutrally rated stimulus words presented four times – once in each of the colours of Red, Blue, Green, and Yellow. This resulted in a set of 20 practice word presentations. These five neutral words were different from those employed in the main experiment. In half (10) of the trials, the stimulus word was displayed for 2000 mSec (non-masked), and in the other half of the trials the stimulus word was displayed for 240 mSec followed by a 'mask' condition lasting 1760 mSecs. Each 'masked' trial therefore lasted for a period of 2000 mSec in total duration.

In the non-masked exposure condition (2000 mSec duration) a trial consisted of the presentation of a coloured stimulus word in capital letters of one centimetre in height at a location in the centre of the visual display unit (VDU). The stimulus word was programmed to remain on the screen until the participant's verbal colour naming response activated the voice key. When this occurred, the screen blanked for a period of 2000 mSecs after which the next trial would commence.

In the 'masked' exposure condition (240-mSec duration plus 1760 mSec mask), a trial consisted of the presentation of a coloured stimulus word in capital letters of one centimetre in height at a location in the centre of the visual display unit (VDU). However, at a point precisely 240 mSec after the presentation of the stimulus word, a patterned 'mask' replaced the stimulus word. The mask consisted of an equivalent length string of graphic characters designed to resemble rotated and inverted letter fragments. The mask was undecipherable in the sense that the pattern of letter fragments did not represent any form of written language. The mask was presented in the same colour as the stimulus word, and the stimulus word/mask condition was programmed to remain on the screen until the participant's verbal colour naming response activated the voice key. When this occurred, the screen blanked for a period of 2000 mSecs after which the next trial would commence.

The primary colours of Red, Green, Blue, and Yellow were selected for inclusion in this task because these colours have been successfully employed in other similar studies (J. M. G. Williams et al., 1996). Whilst there is some evidence suggesting that borderline participants might have a generic preference for the colour red under non-specific demand conditions (Cernovsky, Fernando,

Hussein, & Fernando, 1997) other studies employing Stroop tasks with BPD have not found evidence for colour preference on the part of their BPD cohorts (Arntz et al., 2000; Kunert et al., 2003).

The stimulus words included in the practice trial included CIVILITIES, QUANDRY, ADMITTING, SERIOUSLY, UNARMED. The colour of each displayed word employed on any trial was randomised and controlled by the software. The relationship between the presentation of masked and non-masked stimulus conditions was also randomised and controlled by the software. When the practice trials had been completed, the programme indicated that the practice phase had been completed, and defaulted to the main task window of the microcomputer.

2. Experimental Programme (Stroop)

The experimental programme was designed to present the word lists derived from the studies reported in Chapters Five and Six. The task was identical to the procedure described for the practice task with the exception that the experimental task consisted of 60 words derived from six affect categories delivered at both a supraliminal (non-masked) (2000 mSec) and a subliminal (240 mSecs followed by a 1760 mSec mask) condition. Assessment of each participant took place within a single testing session.

The experimental task included 120 colour naming trials, with each of the 60 stimulus words presented at two levels of stimulus duration - 2000 mSecs, or 240 mSecs plus a 1760 mSec 'mask' in one of four colours (Red, Blue, Green, or Yellow). The structure of the masked and non-masked conditions was identical to that reported for the Practice programme. The colour of the displayed word

employed on any trial was randomised and was controlled by the software. No word appeared in the same colour more than once.

The stimulus words included in the experimental trial were reported in Table 6.3. The colour of each displayed word employed on any trial was randomised and was controlled by the software. The relationship between the presentation of masked and unmasked stimulus conditions was also randomised and controlled by the software. At the completion of 60 stimulus presentations, the programme paused the trials, advised the participant that the task was at the halfway point, and indicated to the participant that they could re-initiate the task by depressing any key on the computer keyboard. This enabled the participant to take a rest break if they so desired. At the completion of 120 experimental trials, the programme indicated that the experimental phase had been completed, and defaulted to the main task window of the microcomputer, thus ending the Stroop task.

In both exposure conditions, the computer calculated the duration in milliseconds between the onset of the stimulus word and the detection of the participant's response. Each trial was recorded individually, and saved onto the hard drive of the microcomputer. If the participant did not respond or, more likely, the recording system failed to detect the participant's response, the system would 'Time Out' at 5000 mSecs. If this occurred, the displayed word would remain on the screen until the participant either responded again, the participant spoke into the microphone, or the 'Return' key of the computer was depressed. Once this occurred, the task would continue. The data for this trial was recorded as a 5000 mSec latency, thus ensuring that the particular trial could be identified as a 'time out' trial. The effects of random noises issued by the participant such

as coughs, grunts, or sighs were expected to be randomly distributed across participants and conditions.

The masking procedure reported above was designed to assess responses in BPD participants to affect-related stimuli presented either subliminally or supraliminally. Previous work suggested that colour-naming response-latencies of 2,000 mSecs would be above, and 240 mSecs would be below the detectable sensory threshold (Locke, MacLeod, & Walker, 1994). Therefore, presentations of 240 mSecs were employed to prevent conscious awareness of a stimulus word from occurring without preventing semantic processing.

3. Conversion Programme (Convert)

The data output of the programme was organised as an output file for each participant, and was listed in the main window of the programme as a programme file icon. In order for the file to be read, a conversion programme called 'Convert' was initiated for each data file. This required the specified file to be named, and then renamed to reflect the conversion of the data. The new file was then able to be loaded into a Windows-based platform, and read using a 'WordPad' Text Processor. The data in these files was organised into a printout which displayed all words employed according to their affect category, whether the data was related to the supraliminal (2000 mSec) or the subliminal (240 plus masking) condition, and then finally the response latency for each specific word.

7.4. PROCEDURE

Participants were seated at the monitor, and a headset with microphone was placed on their head. The headset had a flexible-arm microphone head that enabled the adjustment of the microphone in order to ensure participant comfort and proximal location of the microphone to the participant's mouth in order to

ensure accurate recording of the task. The participant was advised of the task demands for this component of the study via the following instructions.

The Stroop Protocol consisted of the following steps:

1. Description of the Task

The task requirements were described to the participant as follows:

'The following task is known as the 'Emotional Stroop' Task. You will already know about this task because it has been described to you in the Information Sheet provided to you at the commencement of the study. The experimental task involves the presentation of 120 words in upper case lettering, one at a time, on the computer screen in front of you. The words will be presented in one of four different colours: Red, Green, Blue, or Yellow. Your task is to name the colour that the word is presented in.

Sometimes the word will be presented for a comparatively long time, and at other times it will be presented for a short time with what is termed a 'mask' after it. Whichever of these two conditions is presented to you, the colour of the word, or the colour of the word and the following mask will always be in the same colour for the duration of the presentation of the word. The task is divided into two parts: the first component is a practice exercise which will enable you to become accustomed to the requirements of the task. This practice trial will be relatively brief, but is identical to the experimental trials. After this has been completed, the experimental trials will commence. The practice component of the task takes approximately five minutes to complete, and after this is completed we will commence the experimental task. Please let me know when you are ready, and we will commence the practice trials.'

2. Practice Task

At the completion of the instructions, the experimenter accessed the practice programme of the Stroop. Each trial commenced with the instructions:

'NAME THE COLOUR THE FOLLOWING WORD IS DISPLAYED IN.'

These instructions were presented in white characters in the top half of the screen for a period of 2000 mSec followed immediately by a 'fixation point' of four white stars in the centre of the screen. The white stars served as a 'marker' which indicated the location where the stimulus word would be presented. The stars were displayed for 2000 mSec. At this point, each colour stimulus word was presented separately in either the masked or non-masked condition. The participant then responded by naming the colour of the stimulus word. This procedure was completed for all 20 practice trials. At the end of the practice presentations, a white display stated:

'THIS COMPLETES THE PRACTICE ASPECT OF THE TASK. THE TASK COMMENCES SHORTLY.'

This signalled the end of the practice programme.

3. Experimental Task

The experimental task commenced by initiating the 'Stroop' icon. The task commenced with the following instructions:

'The following task is the experimental component of the 'Emotional Stroop' Task. You will already know about this task because you have completed the Practice component of the task. The task involves the presentation of 120 words in upper case lettering, one at a time, on the computer screen in front of you. The words will be presented in one of four different colours: Red, Green, Blue, or Yellow. Your task is to name the colour that the word is presented in.

Sometimes the word will be presented for a comparatively long time, and at other times it will be presented for a short time with what is termed a 'mask' after it. Whichever of these two conditions is presented to you, the colour of the word, or the colour of the word and the following

mask will always be in the same colour for the duration of the presentation of the trial. The task you are now being asked to complete is the experimental component of the task. These experimental trials are identical to the practice trials you have just completed. This component of the study takes approximately 18 minutes to complete, and there is an opportunity to break at the mid point in the task. Please let me know when you are ready, and we will commence the trials'.

When the participant reported that they were ready to commence the task, the programme was initiated and the computer displayed the following instructions:

'THE TASK COMMENCES NOW. YOU ARE REQUIRED TO NAME THE COLOUR OF THE WORDS WHICH ARE PRESENTED TO YOU'.

This was presented for 2000 mSec followed by a 2000 mSec Blank screen. At that point, the stimulus series commenced with the instructions:

'NAME THE COLOUR THE FOLLOWING WORD IS DISPLAYED IN'.

This instruction was followed immediately by a fixation point of four white stars which oriented the participant to the location where the task stimuli would be presented. Once the task commenced, each task-stimuli proceeded automatically. At the halfway point of the programme (i.e., after 60 stimulus presentations) there was an opportunity for the participant to have a rest break if required. The programme stated:

'THIS IS A REST BREAK. TO CONTINUE, HIT THE ENTER KEY'.

When the participant was ready to resume the trials, the task was re-initiated by pressing the 'Enter' key on the computer keyboard. At the completion of the 120 trials, the computer advised that the trials were over.

7.5. DATA OUTPUT

Data output was organised in the following manner:

1. The data for stimuli presented at the 2000 mSec level was provided initially, and the data for stimuli presented at the 240 mSec level was provided secondly.
2. The data was presented according to affect categories. These were: Anger Words, Sadness Words, Anxiety Words, Shame Words, Neutral Words, and Shame Words.
3. Each word employed in the task was presented with the specific colour-naming response latency associated with it.

The data was then converted into 'WordPad' format and then transferred into Windows compatible applications using the Convert programme described previously.

7.6. STOP-SIGNAL PARADIGM

Response inhibition was examined through a paradigm known as the Stop-Signal Task. This task was originally developed by Logan (1985; 1994) in order to provide a theoretical account of the stopping process. The reader is referred to Section 3.6.1 for explication of Logan's theory of stopping, and the race model of inhibition that informs this theory of stopping. The version employed in this study has been described in detail elsewhere by Badcock et al. (2002).

7.6.1. Experimental Hardware

The Stop-Signal Task operated on a free-standing 486DX personal computer that was supported by MS-DOS software. The computer itself consisted of a standard size keyboard, and a 15" colour monitor. The Stop-Signal programme was loaded onto the 'C' Drive of the computer. A 'guard' was placed over the keyboard of the computer and two holes had been cut into the guard immediately over the location of the 'X', and the 'O' keys. This prevented the participant depressing irrelevant keys, and improved the likelihood of correct responding by the participants.

7.6.2. Experimental Software

The Stop-Signal Task was written in MS-DOS computer language. When the computer that contained the programme was initiated, it commenced by showing a Microsoft Windows 3.1 operating window. In order to operate the programme, the 'MS-DOS Prompt' icon was initiated which closed the Windows operating system.

The programme then utilised a series of prompts in order to initiate the programme. Once these prompts had been initiated, the programme required the case to be coded by providing a unique code-identification for each case. A participant identification number was then entered into the programme, and at that point, the programme commenced.

The 'go-task' stimuli consisted of the random, serial presentation of upper case letters (either an 'X' or an 'O') in the centre of the computer screen. A fixation or 'orienting' point in the centre of the screen always preceded the presentation of the 'go-task' stimuli. The purpose of the fixation point was to assist the participant to orient to location of the screen where the 'X' or the 'O'

stimulus would be presented. The stop-signal was randomly presented on 25% of the 'go-signal' trials, and consisted of a 100 mSec, 1000 Hz tone.

Response bias was also controlled for by changing the keyboard key that identified whether the 'go-task' stimuli was an 'X' or an 'O'. This was done at the commencement of the participant's trials by nominating which of the two keys represented the 'X' or the 'O'. A 'sticky label' was then placed adjacent to the key to indicate which key was to be depressed in order to identify the relevant stimulus.

The data output for each case was saved according to a code number that was provided at the start of each participant's trial. The output file was saved to a sub-file under the stop-signal programme on the 'C' drive on the computer. This file could then be read as a Microsoft Text File, and introduced into other Windows-based formats.

7.6.3. Procedure

The commencement of the Stop-Signal Task began with the experimenter instructing the participant as follows:

'The following task is known as the 'Stop-Signal Paradigm.' You will already know about this task because it has been described to you in the information sheet provided to you at the commencement of this study. The task is divided into two parts: the first component is a practice component which will enable you to become accustomed to the requirements of the task. This practice trial will be relatively brief, and will help you to get used to the requirements of the task. After this has been completed, the proper trials will begin. This component of the study takes approximately 30 minutes to complete, and there is opportunity to break at a number of points throughout the task. Please let me know when you are ready, and we will commence the practice trials'.

At this point, the Stop-Signal programme was initiated, and the experimenter read out the following task instructions to the participant. The task instructions were also presented on the screen as the first component on the Stop-Signal programme. This enabled the participant to read the instructions at the same time as they were being read aloud to the participant. The programme itself consisted of two components: a practice phase, and the experimental trial phase. The initial phase of the practice programme commenced with a set of instructions that referred to the practice component of the task. These instructions were read aloud to the participant. The instructions were:

'In this task you'll be shown a series of characters presented one at a time in the centre of the screen. Your task is to indicate whether each character is an O or an X by pressing the corresponding key on the keyboard. Sometimes the computer will beep while the character is presented. This will be important later, but for the moment just ignore it. Rest a finger of one hand on one of the response keys and a finger of the other hand on the other response key. Respond as quickly and as accurately as possible'.

The participant was then advised that the task would commence as soon as they pressed one of the two response keys. The participant then completed the practice phase of the task. At the completion of the practice phase, the participant was provided with an additional set of instructions which referred to the experimental trial. These were also read out to the participant whilst they also read them on the screen. The instructions were:

'That was the end of practice on this task. The following trials will be the same, only now we want you to listen as well for the beeps that the computer makes when a character is presented. Respond as quickly and as accurately as possible, but DO NOT respond when the beep

occurs. The computer varies the timing of the beep. Some beeps will occur so early that you will always be able to stop, and some so late that you will never be able to stop. Stop if you can, but don't worry if you can't. Don't let the beeps interfere with your performance on the task. Don't delay your responses in order to improve your chances of stopping'.

To this set of instructions, the experimenter added:

'Please commence when you are ready'.

The programme was divided into nine 'blocks' of 48 trials with an equal number of 'X' and 'O' stimuli in each block. The participant commenced each trial by depressing either the 'X' or the 'O' key. Once the block commenced, the trials continued automatically until the block was completed. Therefore, participants were required to be vigilant in order to ensure that stimulus presentations were responded to. When all blocks of trials were concluded, the programme automatically terminated, and the participant was advised that this task was completed. The task took approximately 30 minutes to complete.

7.6.4. Data Output

The race model of inhibition predicted that the probability of inhibition is conditionally dependent upon the speed and variability of the 'go' process (Logan, 1994). Six stop-signal delays (SSD) were included in the current study which was derived from the participants' mean reaction time (MRT) scores. Therefore, the relevant SSD's were (MRT-0) mSecs, (MRT-100) mSecs, (MRT-200) mSecs, (MRT-300) mSecs, (MRT-400) mSecs, and (MRT-500) mSecs, respectively. At an (MRT-0) mSecs, the stop-signal was presented according to the estimated time that the response to the 'go' task would be expected. Under

circumstances where SSD was less than zero, SSD was set at zero. MRT was also calculated during the first practice block, and was then used to set the delay for the first response block. Response latencies derived in the first testing block were then used to set the delay for the second response testing and so on. This method was employed for each subsequent block of trials. The stop-signal was presented twice at each of the six delay levels per block (18 trials for each SSD), and each stop-signal occurred equally frequently with each stimuli. The relationship between task stimuli, stop-signals, and stop-signal delays were randomly organised.

For each participant, a number of different measures were produced by the programme. Table 7.1 outlines the data output for each participant on the Stop-Signal programme.

Table 7.1: Data Output Provided by the Stop-Signal Paradigm

1. Stop-Signal Mean Reaction Time (MRT)
2. Stop-Signal Reaction Time (SSRT)
3. Number of Errors (X & O)
4. Percentage Errors (X & O)
5. Number of Non-Responses (NNR)
6. % of Non-Responses
7. Number of Non-Responses @ 0, 100, 200, 300, 400, & 500mSec Delay
8. SSRT @ 0, 100, 200, 300, 400, & 500mSec Delay
9. ZRFT @ 0, 100, 200, 300, 400, & 500mSec Delay

Logan (1994) argues that analysis of MRT and SSRT provides the level of analysis necessary to determine inhibitory capability. In addition, it was decided to also examine the Number of Non-Responses @ 0, 100, 200, 300, 400, & 500 mSec delay in order to more closely examine response-inhibition in this study. This data, along with the analysis of all hypothesised executive functions in BPD is reported in Chapter Eight.

SECTION IV: ASSESSMENT OF EXECUTIVE FUNCTION IN BPD

CHAPTER EIGHT: ASSESSMENT OF EXECUTIVE FUNCTION IN BPD

8.1. OVERVIEW

The studies reported in earlier sections of this project provided the empirical basis for the execution of the current study. The study reported in Section Two provided psychometric support for the use of the MCMI-III as an 'instrument of first detection' in diagnosing BPD. The studies comprising Section Three were designed to construct an Emotional Stroop task, which was developed in order to test Hypothesis Three.

The objective of the current study was to examine selected aspects of the multidimensional developmental neuropsychological model of BPD outlined in Chapter Three. The study commenced in September 1999, and concluded in May 2002.

The aims and hypotheses of the current study are described in Section 8.2 The participants included in the study are reported in Section 8.3, the Procedure is reported in Section 8.4, and the Results in Section 8.5. A brief conclusion is outlined in section 8.6, and this provides the basis for the Discussion which is included in Chapter Nine.

8.2. AIMS AND HYPOTHESES

The principal aim of the study was to examine selected aspects of the multidimensional developmental neuropsychological model of BPD described in Chapter Two. As a result of the literature reviews contained within Chapters One, Two, and Three, four hypotheses were formulated and tested in the present study.

First, on the basis of the available neuropsychological data (Bazanis et al., 2002; Burgess, 1990, 1991; Cornelius et al., 1989; Dinn et al., 2004; Driessen et

al., 2000; Judd & Ruff, 1993; Kunert et al., 2003; Kurtz & Morey, 1999; O'Leary et al., 1991; Sprock et al., 2000; Swirsky-Sacchetti et al., 1993; van Reekum, Conway et al., 1993) it was predicted that borderline participants would return poorer scores on measures of working memory when compared with controls. Second, on the basis of various reviews of the literature (Bazanis et al., 2002; Links et al., 1999; Zanarini, 1993) it was predicted that borderline participants would demonstrate impaired response inhibition as measured by the Stop-Signal Paradigm. Third, based on previous studies of the role of the Emotional Stroop in assessing clinical disorders (J. G. Beck et al., 2001; Bentnall & Kaney, 1989; Cooper et al., 1992; Kinderman, 1994; Motta, Suozzi, & Joseph, 1994; J. M. G. Williams et al., 1996), and also in examining BPD (Arntz et al., 2000; Swirsky-Sacchetti et al., 1993), it was predicted that borderline participants would return delayed negative-word colour-naming response latencies and also demonstrate interference effects on an 'Emotional Stroop' task. Finally, on the basis of selected clinical literature (Grotstein, 1987), and neuropsychological data (Burgess, 1990; Cornelius et al., 1989; Dinn et al., 2004; Judd & Ruff, 1993; O'Leary et al., 1991; D. J. Stein, Hollander et al., 1993; Swirsky-Sacchetti et al., 1993) it was predicted that borderline participants would return poorer scores on problem solving measures when compared to controls.

The hypotheses of the study can be re-summarised as follows:

1. BPD participants will demonstrate impairments to working memory compared to controls;
2. BPD participants will demonstrate impaired response inhibition compared to controls;

3. BPD participants will demonstrate impaired affective attentional bias compared to controls;
4. BPD participants will demonstrate impaired problem solving capacity compared to controls.

8.3. PARTICIPANTS

The study included three groups of participants. These included a BPD group, a Depressed comparison group, and a Medical comparison group. The following sub-sections describe the recruitment process for each group. In addition, a rationale is provided for the use of differential recruitment sources for the BPD group, and a rationale is also provided for the inclusion of two comparison groups.

8.3.1. Recruitment of BPD Participants

BPD participants were recruited from three sources. These included a cohort of BPD participants recruited through a newspaper article, a cohort of BPD participants recruited through the Mental Health Directorate of Fremantle Hospital, and a cohort of BPD participants recruited through the Department of Infectious Diseases, Fremantle Hospital.

Newspaper Recruitment of BPD Cases

A newspaper article was included in the September 18, 1999 edition of the 'Sunday Times' newspaper. The article described the nature of the study, and called for BPD volunteers. It also included DSM-IV/DSM-IV-TR criteria to assist readers to identify relevant diagnostic criteria. A copy of this article is included as Appendix XIII. 14 respondents volunteered for inclusion in the study using this method. Each respondent was interviewed at this point by the use of a telephone screening instrument in order to exclude participants with confounding

factors that might adversely affect the outcome of the study (Appendix XIV). Of the original 14 respondents, two were excluded because they reported a history of head injury or pre-existing major psychiatric illness, four were excluded from the study because they were not contactable or failed to return phone calls, and three were excluded because they did not meet BPD criteria upon interview. Five participants met criterion for BPD, and were invited to take part in the study. Of these, four completed the requirements of the study, and one participant completed the diagnostic component of the study, but failed to complete any of the experimental components of the study.

Recruitment of BPD Cases Through the Mental Health Directorate, Fremantle Hospital

Participants were also recruited from the inpatient 'Green Team' of the Mental Health Directorate, Fremantle Hospital. The author attended weekly Case Conferences of the Green Team during the period May – December 2000 and February – October 2001. Prospective BPD participants were identified through a 'case identification' methodology. This case identification methodology employed BPD criteria as identified by both DSM-IV and DSM-IV-TR (American Psychiatric Association, 1994, 2000) and Gunderson Criteria (Gunderson, 1994; Gunderson & Kolb, 1978; Gunderson et al., 1981; Gunderson & Singer, 1975). As each case was presented, it was evaluated against DSM-IV/ DSM-IV-TR/Gunderson Criteria in order to establish whether the case might meet BPD criteria. In addition, the attending Consultant Psychiatrist was familiar with the objectives of the study and would identify potential participants during the course of each Case Conference. Patients identified in this manner were then

approached for potential recruitment into the study. A total of 28 potential cases were identified during this period via this recruitment source.

Seven cases were eliminated on the advice of the Consultant Psychiatrist because they returned co-morbid diagnoses of Paranoid Schizophrenia (2), Bipolar Affective Disorder (BPAD) (1), or their Substance Abuse Disorder (SAD) (4) was judged to be too severe to meaningfully take part in the study. This yielded 21 cases for potential inclusion in the study.

After identifying potential BPD cases, the researcher contacted the participant directly. Where the participant remained an inpatient, the researcher attended the ward and met with the participant. If the participant had been discharged from hospital, the researcher contacted the participant by phone in order to recruit them into the study. In both instances, the study was described to the participant and their agreement was sought for inclusion in the study. They were further advised that all screening, assessment, and testing would be conducted at a point at least 14 days after discharge from hospital. This was done in order to avoid interfering with their inpatient care and to ensure a consistent methodological approach in that all data collection would occur with participants as outpatients only. Of the 21 potential BPD cases identified using this method, 10 participants were excluded at this point. The reasons for exclusion included refusal to participate (3), documented medical history involving stroke or head trauma, (2), itinerant lifestyle with subsequent difficulties in making contact (2), evidence of borderline IQ (2), and completed suicide (1). This yielded a total of 11 participants who were recruited into the study using this method.

Recruitment of BPD Cases Through the Department of Infectious Diseases, Fremantle Hospital

Participants were also recruited from within the Department of Infectious Diseases at Fremantle Hospital. During the period February-October 2001, all new cases of Hepatitis C (HCV) positive patients referred to the Hepatology service of the Department of Infectious Diseases were contacted for potential inclusion in the study. Sixty-three patients were identified through this process, and were contacted by telephone. The study was described to them, and they were invited to participate (Appendix XV). Of the original 63 contacts, 10 refused to participate, and 38 were reported to be in receipt of interferon therapy at the time of testing. These 38 participants were excluded because it was possible that interferon would act as a confounding factor because of its purported depressive qualities (Zdilar, Franco-Bronson, Buchler, Locala, & Younossi, 2000). Of the remaining 15 participants, 12 were excluded because they failed to meet BPD criteria, and three were included because they were found to meet BPD criteria. Figure 8.1 illustrates the procedure employed to recruit the BPD group.

Figure 8.1: Recruitment Pathway for BPD Cases

NEWSPAPER RECRUITMENT	MENTAL HEALTH DIRECTORATE	DEPARTMENT OF INFECTIOUS DISEASES
Initial Contacts (N=14)	Initial Cases Identified/ Contacted (N=28)	Initial Cases Identified/ Contacted (N=63)
↓		
Exclusion Via Head Injury or Psychiatric Disorder (N=2)	↓	↓
↓		
Not Contactable (N=4)	Eliminated on Advice of Consultant Psychiatrist (N=7)	Eliminated: Commenced Interferon Therapy (N=38)
↓		
Assessed as Not Meeting BPD Criteria (N=3)	↓	↓
↓	Excluded Through Refusal to Participate	Refused to Participate: (N=10)
Assessed as Meeting BPD Criteria (N=5)	or Other Identified Confounding Factors (N=10)	15 Cases Assessed Not Meeting BPD Criteria (N=12)
↓		
BPD Cases Withdrawing From Study at This Stage (N=1)	↓	↓
↓		
TOTAL BPD CASES VIA PATHWAY (N=4)	TOTAL BPD CASES VIA PATHWAY (N=11)	TOTAL BPD CASES VIA PATHWAY (N=3)

TOTAL BPD CASES IN STUDY = 18

8.3.2. Recruitment of Depressed Control Participants

A mood disordered control group was also recruited into the study. Participants were recruited exclusively from the 'Green Team' of the Department of Psychiatry, Fremantle Hospital. The rationale for the recruitment of this control group was justified for two reasons. Firstly, some authors believe that BPD is a variant of affective spectrum pathology (Akiskal, 1981), and inclusion of this group was a method by which a more complete control for the potential effects of mood disorder could be assured. Secondly, it is possible that the findings of the study might be an artifact of 'psychiatric caseness' rather than a more specific effect associated with BPD in the experimental group. The inclusion of a Depressed Control Group which experienced a similar inpatient history and mood profile was employed as a means of controlling for the influence of these factors. In this way, potential confounding factors associated with psychiatric disturbance such as general distress, frank mood disorder, length of inpatient stay, and medication usage could also be better controlled for in the design of the study.

Participants were identified through a 'case identification' methodology. This procedure identified patients with DSM-IV/DSM-IV-TR diagnoses of Major Depression, Dysthymia, Anxiety and/or Panic Disorder, or Situational Crisis as having a high probability for meeting Depressive criteria. A total of 47 potential cases were identified using this recruitment source. 33 were eliminated on the advice of the consultant psychiatrist because of co-morbid diagnoses of Paranoid Schizophrenia (SCZ) (8), Bi-Polar Affective Disorder (BPAD) (12), Psychotic Depression (2), Puerperal Psychosis (1), or their Substance Abuse

Disorder (SAD) (10) was too severe to meaningfully take part in the study. This yielded a total of 14 cases, of which three refused to participate. As a result, a total of 11 participants became available for diagnostic assessment. Figure 8.2 illustrates the procedure employed to recruit the Depressed Control group.

Figure 8.2: Recruitment Pathway for Depressed Control Cases

Initial Cases
Identified and Contacted
(N=47)

↓

Eliminated on
Advice of
Consultant Psychiatrist
(N=33)

↓

Excluded Through
Refusal to Participate
(N=3)

↓

TOTAL CASES
VIA PATHWAY
(N=11)

TOTAL DEPRESSED CONTROL CASES IN STUDY = 11

8.3.3. Recruitment of Medical Control Participants

Participants in this group were recruited exclusively from the Genitourinary Medicine (GUM) clinic within the Fremantle Hospital Department of Infectious Diseases during the period February 1, 2001 to December 30, 2001. The GUM patients recruited into the study were all medically well and were defined as ‘asymptomatic’ patients who had presented for a general genitourinary

medical screening. All of the patients in this group were attending either for PAP smear testing, or for general sexual health screening arising out of the commencement of a new, intimate relationship.

Participants were initially identified by the clinical nurse practitioners who reviewed each participant at first medical presentation. Patients identified by the nurse practitioner as potential candidates for the study (age: 18-59; medically asymptomatic; not obviously suffering from a major psychiatric or emotional disorder) were approached in the first instance by the nurse practitioner for agreement to be recruited into the study. If the patient refused to participate, they continued to be offered the relevant treatment as usual. Data was not available regarding patients who refused involvement at this stage. If the patient agreed to participate in the study, their name was passed to the researcher who contacted the participant independently and invited them to participate in the study. No participants were in receipt of medical and/or psychiatric treatment at the time of the study. Appendix XVI outlines the communication procedure undertaken for recruitment of this sample.

The rationale for the inclusion of this comparison group was that they represented an essentially normal group of participants who were able to be recruited through the same 'capture area' as the other groups. In this case, the capture area was defined as patients seeking clinical services from Fremantle Hospital. 12 participants were identified through this procedure for recruitment into the study. When contacted, two participants refused to be involved in the study, and 10 participants were recruited as Medical Controls. Figure 8.3 illustrates the procedure employed to recruit the Medical Control group.

Figure 8.3: Recruitment Pathway for Medical Control Cases

Initial Cases
Identified and Contacted
(N=12)

↓

Excluded Through
Refusal to Participate
(N=2)

↓

TOTAL CASES
VIA PATHWAY
(N=10)

TOTAL MEDICAL CONTROL CASES IN STUDY = 10

8.4. PROCEDURE

Setting

All participants were tested as outpatients at the Department of Infectious Diseases, Fremantle Hospital. Participants were tested individually in a room located within the Department. The room was furnished with armchairs to complete the interview components of the study, and two desks were co-located in the room to complete the information processing and neuropsychological testing components of the study.

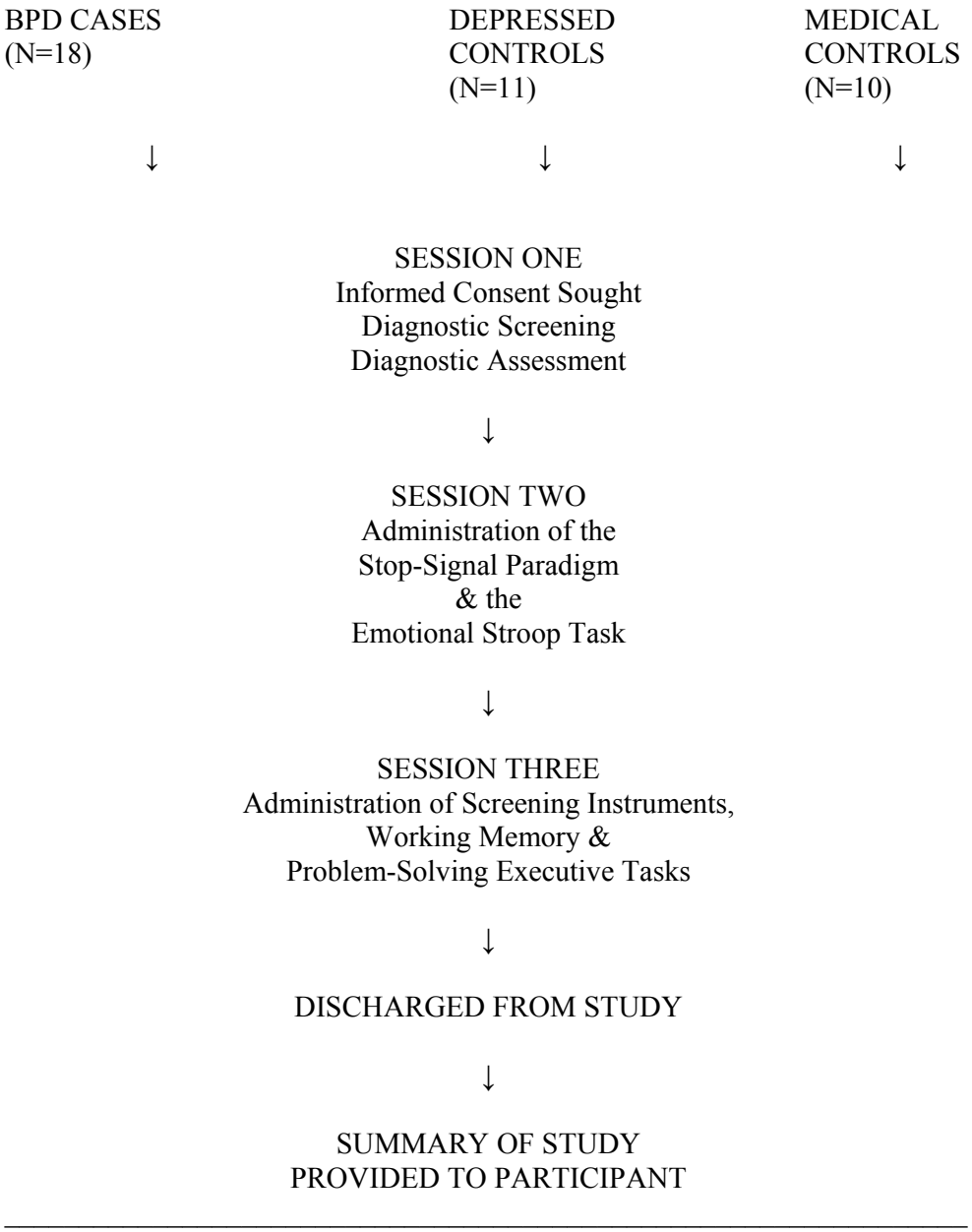
The testing room was a climate and light controlled, acoustically secure room measuring approximately 4 x 3 metres. The light source to the room consisted solely of the delivery of electrical supply light - the room did not have

a natural lighting source (windows). The light source itself consisted of two components - an 'essential supply' which is a guaranteed source of supply designed to maintain power in the event of a failure of the general electricity supply, and an additional 'auxiliary' supply. For 'normal' levels of lighting (i.e., a light level approximating an acceptable level of office lighting) both the auxiliary and essential supply were required.

During the diagnostic interview and cognitive testing components of the study, both the auxiliary and essential light supply were utilised. During the Stroop and Stop-Signal Tasks, only the Emergency supply was used. This reduced the light intensity in the room in order to enable the computer screens to be perceived in an optimal manner. Participants were advised of the intention to reduce light intensity in the room prior to the commencement of the Stroop and Stop-Signal Tasks, and their agreement was sought before this condition was implemented. Room temperature was held at a constant 24°Celsius throughout all components of testing. The room was 'laboratory like' in quality.

Because of the complexity of allocation to groups, and the number of levels of assessment involved in the study, Figure 8.4 outlines the steps taken in executing the procedure of the study. The reader is referred to Figures 8.1 to 8.3 for a description of the procedures for allocation to the specific groups.

Figure 8.4: Algorithm for Admission of Participants Into Study Four



The data collection took place over the course of three testing sessions. The contents of each session are outlined briefly below.

8.4.1. Session One: Informed Consent, Screening, and Diagnostic Assessment

Session One involved obtaining informed consent for participation in the study, screening each participant for continuation in the study, and administering diagnostic instruments in order to confirm the allocation of participants to groups. This session required approximately two hours for its completion. The procedural steps involved in this session are reported below.

Session one commenced with each participant providing written informed consent for inclusion in the study. Each participant was initially provided with an Information Sheet (Appendix XVII) which outlined the nature of the study, and a Consent Form (Appendix XVIII). Participants were required to read the Information Sheet prior to signing the Consent Form. After signing the Consent Form, participants were formally admitted into the study. Additional information not contained within the Information Sheet was provided by the experimenter where the participant required it.

A preliminary screening assessment was then conducted on each participant (Appendix XIX). The screening assessment was conducted in order to screen for handedness, alcohol and drug use history, and to briefly review each participant's neurological history. Specific inquiry was made in order to identify the presence of epilepsy, a history of head injury, or other relevant neurological conditions including convulsions and encephalitis and/or loss of consciousness for a period greater than five (5) minutes at any time in their life. In addition, each participant was also screened for the presence of any co-morbid major psychiatric illness, such as Bipolar Affective Disorder, Schizophrenia, or Organic Psychosis. This was also confirmed by reviewing the hospital chart where

available for any evidence of medical conditions likely to affect performance upon these tasks.

A colour screening test was also developed in order to screen for difficulties in the perception of colour. The accurate perception of colour was important in the successful execution of the experimental tasks such as the Emotional Stroop task and the Wisconsin Card Sorting Test (WCST). Colour 'swatches' consisting of four 2x2 cm coloured cardboard forms were fastened to a steel ring for ease of use. The cardboard forms included the colours of Red, Blue, Green, and Yellow. Participants were asked to name the colour of each swatch. Participants were eliminated from the study if they failed to successfully name all four colours contained on the swatches.

In addition, Participants from any of the three groups were screened out if they met the following criteria:

1. English was not the participant's first language;
2. The participant reported a history of neurological trauma, neurological illness, or brain damage including convulsions and encephalitis and/or loss of consciousness (LOC) for a period greater than five (5) minutes at any time in their life;
3. The participant reported a history of serious psychiatric disorder defined as Bipolar Affective Disorder, Schizophrenia, Schizoaffective Disorder, Organic or Metabolic Psychosis, or Psychosis 'Not Otherwise Specified' (NOS);
4. The participant reported a history of significant gross or fine motor impairment;
5. If at the time of testing, the participant had failed to abstain from the use of alcohol or illicit drugs for a period of at least 72 hours;

6. The participant had received ECT in the previous 12 week period;
7. The participant reported colour blindness or failed the colour perception screening task as described above;
8. The participant was not exclusively right handed.

No participants were eliminated at this stage of the study, and each participant was then administered the Millon Clinical Multiaxial Inventory (3rd Edition) (Millon et al., 1994), and the Diagnostic Interview for Borderlines – Revised (Zanarini et al., 1989) (Appendix V). The instruments were not scored until the diagnostic examination had been concluded. This was done in order to provide some measure of 'blindness' with regard to the DIB-R interview protocol. After completing the diagnostic interview, the instruments were scored and interpreted. All participants were advised of their results, and the group which each participant was to be allocated to was communicated to the participant. BPD participants were formally advised that they met study criteria for BPD.

The criteria for allocation to groups involved the following: BPD participants were required to achieve a scaled score of at least 85 on Scale C of the MCMI-III **and** a Scaled Score of at least eight (8) on the DIB-R Total Score. Depressed Controls were required to return a Scaled Score of less than 85 on Scale C of the MCMI-III. In addition, each participant was also required to score in excess of a Scaled Score of 85 or more on either Scale D (Dysthymia) or Scale CC (Major Depression) of the MCMI-III. The reason for the requirement of scaled scores in excess of 85 for Scale D and Scale CC was that this provided further empirical support that the participants in this group were a non-borderline, depressed comparison group.

It was understood that it was possible that the DIB-R Total Scaled Score for the Depressed Control group might be in excess of eight, suggesting that the participant met BPD criteria. However, the diagnosis of BPD in this study required achievement of criterion on both Scale C of the MCMI-III (Scaled Score > 85) **and** the DIB-R (Scaled Score > 8). Whilst this decision raises questions with regard to the discriminant validity of the DIB-R, this reflects the current status with regard to the diagnosis of BPD where there remains a debate about whether it reflects a variant of affective disorder or a discrete disorder in its own right (Akiskal, 1981; D. F. Klein, 1975, 1977; Paris, 1999). Achievement of criterion on one instrument (MCMI-III or DIB-R) resulted in automatic default to the Depressed Comparison group as long as the participant achieved criterion for one of the mood disorder scales (Scale D or Scale CC) on the MCMI-III. In reality, if the participant achieved BPD criterion on the MCMI-III, there was a high probability that they would achieve criterion on the DIB-R. The difficulty occurred in those cases where BPD criterion was not achieved on the MCMI-III, but was achieved on the DIB-R. In these cases, participants were allocated to the Depressed Control condition because they did not meet the study criterion for allocation to the BPD group, but did meet study criteria for allocation to the depressed condition.

Nonetheless, this issue was also examined empirically in a series of subsidiary analyses. The findings of these subsidiary analyses which involved the removal of 'high scoring' DIB-R Depressed Controls (i.e., DIB-R Scores > Eight) from Between Groups analyses will be briefly reported in Section 8.5.7.

Medical Controls were required to return a Scaled Score on Scale C of the MCMI-III of less than 85, and a Scaled Score on the DIB-R of seven or less.

In addition, Medical Controls were required to return MCMI-III Scale D (Dysthymia) and Scale CC (Major Depression) Scaled Scores of less than 85. The requirement for Scaled Scores below 85 on Scale D and Scale CC of the MCMI-III was that this provided empirical support that the group was a non-borderline, non-depressed comparison group.

At the completion of Session One, participants were reminded that they were required to attend for two more testing sessions. A further two appointments were then made with the participant in order to complete the requirements of the study. Session Two took place a minimum of one week after the completion of Session One.

8.4.2. Session Two: Administration of the Emotional Stroop Task and the Stop-Signal Paradigm

Session Two involved the administration of a measure of mood, and the administration of the Stop-Signal Task to examine Hypothesis Two, and the Emotional Stroop task to examine Hypothesis Three. This session required approximately one and a half hours for its completion. The procedural steps involved in this session are reported below.

Session Two commenced with each participant completing the Positive and Negative Affect Scale (PANAS) (Watson, Clark, & Tellegen, 1988). After completing the PANAS, each participant was first administered the Stop-Signal Paradigm and then the Emotional Stroop Task. The tasks operated on separate computers placed beside each other with the participant seated approximately 60 cm in front of the respective monitors. For this component of the study, the experimenter sat behind the participant in order to minimize distraction on the task. As reported in Section 8.4, the lighting for this component of the testing

was reduced to ‘essential supply’ in order to allow ease of perception of material on the computer screen. These tasks required approximately 1.5 hours to complete. At the end of this aspect of testing, the participant was reminded that that they were required to complete one final testing session. Session Three took place a minimum of one week after the completion of Session Two.

8.4.3. Session Three: Administration of Screening Instruments, Working Memory and Problem-Solving Executive Tasks

Session Three involved the re-administration of a measure of mood, and the administration of the neuropsychological executive tasks designed to examine Hypotheses One and Four as outlined in Section 8.2. This session required approximately two and a half hours for its completion. The procedural steps involved in this session are reported below.

Session Three commenced with each participant completing the Positive and Negative Affect Scale (PANAS) (Watson et al., 1988). After completing the PANAS, the neuropsychological testing component of the study was conducted. At the completion of the third session, participants were discharged from the study, and thanked for their participation. They were further advised that upon completion of the study, a written summary of the results would be provided to them. This is included as Appendix XX.

A number of measures were administered in Session Three, and their order of presentation is provided below. For ease of understanding, each reported measure is located under one of the three headings. The first heading (Screening Measures) refers to those instruments employed in order to screen participants and where necessary, exclude participants from further involvement in the study. The second heading (Measures of Executive Functioning) refers to those

instruments employed in order to test the Hypotheses Three and Four as reported in Section 8.2. This section is further subdivided into two subsections: those instruments examining working memory, and those instruments examining problem solving ability. Table 8.1 describes the tasks included in Session Three and Appendix III provides information concerning the tasks and the scoring procedures employed with each instrument.

Table 8.1: Screening and Executive Tasks Administered in Session Three

---

SCREENING MEASURES

1. The Positive and Negative Affect Scale (PANAS) (Watson et al., 1988)
2. National Adult Reading Test (NART) (Nelson, 1982).
3. St. Lucia Word Recognition Test (Andrews, 1973)
4. Digit Symbol (DS) (Wechsler, 1981)
5. Quick Test (QT) (Ammons & Ammons, 1962).

ASSESSMENT OF WORKING MEMORY

6. Logical Memory (LM) (Wechsler, 1987) (Immediate and Delayed – 20 Minute Conditions)
7. Visual Reproduction (VR) (Wechsler, 1987) (Immediate and Delayed – 20 Minute Conditions)
8. Paired Associates Learning (PAL) (Wechsler, 1987) (Immediate and Delayed – 20 Minute Conditions)
9. Digit Span (DSp) (Wechsler, 1981)
10. Visual Memory Span (VMS) (Wechsler, 1987)

ASSESSMENT OF PROBLEM-SOLVING

1. Complex Figure of Rey (Rey Figure) (Lezak, 1995) (Immediate and Delayed – 20 Minute Conditions)
2. Controlled Oral Word Association Test (COWAT) (FAS) (Spreen & Strauss, 1991)
3. Similarities (Wechsler, 1981)
4. Wisconsin Card Sorting Test (WCST) (Heaton et al., 1993)
5. Austin Maze (Walsh, 1978)
6. Tower Of London (TOL) (Shallice, 1982)
7. Tower Of Hanoi (TOH) (Simon, 1975)

8.5. RESULTS

The results section is divided into three subsections. Section 8.5.1 analyses DIB-R and MCMI-III diagnostic data, and commences by analysing MCMI-III and DIB-R data on the BPD subgroups. This analysis was undertaken because BPD participants were drawn from three sources. As a first step, it was important to determine if there was diagnostic homogeneity between the three BPD subgroups. The analysis demonstrated that sufficient diagnostic homogeneity existed between the three subgroups to permit collapsing them into one BPD category.

A second set of analyses comparing MCMI-III and DIB-R data for BPD, Depressed Controls, and Medical Controls was then undertaken. This analysis was undertaken to confirm diagnostic differences between the groups. The findings of this analysis demonstrated that there were diagnostic differences between the groups and this provided the justification for maintaining the groups as separate entities.

Section 8.5.2 examines the hypotheses originally articulated in Sections 3.10 and 8.2 respectively. Hypothesis One analysed working memory data for the BPD, Depressed Controls, and Medical Controls and is reported in Section 8.5.3. The analysis revealed no significant differences between the groups. Hypothesis Two analysed Response Inhibition data for BPD, Depressed Controls, and Medical Controls and is reported in Section 8.5.4. This analysis also revealed no significant differences between groups. Hypothesis Three analysed affective-attentional bias data for the BPD, Depressed Controls, and Medical Controls and is reported in Section 8.5.5. The analysis revealed a number of significant differences between groups, and these are detailed within Section 8.5.5.

Hypothesis Four analysed Problem Solving data for the BPD, Depressed Controls, and Medical Controls and is reported in Section 8.5.6. This analysis also returned no significant differences between groups.

8.5.1. Analysis of DIB-R and MCMI-III Data

This section reports the analyses of two data sets. First, an analysis of the diagnostic data from three BPD subgroups is reported. Second, an analysis of the diagnostic data from the BPD Group, Depressed Control Group, and the Medical Control Group is then reported.

Analysis of BPD Subgroup MCMI-III and DIB-R Data

The rationale for analyzing BPD subgroups diagnostic data has been reported in Section 8.5. Preliminary analyses were conducted on the MCMI-III and DIB-R diagnostic data in order to assess whether the subgroups differed significantly from each other. Diagnostic homogeneity between the subgroups would permit collapsing the three BPD subgroups into one group.

Sample Means

Table 8.2 reports data on the age ranges, gender distribution, occupational, marital, and educational status for BPD Subgroups.

Table 8.2: Demographic Data for the IDD, Psychiatry, and Newspaper BPD Subgroups

	IDD	MENTAL HEALTH DIRECTORATE	NEWSPAPER
	(N=3)	(N=11)	(N=4)
AGE			
10-19	0	1	0
20-29	1	4	1
30-39	0	1	1
40-49	2	4	1
50-59	0	1	1
Mean	38.00	33.27	40.25
SD	8.72	11.35	11.03
GENDER			
Male	1	0	0
Female	2	11	4
OCCUPATIONAL STATUS			
Professional	0	1	1
Managerial	0	0	0
Technical	0	0	0
Clerical/Sales	1	3	1
Skilled Labour	0	0	0
Semi-Skilled Labour	0	0	1
Unskilled Labour	0	0	0
Student	0	1	0
Home Duties	1	2	1
Unemployed	1	4	0

Table 8.2: (Continued) Demographic Data for the IDD, Psychiatry, and Newspaper BPD Subgroups

	IDD	MENTAL HEALTH DIRECTORATE	NEWSPAPER
	(N=3)	(N=11)	(N=4)
MARITAL STATUS			
Married	0	1	1
Divorced	1	3	0
Separated	0	0	1
De Facto	1	1	0
Single	1	6	2
EDUCATIONAL STATUS			
1 = Completed Yr 10	0	7	2
2 = Completed Yr 12	3	3	1
3 = Completed Degree	0	1	1
4 = Completed Postgraduate Degree	0	0	0
Mean Years Education	12	11.45	12
SD	0.0	1.97	2.16

A series of one-way ANOVAs were conducted on the BPD subgroups' demographic data because of insufficient cell size to justify alternative statistical analysis (Keppel, 1991)[40]. Two demographic variables (age and years of education) provided continuous data, and could therefore be tested using oneway ANOVA for differences across the three groups. Neither age ($F(2,14) = 0.68$, ns) nor years of education ($F(2,14) = 0.18$, ns) produced a significant effect.

Diagnostic Data

Table 8.3 reports the sample means, standard deviations and F-test scores for the three BPD subgroups on the DIB-R Scaled Scores. Table 8.4 reports the sample means, standard deviations and F-test scores for the three BPD subgroups on the MCMI-III Validity and Clinical Personality Scaled Scores, and Table 8.5 reports the sample means, standard deviations and F-test scores for the three BPD subgroups on the MCMI-III Clinical Syndromes Scales Scores, respectively.

[40] It should be noted here that Bonferroni adjustments were not undertaken throughout the study. The scope of the study was such that it was adjudged as preferable to risk Type I error rather than close off potentially promising lines of research as a result of error adjustment.

Table 8.3: Sample Means, Standard Deviations, and F Statistics for DIB-R Scaled Scores for BPD Subgroups

	IDD		MENTAL HEALTH DIRECTORATE		NEWSPAPER		F
	N = 3		N = 11		N = 4		
DIB-R	Mean	S.D.	Mean	S.D.	Mean	S.D.	
DIB-R Affect Scaled Score	2.00	0.00	2.00	0.00	2.00	0.00	-
DIB-R Cognition Scaled Score	1.33	0.58	1.91	0.30	1.75	0.50	2.53
DIB-R Impulse Scaled Score	3.00	0.00	2.82	0.40	2.75	0.50	0.36
DIB-R Interpersonal Scaled Score	2.67	0.58	2.91	0.30	2.75	0.50	0.56
DIB-R Total Score	9.00	1.00	9.64	0.67	9.25	0.50	1.18

Note: All F-Ratio's had *df* = 2,14, and were ns. The F for the DIB-R Affect Scaled Score could not be calculated because of zero variance.

Table 8.4: Sample Means, Standard Deviations and F Statistics for MCMI-III Validity, Clinical Personality, and Severe Personality Pathology Scaled Scores for BPD Subgroups

	IDD		MENTAL HEALTH DIRECTORATE		NEWSPAPER		F
	N = 3		N = 11		N = 4		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
MCMI-III							
Scale X (Disclosure)	83.67[ab]	9.87	90.18[a]	7.70	76.50[b]	5.80	4.81*
Scale Y (Desirability)	28.00	12.12	20.91	16.12	35.00	14.97	1.28
Scale Z (Debasement)	78.00[a]	10.15	93.27[b]	5.95	85.00[b]	3.27	7.95**
Scale 1 (Schizoid)	74.33	19.03	78.27	8.57	75.25	16.09	0.17
Scale 2A (Avoidant)	88.00	10.15	82.73	15.30	77.75	20.50	0.36
Scale 2B (Depressive)	85.67	4.04	91.55	6.65	91.50	11.27	0.75
Scale 3 (Dependent)	89.67	13.50	79.82	7.28	82.50	11.48	1.34
Scale 4 (Histrionic)	18.00	6.24	12.18	17.09	31.50	19.07	2.01
Scale 5 (Narcissistic)	35.00	21.93	26.82	21.68	33.75	3.78	0.32
Scale 6A (Antisocial)	74.00	14.00	60.00	12.34	68.00	11.40	1.76
Scale 6B Aggressive)	60.00	5.57	66.00	20.56	67.50	3.87	0.19
Scale 7 (Compulsive)	33.00	12.49	23.64	15.84	37.75	23.73	1.10
Scale 8A (Passive-Aggressive)	78.00	7.00	79.64	9.24	74.50	1.92	0.60
Scale 8B (Self-Defeating)	77.33	6.66	85.00	10.65	79.50	9.49	1.03
Scale S (Schizotypal)	75.33	9.87	76.45	13.95	67.50	4.73	0.81
Scale C (Borderline)	93.33	9.71	92.64	4.32	89.25	3.86	0.71
Scale P (Paranoid)	70.00	7.21	76.36	16.18	52.00	34.71	2.06

Note: All F-Ratio's had *df* = 2,14. *p<0.05, **p<0.01; Differential superscripts demarcate the location of significant between-groups differences

Table 8.5: Sample Means, Standard Deviations, and F Statistics for MCMI-III Clinical Syndromes Scaled Scores for BPD Subgroups

	IDD		MENTAL HEALTH DIRECTORATE		NEWSPAPER		F
	N = 3		N = 11		N = 4		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
Scale A (Anxiety)	82.67	6.66	92.09	9.95	80.75	2.22	3.29*
Scale H (Somatoform)	41.33[a]	36.68	84.00[b]	14.39	70.25[ab]	2.36	6.84**
Scale N (Bipolar)	62.67	3.51	64.64	17.47	65.75	4.19	0.04
Scale D (Dysthymia)	70.33	19.55	91.00	11.67	80.75	13.12	3.16*
Scale B (Alcohol Dependence)	65.67	4.93	64.64	9.82	69.25	9.07	0.37
Scale T (Drug Dependence)	96.00[a]	22.72	55.00[b]	12.34	67.50[ab]	10.54	10.40***
Scale R (PTSD)	72.33	3.22	86.55	21.04	69.50	7.94	1.76
Scale SS (Thought Disorder)	67.33	8.08	78.73	14.1	67.00	1.41	2.03
Scale CC (Major Depression)	58.67[a]	19.42	97.64[b]	8.14	90.00[b]	7.07	17.14***
Scale PP (Delusional Disorder)	48.00	23.065	60.36	60.36	49.75	49.75	0.30

Note: All F-Ratio's had *df* = 2,14. *p<0.1, **p<0.01, ***p<0.001; Differential superscripts demarcate the location of significant between-groups differences

The analysis revealed no significant differences between the BPD subgroups on any of the DIB-R Scaled Scores. Therefore, no post hoc analyses were conducted on the DIB-R subgroup data. There were, however, a number of significant differences between the BPD subgroups on selected MCMI-III Scaled Scores. In addition, Scale A (Anxiety) and Scale D (Dysthymia) produced differences that approached significance, but these will not be pursued further because of concerns about alpha-inflation due to the large number of non-independent tests conducted.

The significant differences between the groups can be summarized as follows:

1. The IDD BPD subgroup returned significantly higher scores than both the Mental Health Directorate and the Newspaper BPD subgroups on Scale T (Drug Dependence);
2. The Mental Health Directorate BPD subgroup returned significantly higher scores than the IDD BPD subgroup only on Scale Z (Debasement), Scale H (Somatoform), and Scale CC (Major Depression);
3. The Newspaper BPD subgroup returned significantly higher scores than the Mental Health Directorate BPD subgroup on Scale X (Disclosure);
4. The Newspaper BPD subgroup also returned significantly higher scores than the IDD BPD subgroup on Scale CC (Major Depression).

A number of explanations are offered to account for the differences between the respective subgroups.

The significant differences detected between the Mental Health Directorate BPD subgroup and the IDD BPD subgroup on Scales Z (Debasement), H (Somatoform), and CC (Major Depression) is probably best

interpreted as an artifact of negative mood. The Mental Health Directorate BPD subgroup had been recruited from a cohort of patients who had recent inpatient admissions primarily for Major Depression. It is likely that residual, negative affect associated with these admissions was reflected in elevated scores on the propensity for negative self-evaluation (Debasement), the experiencing of emotions via sensory modalities (Somatoform), and in scores on Major Depression. Therefore, it would be reasonable to expect that the Mental Health Directorate BPD subgroup would have more severe mood based scores when compared with the IDD subgroup and would be reflected in elevated scores on Scales Z (Debasement), H (Somatoform), and CC (Major Depression). The significant differences on Scale CC (Major Depression) between the IDD BPD subgroup and the Mental Health Directorate subgroup will be further commented on later as the IDD BPD subgroup and the Newspaper BPD subgroup were also significantly different from each other.

It is also likely that the IDD BPD subgroup would return significantly higher scores on Scale T (Drug Dependence) than the Mental Health Directorate and Newspaper BPD subgroups because the IDD BPD subgroup was drawn from patients presenting for treatment for Hepatitis C through the Hepatology service of IDD. It is well known that in excess of 90% of patients contract Hepatitis C through the pathway of injecting drug use (IDU) (Zdilar et al., 2000). This referral artifact is likely to be reflected in elevated scores on Scale T of the MCMI-III for this cohort.

In general, the Newspaper BPD subgroup returned a diagnostic profile similar to the Mental Health Directorate BPD subgroup. The only detectable difference between the two groups was that the Newspaper BPD subgroup

returned a significantly higher score than the Mental Health Directorate BPD subgroup on Scale X (Disclosure). This suggests that the Newspaper BPD subgroup may have underreported the severity of their symptoms. There are a number of possible explanations for this finding. First, the Mental Health Directorate BPD subgroup was likely to be more familiar with hospital protocols than the Newspaper BPD recruited subgroup, and therefore more familiar and probably more experienced with the disclosure processes typical of this type of setting (a tertiary teaching hospital environment). In addition, the Mental Health Directorate BPD subgroup was essentially a 'help seeking' cohort recruited in part out of their desire to receive clinical assistance. It is probable under this circumstance that their disclosure preparedness would be greater than for participants who were not seeking clinical assistance but who responded as a result of contact through media sources. Furthermore, the Mental Health Directorate BPD subgroup was also likely to be a more acutely unwell group. As a result, it is probable that they had a greater preparedness to openly disclose the current state they were experiencing. Finally, the Mental Health Directorate BPD subgroup was probably more acutely distressed, and as a result it is possible that they would overemphasize the severity of their distress. The differences between the subgroups on this scale is a diagnostically important contrast, but in view of the similar returns on a number of other dimensions, the differences in subgroup returns is insufficient to warrant elimination of one of these subgroups from further inclusion in the study.

Finally, the analysis also returned a significant difference between the IDD BPD subgroup and both the Newspaper BPD and the Mental Health Directorate BPD subgroups on Scale CC (Major Depression). One possible

explanation is that this finding represents a difference in the nature of borderline experience. There is evidence in the literature suggesting that different types of borderline cases might exist (Andrulonis et al., 1982; Clarkin & Kernberg, 1993; Grinker et al., 1968; Rusch et al., 1992). One of the subtypes referred to in the literature is associated with those borderline cases experiencing frank mood disorder (Grinker et al., 1968). Clinically, the evidence suggests that the IDD BPD subgroup was not depressed (Mean CC Scaled Score = 58.67), whereas both the Mental Health Directorate BPD subgroup and the Newspaper BPD subgroups were (Mean CC Scaled Scores = 97.64, and 90.00 respectively). It is possible that the differences between subgroups on Scale CC reflect evidence of these different typologies of borderline experience, with the Mental Health Directorate and Newspaper BPD subgroups reflecting affectively dysregulated BPD subtypes, and the IDD BPD subgroup reflecting a non-affectively dysregulated subtype.

A second related explanation is that there are probably a number of 'pathways' into the diagnosis of BPD, and some of these pathways will involve recruiting borderline participants who do not experience mood-disorder phenomena. BPD diagnosis involves 'polythetic' criteria, so it is possible to meet BPD criterion but not have a diagnosable mood disorder (Widiger et al., 1992). Clearly, this represents a direction for future research.

It should also be noted that the significance level between the IDD BPD subgroup and the Newspaper BPD subgroup on Scale T was precisely 0.05. Whilst such a finding has been regarded historically as non-significant, it can be argued that this result is very close to significance. Viewed collectively, the BPD subgroup analyses on Scale T suggest that the returns of the IDD subgroup were

markedly different from the other two subgroups. This finding makes sense when it is recalled that the IDD BPD subgroup was drawn from a Hepatitis C cohort. There is a strong association between Hepatitis C and a history of injecting drug use (Zdilar et al., 2000). Under these circumstances, it is understandable that this subgroup would return elevated Scale T (Drug Dependence) Scaled Scores.

It could be argued that the significant differences between subgroups provide a rationale for eliminating the IDD BPD subgroup from further inclusion in the analysis. There are a number of reasons why eliminating the IDD BPD subgroup was not justified. First, on all of the BPD measures no differences were detected between the subgroups. Second, the measures where significant differences were found were found on either 'state' or 'response bias' measures. None of the other 'trait' (personality) measures on the MCMI-III yielded significant differences between the subgroups. This finding suggests that the differences between the subgroups is not associated with 'personality' based (Axis II) variables. Rather, the differences that the MCMI-III detected appear to be associated with the acute affective state of the respondents, and these appear to be dissociable from the trait based dimensions involved in the assessment of personality disorder. The absence of significant differences in age and educational status as well as the generally similar diagnostic profiles of the three BPD subgroups provide further support to justify collapsing the three BPD subgroups together into one group. For these reasons, it is argued that there is a sufficient basis to justify collapsing the three BPD subgroup data sets together.

Analysis of BPD, Depressed, and Medical Control MCMI-III and DIB-R Data

The previous section provided evidence to justify collapsing the three BPD subgroups into one BPD group. This section compares the DIB-R and MCMI-III diagnostic data between the aggregated BPD group, Depressed, and Medical Control Groups, and provides empirical demonstration of significant diagnostic differences between the three groups.

Sample Means

Table 8.6 reports sample age means and standard deviations as well as gender, occupational, marital, and educational status for the BPD, Depressed Control, and Medical Control Groups.

Table 8.6: Demographic Data for the BPD, Depressed, and Medical Control Groups

	BPD (N=18)	DEPRESSED CONTROL (N=11)	MEDICAL CONTROL (N=10)
AGE			
10-19	1	0	0
20-29	6	5	4
30-39	2	4	2
40-49	7	1	2
50-59	2	1	2
Mean	35.61	33.00	37.11
SD	10.76	7.73	12.60
GENDER			
Male	1	1	1
Female	17	10	9
OCCUPATIONAL STATUS			
Professional	2	2	0
Managerial	0	1	2
Technical	0	1	0
Clerical/Sales	5	0	4
Skilled Labour	0	4	1
Semi-Skilled Labour	1	0	0
Unskilled Labour	0	1	0
Student	1	0	3
Home Duties	4	1	0
Unemployed	5	1	0

Table 8.6: (Continued) Demographic Data for the BPD, Depressed, and Medical Control Groups

	BPD (N=18)	DEPRESSED CONTROL (N=11)	MEDICAL CONTROL (N=10)
MARITAL STATUS			
Married	2	2	2
Divorced	4	1	3
Separated	1	2	0
De Facto	2	3	1
Single	9	3	4
EDUCATIONAL STATUS			
1 = Completed Yr 10	10	4	3
2 = Completed Yr 12	6	4	2
3 = Completed Degree	2	3	4
4 = Completed Postgraduate Degree	0	3	1
Mean Years Education	11.67	13.36	13.50
SD	1.78	3.56	3.56

There were no significant differences between the three groups for age ($F(2,35) = 0.24$, ns) or years of education ($F(2,35) = 2.22$, ns). Table 8.7 reports the means, standard deviations, and F statistics for the DIB-R analyses for BPD, Depressed, and Medical Controls. Table 8.8 reports the means, standard deviations, and F statistics for the MCMI-III Validity and Clinical Personality Scaled Scores for BPD, Depressed, and Medical Controls, and Table 8.9 reports the means, standard deviations, and F statistics for the MCMI-III Clinical Syndrome Scaled Scores for BPD, Depressed, and Medical Controls.

Table 8.7: Sample Means, Standard Deviations, and F Statistics for DIB-R Scaled Scores for BPD, Depressed, and Medical Control Groups

	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	N = 18		N = 11		N = 10		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
DIB-R Affect Scaled Score	2.00[a]	0.00	2.00[a]	0.00	0.50[b]	0.71	66.92*
DIB-R Cognition Scaled Score	1.78[a]	0.43	0.91[b]	0.70	0.00[c]	0.00	46.57*
DIB-R Impulse Scaled Score	2.83[a]	0.38	2.09[b]	1.14	0.70[c]	1.16	19.15*
DIB-R Interpersonal Scaled Score	2.83[a]	0.38	1.73[b]	1.19	0.70[c]	1.16	103.79*
DIB-R Total Score	9.44[a]	0.70	6.73[b]	1.56	1.90[c]	2.64	68.95*

Note: All F-Ratio's had *df* = 2,35. *p< 0.001; Differential superscripts demarcate the location of significant between-groups differences

Table 8.8: Sample Means, Standard Deviations, and F Statistics for MCMI-III Validity, Clinical Personality, and Severe Personality Pathology Scaled Scores for BPD, Depressed, and Medical Control Groups

	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	N = 18		N = 11		N = 10		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
MCMI-III							
Scale X (Disclosure)	86.06[a]	9.26	74.73[a]	6.45	37.30[b]	19.98	51.27***
Scale Y (Desirability)	25.22[a]	15.67	35.09[a]	16.68	70.50[a]	22.74	20.92***
Scale Z (Debasement)	88.89[a]	8.47	83.27[a]	9.04	31.30[b]	28.31	44.92***
Scale 1 (Schizoid)	76.94[a]	11.60	73.64[a]	11.81	36.30[b]	24.33	23.20***
Scale 2A (Avoidant)	82.50[a]	15.32	78.36[a]	22.08	26.70[b]	31.16	22.48***
Scale 2B (Depressive)	90.56[a]	7.45	86.91[a]	12.55	24.20[b]	28.62	57.29***
Scale 3(Dependent)	82.06[a]	9.45	82.73[a]	7.40	26.80[b]	24.18	67.25***
Scale 4 (Histrionic)	17.44[a]	17.46	31.45[a]	26.72	80.70[b]	26.86	25.49***
Scale 5 (Narcissistic)	29.72[a]	18.71	31.73[a]	31.84	67.20[b]	20.84	3.98*
Scale 6A (Antisocial)	64.11[a]	12.94	53.00[ab]	16.67	47.00[b]	20.47	9.05**
Scale 6B Aggressive)	65.33[a]	16.16	51.82[ab]	19.06	35.60[ab]	27.42	6.97**
Scale 7 (Compulsive)	28.33[a]	17.45	54.82[b]	9.86	57.30[b]	11.22	18.42***
Scale 8A (Passive-Aggressive)	78.22[a]	7.83	65.82[a]	20.04	24.70[b]	19.83	39.30***
Scale 8B (Self-Defeating)	82.50[a]	9.49	86.64[a]	7.09	20.90[b]	25.52	67.92***
Scale S (Schizotypal)	74.28[a]	11.99	68.55[a]	12.58	17.90[b]	25.10	40.96***
Scale C (Borderline)	92.00[a]	5.20	73.55[b]	7.30	18.40[c]	23.88	103.79***
Scale P (Paranoid)	69.89[a]	21.80	59.64[a]	29.84	20.90[b]	23.32	13.04***

Note: All F-Ratio's had *df* = 2,35. *p<0.05, **p<0.01, ***p< 0.001; Differential superscripts demarcate the location of significant between-groups differences

Table 8.9: Sample Means, Standard Deviations, and F Statistics for MCMI-III Clinical Syndrome Scaled Scores for BPD, Depressed, and Medical Control Groups

	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	N = 18		N = 11		N = 10		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
Scale A (Anxiety)	88.00[a]	9.62	80.73[a]	23.46	21.10[b]	30.81	35.67***
Scale H (Somatoform)	73.83[a]	23.18	84.00[a]	14.97	26.00[b]	23.81	22.47***
Scale N (Bipolar)	64.56[a]	13.60	54.18[ab]	25.20	45.80[b]	18.52	3.39*
Scale D (Dysthymia)	85.28[a]	14.86	88.45[a]	13.97	15.30[b]	18.80	76.42***
Scale B (Alcohol Dependence)	65.83[a]	8.82	58.09[ab]	11.42	39.40[b]	30.99	7.21**
Scale T (Drug Dependence)	64.61[a]	20.14	55.55[ab]	18.78	40.50[b]	20.33	4.76*
Scale R (PTSD)	80.39[a]	18.35	72.82[a]	21.78	18.50[b]	23.60	30.57***
Scale SS (Thought Disorder)	74.22[a]	12.60	69.91[a]	8.73	14.90[b]	22.19	56.72***
Scale CC (Major Depression)	89.44[a]	17.40	94.18[a]	13.72	21.50[b]	22.73	55.97***
Scale PP (Delusional Disorder)	55.94[a]	29.25	36.55[ab]	31.22	13.20[b]	19.33	7.73**

Note: All F-Ratio's had *df* = 2,35. *p<0.05, **p<0.01, ***p< 0.001; Differential superscripts demarcate the location of significant between-groups differences

The results of these analyses suggest that significant differences between the groups were reported for all DIB-R Scaled Scores, all MCMI-III Clinical Personality Pattern Scaled Scores, and all MCMI-III Clinical Syndrome Scaled Scores.

The significant differences between the BPD, Depressed, and the Medical Control groups can be summarised as follows:

1. The BPD subgroup returned significantly higher scores on all measures when compared with the Medical Control group with the exception of Scales Y (Desirability) and 5 (Narcissistic). On these two scales, the Medical Control participants returned significantly higher scores than the BPD participants. These results suggest that the two groups are characterised by considerable difference. It further suggests that the Medical Control group represents a satisfactory comparison group to the BPD group.
2. The BPD group returned significantly higher scores when compared with the Depressed Control group on three of the five DIB-R Scales (Cognition, Interpersonal Relationships, and Total Scaled Score), and on Scale C (Borderline) of the MCMI-III. The Depressed Control group returned a significantly higher MCMI-III Scale 7 (Compulsive) Scaled Score. On all other measures, no significant differences were detected between the BPD and Depressed Control groups. The differences between the groups were associated primarily with differences in BPD measures with the exception of the DIB-R Affect Scaled Score and the DIB-R Impulsive Scaled Scores. These findings suggest that the BPD and Depressed Control group have a similar mood-disorder profile, but differ on salient measures of BPD status. The similarities in mood-disorder profile between the BPD and Depressed

controls also has significant implications for the interpretation of the Stroop findings and will be more fully considered in Chapter Nine. Furthermore, the lack of significant difference on the DIB-R Impulse Scaled Scores is likely to have important clinical and the theoretical implications for understanding the nature of BPD. This issue will be further examined in Section 8.5.4 and again in the Discussion in Chapter Nine.

3. The Depressed Control group returned significantly higher scores than the Medical Control group on all measures with the exception of the DIB-R (Interpersonal Scaled Score) and the MCMI-III Scales 7 (Compulsive), T (Drug Dependence), and PP (Delusional Disorder). The Medical Control group returned significantly higher results than Depressed Controls on MCMI-III Scales Y (Desirability) and Scale 5 (Narcissistic). It is likely that the elevated scores in the direction of the Medical Control group for MCMI-III Scales Y and 5 are probably best interpreted as an attempt at dissimulation (denial of psychopathology) by this group.

In summary, there are a large number of significant differences between the Depressed Control group and the Medical Control group. These differences are observed in terms of both mood and personality variables. The differences are likely to reflect a greater degree of general morbidity on the part of the Depressed Controls. As a result, inclusion of the Depressed Control group probably represent good contrast groups for examining the effects of mood and personality in this study.

Overall, the current analyses provide sufficient empirical evidence to justify maintaining the groups as they are currently configured. The evidence clearly suggests that there are differences between the Medical Control group

and the BPD group on the one hand, and the Medical Control group and the Depressed Control group on the other. The data further suggests considerable diagnostic overlap between the BPD group and the Depressed Control group on salient Axis I variables, and a number of non-BPD Axis II dimensions. Significant differences were detected between the BPD and Depressed control group on salient BPD diagnostic variables including the two principal factors used to diagnose BPD – DIB-R Total Scaled Score and the MCMI-III Scale C (Borderline) Scaled Score. This finding is commensurate with the literature which has often reported diagnostic overlap and at times problematic differential diagnosis between depressive states and BPD (Gold & Silk, 1993; Paris, 1999). This is an ongoing debate with little prospect of adequate resolution in the short-term. For now, the best resolution involves recognising this phenomenon and controlling for it through the use of an adequate research design. This has been attempted in the present study.

8.5.2. Analyses of Hypotheses

Preliminary Analysis of Confounding Variables

A series of oneway ANOVAs were conducted on a number of the screening measures described in Section 8.4.3. These included the Positive and Negative Scales of the PANAS from Sessions Two and Three, as well as the NART, St Lucia, Digit Symbol subtest of the WAIS-R, and Medication Equivalence Scores. Table 8.10 reports the means and standard deviations for each group on the above listed dependent variables.

Table 8.10: Sample Means and Standard Deviations for Hypothesized Confounding Variables for BPD, Depressed, and Medical Control Groups

	BPD N = 18		DEPRESSED CONTROL N = 11		MEDICAL CONTROL N = 10		F STATISTIC
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
SESSION TWO PANAS NEGATIVE	22.33[a]	10.10	15.27[ab]	5.20	11.00[b]	1.15	8.01***
SESSION TWO PANAS POSITIVE	21.94[a]	6.80	24.09[ab]	8.93	29.40[b]	5.08	3.56*
SESSION THREE PANAS NEGATIVE	20.73[a]	9.15	15.00[ab]	8.02	10.9[b]	1.29	5.45**
SESSION THREE PANAS POSITIVE	23.87	8.19	22.09	9.39	30.2	7.36	2.73
MEDICATION EQUIVALENCE	2.44[a]	3.05	2.82[ab]	2.18	0.00[b]	0.00	4.43*
BED DAYS	19.67	29.62	21.91	24.57	0.00	0.00	2.72
NART FSIQ	112.71	5.37	112.27	5.87	114.80	4.94	0.67
ST LUCIA	6.48	4.40	8.64	6.36	4.90	2.92	1.69
QUICK TEST	97.53	10.72	100.36	6.80	103.20	7.90	1.22

df= 2,35; *p<0.05, **p<0.01, **p<0.001, ***p<0.0005; Differential superscripts demarcate the location of significant between-groups differences

The results of the analysis suggest that no significant differences were returned for the Bed Days, NART FSIQ, St. Lucia, Quick Test, and Session Three PANAS Positive Affect Scales. Significant differences were returned for Session Two PANAS Positive Affect Scales, Session Two and Three PANAS Negative Affect Scales, and for Medication Equivalence Ratings.

The results of this analysis can be summarized as follows. Significant differences in PANAS ratings were observed between the BPD group and the Medical Control Group on both positive and negative measures of affect. The BPD group returned greater ratings of negative mood, and lower ratings of positive mood than Medical Controls in Session Two. Of note, the BPD and Depressed Control group did not differ significantly from each other on measures of either negative or positive mood at Session Two. This finding suggests that the inclusion of a Depressed comparison group is important in assessing whether differences between groups are associated with the effects of BPD in contrast to the effects of depressed mood. The BPD group also returned significantly greater ratings of negative mood than Medical Controls in Session Three.

Second, the analysis of medication equivalence ratings suggested that the BPD and Depressed Control groups consumed significantly more sedating medication than the Medical Control Group. No significant differences in sedating medication use between the BPD and Depressed Control group was found.

Because both the BPD and the Depressed Control Group returned similar mood ratings in Sessions Two and Three, it was decided that covariate analysis would not be employed using mood at the time of testing as a covariate. Whilst this might be controversial, it is argued that the inclusion of a Depressed, non-

BPD comparison group represents an acceptable level of methodological control for the influence of mood at the time of testing. It is argued that if differences emerge between the Depressed Control group and the BPD, then it is unlikely that the difference could be accounted for by the effects of mood at the time of testing.

8.5.3. Analysis of Hypothesis One: Impaired Working Memory in BPD

A Variety of measures of memory were employed to test for evidence of impaired working memory in BPD. A series of oneway ANOVAs were employed to detect differences on measures of working memory between the BPD and the Depressed and Medical Control groups. Table 8.11 reports the means, standard deviations, and F statistics for the respective memory tests employed in the study.

Table 8.11: Sample Means, Standard Deviations and F Statistics for Working Memory Tasks for BPD, Depressed, and Medical Control Groups

	BPD N = 15		DEPRESSED CONTROL N = 11		MEDICAL CONTROL N = 10		F
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
VISUAL REPRODUCTION I	34.80	5.20	36.45	4.52	36.10	2.33	0.52
VISUAL REPRODUCTION II	27.93	11.27	33.18	7.30	33.50	4.20	1.71
LOGICAL MEMORY I	21.33	6.42	22.36	8.49	20.50	4.97	0.20
LOGICAL MEMORY II	16.80	5.94	16.64	8.09	17.00	4.71	0.008
PAIRED ASSOCIATES LEARNING I	14.47	6.57	16.73	3.98	15.10	3.90	0.61
PAIRED ASSOCIATES LEARNING II	6.13	2.30	6.64	1.43	6.70	1.34	0.38
VISUAL MEMORY SPAN	13.93	1.98	15.55	2.73	15.00	2.58	1.53
DIGIT SPAN SCALED SCORE	10.00	2.24	9.18	2.36	8.60	2.37	1.15

df=2,35; *$p<0.05$

The analysis of measures of working memory revealed no significant differences between the groups. Therefore, no post hoc analyses were conducted on the data. These findings suggest that BPD participants do not experience impairments in working memory.

8.5.4. Analysis of Hypothesis Two: Impaired Response Inhibition in BPD

The Stop-Signal paradigm was employed to directly test for evidence of impaired response inhibition in BPD. The data was analysed in the manner recommended by Logan (1994). First, Mean Reaction Time (MRT) scores were compared, and this was followed by an examination of Stop-Signal Reaction Times (SSRT). Because the SSRT is an averaged index of the ability of participants to inhibit behaviour averaged across all stop-signal delay conditions, a third level of analysis was also included which analysed the number of non-responses (NNR) of participants at each level of stop-signal delay.

Sample Means

Table 8.12 reports the means, standard deviations, and F statistics for the MRT, SSRT and for NNR at each level of stop-signal delay for BPD, Depressed, and Medical Controls.

Table 8.12: Stop-Signal Paradigm Mean Reaction Time (MRT), Stop-Signal Response Times (SSRT)and Number of Non-Responses at 0, 100, 200, 300, 400, and 500 mSecs Delay Means, Standard Deviations, and F Statistics for BPD, Depressed, and Medical Control Groups

	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	N = 18		N = 11		N = 10		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
Mean Reaction Time	560.61	110.44	587.82	160.40	533.80	132.94	0.44
Stop-Signal Reaction Time	156.27	84.91	189.45	126.58	172.91	37.56	0.47
Non-Responses@ 0mSec Delay	1.39	1.54	2.36	1.69	0.90	1.10	2.07
Non-Responses@ 100mSec Delay	3.28	3.04	4.09	3.33	2.80	1.99	0.54
Non-Responses@ 200mSec Delay	6.67	4.21	8.64	4.48	7.20	1.99	0.90
Non-Responses@ 300mSec Delay	11.28	4.98	12.55	3.72	13.50	1.65	1.03
Non-Responses@ 400mSec Delay	13.56	4.89	14.36	3.14	16.00	1.16	1.34
Non-Responses@ 500mSec Delay	13.89	5.09	14.73	3.41	17.00	1.05	2.00

df=2,35; *p<0.05

In view of the recommendations of Logan and Cowan (1984) and Logan (1994), these findings suggest that BPD participants did not return deficits in their capacity to inhibit responses. However, because SSRT returns represent an index score averaged across the six levels of stop-signal delay, an additional level of analysis examining the number of non-responses (NNR) at each level of stop-signal delay was also undertaken. One-way ANOVAS were then conducted on the frequency of non-responses at each level (0 mSec, 100 mSec, 200 mSec, 300 mSec, 400 mSec, and 500 mSec) of stop-signal delay. The analysis of the Stop-Signal Numbers of Non-Responses at all levels as well as the Stop-Signal Mean Reaction Time (MRT) revealed no significant differences between the groups. These findings suggest that response inhibition is not deficient in BPD.

BPD DIB-R Impulse Subscale Analysis of Stop-Signal Responses

Because impulsivity is thought to be a central diagnostic feature of BPD, a further analysis was conducted in order to examine whether there was evidence of differences in inhibition on the part of self-reported low-impulsive BPD participants versus high-impulsive BPD participants on the stop-signal task. BPD participants were allocated to a high-impulsivity or low-impulsivity DIB-R Impulse Group on the basis of their DIB-R Impulse Scale Raw Score. The DIB-R Impulse Scale Raw Score rather than DIB-R Impulse Scaled Scores were used because DIB-R Impulse Scaled Scores did not discriminate sufficiently between participants. The mean raw score for the group (7.39) was used rather than the median score for the group (8.0) because it permitted an easier allocation of participants to groups. Table 8.13 reports the means, standard deviations, and F statistics for the high and low DIB-R Impulse Subscale BPD sub-groups.

Table 8.13: High BPD DIB-R Impulse Subscale Sub-Group vs. Low BPD DIB-R Impulse Subscale Sub-Group on MRT and SSRT Scores

	BPD High N = 11		BPD Low N = 7		
MRT (mSecs)	Mean	SD	Mean	SD	F
	594.36	100.45	507.57	111.23	2.94
SSRT (mSecs)	Mean	SD	Mean	SD	F
	154.44	104.86	159.16	45.65	0.01

df=1, 14; *p<0.05

The analysis of a high BPD DIB-R Impulse Subscale versus a low BPD DIB-R Impulse Subscale group again revealed no significant differences between the groups. These findings suggest that despite differentiating BPD participants into high versus low impulsivity groups on the basis of DIB-R Impulse Raw Scale returns, no differences in the capacity to inhibit behaviour was observed.

Collectively, the non-significant results on MRT, SSRT, and NNR at all levels of Stop-Signal delay suggest that BPD is not characterised by deficits in response inhibition. These findings have considerable implications for a fundamental theoretical understanding of BPD, and will be elaborated upon extensively in Chapter 10. Because there were no significant differences between the groups, no further analyses of the data was undertaken.

Comparison of Stop-Signal Results with Similar Studies

One final analysis was undertaken which examined the comparability of returns on the Stop-Signal Task in this study with two other studies employing

somewhat similar measures of response inhibition (Dinn et al., 2004; Kunert et al., 2003). The Dinn et al. (2004) (Study One) and Kunert et al. (2003) studies reported Mean Reaction Time (Go data) on 'Go/NoGo' tasks, and these findings are reported in Table 8.14. Their studies did not report Stop-Signal Reaction Time (SSRT) in a manner that was directly comparable to the results returned in the present study. Because this data is a cross-study comparison, the means and standard deviations for Dinn et al. (2004) (Study One), Kunert et al. (2003), and the present study are reported.

Table 8.14: Comparison of the Means for Mean Reaction Time Data (Go Data) for the Current Study and the Kunert et al. (2003) and Dinn et al. (2004) Studies

CURRENT STUDY

	BPD	DEPRESSED CONTROL	MEDICAL CONTROL
Mean MRT Scores (mSecs)	560.61	587.82	533.80

KUNERT et al. (2003) (mSecs)

BPD		CONTROL	
MEAN	S.D.	MEAN	S.D.
529.70	52.40	544.60	68.70

DINN et al. (2004) (Study One) (mSecs)

	BPD		CONTROL	
	MEAN	S.D.	MEAN	S.D.
Condition One	340.00	83.10	296.00	40.7
Condition Two	513.00	106.80	444.00	36.70
Condition Three	508.00	78.30	425.00	44.90

This data suggests there are similarities in at least one component of the respective stop-signal and Go/NoGo tasks – the speed of initial response to the 'go-task' signal for the Kunert et al. (2003), and two of the three conditions of the Dinn et al. (2004) clinical study. The similarity of these results provides some validity to the findings returned in the present study.

8.5.5. Analysis of Hypothesis Three: Impaired Affective Attentional Bias in BPD

The Stroop Task was employed to test for evidence of 'affective-attentional bias' in BPD. The data was analysed in two ways. First, a between-groups colour-naming response latency analysis was undertaken. This was conducted using a series of oneway ANOVA's between the BPD, Depressed, and Medical Control groups. Second, a between-groups 'interference analysis' was undertaken in order to control for disruption due to the general effects of emotional words which tends to affect all groups (J. M. G. Williams & Broadbent, 1986), and as a test of emotional interference.

Comment is offered here regarding the decision to employ oneway ANOVA in contradistinction to two-way ANOVA. It is understood that given the design of the study that a more technically correct analysis would involve the use of a 3x6x2 ANOVA, but there are two issues that contraindicate the employment of this mode of analysis. First, the sample size employed in the current study suggested that it was highly unlikely that a 3x6x2 ANOVA would realise significant differences. Second, within-group means for each category of affect at both supraliminal and subliminal stimulus presentations were very similar. This observation suggested that it would be highly unlikely that any significant interaction effects would have been returned by using a 3x6x2 ANOVA. In

addition, a number of other studies have been conducted on similar data sets with BPD samples employing t-tests (Kunert et al., 2003), ANCOVA (Sprock et al., 2000), and non-parametric univariate analyses (Wilcoxon Rank Sum Test) (Swirsky-Sacchetti et al., 1993). For these reasons, a decision was taken to analyse the data using oneway ANOVA.

Analysis of Colour-Naming Response Latencies: Preliminary Data Management

Prior to the analysis of Stroop data, the data set was adjusted in order to control for the possible effects of random interference with the recording process. This included the effects of such factors such as throat-clearing or other non-intentional responses. The data was adjusted using the following procedure.

1. For each affect category, the Mean Reaction Time by group was established for presentations at both 2000 and at 240 mSec latencies. Where an individual colour-naming response for any word was more than three standard deviations from the mean in either direction, it was replaced with the mean score for the specific word for the particular group the participant belonged to. This procedure was conducted separately for each word for each group of participants.
2. Each participants mean score for each affect category at each level of presentation (2000 mSecs and 240 mSecs) was then calculated.
3. The procedure was repeated for all affect categories (Anger, Sadness, Shame, Anxiety, Neutral, and Joy) at both 2000 and 240 mSecs.

This resulted in Mean replacement group data for each affect category at both 2000 and 240 mSecs.

Sample Means

Table 8.15 reports the means and standard deviations and F Tests for Stroop Colour-Naming Response Latencies for each of the three groups in each of the six categories of affect (Anger, Sadness, Fear, Shame, Neutral, and Joy) at two levels of stimulus presentation (2000 mSec and 240 mSec).

Table 8.15: Means, Standard Deviations, and F Statistics for Stroop Colour-Naming Response Times for BPD, Depressed, and Medical Control Groups at Supraliminal (2000 mSec), and Subliminal (240 mSec) Presentation

	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	N = 18		N = 11		N = 10		
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
ANGER @ 2000 mSecs	951.00 [a]	306.58	722.95[b]	152.40	727.00[ab]	144.40	4.42*
SADNESS @ 2000 mSecs	987.33[a]	339.08	728.73[b]	131.29	705.70[b]	137.30	5.54**
ANXIETY @ 2000 mSecs	981.33[a]	395.14	734.00[a]	142.79	708.10[a]	91.29	4.03*
SHAME @ 2000 mSecs	1036.28[a]	400.32	711.27[b]	175.59	696.20[b]	92.73	6.20**
JOY @ 2000 mSecs	1049.82[a]	369.37	740.71[b]	177.47	732.17[b]	168.21	5.92**
NEUTRAL @ 2000 mSecs	965.61[a]	307.20	727.64[b]	137.41	681.7[b]	93.50	6.06**
ANGER @ 240 mSecs	1007.67[a]	316.32	684.64[b]	115.24	739.50[b]	169.08	7.49**
SADNESS @ 240 mSecs	1011.11[a]	368.12	739.27[b]	146.64	720.50[b]	118.75	5.21*
ANXIETY @ 240 mSecs	965.13[a]	284.34	725.64[b]	180.28	704.30[b]	128.60	5.91**
SHAME @ 240 mSecs	977.50[a]	318.68	698.73[b]	171.25	782.20[ab]	129.89	4.89*
JOY @ 240 mSecs	898.36[a]	205.08	749.09[a]	141.79	753.90[a]	145.31	3.41*
NEUTRAL @ 240 mSecs	978.17[a]	251.19	737.27[b]	146.02	754.90[b]	165.16	6.18**

df=2,35; *p<0.05, **p<0.01; Differential superscripts demarcate the location of significant between-groups differences

The findings of this level of analysis can be summarized as follows:

For Stimulus Words Presented Supraliminally (2,000 mSecs)

The BPD group returned significantly longer Colour Naming Response Latencies when compared to Medical Controls on four of the six categories of affect (Sad, Shame, Joy, & Neutral) and on five of the six categories of affect when compared to Depressed Controls (Anger, Sad, Shame, Joy, & Neutral). There were, however, anomalous data returns with regard to the multiple comparison analysis for Anxiety words at the 2,000 mSec presentation. The F-test returned a significant between groups score ($F = 4.03$; $p < 0.05$). The subsequent multiple comparison analysis reported no significant differences between the groups. The Medical Control and the Depressed Control groups were not significantly different from each other on any of the six affect categories. In summary, the findings of this component of the study suggest that BPD participants took significantly longer to colour-name stimuli than either the Depressed or Medical Controls.

For Stimulus Words Presented Subliminally (240 mSecs Followed by a Masking Condition)

The BPD group returned significantly longer response times when compared to Medical Controls on four of the six categories of affect (Anger Sad, Anxiety, & Neutral) and on five of the six categories of affect when compared to Depressed Controls (Anger, Sad, Anxiety, Joy, & Neutral). There were, however, anomalous data returns with regard to the multiple comparison analysis for Joy words at the 240 mSec presentation. The F-test returned a significant between groups score ($F = 3.41$; $p < 0.05$). The subsequent multiple comparison analysis reported no significant differences between the groups. The Medical Control and

the Depressed Control groups were not significantly different from each other on any of the six affect categories at either level of stimulus presentation. In summary, the findings of this component of the study suggest that BPD participants took significantly longer to colour-name stimuli than either the Depressed or Medical Controls.

The analysis of Colour-Naming Response Latency Stroop data indicated that there were consistent differences between BPD and the Depressed Controls across five categories of affect at both the 2000 mSec and 240 mSec presentation. Significant differences were also returned between the BPD and Medical Controls on four of the six categories at both the 2000 mSec and 240 mSec presentations.

Colour-Naming Interference Analysis

A second level of Stroop analysis known as an 'Interference Analysis' was subsequently conducted in order to control for disruption due to the general effects of the emotionality of the word stimuli (J. M. G. Williams & Broadbent, 1986). This analysis was conducted using the following approach:

1. For each case, mean scores were calculated for each affect category at both the supraliminal and subliminal levels. This process yielded a total of 12 separate mean scores at two levels of stimulus presentation (2000 mSecs, and 240 mSecs respectively);
2. For the affect categories of Anger, Sad, Shame, Anxiety, and Joy, the mean Neutral score associated with the same level of stimulus presentation (the mean 2000 mSec Neutral score for words presented at the supraliminal level, and the mean 240 mSec Neutral score for words presented the subliminal level) was subtracted from each participant's mean score for all other

categories of affect. This yielded a series of 10 adjusted affect scores (Anger, Sad, Shame, Anxiety, and Joy at 2000 mSecs and 240 mSecs respectively) for each participant.

These adjusted affect scores were then subjected to a between groups re-analysis using oneway ANOVA. Table 8.16 reports the means and standard deviations for the modified Stroop Interference Scores for the five categories of affect (Anger, Sad, Shame, Anxiety, and Joy) at two levels of stimulus presentation (2000 mSecs and 240 mSecs).

Table 8.16: Stroop Interference Scores Means, Standard Deviations, and F Statistics for BPD, Depressed and Medical Controls for Supraliminal (2000 mSecs) and Subliminal (240 mSec) Presentations

	SUPRALIMINAL PRESENTATIONS (2000 mSecs)						
	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
ANGRY	-15.00	306.58	45.00	144.40	-5.00	152.39	0.22
SAD	21.33	339.08	23.40	137.00	0.73	131.29	0.37
SHAME	70.28	400.32	4.20	104.90	-16.73	175.59	0.34
ANXIETY	30.89	394.17	26.10	91.29	6.00	142.79	0.03
JOY	83.78	369.51	50.20	168.24	71.55	257.81	0.04
	SUBLIMINAL PRESENTATIONS (240 mSecs)						
	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
ANGRY	29.83	316.28	-15.50	169.08	-52.36	115.24	0.41
SAD	82.39	467.94	-34.50	118.75	2.27	146.64	0.44
SHAME	-0.50	318.68	27.20	129.89	-38.27	171.25	0.19
ANXIETY	-12.89	284.32	-50.70	128.60	-11.36	180.28	0.11
JOY	-5.44	366.19	-1.10	148.58	12.09	141.79	0.01

df:2,35; *p<0.05

The results of this analysis did not reveal significant differences between the groups, suggesting an absence of affective interference on the Stroop task. The findings of this component of the study suggest that there were significant between-groups colour-naming response latency differences, and a non-significant between-groups 'interference effect'. The implications of these findings will be explored in greater detail in Chapter Nine.

8.5.6. Analysis of Hypothesis Four: Impaired Problem Solving in BPD

A variety of measures were employed to test for evidence of impaired problem-solving in BPD. A series of between-groups oneway ANOVAs were conducted to test for differences on all measures of problem solving between the BPD, Depressed and Medical Control groups with the exception of the Austin Maze analyses.

The Austin Maze analyses were conducted independently of the analyses of the other problem-solving data because of the requirements for a repeated-measures analysis. Because of the large number of trials conducted on a comparatively small number of participants (and therefore the inherent risk of Type I error), it was decided to conduct a 3x3 Repeated Measures ANOVA (Groups by Time). The time points included in the analysis included trials one, five and 10 of the task.

Sample Means

Table 8.17 reports the means, standard deviations, and F statistics for the problem-solving tests employed in the study. Table 8.18 reports the means, standard deviations, and F statistic for Trials One , Five, and 10 of the Austin Maze.

Table 8.17: Sample Means, Standard Deviations and F Statistics for Problem Solving Tasks for BPD, Depressed, and Medical Control Groups

	BPD N = 15		DEPRESSED CONTROL N = 11		MEDICAL CONTROL N = 10		F
	Mean	S.D.	Mean	S.D.	Mean	S.D.	
DIGIT SYMBOL SCALED SCORE	9.60	2.75	9.91	2.77	11.20	1.75	1.27
FAS-F	14.20	4.13	12.45	4.06	12.90	3. 07	0.73
FAS-A	12.07	3.39	10.73	3.69	10.90	3.18	0.60
FAS-S	14.87	4.69	13.18	4.75	14.90	3.96	0.54
FAS-ANIMALS	21.13	4.44	21.18	6.01	23.20	2.20	0.73
SIMILARITIES SCALED SCORE	8.13	3.07	8.91	1.92	9.00	1.16	0.54
REY FIGURE I	33.00	3.00	32.73	3.69	32.50	3.98	0.06
WCST NUMBER CATEGORIES	4.67	1.95	5.64	1.21	5.90	0.32	2.60
WCST TRIALS TO COMPLETE FIRST CATEGORY	24.93	31.71	11.91	2.43	11.10	0.74	1.84
TOWER OF LONDON	27.20	11.12	33.64	10.43	31.20	12.60	1.07
TOWER OF HANOI	25.40	19.86	34.00	23.66	31.70	20.80	0.57

df: 2,35; *p<0.05

Table 8.18: Sample Means, Standard Deviations and F Statistics for Austin Maze Trials One, Five, and 10 for BPD, Depressed, and Medical Control Groups

	BPD		DEPRESSED CONTROL		MEDICAL CONTROL		F	
	N = 15		N = 11		N = 10			
	Mean	S.D.	Mean	S.D.	Mean	S.D.	TIME	31.27*
TRIAL I	18.20	8.21	14.27	4.32	14.10	3.72	TIME X	
TRIAL V	10.47	10.1	3.73	3.35	5.60	5.04	GROUP	0.77
TRIAL X	6.60	9.10	1.64	2.88	3.50	3.50		

df: 2,35; *p<0.0005

The analyses of problem-solving tasks reported in Table 8.17 all returned non-significant findings. The repeated measures ANOVA conducted on the Austin Maze data returned a significant effect for Time, but a non-significant Group x Time interaction. Viewed collectively, these findings do not support the view that BPD participants experience deficits in problem-solving ability.

8.5.7. Subsidiary Analyses

A number of subsidiary analyses were also conducted in order to address specific issues of methodological concern that might otherwise compromise the integrity of the study. Two issues were identified that required further investigation. These issues included a consideration of diagnostic differences between Study Two and Study Four BPD cohorts, and reanalyzing the data from the current study with those Depressed controls returning high DIB-R scores (scaled scores greater than eight) eliminated from the re-analysis.

Comparison of Study Two and Study Four BPD Participants

A subsidiary issue that will be considered in greater detail in Chapter Nine concerns the comparability of the BPD samples between Studies Two and the present study. This is an important issue because the BPD group employed in Study Two (Affective and Semantic Representations in BPD) was used to elicit information concerning salient affective experience. This data was then employed to develop the Stroop task employed in the present study. Therefore, if there were significant differences in the nature of the respective BPD samples, this might constitute a methodological flaw whereby the affective experiences presumed to be relevant for Study Four participants might not in fact be the case.

As a result, it was decided to compare the diagnostic data for the BPD samples for Studies Two and Four. Because the means and standard deviations

for these samples have been reported previously (Table 5.3; Tables 8.7 to 8.9 inclusive), they will not be re-reported here. Table 8.19 reports the oneway ANOVA's on DIB-R Scaled Scores, MCMI-III Validity Scales, Clinical Personality Patterns, and Clinical Syndrome returns for Study Two and Study Four BPD Groups.

Table 8.19: Study Two and Study Four DIB-R and MCMI-III Scaled Score BPD Oneway ANOVA's

	F
DIB-R AFFECT SCALED SCORE	0.50
DIB-R COGNITION SCALED SCORE	0.09
DIB-R IMPULSE SCALED SCORE	0.31
DIB-R INTERPERSONAL SCALED SCORE	0.01
DIB-R TOTAL SCALED SCORE	0.002
SCALE X (DISCLOSURE)	0.09
SCALE Y (DESIRABILITY)	7.64*
SCALE Z (DEBASEMENT)	3.81
SCALE 1 (SCHIZOID)	2.25
SCALE 2A (AVOIDANT)	1.78
SCALE 2B (DEPRESSIVE)	2.16
SCALE 3 (DEPENDENT)	0.74
SCALE 4 (HISTRIONIC)	1.96
SCALE 5 (NARCISSISTIC)	0.57
SCALE 6A (ANTISOCIAL)	2.05
SCALE 6B AGGRESSIVE)	2.83E-05
SCALE 7 (COMPULSIVE)	0.31
SCALE 8A (PASSIVE-AGGRESSIVE)	0.001
SCALE 8B (SELF-DEFEATING)	1.82
SCALE S (SCHIZOTYPAL)	0.09
SCALE C (BORDERLINE)	1.70
SCALE P (PARANOID)	0.04
SCALE A (ANXIETY)	2.13
SCALE H (SOMATOFORM)	0.06
SCALE N (BIPOLAR)	1.58
SCALE D (DYSTHYMIA)	0.34
SCALE B (ALCOHOL DEPENDENCE)	1.55
SCALE T (DRUG DEPENDENCE)	0.79
SCALE R (PTSD)	0.03
SCALE SS (THOUGHT DISORDER)	0.09
SCALE CC (MAJOR DEPRESSION)	1.14
SCALE PP (DELUSIONAL DISORDER)	0.69

df: 1,26; *p<0.05

The results of these analyses suggest a similar diagnostic profile for both BPD samples. The only scale that returned significant differences was Scale Y (Social Desirability) suggesting that participants in Study Two were more likely to present their responses in a favorable light when compared with Study Four participants. The implications of these findings will be considered in greater detail in Chapter 10.

Elimination of 'High DIB-R' Depressed Control Participants

Section 8.4.1 indicated that some Depressed Control participants might potentially return a DIB-R Total Scaled Score of eight or more, suggesting that they might in fact meet BPD criterion. This issue was addressed in part by clarifying that the study criterion for BPD involved an MCMI-III Scale C Scaled Score of 85 and a DIB-R Total Scaled Score of Eight or more. Despite this, half (n=5) of the Depressed Control sample returned DIB-R Total Scaled Scores in excess of eight. Because of the potential confounding effect of having Depressed Controls with a DIB-R Total Scaled Score in excess of eight, it was decided to re-execute all of the analyses reported above with those Depressed Control participants with 'high' DIB-R Total Scaled Scores (i.e., eight or more) eliminated from the re-analyses. Therefore, five of the initial 10 Depressed Control participants were eliminated from the reanalysis. New between-groups analyses of Working Memory, Stop-Signal, Stroop, and Problem-Solving were conducted on the BPD and Medical Control, and a modified Depressed Control Group.

The reporting of all of the re-analyses is too voluminous to include here. The findings however, were entirely consistent with the findings previously reported. In summary, the re-analyses found no significant differences between

the groups on measures of Working Memory, Stop-Signal Mean Reaction Time (MRT), Stop-Signal Reaction Time (SSRT), Stop-Signal Number of Non-Responses (NNR), or on any of the Problem Solving Measures. The Stroop re-analyses returned the same significant Between Groups differences on Colour-Naming Response Latencies, and non-significant differences on a subsequent Interference Analysis.

These analyses suggest that the elimination of Depressed participants with high DIB-R Total Scaled Scores did not alter the findings of the study reported in the previous sections. No evidence was found to indicate that the inclusion of these participants in the Depressed control group confounds the findings of the study as originally reported.

8.6. CONCLUSIONS

The findings of the study can be summarised as follows:

1. Three BPD subgroups were recruited into the study. Because the mode of recruitment of each of the sub-groups was different, a preliminary analysis was conducted in order to establish whether there were significant diagnostic differences between the groups.
2. The BPD subgroup analysis demonstrated sufficient diagnostic homogeneity to permit the collapsing of the three subgroups into one group for between-groups analysis with the Depressed and Medical Control Groups.
3. Diagnostic analysis of the BPD, Depressed Control, and the Medical Control Groups demonstrated sufficient diagnostic heterogeneity to permit analysing them as separate groups.

4. Analysis of Hypothesis One – Impaired Working Memory in BPD - revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups.
5. Analysis of Hypothesis Two – Impaired Response Inhibition in BPD – revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups.
6. Analysis of Hypothesis Three – Impaired Affective Attentional Bias in BPD – revealed significant and consistent differences between the BPD group and the Depressed Control Group on the one hand, and the BPD and the Medical Control Group on colour-naming response latencies. No differences were found between the Depressed Control Group and the Medical Control Group on this measure. This finding provides some support for the hypothesis of affective-attentional bias in BPD.
7. An 'Interference Analysis' of Stroop scores returned no significant differences between the groups suggesting that all groups were equally disrupted by the emotionally valence of stimulus words.
8. Analysis of Hypothesis Four – Impaired Problem Solving in BPD - revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups.

The results of this study will be considered in detail in Section V. The theoretical, clinical, and policy implications of the findings will be reviewed within the context of evaluating the methodological limitations of the studies which comprise this thesis.

SECTION V: DISCUSSION

CHAPTER NINE: DISCUSSION

9.1. OVERVIEW

This chapter commences by briefly reiterating the developmental neuropsychological model of impaired executive functioning outlined in Chapter Three (Section 9.2). The chapter then summarises the findings of the project, placing these findings within the context of the established body of knowledge on BPD, and then identifies the implications for future research and practice. In order to accomplish this, Section 9.3 provides a summary of the findings of the studies and Section 9.4 provides an interpretation of the hypotheses and principal findings of the project. Section 9.5 provides a conceptual and methodological review of the project, and Section 9.6 considers some of the implications for future research. Section 9.7 considers the implications for clinical practice, and Section 9.8 addresses the public policy/early intervention implications of the study findings. Section 9.9 offers concluding remarks and some reflections with regard to the contextual and philosophical issues concerning BPD.

9.2. A MULTIDIMENSIONAL DEVELOPMENTAL NEUROPSYCHOLOGICAL THEORY OF EXECUTIVE DISORDER IN BPD

A multidimensional developmental neuropsychobiological model of BPD was proposed. The model suggested that BPD involves a number of impaired executive functions including working memory, behavioural inhibition, affective-attentional bias, and complex-problem solving ability. These impaired executive functions were argued to represent the cognitive manifestations of underlying deficits in orbitofrontal-corticolimbic pathways of the Central Nervous System (CNS). These deficits were thought to occur as a result of the

influence of a number of independent risk factors that included a genetic and psychobiological predisposition to BPD, early loss and/or separation, parental and/or family psychopathology, impaired parental bonding and/or attachment pathology, and trauma usually in the form of child abuse and/or neglect. The interaction of these factors was argued to result in the failure of an 'experience-dependent' maturation of orbitofrontal-corticolimbic networks. The failure of these networks to mature appropriately was argued to result in the neuropsychological architecture of BPD and the resultant impaired executive impairments hypothesised to characterise the disorder.

The proposed model argued that the executive functions of working memory, behavioural inhibition, affective-attentional bias (affect regulation), and problem solving shared interdependent relationships with each other, and acted in a 'co-operative' or 'seamless' fashion in order to effectively regulate transactions between the person and the environment. Impairment in one domain of executive functioning was argued to have the potential to contribute to impairment in other domains of executive functioning. For example, the inability to effectively regulate affective states was argued to result in episodes of affect dysregulation which in turn could provoke behavioural dysregulation which in turn could provide the basis for 'impulsive' acting out. Similarly, failure to successfully execute a problem solving sequence could lead to affective dysregulation which in turn could lead to 'impulsive' behavioural enactments as a means of restabilizing a dysregulated affective-attentional system.

The proposed model did not assume one predominant causal pathway for BPD. Instead, BPD was viewed as a final common pathway for a number of independent risk factors. The proposed model was also argued to be consistent

with the cognitive perspectives of Beck (A. Beck et al., 1990), Young (Young, 1990), as well as the biosocial-cognitive perspective of Linehan and colleagues (Heard & Linehan, 1993; Linehan, 1993; Linehan et al., 1991; Wagner & Linehan, 1997). The model was also argued to be consistent with the various evidence-based psychoanalytic theorists who emphasise identity diffusion (Clarkin et al., 2004; Clarkin et al., 1999), disturbed self-systems (Monsen et al., 1995; J. Stevenson & Meares, 1992, 1999), or attachment-based difficulties (Bateman & Fonagy, 1999, 2004) as central causal explanations for the genesis of BPD.

9.3. SUMMARY OF THE FINDINGS OF THE STUDIES

The significant findings from each of the studies comprising this project are briefly summarised below.

9.3.1. Study One: Validity Study of the MCMI-III

The objective of the first study was to assess whether Scale 'C' of the Millon Clinical Multiaxial Inventory (MCMI-III) was a valid measure for detecting BPD. This was regarded as a crucial issue in the development of the sequence of reported studies as the MCMI–III was employed throughout the project as an 'instrument of first detection' for identifying BPD.

As a result, the first study conducted in this project consisted of assessing the validity of the Borderline Scale (Scale C) of the MCMI-III. This was done by prospectively recruiting patients awaiting psychotherapy from the author through a clinical psychology clinic at Fremantle hospital. Each consecutively presenting patient was administered the MCMI-III (Millon et al., 1994), and the Diagnostic Interview for Borderlines Revised (DIB-R) (Zanarini et al., 1989). The administration of these instruments was conducted in such a manner that the

interviewer was blind to the participants' scores on either instrument at the time the instruments were administered. The findings of this study suggested an acceptable validity for Scale C of the MCMI-III, with the data suggesting that it had both the capacity to correctly identify cases of BPD and correctly reject non-BPD cases. This finding justified the continued use of the MCMI-III throughout the project as an 'instrument of first detection' for diagnosing BPD.

9.3.2. Study Two: Affective and Semantic Representations in BPD

The objective of this study was to identify specific affective categories for inclusion in an 'Emotional Stroop' task. This was undertaken by interviewing 11 BPD participants and identifying specific categories of affective experience. All participants were required to confirm the experience of the affect category for it to be endorsed for use in the project.

One of the more important findings of this study was the response of BPD participants to the receipt of advice confirming their BPD status. Participants reported positive experiences with regard to the provision of diagnostic information on BPD, and indicated that it was a beneficial experience to be advised of this diagnosis. This finding has implications for diagnosis and assessment, and will be commented upon further in Section 9.7.1.

The primary outcome of this study was that a total of four affect constructs were reliably reported across all 11 participants. These affects were: Anger-Rage, Distress-Anguish (Sadness), Shame-Humiliation, and Fear-Terror. These constructs were then employed in a subsidiary study (Affect Category Judgement Task) in order to elicit specific word representations of each of these generic constructs. Furthermore, it was also decided to include categories of Neutral affect in order to conduct an 'interference analysis' (J. M. G. Williams et

al., 1996), and Joy words were also included in order to more fully examine an affective-attentional bias hypothesis in BPD.

Another significant finding from this study was the identification of 'affect blends'. This phenomenon refers to experiences reported by a number of participants of multiple, co-occurring negative affects. These often appeared to be associated with specific, adverse experiences. This finding suggested that BPD participants might experience a more general regulatory impairment which transcends or overrides difficulties with specific, discrete affects. An alternative interpretation of this data suggests the regulatory impairment this data is accessing is more associated with the regulation of arousal rather than specific affects or specific categories of emotion. This observation further suggests that borderlines might experience a general affect regulatory deficit which represents an overlearned response to change or novelty in the social environment, or alternately, reflects the operation of a highly sensitive 'orienting response' in BPD (Sokolov, 1963).

The issue of 'affect blends' is also important because it has the potential to challenge the orthodoxy of the use of 'interference analyses' in Stroop tasks. Interference analyses essentially rely on the presupposition that affect categories are discrete entities, and can be easily distinguished from one another. If this is incorrect, then the capacity for robust and reliable performance of interference analyses on the Stroop task is rendered suspect. This issue will be further addressed in Section 9.4.4.

9.3.3. Study Three: Affect Category Judgement Task

The objective of this study was to develop word lists for each of the nominated affect categories for inclusion in an 'Emotional Stroop' paradigm.

Chapter Five identified the categories of Anger-Rage, Distress-Anguish (Sadness), Fear-Terror, and Shame-Humiliation, as the salient affective states reported by borderline participants. In addition, it was decided to include Joy words as a result of K. F. Stein's (1996) study in which she observed 'rapid cycling' of affective experience in BPD. Neutral words were also included in order to calculate an 'interference' index (J. M. G. Williams et al., 1996). The 'Affect Category Judgement Task' (ACJT) was developed in order to specify and create word lists which reflected the affect categories of Anger, Sadness, Anxiety, Shame, Neutral, and Joy, and was derived from the Dictionary of Affect in Language (DAL) (Sweeney & Whissell, 1984; Whissell, 1989). This task derived 10 words for each category of affect resulting in a total of 60 words that were incorporated into the Stroop Task.

9.3.4. Study Four: Assessment of Executive Function in BPD

The objectives of the project were examined through a number of hypotheses that formed the basis of Study Four. This study found limited support for the original hypotheses, and the findings of the study can be summarised as follows:

1. Three BPD subgroups were recruited into the study. Because the mode of recruitment of each of the sub-groups was different, a preliminary analysis was conducted in order to establish whether there were significant diagnostic differences between the groups.
2. The BPD subgroup analysis demonstrated sufficient diagnostic homogeneity to permit collapsing the three subgroups together to form one BPD group. A subsequent between-groups diagnostic analysis was conducted with the BPD, Depressed, and Medical Control Groups.

3. Diagnostic analysis of the BPD, Depressed Control, and the Medical Control Groups demonstrated sufficient diagnostic heterogeneity to permit maintaining them as separate groups.
4. Analysis of Hypothesis One – Impaired Working Memory in BPD - revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups.
5. Analysis of Hypothesis Two – Impaired Response Inhibition in BPD – revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups.
6. Analysis of Hypothesis Three – Impaired Affective Attentional Bias in BPD – revealed significant and consistent differences between the BPD group and the Depressed Control Group on the one hand, and the Medical Control Group on the other in relation to colour-naming response latencies. No differences were found between the Depressed Control Group and the Medical Control Group on these measures. This finding provided some limited support for the hypothesis of an affective-attentional bias in BPD.
7. An 'Interference Analysis' of the Stroop returns was then conducted by subtracting Neutral scores from each of the other five categories of affect for each participant in each group. The subsequent between-groups analysis returned no significant differences between the groups suggesting that the groups were equally disrupted by emotional valence of the stimulus words.
8. Analysis of Hypothesis Four – Impaired Problem Solving in BPD - revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups.

9.4. INTERPRETATION OF HYPOTHESES AND THE PRINCIPAL FINDINGS OF THE PROJECT

This section examines and synthesises the principal findings of Study Four. The aim of this section is to interpret the data in order to explicate new theoretical understandings concerning BPD. This will assist in further considering the clinical, experimental, and public policy implications of the findings in Sections 9.6 to 9.8. In order to examine the implications of the data, a variety of different interpretations of the data will be offered, and a critical analysis of each will be provided. As a result, the interpretations that will be offered will be consistent with what is currently understood with regard to the developmental neuropsychological perspectives on BPD.

9.4.1. Impaired Working Memory in BPD

There is a limited experimental literature suggesting impaired general memory as well as impaired working memory in borderlines. This study provided no support for the hypothesis that borderlines experience any form of impairments to memory, or of any form of impaired memory system. These findings suggest that whatever difficulties borderlines may experience, their difficulties are not associated with impairments in working memory.

9.4.2. Impaired Response Inhibition in BPD

Impulsivity is considered to be one of the hallmarks of BPD (Siever & Davis, 1991; Zanarini, 1993; Zanarini, Dubo, Lewis, & Williams, 1997). This phenomenon is considered so central to the condition that impulsivity is a key diagnostic criterion in the DSM-IV (American Psychiatric Association, 1994), DSM-IV-TR (American Psychiatric Association, 2000), ICD-10 (A. W. Loranger et al., 1997), Gunderson (Gunderson, 1994; Gunderson et al., 1981; Gunderson

& Singer, 1975), and Kernberg diagnostic systems (Kernberg, 1967, 1975, 1984). The study examined whether the capacity to inhibit responding on a forced choice reaction time task was impaired in BPD. Along with the findings reported by Dinn et al. (2004), Kunert et al. (2003), and Leyton et al. (2001), Hypothesis Two represents one of the first attempts to examine response inhibition in BPD.

This analysis revealed no significant differences between the BPD, Depressed Control, and Medical Control Groups. The data clearly indicated that borderline participants demonstrated levels of response inhibition comparable with controls. This suggests that borderlines cannot be considered 'impulsive' in the manner which they have previously been considered. This finding challenges the long held assumption of impulsivity in BPD, and is highly consistent with the findings reported by Kunert et al. (2003), and is at variance with aspects of the findings reported by Dinn et al. (2004) and Leyton et al. (2001). The implication of this finding suggests that there is an urgent need to reconsider the current theoretical understanding of impulsivity in BPD.

This finding is clinically and theoretically highly significant. The Stop-Signal results are significant because they, along with Kunert et al. (2003), cast doubt on one of the major assumptions of BPD. The assumption that impulsivity is a central feature of borderline pathology has in part shaped clinical theory and practice for many decades. The findings of this study suggest that what has hitherto been understood as 'impulsive' behaviour might now need to be accounted for by some other method of explanation (Zanarini et al., 1997).

This finding is also at variance with the view suggesting there is impaired 'frontal' function in BPD. The role of the frontal lobes in mediating response

inhibition is a well documented phenomenon (Fuster, 1989; Lezak, 1995; Schore, 2003a, 2003b). Accordingly, the absence of evidence of inhibitory impairment provides further empirical evidence suggesting adequate frontal function in BPD. This finding is also consistent with the negative findings reported for the examination of problem-solving capacity as reflected in the tests examining Hypothesis Four.

The absence of deficits in response inhibition in the BPD group requires the concept of impulsivity to be reconsidered. Section 2.2.3 identified five generic, interrelated uses of the term 'impulsive'. They included:

1. The inability to stop or inhibit a prepotent behavioural action or sequence;
2. The use of behavioural sequences occurring in a social or interpersonal context that have either a low probability of controlling or managing environmental variables on the one hand, or are not 'ecologically valid' on the other;
3. The use of various behaviours that are used to regulate emotional states when there is an absence of a more mature or functional mode of regulation available to the person;
4. Evidence of some form of subtle brain impairment suggestive of 'frontal-lobe' involvement, or;
5. The employment of so-called 'mindless' behaviour which is 'irrational' and not amenable to logical explanation.

The findings of this study clearly indicate that the first and fourth explanations – an inability to inhibit a prepotent response, and evidence for subtle brain impairment suggestive of 'frontal-lobe' involvement - are not supported by the stop-signal evidence. Therefore, alternative explanations of so-

called ‘impulsive’ behaviour need to be considered. Some of the more the salient possibilities are considered below.

Explanation One: ‘Response Inhibition’ is an Artifact of Impaired Affect Regulation

There is an abundant clinical literature (Grotstein, 1987; Linehan & Heard, 1992; Westen, 1991; Wilkinson-Ryan & Westen, 2000) and an emerging experimental literature (Arntz et al., 2000; Levine et al., 1997; K. F. Stein, 1996; M. I. Stern et al., 1997; Yen et al., 2002) confirming difficulties in affect regulation in BPD. Whilst this literature has alluded to the existence of behavioural dysregulation in borderlines, there has rarely been reference to an explicitly articulated relationship between affect regulation and behavioural dyscontrol in the literature. The exceptions to this include Zanarini (1997) who hypothesised that behavioural dyscontrol in BPD might reflect an underlying affective disturbance, and Linehan (1993) who argued that parasuicidal acts are often employed as a method for regulating dysphoric affect..

The explanation offered here suggests that there is a direct relationship between the ability to regulate affect on the one hand, and the capacity to inhibit behaviour, on the other. This explanation specifically suggests that so-called ‘impulsive’ behaviour might represent the behavioural component of attempts by borderlines to regulate arousal and/or affective states. This explanation further suggests that when borderline patients experience intense levels of affect or arousal, dysregulation occurs, and behavioural enactments represent one class of activity borderlines utilise in order to re-regulate arousal and/or internal affective states. Therefore, what is often regarded as ‘impulsive’ behaviour might represent the behavioural referent of an arousal or affect regulation impairment.

If this speculation is correct, it necessitates a radical re-evaluation of behavioural dyscontrol syndromes in BPD. More importantly, it suggests that these behaviours might now be understood in the context of their role in affect regulation and re-regulation. This mode of understanding has a long tradition in clinical psychology and psychotherapy, and allows a number of therapeutic approaches to be implemented in the management of this issue (Bateman & Fonagy, 2001; Clarkin et al., 2004; Clarkin et al., 1999; Linehan, 1993; Monsen et al., 1995; Ryle, 2004; J. Stevenson & Meares, 1992).

Explanation Two: 'Behavioural Inhibition' Reflects Attempts to Regulate, Re-regulate and/or Control the Interpersonal Environment

This explanation suggests that what has typically been referred to as disinhibited or impulsive behaviour represents a form of 'functional' behaviour that is employed in order to exact some type of outcome in the interpersonal world. This view suggests that what appears to be dysregulated behaviour actually represents a form of interpersonal regulation designed to extract particular outcomes from the interpersonal world. In the historical literature on BPD, this has often been referred to as 'manipulative' behaviour, although there clearly are potentially many other forms of *ex post facto* explanation available to account for this phenomenon. Clinical impression suggests that there often appears to be an association between what might be referred to as 'manipulative' behaviour on the one hand, and 'impulsive' behaviour on the other (Grinker et al., 1968). This is often observed through patient or third-party self-report, and is also often observed in the behaviour of patients in psychotherapy (Grotstein, 1987).

The use of the term 'impulsive' has often implied that the specific behaviour in question is either 'mindless' (irrational), or serves no functional purpose. However, functional analyses typically illustrate that so-called impulsive acts often result in behavioural advantage to the initiator of the activity. Clinically, it is usually important to understand the functional role that certain classes of behaviour serve for people with BPD. Often, the behaviour in question realises particular outcomes from an unresponsive or negating interpersonal environment that would not otherwise be attained. Therefore, this perspective emphasises the need to understand the functional significance so-called impulsive behaviour serves for the initiator of the action sequence (Koerner et al., 1996).

Explanation Three: 'Behavioural Inhibition' Reflects the Operation of 'What Works'

Explanation Two implied that what has traditionally been referred to as impulsive behaviour serves a functional purpose for the agent of the action. It is possible to conceive of certain classes of hitherto impulsive behaviour as representing operant activities which are highly effective in extracting functional outcomes from the social or interpersonal world.

Again, it is important to emphasise that this perspective does not reflect the notion of 'impulsive' behaviour in the traditional sense. This view does not regard behaviour as occurring 'without thought'. Rather, it describes a class of activity which does not appear to serve a 'psychologically mature' purpose. However, in recasting descriptions of 'impulsive' behaviour in order to analyse its functional consequences, it is often possible to infer that 'impulsive' behaviour has actually been shaped over the course of many years such that it

bestows behavioural advantages to the agent (patient). In this sense, it is reasonable to regard this type of behaviour as exploitative or opportunistic in nature rather than 'impulsive' per sê. Reformulation of this aspect of BPD activity is important because it actually reinforces the notion that the agent in question is in fact exercising 'executive authority' over the commission of behaviour that can be interpersonally exploitative and/or socially inappropriate. The functional significance of the behaviour is that it is strategically effective and bestows social or interpersonal advantage to the initiator of the activity.

This interpretation has significant implications for the management of BPD, particularly in acute, crisis situations. This perspective suggests that so-called 'borderline impulsivity' is often functionally determined and is therefore within the control of the agent. Therefore, this view suggests that maintaining clear and unambiguous parameters for the management of so-called impulsive behaviour is of paramount importance. This is an issue that Kernberg (1984) and Clarkin et al. (1999) have considered in detail. It further implies that because this behaviour has probably been learned, it has the capacity to be modified. This further implies the importance of rigorous formulation and case management practices in the care of borderline patients, and further emphasises the need for clear management and treatment structures emphasising precise behavioural controls and consequences in the management of cases.

The main implication of the finding of an absence of impaired behavioural disinhibition in BPD is the requirement to understand impulsivity in BPD as a potential artifact of some other process. The principal candidate is affect dysregulation, and this is related to a second explanatory factor which involves impairments to self-regulatory function. This finding requires a

reinterpretation of behavioural dysregulation in BPD in order to understand it from a new perspective. It is suggested that this new perspective involves understanding that impulsivity in BPD probably reflects dysfunctional affect regulatory and/or self-management difficulties.

9.4.3. Impaired Problem Solving In BPD

There is a mixed literature suggesting that borderlines experience difficulties with regard to problem solving, and from the broader demand of 'learning from experience'. This study provided no support for the hypothesis that borderlines experience impaired problem-solving capacity. These findings are of particular significance, because a number of the tasks utilised in the study are also known to be associated with frontal-executive function. These findings clearly suggest that when adequate methodological controls are employed to control for co-morbid issues, borderlines do not return scores consistent with impaired frontal-executive functioning. This finding casts doubt on a more recent trend in the literature that has suggested subtle structural brain disturbance associated with frontal lobe impairment in BPD (Schore, 2003a, 2003b). Viewed collectively, the results of the tests of working memory, response inhibition, and problem-solving provide no support for the view that borderlines experience impaired frontal-executive function. This finding is important because it suggests that the difficulties in BPD are probably not accounted for by an exclusively cognitively mediated model of psychopathology.

9.4.4. Impaired Affective Attentional Bias in BPD

The analysis of Hypothesis Three – Impaired Affective Attentional Bias in BPD – will be addressed in two ways. First, comment is offered on the interference analyses, and second, on the colour-naming response latency

findings. The findings of this aspect of the study indicated that significant differences exist between the BPD group on the one hand, and the Depressed and the Medical Control Groups on the other, at both a supraliminal (2000 mSecs) and a subliminal (240 mSecs followed by a 'masking' condition) level for colour-naming response latencies. However, a subsequent 'interference analysis' returned non-significant differences between the groups for either supraliminal or subliminal stimulus-word presentations.

Interference Analysis

The results of the interference analysis suggest that the BPD group was no more disrupted by the emotional valency of the stimulus-words than were the control groups, and as a result, borderlines are not attentionally biased for emotionally valenced material. There are a number of methodological, statistical, and theoretical issues associated with these findings that suggest that this conclusion is unwarranted.

Section 9.5 addresses a range of different methodological issues associated with the Stroop task, and one possibility that accounts for the absence of an interference effect was the effect of a 'strategic override' strategy. The strategic override account provides an explanation regarding why similar latencies were realised for all affect categories including Neutral words in the task. Under these circumstances, it is likely that null interference results would be returned. This was the outcome that was realised in this study. The details of this argument are more fully explicated in Section 9.5.6.

It is important to emphasise however, that an absence of an interference effect is not consistent with a null effect on the Stroop. This is a commonly argued for position, but ignores the broader findings from cognitive neuroscience

literature on the neural mechanisms underpinning performance on these tasks. The use of interference analyses within an emotional Stroop paradigm was originally argued for by Williams & Broadbent (1986) who suggested that this methodology controlled for the disruption that occurs for all participants when confronted by affectively-laden stimuli. They argued that by using the methodology of interference analysis, a more pure measure of interference could be realised. This methodology clearly has plausibility, and has been employed subsequently in a large number of studies (J. M. G. Williams et al., 1996).

The emphasis on interference analyses has resulted in a view suggesting that between-groups differences in colour-naming response latencies are of less importance. Typically, where there is a non-significant interference result, the study is considered to have produced non-significant findings. In the case of this study, this view is challenged because it ignores other important findings from the study that warrant qualification of this interpretation. Foremost amongst these objections is the need to account for the different between-groups response-time findings on the Stop-Signal and Stroop tasks, the similarity of findings for BPD participants with different forms of the Stroop, the similarity of many of the diagnostic features of the BPD and Depressed Controls in the present study, the role of affect identification and affect blends in BPD, and finally, the neuroimaging studies that indicate that Stroop and Stop-Signal type tasks activate similar networks mediated by the Anterior Cingulate Cortex (ACC) (Bench et al., 1993; Luu & Posner, 2003; Pardo, Pardo, Janer, & Raichle, 1990) irrespective of whether the stimuli are affective or non-affective in nature.

Therefore, whilst it is acknowledged that the interference analysis did not return significant findings in the present study, it is not accepted that this

amounts to a non-significant Stroop result. It is argued that the significant differences in colour-naming response latencies between the groups represent a clinically and theoretically significant finding that is independent of the interference returns, and requires explanation in its own right. The following section attempts to do this.

Colour-Naming Response Latency Returns

It is argued that the significant between-groups differences on colour-naming response latencies represent an important independent Stroop finding. The reasons why this result is significant rest upon the following arguments.

1. The results of the Mean Reaction Time (MRT) scores on the Stop-Signal Paradigm in the current study.

   The MRT returns on the Stop-Signal Paradigm and the Colour-Naming Response Latency returns on the Stroop task both represent differing types of a similar class of data – reaction or response time data. In examining both sets of returns, the MRT data returned non-significant between-groups results, whereas the Stroop Colour-Naming Response Latency data returned significant between-groups differences between the BPD group on the one hand, and the Depressed and Medical Controls on the other.

   There were two essential differences in the task demands surrounding these two tasks. First, the Stop-Signal MRT data represents a class of motoric, non-affectively influenced reaction-time data, whereas the Colour-Naming Response Latency data represents a class of verbally mediated, affectively influenced response-time data. Logan (1980; 1985) has argued that reaction or response-time is centrally mediated, and therefore it is unlikely that there will be differences in reaction-time tasks requiring either

verbal or motoric response. In other words, the findings in this study of non-significant MRT and significant Colour-Naming Response Latency returns cannot be understood as the result of the employment of differing modes of response/reaction time measurement. The differences cannot be accounted for by differences in central mediation of response-time. In addition, the differences on the Stroop Colour-Naming Response Latency returns cannot be interpreted as a poorer speed of response on the part of the BPD group. If that were the case, it would be expected that BPD participants would have also returned slower MRT scores on the Stop-Signal Paradigm when compared to the control groups. Therefore, it appears that some other explanation must be provided to account for the delay on colour-naming response latency – it does not appear to be an artifact associated with a deficit in response-time processing.

Second, the Stop-Signal MRT data is 'non-affective', whereas the Stroop Colour-Naming Response Latency data includes positive and negatively valenced stimuli as well as stimuli that were 'non-affective' (i.e., neutrally valenced) in nature. Because the Stroop returns were similar across all categories of affect at both levels of stimulus presentation, the findings suggest that the differences might be associated with the 'response-conflict' nature of the task in contrast to the effect of disruption associated with the affectivity of the task. This interpretation is also consistent with the findings reported by Wagner & Linehan (1999) who reported that there was no evidence of heightened sensitivity to negatively valenced affective cues in their BPD study. This is an issue that will be considered separately later in this discussion.

2. Diagnostic Similarities Between the BPD Group and the Depressed Control Group on Axis I (Acute State) Mood Variables

   The between-groups diagnostic analyses reported in Section 8.5.1 indicated that whilst there were significant differences between the BPD group and the Depressed Control group on salient measures of BPD functioning, there were a number of mood and personality based measures where no significant differences were returned. These included the DIB-R Affect Scaled Score (See Table 8.7), and all MCMI-III scales with the exception of Scale C (Borderline) (See Tables 8.8 and 8.9 respectively). These findings suggest that there were similarities between the BPD and Depressed Control Groups in relation to Axis I (Acute State) variables. Despite the identified similarities, the Depressed Control group returned colour-naming response latencies approximately 200 mSecs faster than the BPD cohort. Given the similarities on 'affect state' variables, it would be reasonable to suggest that both groups would have been expected to return similar colour-naming response latencies to affectively valenced stimuli, and the evidence clearly contradicts this. The significant differences on this measure represent an important finding suggesting a distinctive Stroop profile in BPD.

3. Colour-Naming Response-Latency Differences in the Absence of Interference Findings on the Colour-Word Stroop Task.

   There are two basic forms of the Stroop task - the 'Emotional Stroop' and the 'Colour-Word' Stroop (C. M. MacLeod, 1991; J. M. G. Williams et al., 1996). Whilst they represent different methodologies, both methods have in recent times been accounted for by the parallel-distributed processing model

of J. D. Cohen et al. (1990). The function that is argued to be common to both tasks is that of 'conflict management' (Jones, Cho, Nystrom, Cohen, & Braver, 2002; Luu & Posner, 2003). The implication of this is that the affective features of the task are of lesser importance than is the 'conflict-provocation' nature of the task. Viewed from this perspective, it would be predicted that where both forms of Stroop are employed similar between-groups results would be returned.

The existence of these differing forms of Stroop methodology constitute an important issue in interpreting the findings of the current study, and one study is available that has employed both Stroop methods in studying BPD. Sprock et al. (2000) employed the use of a card-based Stroop task with both colour-word (colour-conflict) and emotion-word (emotional-Stroop) conditions. They found non-significant between-groups results for colour-naming of words in both the emotion-word (emotional-Stroop) and the colour-conflict conditions.

This finding is important because similar results were returned under conditions requiring the colour-naming of affect-words on the one hand, and the colour-naming of colour-words on the other. The finding of a similar pattern of results across Emotional Stroop and Colour-Conflict Stroop tasks suggests either further evidence for a 'strategic override', or that the application of interference analyses in emotional Stroop analyses is unwarranted. Whilst it is possible that a strategic override process also occurred in the Sprock et al. (2000) study, a second candidate explanation emerges out of the neuroimaging research and is associated with the

mediating processes involved in 'response' conflict. This issue is further examined below.

4. The Issue of Affect Identification and 'Affect Blends'.

   Section 5.7 identified two issues that have salience for interpreting the Stroop findings. First, participants in Study Two (Affective and Semantic Representations in BPD) identified difficulties in relation to both the identification and regulation of affective states. One assumption underpinning effective interference analyses on the Stroop is that of accurate affect identification. 'Interference' analyses typically rely upon subtracting the colour-naming response latencies of 'neutrally' valenced words from affectively laden ones in order to obtain measures of 'pure' interference. This approach rests on the assumption that it is possible for the respondent to accurately discriminate between different affect categories. Section 5.7 provided evidence suggesting that this assumption might be violated in the case of BPD because the respondents in this study reported the experience of complex co-occurring affective states (Affect Blends), and difficulties in identifying individual affective states. Furthermore, participants reported difficulties in the identification of discrete categories of affect. Therefore, the emotional Stroop interference methodology has the potential to be seriously compromised if participants experience difficulties in accurately identifying differing affective states as represented by the nominated stimulus words.

5. Neurobiological research on response conflict and the Anterior Cingulate Cortex (ACC)

   Jones et al. (2002) and Luu & Posner (2003) argue that tasks such as the Stroop and Go/NoGo (or Stop-Signal Paradigm in this study) are essentially

'response-conflict' tasks, and they argue that there is consistent evidence from both the neuroimaging literature and computational modelling research suggesting that these tasks are mediated by the Anterior Cingulate Cortex (ACC) (Bench et al., 1993; R. A. Cohen, Kaplan, Moser, Jenkins, & Wilkinson, 1999; Luu & Posner, 2003; Pardo et al., 1990). The important point here is that it is not the issue of affectivity in the task that is of central significance. This literature suggests that it is the 'response-conflict' process inherent in the task that is the significant issue. As a result, this perspective suggests that interference phenomenon might be less important than the underlying function which mediates performance on the task, and that function appears to be response-conflict monitoring.

For all of the reasons cited above, it is argued that Colour-Naming Response Latency returns represent an independent level of Stroop analysis that requires interpretation independent of the non-significant interference findings. This argument implies, though, that the importance of the findings are not exclusively associated with affectivity, but rather with more fundamental neuropsychobiological functions involving attention, 'pre-attention', arousal, vigilance, and response-conflict monitoring processes.

Hypothesis Three also represents one of the first attempts to experimentally ascertain whether BPD participants demonstrated impaired levels of affective-attentional bias. Affective-attentional bias was studied as an analogue for affect regulation as affect regulation itself is a difficult phenomenon to study directly. The remainder of this section will examine various interpretations of the finding of slower Stroop colour-naming response latencies for the BPD cohort.

Alternative Hypotheses Concerning Delayed Colour-Naming Response Latencies

There are a number of factors that have the potential to explain the delayed Stroop Colour-Naming Response Latencies for the borderline cohort. These factors include history of use of drugs and alcohol, effects of mood at the time of testing, baseline IQ, use of sedating medication, word recognition capacity, general level of psychiatric morbidity as measured by number of bed day admissions, and ability to accurately perceive colour. These factors are known to affect both the speed of response in information-processing and also in the performance of neuropsychological tasks. As a result, it is possible that these factors might have influenced the outcome of the study. Pre-examination screening was conducted in order to eliminate any participants who experienced impaired colour perception. Therefore, colour misperception cannot account for the colour-naming response latency differences returned by the BPD group.

Section 8.5.2 reported on the statistical analyses for mood at the time of testing, the sedating effect of medication at the time of testing, general level of psychiatric morbidity as measured by number of bed day admissions, baseline IQ, and word-recognition capacity. These analyses found that it is highly unlikely that a general level of psychiatric morbidity as measured by number of bed day admissions, baseline IQ, or word recognition capacity can account for delayed Stroop responding in BPD. Whilst there was a significant finding for the use of sedating medication between the Medical and Depressed Controls, there was no difference between the BPD and the Depressed Control groups on measures of sedating medication and mood at the time of testing. Because the BPD group demonstrated delayed Stroop returns when compared with the

Depressed Controls, but not on measures of sedating medication or mood at the time of testing, it is unlikely that the Stroop differences are accounted for by the effect of mood status at the time of testing or by the use of sedating medication.

The differences in Stroop colour-naming response latencies cannot be accounted for by any of the variables identified above. A series of alternative interpretations of the data will now be considered. Some of these interpretations will consider the structural and demand characteristics of the Stroop task itself, and other interpretations will examine the cognitive features of BPD participants returns on the Stroop task.

Borderlines Demonstrate Generally Slower Reaction Times

The next possible account of the results suggests that the results on the Stroop paradigm simply reflect a slower reaction time on the part of BPD participants. This is reflected in slower response times to Stroop word primes. If performance on the Stroop task is exclusively viewed as a specific reaction time task, then the slower response times of the borderline group simply reflects one form of slower cognitive processing. The findings cannot, however, be explained as an artifact of a slower cognitive response set. If this were the case, then it would be expected that the Mean Response Time Scores (MRT) returned on the Stop-Signal Paradigm would also be significantly slower in borderlines. The evidence from the Stop-Signal task confirms that there were no significant differences between all groups on this task. This finding suggests that a general reaction/response-time deficit in BPD participants is unlikely to account for the differences in colour naming response latencies on the Stroop task.

Slower Response Times in BPD are a Result of Slower Word- Reading Speeds

This interpretation suggests that borderlines' slower response times on the Stroop are an artifact of a generally slower reading speed. The possibility exists that the delay in response time is an artifact of such a process. It is not possible from the design of this study to ascertain if this hypothesis is correct. This is a useful direction for future research to take.

Borderlines Experience 'Set-Shift' Difficulties in the Execution of the Stroop Task

This interpretation suggests that borderlines have difficulty in cognitively re-orienting to the task of naming the colour of the stimulus word. This difficulty could be interpreted as a 'shift-of-set difficulty'. In this sense, it is possible that this task requirement is similar to one of the task requirements of the Wisconsin Card Sorting Test (WCST), or the transition from naming letters beginning with the letters F, A, or S, to responding with animal names on the COWAT. This hypothesized difficulty in shifting sets then results in a longer response time for borderline participants.

The evidence does not support a specific 'shift-of-set' account because shift-of-set capacity was also assessed using the WCST and to a lesser extent by the COWAT (FAS). On both tasks BPD participants demonstrated the capacity to satisfactorily shift set. Therefore, it is unlikely that a 'shift-of-set' deficit will satisfactorily explain these Stroop returns. However, a related phenomenon is that of 'response-conflict' and it is possible to interpret these findings within that particular paradigm. This issue will be considered at a later point in this section.

Limited Vocabulary in BPD

This interpretation suggests that borderlines have a limited vocabulary. As a result, their delayed response times on the Stroop task reflect a lack of knowledge of the words employed in the study. This is unlikely as the St Lucia test (a test of word reading capacity) found no differences between groups. Word-reading capacity relies upon having access to a relevant and appropriate vocabulary. The findings of the St Lucia suggest that BPD participants have as extensive a vocabulary as the two comparison groups. This finding therefore refutes this hypothesis. Furthermore, a lack of word knowledge might also reflect a difference of years spent in education between groups, and there was no evidence of differential levels of education between groups.

9.4.5. Theoretical Accounts of Differences in Colour-Naming Response Latencies on the Stroop Task

This study found a significant Stroop Colour-Naming Response Latency effect in BPD. The significant BPD Stroop Colour-Naming Response Latencies across most categories of affect at both the supraliminal (2000 mSecs) and the subliminal (240 mSec plus mask) conditions were unexpected. The initial interpretation of this finding would suggest the operation of an aspect of the 'Stroop Effect', but a closer examination of the results mitigates against this interpretation.

It was hypothesised that the colour naming response latency for the BPD group would have been slower for the four categories of 'negative affect' (Anger, Sadness, Anxiety, & Shame), but that the response time for the Neutral and Joy affect categories would be similar for each group. This outcome would have been consistent with the prediction of an Emotional Stroop effect, and would have

provided support for the hypothesis of a negative-affect regulatory deficit in BPD. The finding of significant differences across most categories of affect suggests that there was not a specific 'Emotional Stroop' effect. The generalized nature of the Stroop response suggests that these results cannot be explained in this manner. The following section examines a number of possible explanations for the slower response times on the part of borderlines, and suggests that the findings might be best accounted for by a general distractibility hypothesis associated with a hypervigilant attentional set. There are a number of potential accounts for the Stroop findings which include:

1. A behavioural referent of a specific form of frontal compromise;
2. A behavioural referent of attentional bias;
3. A behavioural referent of high arousal.

These speculations will be considered below.

Evidence of a 'Frontal' Hypothesis in BPD

The finding of non-significant differences on a number of cognitive tasks known to be sensitive to frontal impairment as well as the non-significant findings on the Stop-Signal Paradigm suggests that borderlines do not experience a generalised 'frontal' deficit. In contrast, neuro-imaging research suggests that Stroop tasks are associated with the activation of either the anterior cingulate cortex (ACC) in brain damaged participants (R. A. Cohen et al., 1999), the left anterior cingulate cortex (LACC) (Mouratidis, Bolla, Funderburk, Kimes, & Cadet, 2001) and right anterior cingulate (Bench et al., 1993) in normal participants, and suppression of the left ACC in clinical (depressed) participants (George et al., 1997). The impaired Stroop returns found in this study suggest that there was a suppressed response in BPD participants, and this raises the

possibility that a similar neurobiological/metabolic mechanism that operates for depressed respondents (suppression of the left ACC) might also have occurred for the borderline cohort examined in this study. Clearly, this is speculative as this study did not incorporate neuroimaging data which could confirm this proposition. Despite this, there are a number of converging lines of evidence which provide some degree of support for this speculation.

The ACC is one of two components of a larger structure known as the cingulate gyrus. This structure is located above the corpus callosum, and this structure also contains the posterior cingulate gyrus (PCG). The ACC is concerned with emotional, autonomic, and endocrine regulation, whereas the PCG is concerned with integrating motor output and memory function as well as visuospatial and tactile analysis (Joseph, 1996).

The ACC subserves functions associated with regulating emotional expression and learning, and vocalisation. In addition, the ACC is involved in executing goal directed behaviour, the regulation of endocrine and autonomic activity, and the establishment of long-term attachment and maternal behaviour. Evolutionarily, it appears that the ACC first appeared where maternal behaviour, play, and nursing had a central role in social bonding and attachment (Joseph, 1996). Injury to the ACC results in deficits of maternal behaviour, emotional functioning, and impairments in empathic capacity (Cozolino, 2002; Joseph, 1996). In addition, disorders of affective control are also associated with impairment to this region. This can include impulsivity, disinhibition and hyperactive responses.

Structurally, the ACC is closely interconnected with the septal nuclei, amygdala, hippocampus, hypothalamus, periaqueductal grey matter, limbic

striatum, and other frontal areas. Therefore, the anterior cingulate appears to be an 'association area' involved in the integration of motoric, tactile, autonomic, and emotional material. This area also appears to have the capacity to experience 'psychological pain' (Joseph, 1996).

It appears that the ACC is a supra-modal area responsible for the integration of a variety of motoric and emotional functions. Furthermore, there appears to be a high degree of flexibility and voluntary control within the ACC. The emerging consensus suggests that the ACC is of central importance in the development of maternal behaviour and child care, social relations, and long-term attachments (Schore, 1994). Deficits in mothering and maternal behaviour (Lawson, 2000), social relationships (Westen, Lohr, Silk, Gold, & Kerber, 1990), and long-term attachments (Barone, 2003; Patrick et al., 1994) are well documented deficits in BPD. Therefore, it is possible that these borderline deficits might be mediated by impairments in the development and/or functioning of the ACC. To date, there is limited neuroradiological evidence that suggests that there are impairments in ACC function in BPD (Leyton et al., 2001). Further neurobiological and neuroradiological research is required to confirm this speculation but the findings of this study are interpretable within a paradigm of impaired functioning of the anterior cingulate cortex. This finding cannot be accounted for by frank cortical insult in the present study, thus suggesting that the results are artifactual to the vicissitudes of neural development, metabolic impairment, or inadequate utilisation of this structure.

In related work, Luu & Posner (2003) speculate that the ACC might also be responsible for the integration of complex cognition and simple motor acts on the autonomic nervous system. They further suggest that this association might

represent an important model for studying the mechanisms by which mental processes are integrated with bodily systems. One implication suggests that these processes are involved in the autonomic reactions that signal the need for an adaptive control of behaviour. This provides the clearest link yet in understanding the role of the ACC in mediating Stroop activity. It provides a conceptual link in understanding the Stroop findings as an arousal-mediated outcome, and is also interpretable within a response-conflict paradigm. Finally, the ACC is also involved in determining when strategic rather than autonomic control is required, again emphasising its importance as a mediator in response-conflict functions such as those provoked by Stroop task demands.

An Attentional Bias Hypothesis?

A second interpretation of the Stroop findings suggests that the results reflect a hyper-vigilant attentional set in BPD which is organised in order to attend and/or respond to novel stimuli. Furthermore, it is possible that this might be indicative of a hyperreactive 'orienting response' (Sokolov, 1963). This interpretation is also consistent with the view of Arntz et al. (2000) who concluded that the results of their Stroop study were consistent with the operations of a primitive form of hypervigilance.

This form of attentional bias persists despite evidence from other data in the study suggesting that frontal-regulatory functions remain intact. Considering that the Stroop colour-naming response latency effect occurs despite evidence of adequate 'frontal' function', it suggests that the process activated by the Stroop task has established itself prior to the consolidation of so-called 'frontal-executive' functions. This suggests that the Stroop effect observed in this study has developed prior to the establishment of consolidated executive function, and

this typically concludes by late adolescence (Thatcher, 1991). Therefore, the Stroop Colour-Naming Response Latency results probably reflect a 'hard wired', neuro-psychobiologically based process which pre-dates the establishment of mature frontal-executive function.

The cause of this form of hypervigilance or 'orienting response' in BPD remains elusive, but an obvious candidate factor is early family environment. The combination of maternal over-control and intrusiveness (Bezirganian et al., 1993) abnormal parental bonding (Torgersen & Alnaes, 1992), disordered attachment (Barone, 2003; Patrick et al., 1994), 'bi-parental failure' (Paris, 2003b), or childhood trauma (B. D. Perry, 1997; B. D. Perry et al., 1995), could clearly predispose a vulnerable child with an immature CNS to a hypervigilant attentional set directed towards monitoring a chaotic or unpredictable social/familial environment. In this sense, a hypervigilant attentional set represents an adaptive mechanism designed to both respond to, and protect the child from, unpredictable and/or traumatic experiences.

An Arousal Hypothesis?

A third interpretation of the Stroop results suggests that the findings reflect a particular response to elevated levels of arousal. Viewed from this perspective, the results are not associated with attentional bias to explicitly 'affective' material, but rather reflect an attentional response to highly arousing stimuli. This interpretation is also consistent with the finding of a non-significant interference score, and a significant colour-naming response latency on the Stroop task. The distinction offered here is that the phenomenon elicited in the Stroop task reflects a response to arousing or 'pre-affective' material, in contrast to an 'emotional' response to a 'discrete' affective stimulus. In other words, this

interpretation suggests that BPD participants were responding via a 'primitive' mode of processing of arousal-based phenomena in contrast to a more sophisticated processing of affectively valenced material. The interpretation of the findings in terms of an arousal hypothesis is also consistent with the finding of significant between-group differences on Neutral and Joyful Stroop affect categories.

This interpretation is also consistent with the view that the findings reflect an attentional response to novel stimuli in BPD. However, rather than interpreting the delayed reaction times as an artifact of an 'arousal-affectivity' hypothesis, this interpretation proposes that the delayed Colour-Naming Response Times are an artifact of an 'arousal-novelty' mechanism. The difference between the current and the previously articulated interpretation reflects an attempt to understand precisely what the form of attentional bias in BPD might be. The attentional bias hypothesis suggests that borderlines attend to any stimulus because of a hyperreactive neurophysiologically-based arousal state, which implies that borderlines will attend to any stimulus irrespective of its affective content. The arousal hypothesis suggests that borderlines are attending not specifically to an affectively laden state per sê, but to stimuli in the environment which require that the person shift attention. In other words, the arousal hypothesis suggests that the attentional biasing system evident in BPD is an adaptive mechanism which probably serves a monitoring and self-regulation function.

9.4.6. A Modified Multidimensional Developmental Neuropsychological Model of Impaired Executive Function in BPD

As a result of the findings of this study, a significant modification of the originally proposed multidimensional developmental neuropsychological model of impaired executive function is required. The modified model suggests:

1. BPD is characterised by an intact frontal-executive system;
2. BPD is characterised by a hyperreactive arousal state associated with a hypervigilance to environmental stimuli.

Therefore, when confronted with novel stimuli such as a response-conflict task, borderlines take significantly longer to orient themselves and respond in an appropriate manner. Figure 9.1 describes the developmental features likely to account for the delayed Colour-Naming Response Latencies returned by the BPD group.

Figure 9.1: Modified Multidimensional Developmental Neuropsychological Model of Stroop Colour-Naming Response in BPD

BPD RISK FACTORS						BPD CRITERIA	IMPAIRED STROOP RESPONSE
8. Genetic/ Psychobiological Predisposition		Maturational Failure				Affect Dysregulation →	Affective Attentional Bias
9. Early Loss/ Separation		of a		Development of		Impulsivity	Explained By:
10. Parental Psychopathology		CNS,				Interpersonal Dysregulation	Hyper-
11. Family Psychopathology	→	Distributed	→	BPD	→	Identity Disturbance	Vigilance
12. Impaired Parental Bonding		Regulatory				Fears of Abandonment	Response Conflict
13. Attachment Pathology		System				Social Maladaptation	
14. Trauma: Child Abuse & Neglect						Transient Paranoid Ideation	Excessive Arousal

9.5. CONCEPTUAL AND METHODOLOGICAL REVIEW OF THE PROJECT

This section considers a number of conceptual and methodological issues that limit the generalisability of the findings of the project. One major conceptual issue stems from the use of a categorical approach to studying personality, and this includes problems associated with the validity and reliability of personality disorders. Thereafter, a variety methodological issues associated with specific features of the project are identified.

9.5.1. The Categorical Approach to Studying BPD

The study of personality disorder is based upon the assumptions of a trait-based model of personality (S. C. Cloninger, 1996). This tradition was first enunciated by Allport (1931), who argued that a personality trait is an enduring feature of individual personality. This approach has been elaborated upon within the context of the study of personality disorders by a number of different theorists who have also argued for their own model of a trait-based, personality disorder conceptualisation (C. R. Cloninger, Svrakic, & Przybeck, 1993; Costa & McCrae, 1992; Tellegen, 1993).

S. C. Cloninger (1996) has identified a number of conceptual difficulties with a trait-based formulation of personality. These include:

1. Personality traits are poorly correlated with measurable behaviours;
2. Specific behaviours are determined by a number of co-occurring causes;
3. The measurement of behaviour has an inherent level of unreliability, and;
4. Specific behaviours vary as a result of the situations in which they occur.

As a result, a number of theorists have argued for a model of personality that emphasises a ‘person by situation interaction’ framework rather than the

continued use of a model that has both conceptual and measurement limitations (Mischel, 1968). The limitations of this frame of reference can also be seen in the findings of the long-term follow-up studies of the course of BPD. The evidence reviewed in Section 2.5 clearly suggested that the majority of BPD cases remit by middle age. These findings raise questions regarding whether BPD actually represents an enduring feature of personality. This is a controversial issue and requires further research and ultimately, conceptual refinement. In addition, recent prospective work has found that some cases of BPD remit in very brief periods (Gunderson et al., 2003), further suggesting conceptual difficulties with a trait-based formulation of personality disorder.

In addition to trait-based dimensional conceptions of personality, BPD is also conceptualised as a categorical diagnostic entity. The criticisms of this approach have been enunciated in Section 2.6. These include difficulties in distinguishing BPD from both Axis I and other Axis II disorders, poor inter-rater reliability, poor diagnostic validity, the absence of a coherent theoretical perspective that defines the condition, evidence that the category might be best represented by a 'normative' model of personality, and significant diagnostic overlap with other personality disorder categories (Livesley, 1998).

As a result, BPD has become difficult to operationalise (Tyrer, 1994). Therefore, it is likely that BPD is a heterogeneous disorder with poor predictive ability. This conceptual flaw may have exerted influence throughout the project, and one of the manifestations of this might be associated with the large variances on a number of dependent variables for the BPD group in Study Four. The large variances observed are also probably in part responsible for a number of the non-

significant findings as the BPD variances probably 'overrode' any potential group effect.

9.5.2. General Methodological Issues Associated With the Project

A significant methodological limitation associated with the project involves the relatively small sample sizes included in each study. Whilst this issue can in part be accounted for by the difficulties associated with the recruitment of clinical populations, the use of small samples results in analyses of low power which in turn increase the likelihood of returning non-significant results. Whilst this did not appear to be a significant issue in the validity study conducted on the MCMI-III (Chapter Four) or in the subsequent interview study (Chapter Five), it represents a more problematic issue in Study Four (Chapter Eight). It is possible that the largely non-significant findings reported in this latter study might in part be an artifact of the comparatively small sample sizes with an attendant lack of statistical power.

A second overall methodological limitation involved the utilisation of two instruments for diagnosing BPD. Whilst this was a necessity in the current study, this approach has a number of limitations. First, the project employed diagnostic instruments with differing theoretical underpinnings. As a result, each instrument was unlikely to be assessing the same BPD construct. The MCMI-III was attempting to measure DSM-IV polythetic criteria which include difficulties in emotion regulation, impulsivity, transient psychotic phenomenon, and interpersonal deficits (Appendix II). The DIB-R attempted to measure Gunderson BPD criteria which include deficits in emotion regulation, impulsivity, interpersonal deficits, and cognitive impairment. This approach has implications for establishing satisfactory construct validity for the sample diagnosed through

such a method, and this therefore limits the generalisability of the findings to other BPD samples diagnosed through alternative approaches.

Whilst the concurrent use of two diagnostic instruments to confirm a positive BPD diagnosis represents a legitimate mode of diagnostic practice (Kaye & Shea, 2000), it is clear that this both represents a limitation in the current study but also suggests an area for future research. Specifically, there is an urgent need for the development of more sensitive and specific instruments not only for the diagnosis of BPD, but for diagnosing specific dimensions of the disorder as well as different subtypes of BPD. A 'gold-standard' diagnostic test for BPD is urgently required.

9.5.3. Study One: Validity Study of the MCMI-III

Study One recruited participants prospectively from the author's outpatient clinical psychology clinic at Fremantle Hospital. Participants were recruited and administered the diagnostic instruments in such a manner that the experimenter remained blind to their diagnostic status at the time of data collection.

One methodological issue inherent in the design of the study is that the BPD cases were not recruited in a similar manner to the BPD cases recruited for Studies Two and Four. The BPD cases included in Study One were recruited through an outpatient clinical psychology clinic rather than through an inpatient service of the Mental Health Directorate of Fremantle Hospital. Therefore, there were a number of potentially significant differences between the respective borderline cohorts suggesting that there might be important clinical differences in the constitution of the respective groups. In addition, the BPD sample recruited for Study One differed from the BPD sample recruited for Studies Two and Four

in terms of a number of other equally important dimensions. These included differences in inpatient admission history, and general level of involvement with the psychiatric system which can also be interpreted as an index of psychiatric morbidity.

9.5.4. Study Two: Affective and Semantic Representations in BPD

Study Two recruited participants prospectively from the author's outpatient clinical psychology clinic at Fremantle Hospital. Participants were recruited into the study after meeting formal diagnostic criteria for BPD. This involved returning a minimum Scaled Score of 85 on Scale C (Borderline) on the MCMI-III a minimum Scaled Score of eight (8) or more on the DIB-R. Participants were then informed that they met criterion for BPD, provided with information regarding the diagnosis, advised that a research study was being conducted into the condition, and then requested to consent to involvement in the study.

A number of methodological issues were identified with regard to the conduct of the study. There is some evidence available suggesting that the constitution of the BPD sample in this study differs in important ways from the Study Four BPD sample. One of the assumptions underpinning the use of the Study Two BPD sample was that this sample should be as similar as possible to the BPD sample comprising Study Four. Whilst the DIB-R and MCMI-III data for these samples were similar, it would appear that the location of the study (IDD) selected for a BPD group who did not report a significant inpatient history. Although it is not reasonable to suggest that the participants in Study Two represent an atypical BPD group, it possible that they represent a sufficiently different BPD subtype that questions might be raised concerning the

generalisability of the findings from Study Two to applications in Study Four. This remains possible despite their similar diagnostic returns.

This critique suggests that the same methodology should probably have been used for the recruitment of BPD participants in both studies. This approach would have enhanced the inter-study methodological validity, and reduced the risk the findings of Study Two might have limited applicability to Study Four.

Despite these criticisms, there were methodological checks in place to counteract this potential flaw. It will be recalled that the decision rules for the endorsement of an affect category required that all BPD participants were required to independently verify the affect category for it to be included. Despite this, seven of the 11 BPD participants in the study were recruited from the Mental Health Directorate. Therefore, over half of the BPD sample was derived from a similar source to the sample that comprised the bulk of Study Four. This similarity in recruitment tends to mitigate against the argument that the groups were sufficiently different from each other that it invalidates the findings from Study Two with regard to their application for Study Four.

A final methodological difficulty associated with Study Two concerns the appropriateness of the methodology employed for categorising the discourse into affect categories. It will be recalled that there were two decision rules concerning the derivation of discourse material into affect categories. They were that either the participant would volunteer affect-related material, or alternately that the interviewer would identify affectively laden material and that this category of affect must then be endorsed by the participant. There are two methodological issues that potentially limit this aspect of the study. First, it could be argued that the interviewer shaped the scope, nature, and direction of the interviews and as a

result, obtained results that were desired rather than results that accurately reflected the affective experience of the participants. Second, the interviewer acted as both a generator of transcript material, and also as a judge of the material. In other words, the researcher was not independent to the process and the results of the study. These issues are central to the critiques typically directed against qualitative research paradigms.

There is now a strong qualitative research tradition which is regarded as a legitimate mode of scientific inquiry (McLeod, 2003; Rice, 1992). According to this tradition, acceptable standards of qualitative research require qualitative data to be analysed by the researcher: This method generally requires the researcher to:

1. Immerse themselves by an intensive engagement with the data;
2. Categorise the data by systematically working through the data by assigning coding categories or identifying meaning within the text;
3. Phenomenologically reduce the data by interrogating the meanings that have developed out of engagement with the immersion process;
4. Triangulate the data by sorting through categories and deciding which are central and relevant.

It is argued that this process was adhered to in the methodology of this study, and as a result the study meets acceptable standards of rigour typically associated with qualitative research paradigms.

The reason for the use of this methodology was that it represented a practical and cost-effective method for identifying the salient categories of affect relevant to borderline experience. Consideration was given to employing more sophisticated methodologies such as using independent judges to review the

transcripts in order to generate affect categories for the Stroop study. The selected approach was, however, considered both cost and time ineffective and not likely to have produced results that would have been substantially different in content. The task was a relatively straightforward one of identifying categories of affect, and this method was judged to be methodologically appropriate to realise this goal.

In addition, whilst the categories of affect derived out of this study were informed by the work of Tomkins (1962; 1963; 1991; 1992), they also represent terms of everyday discourse and experience, and could be argued to be 'universal' experiences. Whilst it is accepted that there are potential differences in the structure of the concepts developed by Tomkins, and the meanings of the same terms employed in everyday discourse, their 'psychological distance' is probably not substantial enough to invalidate the manner in which they have been employed in the development of the Stroop task. They do not represent detailed conceptual categories such as have been developed by Arntz et al. (2000) which rely upon a highly elaborated series of theoretical propositions.

It should also be emphasised here that no other Stroop study has reported undertaking the same level of detail to elicit categories for the development of their respective Stroop protocols. Therefore, rather than being seen as a methodological flaw, it is argued that this approach represents an improvement in the standard of development of the Stroop method and actually represents a strength of the study.

9.5.5. Study Three: Affect Category Judgement Task

The Affect Category Judgement Task (ACJT) employed Whissel's (1989) Dictionary of Affect in Language (DAL) in order to identify specific affective

words for inclusion in the Stroop Task. There were a number of methodological issues associated with the execution of this study which included the parsimony of the task, the selection of judges to complete the task, and whether the selected words represent the most 'ecologically valid' examples of words to represent each of the affect categories. Each of these issues will now be commented upon.

First, the ACJT employed the DAL as the dictionary from which words would be drawn for inclusion in the Stroop task. The DAL included over 5,000 words which judges were required to review and decide which affect categories the word best reflected. After this task was completed, the experimenter then reviewed the judges' responses in order to identify words which were both unanimously and unambiguously endorsed by all judges for each category of affect. Whilst this approach probably represents a methodologically sound approach for deriving appropriate affective words, it was not a particularly cost-effective method for obtaining an appropriate word-sample. All of the judges indicated that the task was onerous in terms of the time required for completion. In addition, the guidelines for making judgements concerning allocation to affect categories were at times insufficiently detailed to assist them in making fine discriminations.

A second methodological issue was also associated with the selection of judges to perform the initial task of allocating words to affect categories. The methodology driving this approach was associated with the use of so-called 'expert' judges who were deemed to be expert by virtue of their experience as clinicians. Whilst this approach remains methodologically sound, an alternative methodology could have employed BPD participants to act as judges. The purpose of the task was to validly allocate words to affect categories, and it is

possible that BPD participants might have been able to undertake this task in a way that might have improved the validity of the words selected for inclusion in the task. This point becomes important in view of the third criticism to be outlined – the so-called 'expert' judges allocated words to affect categories that, upon reflection, have dubious associations with the affect category in question. This occurred despite a team of judges making the first allocation to categories, and a second, independent team of judges cross-verifying the validity of words with affect category.

The third methodological issue is associated with the criticism that a number of the words ultimately selected for inclusion in the Stroop task may not validly represent the affect category they have been selected to represent. For example, the Neutral category employed three words – Dipped, Medicine, and Joystick – which could be argued to not be affectively neutral words. The terms 'Dipped' and 'Joystick' have sexual connotations in Australian parlance and this might have had implications for the Medical Control participants who were drawn from clinics where sexual health screening was being conducted. It is also possible that these words had implications for the BPD group because of the increased likelihood of sexual abuse histories amongst this cohort. Similarly, the term 'Medicine' was also included, and it is possible that this was not a neutrally valenced word either for the Depressed Controls, or for the BPD group. In the Anger category, the term 'Bastard' was also included, and whilst it is possible to conceive of this word being used in an 'angry' manner, it is equivocal whether the semantic meaning of the word includes references to the affective experience of anger. The same argument can be mounted for the use of the word 'Funeral' in the Sadness category, and for the word 'Shoplift' in the Shame category.

Another methodological issue emerged in relation to the selection of words for inclusion in the Joy category. Whilst strenuous efforts were made to ensure that words included in each category emanated from different roots, this decision was violated in part in the inclusion of the words 'Joyful' and 'Enjoyment'. Whilst these words do not necessarily emerge from the same root, there is a 'semantic closeness' suggesting too much similarity between these words.

Fourth, the application of the methodology did not result in the employment of the most readily identifiable words associated with each of the affect categories. For example, it is reasonable to argue that words such as Angry or Anger, Sadness, and Shame or Shaming, might have been included. This would have increased the level of face validity inherent in the paradigm.

Despite these objections, it remains likely that the majority of the words selected for inclusion in the Stroop Task were valid because whilst there were between-groups differences in colour-naming response latencies, there appeared to be limited within-groups differences. It is unlikely that these flaws in study design would be sufficient to account for this finding – it remains likely that the within-group findings would be returned whether or not the identified words were included in the paradigm.

9.5.6. Study Four: Assessment of Executive Function in BPD

Study Four was designed to examine the multidimensional developmental neuropsychological model of impaired executive function in BPD as originally outlined in Chapter Three. A number of methodological issues are evident in relation to the recruitment procedures employed in the study, the measurement of working memory, the use of the Stop-Signal paradigm, the use of the Stroop task,

and the measurement of Problem Solving. Each of these issues will now be considered.

Recruitment Issues

The BPD group was recruited from three sources – a Mental Health Directorate BPD group, an IDD recruited BPD group, and a Newspaper recruited BPD group. This recruitment approach represents a potentially important methodological difficulty even though diagnostic analyses were conducted that indicated that the groups were relatively homogeneous. The reason for the use of multiple recruitment sources was to improve sample size in the BPD group in order to improve the power of the study. This is a situation often experienced in conducting clinical research with difficult to engage clinical populations, and represents a legitimate attempt to reconcile methodological rigour with the practicalities of sample recruitment. In this sense, the use of multiple recruitment sources was methodologically acceptable, and can be justified particularly in light of the absence of diagnostic differences between the BPD sub-groups.

The use of a Medical Control group recruited from participants attending a Genitourinary Medicine (GUM) clinic was an attempt to recruit a group of non-symptomatic participants who would approximate a 'normal' control sample. In many respects, the sample represented this as their returns on both the MCMI-III and the DIB-R fell within 'normal' limits.

One of the methodological strengths of the use of this sample was that they represent an essentially normal comparison group who were recruited from the same location (a tertiary hospital) as the other two groups in the study. This represents a methodological strength of the study as similar studies have drawn their comparison groups from sources other than that used for recruitment of

experimental participants. For example, in the Arntz et al. (2000) and Dinn et al. (2004) studies, controls were recruited by advertisement, and in the Kunert et al. (2003) study, participants were recruited from hospital staff. These methods often represent 'convenience' samples, and whilst methodologically acceptable, are not as appropriate as the methodology employed in this study. Despite this, the Medical Control group returned statistically significant MCMI-III elevations on Scale Y (Desirability) on the MCMI-III (M = 70.50, SD = 22.74) when compared to the Depressed Controls (M = 35.09, SD = 16.68) and the BPD groups (M = 25.22, SD = 15.67). Although the Medical Control group returned scores within the normal range for Scale X, the statistically significant elevation suggests that as a group the Medical Control sample were attempting to cast themselves in an overly favourable light with the potential confound that they minimised the severity of their psychological presentation. In other words, the Medical Control sample might have been more psychiatrically morbid than their diagnostic returns initially suggest. These findings have implications for the experimental data returned by this group, and suggest that the Medical Control group probably does not represent a comparison group that can be thought of as a 'normal' control group. This might have some implications for interpreting the large number of non-significant returns realised in this study.

A final methodological issue associated with the recruitment of participants concerns the overall small sample sizes involved in the study. The study included a total of 39 participants, and this, combined with the fact that the effects associated with the experimental tasks were likely to be subtle, provides a methodological explanation for why more significant results were not returned if in fact there were differences between the groups. The nature of the subtlety of

the tasks was such that the sample sizes were probable too small to detect many of the differences that might exist between the groups. These issues, when combined with the diagnostic issues raised with regard to the Medical Control group, assist in understanding why the study realised predominantly non-significant returns.

Methodological Issues Associated With Screening Participants

The general methodological principle of screening the cohort in order to control for the potential confounding effects of a number of different co-morbid risk factors was a methodological strength of the current study. The decision to assess IQ, word reading capacity, colour perception, hospital admission history, sedating medication usage, and mood at the time of testing with sound instruments where appropriate represents a methodological strength of the study. One of the methodological weaknesses of the screening methodology was the failure to employ a formal measure of handedness. The study relied upon the use of a series of questions in order to provide an index of handedness. Future studies should employ a formal measure of handedness.

Examiner Not 'Blind' to Diagnostic Status of the Participants

Another methodological limitation with this study involved the failure of the examiner to be blinded to the diagnostic status of the participants. The risk associated with this is that knowledge of the diagnostic status of each participant might have resulted in shaping of testing performance in order to confirm the hypotheses under examination. There were however, reasonable attempts to control for this influence in tasks where scoring required interpretation. Thus, for tasks where scorer interpretation of the data was required (i.e., in tasks such as the Complex Figure of Rey (Rey Figure), Logical Memory (LM), and

Similarities) blind scoring by an independent rater was conducted in order to control for the potential confounding effect of examiner knowledge of participant diagnostic status.

The best solution to the issue of the need to have the examiner blind to diagnostic status would have been to utilise independent examiners who were blind to diagnostic status of the participants. This would have increased the cost of the project, and required the training of examiners in the tasks used in the study. This would have necessitated training for reliability in administration, and it is questionable whether this would have yielded better quality data as the tasks selected emerge out of a psychometric tradition that emphasises standardised administration in psychological testing. Where tasks required an interpretive function in their scoring, blind scoring by an independent rater was in fact undertaken. It is therefore argued that this decision represents a methodologically sound and clinically pragmatic approach to take in relation to the conduct of clinical research.

Reliance on MCMI-III to Diagnose the Depressed Control Group

In the present study, the importance of the use of semi-structured interviews as an important component in the diagnosis of BPD was emphasised. It will also be recalled that the diagnosis of the mood-disordered control group (Depressed Controls) was effected by a default process whereby if the participant did not meet BPD criteria, they were allocated to the Mood Disordered Control Group (Depressed Controls). The criteria for this involved failing to meet DIB-R and MCMI-III Scale C (BPD) criteria, but meeting MCMI-III criteria for Dysthymia or Major Depression. In practice, the diagnostic criterion for allocation to the Mood Disordered Control Group rested exclusively upon the use

of a self-report methodology (MCMI-III), and this represents a methodological limitation in the design of the study. The study could have been improved by incorporating a semi-structured interview for the diagnosis of Axis I mood disorder for the very same reasons that were enunciated regarding the importance of the use of semi-structured interviews for the diagnosis of BPD. Future studies should incorporate the use of an 'Axis I' diagnostic interview rather than rely upon the use of default and self-report diagnostic methodologies for the diagnosis of Axis I comparison groups.

Use of the DIB-R to Confirm the Diagnosis of BPD

The methodology employed in this project employed the combined use of the MCMI-III and the DIB-R to confirm a diagnosis of BPD. Although the DIB-R is recognised as a legitimate tool for the diagnosis of BPD, it is aligned with Gunderson BPD criterion rather than DSM or ICD criteria (Kaye & Shea, 2000). There is some evidence that the diagnostic criteria associated with Gunderson BPD criteria might be broader than DSM criteria (Gunderson, 1994), and this might in turn have resulted in the BPD condition employed in this study being too heterogeneous with overly extensive diagnostic borders (Tyrer, 1994). The use of this measure might in part explain the large variances returned by the BPD group on most dependent variables, which might in turn explain the failure to differentiate the groups on most of the measures employed in Study Four. Accordingly, the study might have suffered as a result of the use of the DIB-R as the final diagnostic measure used to confirm BPD because it might have employed too liberal a set of criterion cut-offs in contrast to a DSM or ICD formulated BPD diagnosis. Although there is no gold-standard diagnostic tool, and no evidence that there are measures superior to the DIB-R, future research

might benefit from the adoption of an DSM or ICD aligned diagnostic instrument simply because it enables the findings of the study to be more readily generalised to other studies that use DSM or ICD aligned instruments..

Limitations of the Measurement of Working Memory

This aspect of the study employed tasks drawn exclusively from the Wechsler Memory Scale (Revised) (WMS-R) (Wechsler, 1987) in order to assess working memory (WM). WM is an important emerging concept (Grigsby & Stevens, 2000), but there does not appear to be a consensus at this time concerning appropriate measures of WM.

The concept underpinning WM is that of a limited capacity memory store lasting anywhere between two and 20 seconds (Baddeley & Hitch, 1994; Grigsby & Stevens, 2000). Given these parameters, the most practical method for reliably measuring WM was to employ immediate recall tasks from well validated memory tasks such as those comprising the WMS-R. This approach was selected because it was important to assess WM independently, and not as some confounded factor in a more complex executive task.

There were, however a number of methodological issues associated with the tasks selected for assessing WM. These issues are associated predominantly with the issue of salience of the measures for understanding memory in BPD.

The measures that were employed reflect a cognitivist perspective of memory that emphasises the role of 'declarative' memory (N. J. Cohen & Squire, 1980). Declarative memory in turn is generally divided into two subtypes of memory – episodic memory (memory for subjective events occurring in the participant's life), and semantic memory (so-called 'knowledge' or memory for 'facts') (Grigsby & Stevens, 2000). Viewed from this perspective, it is possible

to observe that the WM tasks employed in this study were exclusively associated with the measurement of semantically based knowledge.

It could be argued that the more clinically relevant form of declarative knowledge – episodic memory – was not assessed at all. This is an important point as the clinical literature is replete with examples indicating that it is the domains of affectivity and interpersonal regulation that are highly problematic in BPD (American Psychiatric Association, 1994, 2000; Grotstein, 1987; Linehan, 1993). These domains are more likely to be accessed by episodic memory probes and essentially reflect the operation of what might otherwise be referred to as 'affective' memory. The absence of the assessment of any aspects of affectivity in the working memory systems of BPD participants represents a legitimate direction for further research.

The limitations in the tasks employed to examine working memory also reflect deficiencies in the conceptualisation of working memory. Whilst there are measures of working memory available, many have been used only in paediatric populations, in experimental situations only, and most do not report normative data (Pennington, 1997). Because of these factors, selected tasks from the Wechsler Memory Scale – Revised (WMS-R) (Wechsler, 1987) were believed to be acceptable as a parsimonious method for examining working memory in BPD. Clearly however, other approaches to examining working memory including computerised tasks examining rapid working memory tasks are now called for. This represents an additional future direction for further research.

Limitations of the Stop-Signal Paradigm (SSP)

The SSP was employed in this study to examine response-inhibition in BPD. The SSP was based on a 'race' model of inhibition (Logan, 1994), and it

remains unclear if this model is consistent with the conception of BPD as a disorder of impulsivity.

The issue is further complicated because impulsivity in BPD might occur within the context of 'affective arousal' (Zanarini, 1993), or might in fact be one component of a broader arousal process. This is an important issue because the SSP was unable to elicit whether 'motivational' factors (i.e., affective) factors have salience for this methodology. Put differently, one of the methodological shortcomings of the SSP was that it was an exclusively 'cognitive' task. It failed to examine the capacity of borderlines to inhibit a prepotent response which was affectively or interpersonally determined, and this represents a limitation in the construction of the task. The task could have addressed this issue if affective stimuli were included, and this is an approach which has been employed by Elliot, Runinsztein, Sahakian, & Dolan (2000).

Despite these objections, other studies have used similar methodologies, and have returned similar reaction or 'go' response times (Dinn et al., 2004; Kunert et al., 2003). The reported data from these studies indicated that the MRT for their groups were similar to the returns for this study. These findings provide some assurance of the comparability of the finding of this study, but again suggest that further work is required to clarify the relationship between the more generic conception of 'impulsivity', with the more specific and operationalised notion of inhibition. There is no question that the inclusion of this paradigm in the current study was justified both theoretically and methodologically, and on balance represents a strength of the study. It is however, also important to emphasise that additional inhibition paradigms need to be developed to further examine the issue of 'impulsivity' in BPD.

Limitations of the Stroop Task:

An 'Emotional Stroop' task was employed in the current study in order to examine evidence for an 'affective-attentional' bias in BPD. This method was employed as a way to examine the more generic conception of 'affect dysregulation' in BPD. It was argued earlier that affect regulation is not directly assessable, and the use of the Stroop method was proposed as an analogue of affect regulation. It was hypothesised that colour-naming response latencies and interference indices reflected BPD reactivity to affectively laden stimuli which could in turn be seen to be an index of affect regulation. There were a number of methodological issues unique to both the construction of the task and the execution of the Stroop procedure, and these will be selectively reviewed here. The methodological issues identified in the Stroop task include:

1. The assumption that 'discrete' affects operate in both the Stroop task and in human cognition;
2. The failure of the task to include other components that would more completely examine attentional bias in BPD, and;
3. The risk that a methodological artifact in the delivery of the categories of affect words might account for the failure to realise a significant 'interference index'.
4. Whether the 'subliminal' presentation of words were truly presented at subliminal levels of activation.

The Limitations of a 'Discrete' Emotions Perspective

An assumption underpinning the development of the Stroop task in this study involved the employment of a 'discrete' emotions model. This perspective understands that 'basic' affects are separate and distinguishable from one

another. Whilst this view has inherent appeal, it been challenged in some quarters (Ortony & Turner, 1990).

Ortony & Turner (1990) challenge the conception of 'basic emotions' theory. They argue that basic emotions theories conceive of emotions as either biologically or psychologically 'primitive'. The 'biologically primitive' approach understands that emotion can be understood by comprehending its evolutionary significance and examining the biological underpinnings of emotion. The objective of this approach is to understand the functional significance of the emotion for the individual and the species.

In contrast, the 'psychologically primitive' view argues that a small number of emotions exist out of which all other emotions are 'built'. As a result, it then becomes possible to study these basic emotions as an end in themselves, but also these basic emotions can be employed as 'primitives' in the study of non-basic emotions by developing a 'combinatorial' model of emotion.

Ortony & Turner (1990) argue that there appears to be a significant lack of consensus regarding the constitution of basic emotions. They note significant inconsistencies in the constitution of a number of lists of basic emotions, and more importantly, question the inclusion of a number of emotions in basic emotions lists. For example, they note that 'surprise' is included in a number of basic emotions lists. They argue however, that it is not self-evident that surprise is an emotion because emotions are usually considered to be 'affectively valenced' states and they doubt that this is the case for a state such as surprise. Ortony & Turner further note that affective valence is considered to be a necessary condition for a state to be an emotion, but this view excludes the possibility that an emotion can be affectively neutral. They argue that under this

reconfiguration, surprise might be viewed as a cognitive state rather than an emotion.

Ortony & Turner (1990) also note that basic emotions construed as 'biological primitives' rest heavily upon evidence of neurophysiological and anatomical data, and the literature linking specific emotions with distinctive, universal facial expressions. They note that there is limited evidence for hardwired neural circuitry for specific emotions, but suggest that there is evidence for circuitry for emotion in general. Furthermore, they argue that characteristic facial expressions apply not only to basic emotions, but also to a number of states that have been explicitly rejected as constituting emotion.

Ortony & Turner (1990) also note that the 'psychological primitives' view of basic emotions rests on the assumption that they are psychologically irreducible constructs. They argue that the main criterion focuses on the interrelationship of the emotions rather than on the nature of the eliciting conditions. In this sense, an emotion is regarded as basic if it contains no other emotion as a component. This is problematic also in the present study as a number of BPD participants reported experiencing 'affect blends' with regard to affect categories that are considered to constitute 'basic' emotions (See Section 5.7). Ortony and Turner also identify the issue of 'ontogenetic primacy' as challenging the conception of basic emotions. The issue here is to do with reducibility: some basic emotions appear to be more basic than others. They note that a number of emotions rely upon the pre-existing operation of other emotions in order for the 'emotion' to be effective, and in addition, different emotions emerge out of configurations of differing emotional appraisals. This latter point also does not require one emotion to be 'more basic' than the other.

The Limits of an 'Emotional Attentional-Bias' Paradigm

A second methodological issue associated with the Stroop task was the decision to examine 'affective-attentional bias' only, and to exclude from consideration the larger consideration of the role of a more generic attentional bias mechanism. As a result, a 'colour conflict' Stroop component was not included in the design. It remains unclear whether the Colour Naming Response Latencies elicited in the Stroop task were associated with affective issues alone, or were associated with some other form of attentional process as has been suggested by other work (Luu & Posner, 2003). The inclusion of a colour-conflict component Stroop might have strengthened the methodological integrity of the study.

The results on the Stroop task indicated that BPD cases returned significantly longer colour-naming response latencies than controls, but that when an 'interference' analysis was conducted, no significant differences between the groups was realised. This was a surprising finding, and one explanation for this outcome might be associated with a methodological artifact in the design of the Stroop task. The absence of a significant interference result is accounted for by an absence of a significant within-groups difference between Neutral word colour-naming responses and affective word colour-naming responses. This finding raises a number of issues, and these will be systematically addressed in Section 9.6. Despite this however, one explanation for the absence of within-group differences on colour-naming response latencies is associated with a methodological artifact related to how Neutral words were delivered in the task.

Specifically, the Stroop task was designed in such a manner that words from each of the six affect categories were randomly presented across the course of the Stroop trial at both supraliminal and subliminal levels of presentation. There are a variety of methodologies employed in the Stroop literature with regard to how Neutral words are delivered. Some studies present Neutral words as an independent group of trials prior to the delivery of emotion-category words (Arntz et al., 2000; C. MacLeod & Hagan, 1992), and other studies present Neutral words randomly throughout the course of the Stroop trial (J. G. Beck et al., 2001). Therefore, it remains equivocal as to whether a randomly delivered versus blocked-trial of Neutral stimulus words in the stimulus array explains the results obtained.

Because Neutral words were randomly presented amongst affectively-laden words, one possible explanation for similar Neutral latencies might be associated with the priming effects of one word upon another. There is some evidence that priming can persist over a number of intervening items such that individual Stroop items might be affected by such priming. However, Williams et al. (1996) have examined this issue in relation to a number of clinical disorders (anxiety, depression, & PTSD) and concluded that colour-naming interference is not accounted for by inter-item priming.

Therefore, it does not appear that the absence of an interference effect can be accounted for by the organisation of stimulus words in the stimulus array. Williams et al. (1996) also note that Stroop interference does not routinely occur, and speculate that a ‘strategic override’ effect can account for this phenomenon. The parallel-distributed processing model (J. D. Cohen et al., 1990) can be used to override attentional bias by increasing the effort made in naming colour. The

'signature' for this phenomenon according to Williams et al. (1996) is a general speeding of response on all categories including Neutral ones. This finding appeared to operate in the current study – there was evidence of a general speeding of responses across all affect categories including the Neutral word category. This interpretation suggests that the participants in the study might have overridden the task demand of the method by allocating additional resources to colour-naming such that they overrode the usual interference effect observed in this task.

It is also possible that the strategic override effect that is hypothesised to have affected interference scores might itself be an artifact of the Stroop Task instructions provided at the commencement of the task. The possible strategic override artifact might be associated with the initial instructions which are repeated here in order to isolate the possible confound. The instructions are included below:

> 'The following task is known as the 'Emotional Stroop' Task. You will already know about this task because it has been described to you in the information sheet provided to you at the commencement of this study. The task involves the presentation of 120 words in upper case lettering, one at a time, on the computer screen in front of you. The words will be presented in one of four different colours: Red, Green, Blue, or Yellow. Your task is to name the colour that the word is presented in.

It is possible that participants might have interpreted these instructions to mean that they should eliminate any possibility of reading the words in the task by allocating additional resources to colour-naming in order to override attentional bias. Whilst it is possible that the final two sentences of the task

instructions might have created a methodological artifact which resulted in a strategic override for some participants, it is impossible to ascertain if this is the mechanism which resulted in similar colour-naming response latencies for neutral as well as non-neutral words for all participants. It does however, remain a possibility which can explain the pattern of Stroop findings found in this study.

A second factor that might also account for the absence of an interference effect is associated with the capacity to discriminate between different affect categories. Section 5.7 reported discourse evidence suggesting that BPD participants experienced significant difficulties in identifying differing affective states. It is therefore possible that all participants did not differentiate between the differing word categories in the Stroop task. This might in part explain why the latencies for the Neutral words were similar to the other affect categories. If there is a difficulty with discriminating affective valence, then it is highly likely that this will be reflected in an absence of interference difference between different categories of affect.

Methodological Strengths of the Stoop Method Employed in the Study

Whilst there has been consideration of the possible methodological limits of the Stroop task, the task was also methodologically sound in a number of ways. Some of the methodological strengths of the task include the following.

First, in the construction of the Stroop task, a decision was made to employ a computerised rather than a card based Stroop. Despite various arguments suggesting that card and computer based Stroop methodologies yield similar findings (J. M. G. Williams et al., 1996), it was adjudged that using a computer based methodology would yield a more accurate set of results in which fewer methodological artifacts would prevail. A second methodological strength

in the design of the Stroop task involved the use of interviews with BPD participants in order to elicit affect categories, and the use of the DAL to select words using pre-rated affect ratings of these words in order to ensure sound affect properties for the words employed in the task. Third, another methodological strength of the design of the Stroop task involved the use of independent judges to select word arrays for the task. Other studies (Arntz et al., 2000) failed to employ similar methodological strategies in the design of their protocols, and in this regard, it is argued that this study represents a methodological improvement in the development of Stroop protocols with this population.

Other methodological strengths in the design of the Stroop included ensuring there were no differences between the affect categories in terms of stimulus-word length, the use of a colour perception check with participants in order to ensure that the results would not be compromised by a colour perception deficit, the use of screening instruments in the form of the St Lucia to control for differential word reading capacity, and the use of the NART to control for IQ using a word based mode of IQ assessment. The use of a word-based IQ assessment in the form of the NART was deemed especially suitable as it required the same modality of assessment (the reading of words) as the Stroop task.

The inclusion of Joy affect words can also be seen as a methodological strength of the study. The decision to include this category of words was driven by a combination of empirical evidence and clinical experience which suggested that the difficulties in BPD might be associated with a general difficulty with

affect regulation rather than a difficulty with the processing of specific, discrete affects.

On balance, the Stroop task developed for inclusion in the project is argued to be both a conceptually and methodologically sound tool that was generally well executed. The findings from this aspect of the study returned results indicating that BPD participants were slower in colour–naming but that a hypothesised interference effect was not realised. The theoretical and clinical implications of this finding will be considered more thoroughly in the following sections.

Limitations of the Measurement of Problem Solving

The study employed a number of tasks in order to examine problem-solving in BPD. These analyses failed to return significant findings, suggesting that there are no problem-solving deficits in BPD. This is consistent with the findings of other studies (Kunert et al., 2003; Sprock et al., 2000), and at variance with others (Bazanis et al., 2002; Dinn et al., 2004).

There are however, a number of theoretical and methodological issues associated with this approach. The fundamental issue inherent in this study is the question of whether the tasks are a valid representation of the types of problem-solving difficulties experienced in BPD.

There are two features to this. First, the tasks employed are not necessarily representative of 'real world phenomena' which are thought to be problematic in BPD. In this regard, the tasks do not enjoy a high degree of 'ecological validity' (Cripe, 1996). More importantly however, it is possible that the tasks were not sufficiently challenging, and therefore lacked sufficient power to elicit differences between the groups. For example, the TOL and the TOH

employed problem-solving levels that were initially developed for use with a paediatric and adolescent population (Humes, Welsh, Retzlaff, & Cookson, 1997). Therefore, the level of difficulty of the tasks may not have been sufficiently demanding to elicit differences between the groups. This critique cannot however, be applied to most of the problem-solving tasks used in the study as they have a long history of use with adult populations. Therefore, the null findings encountered in this study with these tasks are unlikely to be completely explained by the employment of overly simple task demands.

It is possible to offer a similar critique of the problem-solving component of the study as was made for the WM component of the study: the clinical literature considers that the difficulties associated with BPD are largely relational and affective in nature. As a result, experimental tasks that explore cognitive functions in isolation (i.e., without including affective/relational variables) are likely to return non-significant findings. It is likely that the results found in this study are in part a result of the examining cognitive factors at the expense of more affectively valenced ones.

'Ecological Validity' of the Study

One final issue concerns the 'ecological validity' of the study. Ecological validity refers to the functional and predictive relationship between performance on tasks of executive function (EF) and the patient's behaviour in a number of 'real-world' settings (Sbordone, 1996). One of the assumptions associated with the use of the EF tasks in Study Four was that that they assessed the cognitive, affective, and behavioural functions relevant to 'real-world' functioning in BPD.

Cripe (1996) has argued that there is often a discrepancy between the performance on neuropsychological tasks of executive function, and the 'reality'

of the patients maladaptation. Amongst the many reasons for the disparity between the patient's situation and their test performance is the issue of the validity of executive function. This is an important issue because the findings of this project demonstrated that there was little evidence of impairment of executive function in BPD, yet the immediate histories of the majority of the BPD sample were characterised by significant maladaptation – most of the cohort had experienced a recent psychiatric inpatient admission. In addition, the transcripts of the BPD cohort reported in Chapter Five provide numerous examples of significant emotional and interpersonal impairment. In addition, the majority of BPD participants in both studies reported long-standing histories of emotional, relational, and occupational impairment.

From this perspective, the findings emerging from this study are consistent with the anomaly of executive functions eluding measurement (Kolb & Whishaw, 1985, 2003). It is possible that this effect influenced the outcomes found in the present study. Lezak (1982; 1995) has also argued that one of the reasons for this is associated with the conceptualisation of executive functions as 'supramodal' entities. This occurs in part because typical measures of executive function are too specific, and thus do not 'capture' the nature of executive deficits. In addition, Lezak argues that most testing situations are far too controlled, and as a result, executive deficits elude detection because of the strictly controlled demand characteristics of the testing situation. It is possible that the executive tasks employed in the present study did not examine the appropriate level of presumed executive deficit in BPD.

Despite these issues, Cripe (1996) suggests that there is a more fundamental reason for the absence of ecological validity in the examination of

executive function. This is referred to as the 'mind-data' problem, and is essentially an artifact of the use of an empiricist scientific paradigm. Cripe argues that test returns are reductionistic symbolic representations of real events, and as these real events become more complex, the capacity of a test result to accurately reflect these real events becomes significantly degraded. Cripe argues that the human mind and its actions constitute a complex, dynamic, interactive system and the measurement of this system requires methods that are sensitive to this complexity. Cripe further argues that neuropsychology has pursued a path of reductionism as a means for coping with complexity. This in turn requires the participant to perform a structured task over time. The result is an abstract symbol (nominal or, at best, ordinal data) of some part or aspect of the reality that was measured. Cripe argues that there are serious limitations imposed on complex realities when a reductive scientific paradigm is employed. Because executive functions represent complex dynamic processes, their observation is severely limited by the use of a reductionist paradigm. This, Cripe argues, is why executive function measurement is often illusory in the standard testing situation. The findings of the present study might well have been affected by the influence of the 'mind-data' problem. This represents a potentially significant conceptual limitation to the project and the findings that can be inferred from it. The implication of this critique suggests that future research might employ more qualitatively based, descriptive, 'real-world' paradigms in order to examine the nature of executive deficits in BPD.

9.6. IMPLICATIONS FOR FUTURE RESEARCH

This section considers the implications of the findings of this study for future research. It is organised into two sections. The first section addresses the

implications for future theoretical research on BPD, and the second section is concerned with implications for future clinical research on BPD.

9.6.1. Implications for Future Theoretical Research

One of the major implications for future research in BPD involves the need to reconsider the method of measurement of the disorder. Whilst there remains a need to develop more sensitive and specific diagnostic instruments, one of the current controversies in BPD diagnosis involves the continuing debate regarding the merits of categorical versus dimensional diagnosis (Widiger, 2000; Widiger et al., 1992). The continuing use of a categorical approach to diagnosis in BPD will see the continuation of the use of arbitrary criterion cut-offs in diagnosis, a continuation of the use of polythetic criteria which will in turn lead to an increased heterogeneity of the disorder, and diagnostic confusion in relation to both 'state' disorders and other personality disorders.

This study employed two independent, categorical diagnostic measures for detecting BPD – the MCMI-III and the DIB-R. This approach is argued to be inconsistent with the methods typically employed by clinicians for making BPD diagnoses (Westen, 1997). Therefore, future theoretical research requires the development of diagnostic instruments which more accurately reflect methods used to detect BPD in clinical situations as well as incorporating the benefits that might accrue from the incorporation of a dimensional approach to diagnosis. One issue that has received scant attention in the literature is the role of the examiner as an active rather than passive participant in the diagnostic process. To date, the semi-structured interview instruments employed to diagnose BPD are 'passive' in the sense that they rely upon the respondent admitting or confirming evidence of diagnostic criteria for the diagnosis to be confirmed. It is possible therefore for

a respondent to fail to meet criteria for BPD simply because they deny the presence of features of BPD. This can occur despite the impressions and observations of the examiner. More research needs to be directed toward the development of instruments that reliably incorporate the interviewer's knowledge and experience of the patient as legitimate components of the diagnostic process. These developments should lead to greater efficiency and validity in the making of the diagnosis, and in turn should lead to an increased consideration of personality disorder diagnosis in clinical settings.

At this time, there are a number of instruments available for diagnosing BPD. The current standard does not suggest that one instrument enjoys diagnostic superiority (Kaye & Shea, 2000). No obvious solution to this dilemma exists at this time, but in reflecting on the future of the DSM Axis II, Oldham & Skodol (2000) suggested that the current categorical system be retained, but that the number of different categories of personality disorder should be reduced and stratified. In so doing, Oldham & Skodol also argue that it is possible to incorporate dimensional ratings within an otherwise categorical system. Oldham & Skodol argue that this remains necessary because to move to an exclusively dimensional-based model would be too discrepant from the extant medical and clinical tradition.

Future theoretical (and clinical) research also needs to specify the type of BPD that is being studied, and also needs to specify the dimensions employed in making the borderline diagnosis. For example, it may be appropriate in future to distinguish between BPD participants who present with co-morbid Major Depression from those who do not report histories of depressive affect. Similarly, another important dimension to consider in future studies of BPD is whether

participants meet criterion for Post Traumatic Stress Disorder (PTSD) (American Psychiatric Association, 1994, 2000). Alternatively, the borderline subtypes of Grinker, et al. (1968) still retain clinical merit as they inherently describe differential functional capacities on the part of borderlines. In addition, the Grinker et al (1968) typology also suggests differing types of clinical issues that are likely to be encountered with each borderline subtype. Finally, another solution to this dilemma might include reporting data on each borderline research participant whereby each participant has information provided describing the co-morbid diagnoses they present with. Future studies should therefore specify co-varying clinical phenomena and the dimensions or typologies of BPD being employed.

An alternative approach would be to use the equivalent of a 'Welsh Code' (Dahlstrom et al., 1972), type of rating system for BPD subtype diagnosis. This proposal has some similarities to the method proposed by Oldham & Skodol (2000). This practice would see the employment of a dimensional rating system for each BPD criteria, with cut-offs indicating whether the participant meets criterion on each specific diagnostic dimension of the BPD diagnosis. In the DSM-IV/DSM-IV-TR systems (American Psychiatric Association, 1994, 2000), one participant might achieve the criterion of attaining BPD status on all of the 'affective' dimensions as well as the 'impulse criterion', whereas another participant might meet criterion on all of the affective dimensions as well as the criterion for transient psychotic episodes under stress, but does not meet criterion on the impulsive dimension. The clinical 'feel' and the psychological phenomenology of each of these participants are likely to be very different. By developing a dimensional scoring protocol with an embedded coding system in

the context of a categorical diagnostic system, a more sophisticated description of differing types of BPD could be achieved.

Further research on the role of arousal, priming, and hypervigilance in BPD is also required. In addition, there is a need to better understand the relationship between implicit and explicit processes in the development of borderline conditions. Most importantly, the findings of this study suggest that there is an urgent need to identify other implicit processes operating in BPD. Further research should emphasise the use of information processing paradigms, as at least some of the significant issues in BPD are likely to operate as implicit processes. This orientation emphasises the need to study functions that have typically been subsumed under the rubric of the 'cognitive unconscious'. Finally, there is an urgent need for further neuroradiological investigation BPD in order to identify the neural basis of the implicit processes occurring in BPD. This approach will also require further neuroradiological investigation to identify the specific pathways involved in the forms of priming identified in this study. This will hopefully lead to the development of specific pharmacotherapies and information-processing technologies to either interdict or modify these specific implicit processes. It will also assist in developing an understanding of the psychobiological development of BPD, and provide direction for the development of more sophisticated psychotherapies to treat this condition.

9.6.2. Implications for Future Clinical Research

The findings of the current study have a number of the implications for future clinical research. These include the modification of explicit processes, the psychotherapeutic modification of implicit processes, and the use of information processing paradigms to modify implicit processes.

Many of the current evidence-based psychological interventions designed to treat BPD utilise interventions which interrupt and modify explicit processes (A. Beck et al., 1990; Linehan, 1993; Monsen et al., 1995; J. Stevenson & Meares, 1992), although some other approaches might justifiably argue that at least some components of their approach address implicit phenomenon (Bateman & Fonagy, 2001; Clarkin et al., 1999). The findings of the current study suggest that there is a need to intervene at the level of implicit processes in BPD. There are at least three approaches which might be applied to BPD. The first of these is the development of specific pharmacotherapies or 'neuroceuticals' which might act as antagonists to the development and elaboration of implicit processes. This approach forms the basis for much of the psychopharmacology in this area, but is outside the scope of this thesis and will therefore not be considered further.

The second approach involves the systematic application of well documented and evidence-based procedures which have the potential to modify implicit processes. These might include techniques such as autogenic training, thought stopping, relaxation training, specific forms of hypnosis including EMDR, and specific forms of empirically derived psychotherapeutic transference analysis methods such as the CCRT method (Book, 1998).

A third approach for developing treatments for implicit processes in BPD could utilise information processing paradigms as intervention strategies. There is a long history in both experimental psychology and experimental clinical psychology of the use of information processing paradigms as a methodology for identifying implicit processes. The corollary to this suggests developing information processing paradigms as modes of treatment. This approach could then be used to directly modify the operation of implicit processes. As an

example, there is an abundant literature which describes the role of various learning paradigms in the development of psychopathology. In response, learning theory has been applied in various formats to treat various forms of psychopathology. In particular, various forms of 'relearning' in the form of systematic desensitisation or *in vivo* exposure conditions have been applied to a wide variety of conditions where automaticity of response occurs. It is possible that these types of learning paradigms might also be applied to modify implicit processes using information processing paradigms. One such application might include the repeated administration of a Stroop-like paradigm in a manner consistent with desensitisation or flooding paradigms.

Similarly, it is possible to conceive of the use of information processing paradigms being used to alter automatic processes. The obvious approach would involve using treatment modalities involving the repeated presentation of new stimuli at a subliminal level with the intention of altering implicit schematic processing modalities. There appears to be a very limited literature which examines this possibility, and that which exists is clouded by both methodological and empirical difficulties (Balay & Shevrin, 1988; Reber, 1993). Clearly, there are a number of ethical and methodological issues which need to be addressed prior to the establishment of these approaches as a research programme. The emerging evidence with regard to BPD suggests however, that it is imperative that implicit processes be studied more thoroughly, and interventions developed which address this level of deficit.

There are also a number of specific research implications arising out of the findings of this study. The findings of this study suggest that the difficulties in BPD lie not specifically within the cognitive domain, but rather within an

'affective-attentional' one. Because of the nature of the manner in which this finding was determined (Stroop), it suggests that paradigms emerging out of the 'affective neuroscience' tradition hold promise for further investigation in BPD. In particular, further research is required into the areas of response-conflict and hypervigilance in BPD, and further research into behavioural inhibition employing affective stimuli rather than neutral stimuli is also required.

9.7. IMPLICATIONS FOR CLINICAL PRACTICE

The findings of the studies have a number of implications for clinical practice. These stress the importance of assessment, including the need for accurate diagnostic information, the need for the provision of information and psycho-educative resources, and conceptual and methodological changes to the form and scope of psychotherapy for BPD.

9.7.1. Assessment

The findings reported in Section 5.6.1 found that participants reported the provision of diagnostic information on BPD to be a largely positive experience. This is an important issue, as BPD patients are typically not informed that they meet criteria for the disorder. The participants in this study indicated that knowledge about the diagnosis assisted in making sense of aspects of their self-experience, as they had previously had been led to believe that they suffered exclusively from various Axis I disorders. The provision of information that advised participants that they met criterion for BPD appeared to assist participants to make greater sense of their experience of difficulty as many reported a sense that they believed that their difficulties were more intense and extensive than 'captured' by an Axis I diagnosis alone.

As a result, one of the significant implications for clinical practice is the need to provide accurate diagnostic feedback to persons with BPD and to avoid the often-used strategy of providing them with an Axis I diagnosis only. Clearly, this is a sensitive issue because the use of the term 'personality disorder' has been employed in a pejorative manner for decades. It is also important because the provision of accurate diagnostic feedback allows the person diagnosed with BPD access to a variety of sources of information that can assist in helping them to understand their condition. This includes the nature of the Axis I disorder that in all probability led to their presentation, but also the relationship between their Axis I and BPD diagnosis, and information concerning the *experience* of the difficulties they encounter because of their BPD status. The failure to diagnose BPD does not permit this latter point to be addressed clinically, and therefore does not permit the best standard of care to be provided.

9.7.2. Information Provision and 'Psychoeducation'

One of the other significant implications arising out of the findings reported in Section 5.6.1 suggested that it is important that information concerning BPD be provided to the person. This information should include information about the diagnosis, co-morbidity, and phenomenology of the disorder. In addition, information about the incidence, prevalence, course of the disorder, and finally, treatment information should also be provided.

In addition, Section 5.7 also identified that people with BPD experience significant difficulties with regard to the accurate identification of affective states. This is a fundamental issue because both self and affect regulation are heavily reliant upon the operation of a well-structured and well organised system of affects. If there are deficits in the capacity to recognise and identify affective

states, then it remains highly likely that both self and affect regulation will be impaired.

As a result, one of the implications emerging out of this finding is the need to 'educate' or teach people with BPD to more capably identify affective experiences. One of the practical implications involves developing what might be referred to as a 'language of affect' in persons diagnosed with BPD. This approach essentially involves assisting the person to develop an introspective capacity in order to identify affective states, and then to develop linguistic codes that assist them to articulate these affective states. Whilst this appears to be an obvious intervention, clinical experience suggests that this is a capacity that is deficient in many persons diagnosed with BPD. The importance of this as a primary mode of intervention should not be underestimated. This is an issue also independently identified by Farrell & Shaw (1994).

Study Four found evidence for a colour-naming response latency Stroop effect in BPD suggesting that implicit processes are potentially involved in BPD. Whilst implicit processes do not necessarily equate with 'procedural learning', it is reasonable to suggest that at least some borderline pathology is procedurally learned (Grigsby & Stevens, 2000). It is likely that the interpersonal deficits which characterise BPD are procedurally learned (Grigsby & Hartlaub, 1994). The understanding that a range of procedurally learned processes might underpin borderline pathology is an important factor that could potentially be integrated into psycho-educational programmes for BPD.

This perspective suggests that it is essential to educate BPD patients that the nature of their difficulties have developed over the course of many years, and that a significant component of the disorder might be 'organised' in procedurally

based memory networks (Grigsby & Stevens, 2000). This understanding can then be used to assist BPD patients to understand that when attempts to change are directed at modification through the use of explicit processes only, then the effect of change will be limited and unlikely to result in permanent change. In contrast, procedurally learned routines will only change slowly, and are self-limiting in the degree of change that can be realised. To the extent that implicit (procedural) functions have attained a considerable degree of automaticity, BPD patients must be educated to understand that despite their best intentions, the 'automatic-procedural' nature of much borderline pathology will continue to occur despite conscious intentions to the contrary. This is an important issue to communicate to borderline patients as they are notoriously pessimistic and are easily discouraged from persevering with treatment (Clarkin, 2003; Clarkin et al., 1999; Linehan et al., 1993). By providing borderline patients with a clear rationale concerning the nature of procedural (implicit) change, it might be possible to reduce attrition rates and premature terminations from treatment.

9.7.3. Psychotherapy

The findings of Study Four have significant implications for the nature of future psychotherapeutic practice with borderlines. The specific findings from Study Four that are most likely to affect the nature of psychotherapy practice can be summarised as follows:

The Absence of Impaired Working Memory and Problem Solving

The findings from Study Four suggested that there is an absence of cognitive deficits in BPD. These findings have significant implications for the nature and scope of therapy that might be specifically developed to treat BPD. The fact that these executive functions are intact in BPD suggest that borderline

patients have the ability to recall whole/integrated memory episodes, can engage in acts of reciprocity, can shift-set, take the perspective of the other, employ abstract cognition, and have the capacity to plan and effectively execute future oriented acts.

Therefore, the findings of the study suggest that borderlines have the ability to engage in complex problem-solving activity. This implies that the type of psychotherapy indicated for the treatment of BPD does not need to be modified or simplified to accommodate an impaired cognitive executive. Furthermore, the evidence also suggests that the borderline patient is capable of holding material 'in mind' in order to 'work through' clinical issues. In addition, the non-significant working memory returns contradict the assumption often made that multiple sessions per week of psychotherapy is required because of impaired 'evocative memory' in BPD (Adler & Buie, 1979). It remains the case that treatment models might continue to require multiple-session-per-week therapy, but this must now be accounted for by factors such as 'dose-response' issues in the psychotherapy of BPD (Frank & Frank, 1991). Deficits in mnestic function cannot be used to justify high frequency dose rates of psychotherapy.

The Absence of Impaired Response Inhibition

The finding of adequate inhibitory control in borderlines also provides some reassurance concerning the capacity of this population to engage successfully in psychotherapy. This finding also assists in understanding why the available evidence-based psychotherapies are capable of treating this population. Specifically, the capacity to adequately inhibit behaviour is a crucial factor in successfully engaging in psychotherapy (Frank & Frank, 1991). A number of approaches to psychotherapy share common assumptions with regard for the

need the patient reflect on internal processes, and to integrate affective states into self experience. These acts require the patient to inhibit the desire to *act on* rather than *reflect on* internally mediated experiences salient to their condition (Bateman & Fonagy, 2001; A. Beck et al., 1990; Clarkin et al., 1999; Linehan, 1993; Monsen et al., 1995; Ryle, 2004; J. Stevenson & Meares, 1992). As a result, all of these therapies probably capitalise to some extent on the role that general therapeutic factors contribute to the success of psychotherapy (Frank & Frank, 1991; Orlinsky, Grawe, & Parks, 1994).

More importantly, the findings of an absence of deficits in behavioural inhibition has significant implications for how the issue of 'impulsivity' in BPD is understood and managed in the course of psychotherapy. In earlier sections, various explanations were offered to explain the phenomenon of impulsivity. These included deficits in behavioural inhibition, the selection of operant behaviours with a low probability of success, and the enactment of so-called 'mindless' behaviour (action without thought).

Because deficits in behavioural inhibition are not supported by the available evidence, the alternative explanations outlined above assume greater prominence. What is central to understanding these alternative perspectives is the need to understand the affective and functional properties of 'impulsive' behaviour. It is proposed that 'impulsive' behaviour occurs in BPD because it serves the dual purpose of regulating arousal and/or affect, and enhancing interpersonal effectiveness - it realises outcomes for the subject that would not be realised under alternative circumstances.

This perspective has clear implications for the management of impulsivity in the treatment of BPD. It suggests that the principle task of the

therapist is one of understanding the motivation and purpose of the behaviour. In doing this, it then becomes possible to identify strategies by which the impulsive act can be managed, attenuated, or eliminated as part of the repertoire of the BPD patient.

The Presence of 'Affective-Attentional Bias'

The findings from Study Four suggest that borderlines are probably hypervigilant to external stimuli. Because this finding held at both a supraliminal as well as at a subliminal level, it suggests that either an automatic vigilance process or an implicit attentional mechanism operates in BPD.

This interpretation has significant implications for the treatment of BPD. The available evidence-based psychotherapies for BPD are consistent in that they target the affective, interpersonal, and behavioural features of the disorder. In the main, these approaches do not identify and target implicit processes in psychotherapy. This may be an important future direction in the treatment of BPD. It is possible that the evidence-based models of treatment (Bateman & Fonagy, 2001; Clarkin et al., 2004; Clarkin et al., 1999; Linehan, 1993; Monsen et al., 1995; Ryle, 2004; J. Stevenson & Meares, 1992) might benefit by incorporating aspects of a schematic processing approach (Young, 1990), or Beck's (A. Beck et al., 1990; G. K. Brown et al., 2004) cognitive approach, as these represent two approaches that attempt to address implicit processes in psychotherapy.

Stroop Findings and the Anterior Cingulate

One interpretation of the Stroop findings suggests that they might be an artifact of suppression of anterior cingulate cortical (ACC) function (Bench et al., 1993; Jones et al., 2002; Luu & Posner, 2003; Pardo et al., 1990). Because the

effects of frank brain damage were controlled for in the design of the study, these findings cannot be accounted for by the effects of cerebral trauma. As a result, if the Stroop results could be accounted for by deficits in the functioning of the ACC, then it was probable that this was developmental in nature and a result of either genetic influences, or as a result of 'experience dependent' failures of neural development (Schore, 1994, 2003a, 2003b), or alternately, through some combination of both. Section 9.4.5 further indicated that the ACC was implicated in impairments to long-term attachment, and maternal behaviour and empathic capacity. These deficits are also recognised as central to borderline pathology (Lawson, 2000; Patrick et al., 1994). Therefore, developments in therapy for borderlines also need to target these potential deficits as part of an overall integrated therapy intervention.

The available evidence-based psychotherapies for the treatment of BPD vary significantly with regard to their focus on attachment issues, and maternal and empathic capacity. The Fonagy (Bateman & Fonagy, 2001, 2004), Meares (J. Stevenson, & Meares, R., 1999; J. Stevenson & Meares, 1992), and Monsen (Monsen et al., 1995) groups endorse a treatment approach in which attachment issues appear to be centrally important. In contrast, Linehan's (1993) approach appears to address attachment issues only in a peripheral sense – DBT understands that BPD arises out of the effects of a 'negating' early environment. The Clarkin/Kernberg group (Clarkin et al., 2004; Clarkin et al., 1999; Yeomans et al., 2002) acknowledge the importance of attachment, but attachment themes do not appear to be central to their therapeutic orientation. Similarly, the Beck group (A. Beck et al., 1990; G. K. Brown et al., 2004) acknowledge the role of attachment in BPD psychopathology, but emphasise the importance of schematic

processing in their approach. With the exception of Bateman & Fonagy (Bateman & Fonagy, 1999, 2001, 2004), none of these groups explicitly integrate attachment theory into their treatment approaches. Therefore, it appears that there is scope for the development of treatment approaches for borderlines which assist in the development of more sustaining and secure attachments. One means for achieving this is to incorporate attachment theory principles into evidence-based psychotherapeutic approaches.

Similarly, a number of the available evidence-based psychotherapies for the treatment of BPD focus upon the development of affective regulatory capacity, but not specifically on the development of empathic capacity. The Meares, Monsen, and Linehan groups all emphasise the importance of developing appropriate affect regulation in borderlines (Linehan, 1993; Monsen et al., 1995; J. Stevenson, & Meares, R., 1999; J. Stevenson & Meares, 1992), but do not explicitly emphasise the role of the development of empathic capacity as a treatment goal in BPD. The capacity to develop an empathic stance requires in the first instance, competent affective regulatory capacity (Vaillant, 1997). It therefore appears that one implication of these findings for the development of more comprehensive therapies in BPD is the need to link treatment of BPD more explicitly with the tradition of the psychotherapeutic facilitation of empathic capacity.

The final implication of the association between the Stroop findings and possible ACC involvement suggests the possibility of impairments to parental behaviour in borderlines. This is not an explicit focus in the treatment protocols of any of the aforementioned research groups. Deficits in maternal capacity and maternal behaviour have been noted in borderlines (Feldman et al., 1995;

Lawson, 2000), but this data is interpreted here as suggestive of deficits in parenting ability. The use of the term 'maternal' in this context is not used to suggest that only mothers with BPD should be 'targeted'. The principle applies equally to fathers with BPD. The key issue here is the emphasis on addressing affectional-relational functions in the parent-child relationship, and the need to assist parents with BPD to manage these tasks more satisfactorily.

The implication of this finding suggests that there is a need for the provision of specific interventions that focus upon deficient caregiving and parenting practices. There is an abundant child psychiatric and infant mental health literature that addresses the issue of deficient mothering in the borderline which has yet to be integrated into broader psychotherapeutic approaches for BPD (Bezirganian et al., 1993; Fonagy, Steele, Moran, Steele, & Higgit, 1993; Lawson, 2000; D. N. Stern, 1998).

Hypervigilance

Another interpretation of the Stroop colour-naming response latency findings suggested that these results were an artifact of a hypervigilant attentional set. The specific cause of hypervigilance in BPD is unknown, but good candidate factors appear to be the often reported abuse histories of borderlines (K. R. Silk, Nigg, J.T., Westen, D., & Lohr, N.E., 1997; Zanarini et al., 1997), insecure attachment histories (Patrick et al., 1994), and deficits in parental bonding (Paris & Zweig-Frank, 1993).

One of the implications of the data reported from Study Four also suggested that because all other executive functions were found to be intact, the colour-naming response latency effect probably predates the consolidation of other executive function, suggesting that it was established by adolescence at the

latest (Thatcher, 1991). This further suggests that the causal basis of this effect had its origins in the childhood/adolescent developmental processes of the borderline cohort. If this is the case, then this finding has significant implications for a range of early intervention processes to modify hypervigilance in BPD, and perhaps also to prevent the development of BPD itself. This will be discussed further in Section 9.9.

Despite this, one of the implications of this finding is that borderlines might demonstrate elevated autonomic arousal. Although there is evidence that does not support the view that BPD is characterised by a general affective hyper-responsivity (Herpertz et al., 2000), it nevertheless remains possible to interpret these findings within the framework of an exaggerated arousal/startle response. The suggestion that BPD is characterised by a hypervigilant organisation implicates the autonomic nervous system in BPD. This again confirms the importance of a psychobiological perspective in understanding BPD (Siever & Davis, 1991), and raises the need for the combined use of both psychological and medical treatments in the management of the condition (Soloff, 1989).

Tailoring Psychotherapy to Borderline 'Type'

There is evidence that there are differing borderline typologies (Andrulonis et al., 1982; Grinker et al., 1968; Rusch et al., 1992; Russ et al., 1993), and this, combined with the effects of co-morbidity on the course of the disorder suggest that different clinical issues are likely to be encountered in the therapy of the individual BPD case. One implication of this is that the clinical 'feel' of different borderline patients will differ in part because of the effects of different borderline typologies.

This issue is also important in determining the pace and direction of psychotherapy. Furthermore, it has important implications in assisting psychotherapists to be able to anticipate the types of clinical issues likely to be encountered. At this time, generic forms of treatment (i.e., DBT/TFP) are used for all borderline patients independent of the type of BPD the specific case presents with. It is possible to envisage that for some BPD cases with a greater potential to act in an 'impulsive' manner, a primary therapeutic approach will involve reconstructing impulsive episodes and understanding the motivational factors that precipitated the impulsive act. In contrast, those BPD cases where the subject experiences a greater degree of 'identity diffusion' (Kernberg, 1984) without an impulsive overlay are likely to benefit more from approaches which utilise the development of 'reflective self-functioning' (Fonagy, Steele, Steele, Moran, & Higgit, 1991). Similarly, those BPD cases presenting in a more directly affect-dysregulatory mode are likely to benefit more from an 'affect-validational' approach that assists the patient to develop affect-regulatory capacity (Linehan, 1993).

A further implication therefore suggests that the combination of the effects of attentional bias and the effects of differing borderline typologies mean that adjustments to psychotherapeutic technique will be required. Therefore, psychotherapeutic approaches to treating BPD will need to treat the underlying arousal issues which probably underpin attentional bias. Therapy will also require the use of various 'grounding' procedures which validate the patient's experience (Linehan, 1993). Treatment will also need to focus on 'here and now experience' (Clarkin, 2003; Clarkin et al., 1999), and 'transference

interpretations' will also need to be focussed on present rather than past interactions (J. Stevenson & Meares, 1992).

Finally, therapeutic approaches need to be cognisant of the underlying hard-wired procedurally-learned nature of 'borderline' pathology of which their attentional bias is probably one component. Much of the disturbance in BPD is likely to be encoded in procedural (implicit) cognitive and mnestic systems which are essentially dissociable from declarative (episodic, semantic, or explicit) cognitive and mnestic systems (Grigsby & Hartlaub, 1994; Grigsby & Stevens, 2000).

Another major implication of the findings of this study suggests the need to develop therapies which directly intervene in the implicit processes that appear central to the genesis and maintenance of borderline pathology. This requires clinicians to become familiar with the literature on the 'cognitive unconscious' and to devise therapies employing the findings from this literature. It also suggests a potential role for information processing technologies as adjunctive therapeutic agents in the treatment of implicit processes in borderline pathology. Finally, it also means that the usual expectations that therapy will be brief are probably unrealistic for this cohort and that long-term psychotherapy is likely to be the treatment of choice. There is already some evidence to support this view (Bateman & Fonagy, 2001; Clarkin et al., 1999; Linehan, 1993; J. Stevenson & Meares, 1992).

9.8. IMPLICATIONS FOR PUBLIC POLICY AND EARLY INTERVENTION TO REDUCE THE INCIDENCE OF BPD

One of the central propositions of this thesis suggests that BPD should be understood as a developmental neuropsychobiological disorder. The review of

BPD suggested that whilst there are probable genetic (Parker & Barrett, 2000; Torgersen, 2000) and psychobiological (Siever & Davis, 1991) components to the disorder, there is also evidence that the quality of care in the first years of life (Schore, 2003a, 2003b), the quality of mother-infant attachment (Barone, 2003; Patrick et al., 1994), and the quality of mother-child interaction (Bezirganian et al., 1993), as well as elevated rates of child maltreatment (Mitton et al., 1997; K. R. Silk et al., 1995; K. R. Silk, Nigg, J.T., Westen, D., & Lohr, N.E., 1997), represent significant risk factors for the development of the disorder. Because this perspective emphasises a developmental approach, one implication emphasises the importance of public policy and prevention strategies for reducing the incidence of BPD.

Public Policy Implications

The existence of personality disorders does not appear to be recognised at a public policy level, and they do not appear to be considered as part of National Mental Health Policy. In order for this situation to be addressed, a community debate regarding personality disorders, and BPD in particular, is required. The debate will require further research to be undertaken in order to identify the community-wide effects of this problem. This will require the establishment of appropriate tracking systems for following the developmental pathways of borderline patients. One important aspect of this process is a requirement for estimating the cost burden of BPD to the community. Second, there is a need for a significant community-based education program on BPD. An education programme of the form envisaged will need to target separate groups. The first group would include medical, infant mental health and child psychiatry practitioners, welfare, judicial, criminal justice and allied health professionals. A

key group who are critical to this endeavour include professionals who routinely work with young children – pre-school and kindergarten educators, paediatric and mothercraft nurses, and day-care providers. Secondly, there is also a need to educate the general public about BPD. In each case, these programs will need to identify the risk factor profile associated with the development of BPD. In particular, the importance of risk factors such as childhood abuse and neglect, relationships with caregivers, and the quality of attachment in the development of disordered personality will require attention.

Early Intervention Programmes to Reduce the Incidence of BPD

A second level of public policy development should emphasise the role of community based interventions in order to assist the general quality of care-giving for children, and the role of targeted early intervention with children and families identified as being at risk for the development of disordered self-regulation and consequential personality disturbance. The evidence that early adverse childhood experience (particularly child abuse and disordered attachment) represents a significant risk for the development of BPD in adulthood suggests the need to ensure that adequate child protection systems exist. In addition, there is an urgent need to develop large scale prevention programmes for child maltreatment, and further work is required in preventing the development of insecure attachments between infants and caregivers.

There are a number of strategies that could be appropriately employed in order to address these issues, and they might include interventions for use in day-care, playgroup settings, kindergartens and pre-schools to help children more effectively regulate arousal and consequently, affect. In addition, there is a need to develop programmes to assist parents to communicate affectively with their

children. Schools can also play an important role through the further development of programmes to assist children to develop satisfactory affective communication with each other.

A second area for development suggests the need for ensuring that there are adequately resourced child protection systems to identify and intervene effectively with abusing, neglecting, and 'at risk' families. This public policy measure is centrally important in providing effective structures to address child maltreatment and prevent its most adverse effects from occurring.

Finally, there is an urgent need for direct intervention to occur for mothers known to have BPD and/or depression as these also probably represent significant risk factors for the development of BPD also (Paris, 1999). It is again acknowledged that there are a number of services that work with these populations, but what is required are services that have the capacity to provide ongoing support, case-management, and treatment services over the course of the critical developmental periods of a child's life. These services would also require a greater co-ordination between adult and child psychiatry services, and where appropriate, child protection services also.

9.9. CONCLUDING REMARKS

Despite the various controversies surrounding the entity of BPD, the condition (or something very similar to it) appears to have existed for centuries. Part of the controversy associated with BPD appears to be associated with a phenomenon that is familiar to clinicians but at the same time appears to elude accurate measurement. Faced with this dilemma, one human reaction involves the desire to dismiss the existence of the phenomenon itself, or to reduce it to a measurable set of reliable parameters. The problem with this approach is that it

fails to understand that this disorder has a uniquely subjective and interpersonal phenomenology. Accordingly, it is unlikely that the phenomenon will disappear simply through the desire to wish it out of existence. Clinically, the phenomenon of BPD has too strong a currency for this to occur, and it suggests that the manner of understanding and diagnosing the disorder requires greater elaboration and examination.

It is acknowledged that there is a desire in some quarters to abandon the diagnosis as a result of the difficulties in locating it for scientific scrutiny. This is akin to the ancient Greek proposition that matter was made up of *hyle* – their explanation for atomic and sub atomic matter. The history of remarkable developments in science has always been preceded by an age of scepticism in which the subject under scrutiny has been challenged with regard to its authenticity. From the vantage point of the 21st century, there is no difficulty in accepting the idea that objects exist which cannot be seen by the human eye. This understanding is a relatively recent one, and it requires courage and persistence to maintain a stance in the face of criticism and ridicule because the potential advantages for understanding it can be understood. This is how the study of personality disorders and BPD in particular might be construed.

Therefore, there remains a pressing need to continue to study BPD because this will reduce levels of suffering not just for the person with BPD, but also for those in relationship with a person with BPD. It is also important to continue to study BPD in order to develop services which will reduce the fiscal and social burden of the disorder on the community.

It is acknowledged that there are current controversies with regard to the reliability of the diagnosis, the boundaries of the disorder, and the most

appropriate forms of treatment. These concerns will remain, but it is argued that the future of BPD research will be concerned with increasing community awareness of BPD, preventing its occurrence, understanding its development, and developing more effective and integrated treatments. In particular, further research is required that examines the attentional, motivational, and control systems operating in BPD. In this regard, the role of affective neuroscience will have an increasingly important role to play in the future understanding of this disorder. BPD research has increased exponentially in the past 25 years, and the next quarter century is likely to be an even more exciting era.

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APPENDIX I
ICD-10 CRITERIA FOR
'EMOTIONALLY UNSTABLE PERSONALITY DISORDER – BORDERLINE TYPE'

ICD-10 DIAGNOSTIC CRITERIA FOR EMOTIONALLY UNSTABLE PERSONALITY DISORDER –

IMPULSIVE TYPE (F 60.30)

At Least Three of the Following Must be Present, One of Which Must be Criterion Two (2):

1. Marked tendency to act unexpectedly and without consideration of the consequences (Item 58)

2. Marked tendency to quarrelsome behaviour and to conflicts with others, especially when impulsive acts are thwarted or criticised (Item 30)

3. Liability to outbursts of anger or violence, with inability to control the resulting behavioural explosions (Item 43)

4. Difficulty in maintaining any course of action that offers no immediate reward (Item 11)

5. Unstable and capricious mood (Item 50)

BORDERLINE TYPE (F 60.31)

At least three of the symptoms mentioned in Impulsive Type (F 60.30) must be present, with at least two of the following in addition:

1. Disturbances in and uncertainty about self-image, aims, and internal preferences (including sexual) (Items 5, 6, 7, 25, 56)

2. Liability to become involved in intense and unstable relationships, often leading to emotional crises (Item 26)

3. Excessive efforts to avoid abandonment (Item 48)

4. Recurrent threats or acts of self harm (Item 59)

5. Chronic feelings of emptiness (Item 45)

APPENDIX II
DSM-IV AND DSM-IV-TR CRITERIA FOR BORDERLINE PERSONALITY DISORDER (BPD)

DSM-IV AND DSM-IV-TR CRITERIA FOR BORDERLINE PERSONALITY DISORDER (BPD) (301.83)

A pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Frantic efforts to avoid real or imagined abandonment. Note: Do not include suicidal or self-mutilating behaviour covered in Criterion Five.
2. A pattern of unstable and intense interpersonal relationships characterised by alternating between extremes of idealisation and devaluation
3. Identity disturbance: markedly and persistently unstable self-image or sense of self
4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating). Note: Do not include suicidal or self-mutilating behaviour covered in Criterion 5.
5. Recurrent suicidal behaviour, gestures, or threats, or self-mutilating behaviour
6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)
7. Chronic feelings of emptiness
8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)
9. Transient, stress-related paranoid ideation or severe dissociative symptoms

APPENDIX III
DESCRIPTION OF MEASURES AND THE TESTS USED TO MEASURE HYPOTHESES

SUMMARY OF TESTS EMPLOYED IN THE ASSESSMENT OF EXECUTIVE DISORDERS IN BPD

SCREENING MEASURES

The Positive and Negative Affect Scale (PANAS) (Watson, Clark, & Tellegen, 1988). The PANAS consists of two, 10-item Likert-type mood scales which measure positive and negative affect. The scales enjoy high internal consistency, have low correlations with each other, and are stable over a two-month test-retest period.

The PANAS was used in this study to measure mood state at the time of the assessment, and to control for the possible influence of mood on the execution of other tests in the battery. Respondents were required to complete the PANAS by rating their mood at the time of responding. Each participant provided a positive and negative score on the PANAS. was achieved by summing the scores for each scale.

National Adult Reading Test (NART) (Nelson, 1982).

The NART consists of a list of 50 words listed in order of increasing difficulty. The words are relatively short, but are irregular in the sense that they violate the common rules of English language pronunciation. The participant's task is to read aloud the list of words, and the errors are recorded. WAIS Verbal, Performance, and Full Scale IQ are estimated from the reading score the participant returns.

The NART was employed in this study in order to estimate participant IQ. The NART was scored by counting the number of correctly named words from the word list and referring the raw score to the appropriate table in the manual in order to assess Full Scale IQ.

St. Lucia Word Recognition Test

The St Lucia is an Australian developed word recognition task. The raw scores on the St Lucia were used in this study to assess word recognition capacity, and to control for differential word recognition ability between groups. The variable of word recognition capacity was hypothesized to be a possible confounding factor in the completion of the Stroop task. Therefore, controlling for the possible influence of differential word recognition capacity appeared warranted. The St. Lucia was scored by counting the number of correctly named words each participant nominated from the reading list. Scores on St. Lucia could range between 0 and 50.

Digit Symbol (DS) (Wechsler, 1981)

Digit Symbol is a digit substitution task which requires the respondent to match a number with a paired symbol. The task requires the respondent to pair a number with a specific nonsense symbol and copy it into a legend. The task is a speed and accuracy task with the respondent having a total of 90 seconds to match as many symbols and numbers as possible.

Digit Symbol has traditionally been regarded as a measure of 'General Cerebral Efficiency' and was employed in this study as an index of general quality of cognitive processing. The task was administered and scored according to the principles outlined by Wechsler (1981) which involved counting the number of correctly transferred symbols within a 90 second time-limit. Raw scores were then converted to a scaled score using the transformation table provided by Wechsler (1981). Scaled Scores on Digit Symbol could range between 0 and 20.

The available data on this subtest suggests that DS is the most sensitive measure on the WAIS-R battery to cerebral impairment. DS is generally regarded as having a non-specific sensitivity to cerebral impairment, and is often the first test to show signs of deterioration in the event of major cerebral compromise (Lezak, 1995). For these reasons, Digit Symbol has traditionally been regarded as a measure of 'General Cerebral Efficiency' and was included in this study as an index of general speed and quality of cognitive processing.

The Quick Test (QT) (Ammons & Ammons, 1962).

The QT was originally designed as an intelligence test from which Mental Age and IQ scores could be determined. Subsequent reviews suggest that the QT primarily examines contextually based vocabulary (Lezak, 1995).

The task on the QT involves the participant being shown a card with four pictures on it. The participant's task is to point out the correct picture in response to the examiner nominating a 'prompt' word. Words are scaled in difficulty from 'easy' (age six) through to 'hard' (18+ years). The QT consists of three parallel forms which are roughly equivalent to each other. Form 1 of the QT was selected for use in this study.

Although the QT has been regarded as an IQ test, Lezak (1996) recommends its use as a rapid screen of verbal ability. It was speculated that verbal capacity might operate as a confounding variable in participant's responses to the demands of the Stroop task, and was therefore included as a control for the effects of differential verbal ability in contradistinction to frank IQ. The recommended scoring procedure outlined by Ammons & Ammons (1962) was employed in this study.

ASSESSING WORKING MEMORY AND PROBLEM SOLVING EXECUTIVE FUNCTIONING

Assessment of Working Memory

The following instruments were employed to assess various aspects of working memory.

Logical Memory (LM) (Wechsler, 1987)

LM is a subtest of the Wechsler Memory Scale - Revised (WMS-R) (Wechsler, 1987). LM required the participant to recall two stories read out by the experimenter. The tasks are presented under immediate and delayed (20 minutes) recall conditions. Story A contains 24 memory units, and story B 22 memory units (Lezak, 1995). Participants were credited with one point for each correctly recalled 'idea.' A total score for each story is achieved by summing the correctly recalled ideas for each story. The task was administered under both immediate and delayed (30 minute) recall. The data for each story was then summed in order to provide an LM Total Score. The data was analysed separately for immediate and delayed conditions. LM was employed in this study to assess components of verbal working memory.

Visual Reproduction (VR) (Wechsler, 1987)

VR is a subtest of the Wechsler Memory Scale - Revised (WMS-R) (Wechsler, 1987). In this task four cards are presented to the participant with different designs in increasing levels of complexity. The participant observes each card for a period of ten seconds after which the card is removed from sight and the participant is required to draw the design whilst relying upon immediate recall. VR was assessed under both immediate and delayed (30 minutes) conditions.

The data for each design was summed together to provide a VR Total Score. The scoring of VR was blind-scored by a clinical psychologist with advanced neuropsychological training who was familiar with the VR scoring system as outlined by Wechsler (1996).

There is mixed evidence regarding the capacity of VR to reliably localize organicity, although it is very sensitive to detecting deterioration in dementia. It is therefore not recommended for assessing lateralization of lesion sites, although it appears to be a useful marker in detecting impaired mnestic capacity (Lezak, 1995). VR was employed in this study to assess components of non-verbal working memory.

Paired Associates Learning (PAL) (Wechsler, 1987)

PAL is a subtest of the Wechsler Memory Scale - Revised (WMS-R) (Wechsler, 1987). The PAL task consists of eight word-pairs which participants are required to learn. The task is composed of four easy-to-remember word-pairs, and four hard-to-remember word-pairs. The task was administered under immediate, and delayed (30 minute) conditions. Scoring is based upon the first three trials, although there is provision for an additional three trials to be administered if the participant has not learned the task on the first three trials. The scoring method employed utilised the scoring protocol as outlined by Wechsler (1987), and the output was analysed for immediate and delayed recall conditions. PAL was employed in this study to assess components of verbal working memory.

Digit Span (DSp) (Wechsler, 1981)

DS required the participant to verbally recall strings of digits where the number of digits in each trial increased in number. Because this task required the participant to hold in mind the string of digits, it was adjudged to be assessing a

component of verbal working memory. Digit Span was scored according to the principles outlined by Wechsler (1981), and Scaled Scores were employed in the analysis. DS was employed in this study to assess components of non-verbal working memory.

Visual Memory Span (VMS) (Wechsler, 1987)

VMS is a subtest of the Wechsler Memory Scale - Revised (WMS-R) (Wechsler, 1987). The VMS provides two cards on each of which are eight squares printed in a non-linear sequence. Red squares are included for the task assessing forward span memory, and green squares for the task assessing backward span memory.

VMS requires participants to remember the order of tapping of squares located in an irregular fashion on a board. The task is conducted under two conditions: in the first condition the participant must repeat the same order as the experimenter, and in the second condition the participant must tap the squares in the opposite order to the experimenter. Scoring in both the tapping forward and tapping backward condition involves frequency counting the number of correctly executed trials. VMS was also employed in this study to assess components of non-verbal working memory because the participant is required to hold in mind a sequence of moves before ultimately executing it.

Complex Figure of Rey (Rey Figure) (Lezak, 1995)

The complex figure task was initially developed by Rey in order to examine perceptual organization and visual memory in brain damaged participants. The test consists of a reproduction of the complex figure on a single sheet of paper. The participant's task is to copy the figure onto a blank sheet of paper with coloured felt pens. At the completion of sections of the task, the felt pens are

substituted so that the respective sections are completed in different colours. This enables the sequence of the construction of the drawing to be included when the figure is scored at a later point.

The scoring of the Rey Figure was blind-scored by a clinical psychologist familiar with the Rey-Osterreith scoring system as reported by Lezak (1996). In addition, this judge was also undertaking advanced training in clinical neuropsychology and had received specific independent training in the scoring and interpretation of this procedure. The Rey Figure was employed in this study to assess components of non-verbal working memory.

Assessment of Problem-Solving

Controlled Oral Word Association Test (COWAT) (FAS)

The COWAT consists of three word-naming trials. The set of letters employed in this test, FAS, has been used so extensively that the test is better known as the 'FAS.' The instructions for the test involve the examiner asking the participant to report as many words as they can that begin with the letter F in a one minute time frame. Thereafter, the participant is required to report of as many words as they can that begin with the letters A and then the letter S under the same conditions. The additional task of providing the names of animals was incorporated into the COWAT as there is now a well established research tradition for this practice (Lezak, 1995). This inclusion arose out of research with dementing patients who were unable to associate names with letters, but were able to produce animal names. For the reasons of inclusiveness, this latter component was added to the COWAT.

The COWAT was employed because it has been demonstrated to be a sensitive test of frontal dysfunction. This form of cognitive inflexibility is also associated

with naming disorders and with a reduction in capacity to generate words (Lezak, 1995). The COWAT was employed in this study as a more generic test of executive function associated with the concept of cognitive flexibility.

Participants were instructed to name as many words as they could, beginning with the letter 'F.' They were further advised that they could not use proper names, and could not repeat the same word with a different ending. Each participant was advised that they had 60 seconds to produce as many words as possible. Each word was written down, and a frequency count of correctly articulated words formed the score for the task. Thereafter, the participant was required to respond in the same manner to the stimulus letter "A,' and then to the stimulus letter 'S.' The task was completed by the participant being required to name as many animal names as they could in 60 seconds. The scoring of the COWAT involve summing all of the correct responses for the categories of F, A, and S collectively. These scores were then adjusted for age, gender, and education level. The frequency of responses for the Animals naming trials was analysed separately.

Similarities (Wechsler, 1981)

Similarities is a test of verbal concept formation in which the participant is required to explain what each listed word pair have in common. The word pairs range in difficulty from simple (orange-banana) to difficult (praise-punishment). It is generally regarded as an excellent test of general mental ability and is sensitive to the effects of brain injury regardless of location although there is also some neuropsychological data suggesting that Similarities assesses left frontotemporal function (Lezak, 1995). Its ability to assess concept formation suggests that it is a useful measure to assess executive functions associated with

abstract thinking. Similarities was scored according to the principles outlined by Wechsler (1981), and was used in this study to assess one component of problem-solving executive function.

Wisconsin Card Sorting Test (WCST) (Heaton, 1981)

The original WCST was developed in order to study 'abstract' or 'shift of set' behaviour. The original format of the task utilised 60 cards on which between one to four symbols (triangles, stars, crosses, or circles) were printed in either red, green, yellow, or blue. The task required the participant to match each of the cards in the pack to one of four stimulus cards. These were a red triangle, two green stars, three yellow crosses, and four blue circles. There are three ways in which cards can be matched - by colour, by form (shape), or by the number of symbols on the card (number). The participant is not advised what rule is used for matching, but instead has to determine the rule through hypothesis testing. The participant places each card against one of the four key cards, and is advised if the match is correct. After ten consecutive correct placements, the principle of matching is shifted according to predetermined rules (colour to form to number to colour to form to number) without advising the participant of the shift of set. The test continues until the participant has completed six sets of 10 correct placements.

The WCST can be scored in a number of ways. The most usual method is to score for Number of Categories Achieved, and for Number of 'Perseverative' Errors - the number of times that the participant persists with matching a category when told by the examiner that it is incorrect. The method for scoring employed in this study utilised the administration scoring guidelines employed

by Heaton (1981). This modification of the original WCST utilised two packs of 64 cards, and scored for Categories Achieved and Perseverative Errors.

Whilst the WCST was originally viewed as a test which could detect the presence of frontal lesions, recent research does not support this view of the test. The available evidence suggests that the WCST is sensitive to diffuse dysfunction, but should not be used to identify lesion sites or as a marker of frontal dysfunction. Heaton (1981) regards the WCST as an effective measure of executive function, but not as an instrument sensitive to frontal impairment. The WCST was used in this study to assess one component of problem-solving executive function.

Austin Maze (Walsh, 1978)

The contemporary form of the Austin Maze was first reported by Milner (1965) who used it to differentiate the learning performance of brain injured patients with diverse cerebral lesions from controls. The Austin Maze consists of a 10 x 10 matrix of buttons housed in a sealed box. Each button is connected to a 240v electrical supply, and each button when depressed activates either a red or a green light. The objective is to learn a 28-choice pathway on a trial and error basis. Depressing a button on the pathway activates a green light, and depressing a button which is not on the pathway activates a red light. A counter built into the casing of the maze automatically count errors committed by participants. In Australia, the failure to attain errorless performance has often been interpreted as providing evidence of frontal-lobe impairment (Bowden & Smith, 1994; Walsh, 1978).

Participants were instructed to follow the following rules in completing the task:

1. Participants were to proceed in one button steps. Participants were permitted to move in the vertical or horizontal plane, but could not move diagonally on the matrix;
2. Where an incorrect (red) button had been depressed, participants were required to return to, and depress, the immediate previous correct-pathway (green) button, but no further;
3. Participants were instructed to not hold down the button for very long as this increased the chances of 'double-pressing' the button. In the case of an incorrect button, this would increase the likelihood that they would inflate the participants' error score.

Participants were instructed to traverse the maze until they had achieved the criterion of one error-free trial, or had unsuccessfully completed 10 trials.

In a re-examination of a number of data sets on the Austin Maze, Bowden & Smith (1994) note that 10 trials appears to be the optimal cut-off point for the number of trials participants should complete. Furthermore, Bowden & Smith report that the evidence supporting the Austin Maze as a test of prefrontal lesions is lacking. They suggest however, that it might be sensitive to compromise in functional neurotransmitter systems which have cortical representation in the prefrontal region. The Austin Maze was used in this study to assess one component of problem-solving executive function.

Tower Of London (TOL) (Shallice, 1982)

The TOL was employed to directly assess the central executive function of problem solving. Humes et al. (1997) provide a test protocol requiring the solution of 15 problems at six levels of difficulty. This protocol was used in the present study. The levels of difficulty refer to the number of moves required to

successfully solve the problem. The rules for the TOL include: (1) Participants can only move one ball at a time; (2) the moved ball can only be on a peg or in the participants hand, not on the table; the length of the pegs requires that only one ball can be placed on the shortest peg, two balls on the middle peg, and all three balls on the tallest peg; (3) participants must be told the number of moves required to solve the problem. The task commences from the same start state for each trial, and a second model of the TOL is constructed by the examiner prior to the participant's trial in order to demonstrate to the participant the final design. The participant is not shown the sequence of the moves required for successful execution, simply the required final outcome. Scoring for the TOL was done using the scoring protocol reported by Humes et al. (1997) which involved 6 points on the first attempt, 4 points on the second attempt, 2 points on the third attempt, and 0 points for no solution within the time limit. Total scores for the TOL could range from zero to 90.

Tower Of Hanoi (TOH) (Simon, 1975).

The TOH is a disk transfer task which includes a base with three equal length pegs spaced equidistantly with four graduated plastic disks measuring six, eight, 10, and 13 cm in diameter respectively. In the version of the TOH employed in this study, the base was expanded in order provide an additional row of three pegs with disks in order set up the goal configurations for the participant to follow. The requirement of the task is that the front row of disks must be moved one at a time on the three pegs to duplicate a goal configuration on a second row of pegs. In this version of the TOH, participants were required to: not place larger disks on top of smaller ones, only move one disk at a time, and a disk was only able to be in the participants hand or on a peg at any time.

The assessment consisted of the administration of a 12 problem set of arrangements reported by Humes et al. (1997). These included:

1. Three, three-disk ‘tower ending’ goals (5, 6, and 7 move problems)
2. Three, three-disk ‘flat ending’ goals (5, 6, and 7 move problems)
3. Three, four-disk ‘tower ending’ goals (7, 11, and 15 move problems)
4. Three, four-disk ‘flat ending’ goals (7, 11, and 15 move problems)

‘Tower ending’ problems are defined as tasks where all disks are placed on one peg, and the endings were alternated between tower and flat ending goals. ‘Flat ending’ problems were defined as tasks where the disks were dispersed across the pegs. Participants were permitted six trials per problem with a maximum of 20 moves per trial to solve each problem correctly. The problem had to be correctly solved in two consecutive trials for the participant to receive points and proceed to the next problem. If the participant failed to solve a problem twice in succession, testing on the TOH was terminated, and the participant received zero points for all subsequent problems in the TOH series.

Scoring on the TOH was calculated by the assignment of points based on the number of trials required for the correct execution of two consecutive solutions:

1. Six Points: Trials 1 & 2
2. Five Points: Trials 2 & 3
3. Four Points: Trials 3 & 4
4. Three Points: Trials 4 & 5
5. Two Points: Trials 5 & 6
6. Zero Points: No Consecutive Correct Points

The total score on the TOH could range between 0 and 72 points.

APPENDIX IV
PERSONAL COMMUNICATION FROM MARY ZANARINI REGARDING THE DIB-R

-----Original Message-----
From: ctheunis@murdoch.edu.au [mailto:ctheunis@murdoch.edu.au]
Sent: Friday, 11 December 1998 10:19 AM
To: chris.theunissen@health.wa.gov.au
Subject: Re: Diagnostic Interview for Borderlines - Revised.

>Date: Mon, 18 May 1998 13:10:11 -0400
>From: mzanarin@warren.med.harvard.edu (mary zanarini)
>Subject: Re: Diagnostic Interview for Borderlines - Revised.
>To: ctheunis@murdoch.edu.au (chris theunissen)
>Content-Description: cc:Mail note part
>
> Dear Chris,
>
> I have a concern that the DIB-R version that you have may be an out of
> date one. I found out recently that people have been contacting John
> Gunderson's secretary about the DIB-R and she was sending out one that
> was dropped from use about a decade ago. If you would fax my lab
> (617-855-3580) with a brief letter about your plans to use the DIB-R,
> I will have the latest version sent to you. There is no need for a
> formal permission process or to pay any money. Training is not
> mandatory but is advisable. If you have funds for training, I could
> set up a program of training for you and/or your staff.
>
> I hope this is helpful.
>
> Mary Zanarini
>
>
>_____ Reply Separator
_____
>Subject: Diagnostic Interview for Borderlines - Revised.
>Author: ctheunis@murdoch.edu.au (chris theunissen) at HMS-Internet
>Date: 5/15/98 3:03 PM
>
>
>Dear Dr. Zanarini,
>
>Chris Theunissen is my name. I attempted to correspond with you some two
>years ago re: the DIB-R, but I suspect that you did not receive my letter.
>
>I am a Senior Clinical Psychologist with the University team attached to
>Fremantle Hospital Dept of Psychiatry. This is an affiliated teaching
>hospital attached to the University of Western Australia.
>
>I am in the process of conducting research into Borderline Personality
>Disorder, and am interested in using the DIB-R for the purposes of
>diagnosing BPD. I understand that you have been central in the development
>of this instrument, and therefore I am writing to ask the following:
>
>1. What permission do I need to get to use the instrument?
>
>2. Is there a requirement for training or licensing in order to use the DIB-R?
>
>3. Do I need to purchase copies of the DIB-R? There are copies of the DIB-R
>floating around here in Australia, but they appear to be pirated copies.
>
>I would appreciate your correspondence on these matters as I am anxious to
>commence my research.

>
>On a more personal note, I have read your research over the years and I
>think it has been a most useful contribution to a very difficult area of
>clinical work. With this in mind, I wpould appreciate maintaining a low
>level of correspondence with you if that's o.k. I have no doubt that you are
>inundated with e-mail and correspondence, so responding must become a bit of
>a pain after a while.Many thanks for your attendance to this matter.
>
>Cheers.
>
>Chris.
>
>

APPENDIX V

DIAGNOSTIC INTERVIEW FOR BORDERLINES – REVISED (DIB-R)

REVISED DIAGNOSTIC INTERVIEW FOR BORDERLINES

(DIB-R)

John G. Gunderson, M.D.

And

Mary C. Zanarini, Ed.D

McLean Hospital
Harvard Medical school

For further information concerning the DIB-R, contact Dr. Mary C. Zanarini at McLean Hospital, 115 Mill St, Belmont, MA 02178. Revised: September 1983. Modified February, 1992

DESCRIPTION

The revised DIB is a semistructured interview that collects information in four areas thought to be of diagnostic importance for Borderline Personality Disorder: affect, cognition, impulse action patterns, and interpersonal relationships. It rates 97 items concerning the way that the patient has felt, thought, and behaved during the past two years. The patient is the sole source of information for the vast majority of these items, but a small number permit the use of an additional data source as well. The interview is further divided into 24 subsections and the information gathered from 22 of these subsections is used to rate 22 capitalized statements called SUMMARY STATEMENTS. Each of these statements represents an important diagnostic criterion for Borderline Personality Disorder and is used to assess the presence or absence of this disorder. Information from the other two subsections weighs negatively against a borderline diagnosis (items # 24 and 58) and is used in determining the patient's final score in the affect and cognition sections respectively.

INSTRUCTIONS

Probe further if the patient has misunderstood a question or has given an answer that seems incomplete, contradictory, or untrue. Also probe further if a specified set of questions provides insufficient information to rate a summary statement.

Circle the number that represents the best answer to a question or Summary Statement. Unless otherwise specified, all questions and Summary Statements are rated: 2=YES, 1=PROBABLE, 0=NO. If a question is not applicable, write N.A. to the right of it's scoring set.

For each section, add the Summary Statement Scores to obtain a SECTION SCORE.

Convert the Section Score to a SCALED SECTION SCORE of 0-2 or 0-3 by following the directions provided at the end of that section.

Total the Scaled Section Scores to obtain an overall revised DIB SCORE of 0-10.

Use the following guidelines when making a diagnostic assessment at the end of the interview: a revised DIB score of eight or more is considered indicative of Borderline Personality Disorder, while a revised DIB score of seven or less is considered indicative of another clinical syndrome.

BACKGROUND INFORMATION

1. Patient's Code Number: _____

(Patient's Name: _____)

2. Status At Time of Interview:
1. Inpatient
2. Outpatient
3. Nonpatient
_____

(Date of Interview: _____)

(Institution: _____)

(Interviewer's Name: _____)

3. Age: _____

4. Sex: 1. Male 2. Female _____

5. Marital Status: 1. Never Married 2. Ever Married _____

6. Race: 1. White 2. Nonwhite _____

7. Education: Years of Completed Schooling _____

8. Occupation:
01. Professional
02. Managerial
03. Technical
04. Clerical/Sales
05. Skilled Labor
06. Semiskilled Labor
07. Unskilled Labor
08. Student
09. Houseperson
10. None
_____

9. Hollingshead-Redlich Social Class: 1-5 _____
(This rating should be based on the education and occupation of the head of the household in which the patient resides if he or she is not financially self-sufficient.)

Before we begin, I want to point out that most of the questions in this interview pertain to the past twp years of your life or in other words, the period since (APPROPRIATE MONTH, DAY, AND YEAR). I also want to point out that I'm mainly interested in learning about feelings, thoughts, and behaviours that have been typical for you during this two year period. However, I will be asking you a number of questions about specific behaviours that you may have engage in only when you were particularly upset or in crisis.

AFFECT SECTION

During the past two years, have you…

Depression

1. …felt quite down or depressed a lot of the time? (2,1,0)

2. …had any periods when you were very depressed every day for two weeks or more? (2,1,0)

3. S.1 THE PATIENT HAS HAD A CHRONIC LOW-GRADE DEPRESSION OR EXPERIENCED ONE OR MORE MAJOR DEPRESSIVE EPISODES 2,1,0

4. …felt helpless for days or weeks at a time? (2,1,0)

5. How about hopeless? (2,1,0)

6. Worthless? (2,1,0)

7. Extremely guilty? (2,1,0)

8. THE PATIENT HAS SUSTAINED FEELINGS OF HELPLESSNESS, HOPELESSNESS, WORTHLESSNESS, OR GUILT 2,1,0

Anger

9. …felt very angry a lot of the time? (2,1,0)

10. How about furious or enraged? (2,1,0)

11. …often been sarcastic? (2,1,0)

12. How about argumentative? (2,1,0)

13. Quick tempered? (2,1,0)

APPENDIX VI
‘OWNING’ ONE’S PERSPECTIVE

OWNING ONE'S PERSPECTIVE

Preamble

The guidelines on 'owning one's perspective' (Elliot et al. 1999), require the researcher to specify their theoretical orientation and their personal anticipations regarding the data in advance, and how it might be modified across the course of the study. This is referred to as 'owning one's perspective'. Owning one's perspective also requires the researcher to communicate their values, interests, and assumptions. The remainder of this appendix articulates my perspective on BPD to the extent that I am consciously aware of it. This section will be written in the first person as recommended by Elliot et al. (1999) as a means of locating myself within the subjective matrix of material to be reported.

Locating Myself Within the Subjective Matrix of the Material

I am a clinical psychologist who has practiced in a full-time capacity for a period of 20 years. During that period, my clinical experience has included practice in outpatient and inpatient adult psychiatry, 'child protection' experience, establishing a child sexual abuse treatment programme, medical psychology, and paediatrics. In 1999, I accepted a tenured position at Edith Cowan University on a half-time basis. Since then I have combined clinical practice with teaching and research. In addition, I maintain a small private practice in which I practice long-term, psychoanalytically informed psychotherapy.

My original training in clinical psychology (University of Western Australia) emphasised a cognitive-behavioural orientation within a 'scientist-practitioner' paradigm. In addition to this paradigm, the programme also included exposure to psychoanalytic theory, structural and strategic systems theory, and person-centred child psychotherapy. Since graduation I have been influenced by, and

received advanced training in, contemporary models of psychoanalytic practice which have emphasised self-psychological (Kohut, 1977, 1984) and object-relational (Klein 1957) perspectives. In addition, other clinical influences have included ego psychology (Kernberg, 1975, 1984) attachment theory Bowlby (Bowlby, 1969; 1973) and a specific theory of emotions referred to as 'Script Theory' (Tomkins, 1962, 1963, 1991, 1992).

In summary, my clinical approach is a developmental psychodynamic one which attempts to understand and treat the experienced self of the subject through the application of a method of relatedness referred to as 'empathic attunement'. This approach emphasises the need to understand the transactions of affectively valenced material between patient and therapist, or in this case subject and interviewer.

The implications of this theoretical grounding have resulted in an approach to engaging with clinical (interview) material in the following manner:

1. Clinical (interview) material was listened to with an 'ear' for affective content. There were as I saw it, two principal components to this. Firstly, the semantic content of the interview is monitored for evidence of affectively based material. This could either be overt in the sense that the subject of the investigation volunteered semantic information concerning their affective experience, or alternatively, affectively valenced material is obviously implied or overtly stated by the semantic content of the interview material. This approach is driven by my understanding of Tomkins affect theory which proposes a distinct number of specific categories of affect which are emergent at birth and mature and develop in response to appropriate gradients of affective attunement from the infant's caregivers (Stern, 1985).

One implication of this approach is the capacity to examine ‘affect regulation’. What is of particular interest to me is the examination of how the participant ‘regulates’ affect. Clinically, the more important issue is examining difficulties in the subject's ‘regulation’ or management of affect. Difficulties in regulating affect are clinically demonstrable often when the subject appears to use some form of behavioural enactment in order to manage a difficult or distressing affective state. This is understood as a sign of lack of regulatory capacity, and also as a lack of capacity to employ more sophisticated symbolisation or representational strategies to modulate or regulate affect. I attempt to understand what function the specific behaviour serves with regard to affect regulation, and to what extent the subject is consciously aware of their affect state when behaving (acting out) in such a manner.

2. Interview material is understood to be organised by the subject within a developmental context. The implication of this understanding suggests that:

a. Material which appears to relate exclusively to a current event or process is understood to be influenced by past relational experiences. As a result, the capacity to relate to others, and to develop ‘models in mind’ for relating to others develop progressively over the course of the subject’s life. This in turn means that these modes are slow to change, and current interpersonal arrangements have earlier relational referents.

b. Whilst material might be provided within the context of the current time-space-place frame of reference of the subject, it does not necessarily mean that the material is psychologically organised in this manner. For example, whilst an adult subject might report a traumatic event from their past, it might be reported ‘as if’ the person is currently experiencing the event.

My clinical interest in borderline phenomena was shaped by early professional experiences as a newly qualified clinical psychologist. A number of my early cases involved people reporting a range of difficulties including mood disorder and so-called 'disorders of volition'. This latter category included people who experienced difficulties in adjusting to the demands of adult life, experiencing a sense of internal/subjective emptiness, and/or feeling that their life lacked a sense of overarching meaning or purpose.

It was my experience that my original training in clinical psychology did not prepare me to manage clinical material of this nature. The research literature of the time (early 1980's and largely cognitive-behavioural in nature) did not specifically address these themes, and provided little advice concerning the types of interventions that might assist people with these difficulties.

As a result, I began to explore other literatures and received clinical supervision from senior colleagues who were experienced in managing these types of cases. I came to understand that often these were cases associated with ‘borderline’ pathology. It became clear that the psychiatric literature addressed (in an objective manner) the issues associated with personality disorders and more specifically, BPD. The psychoanalytic literature of the time also explored the subjective experience of what might be termed the ‘borderline spectrum disorders’ and offered a substantial body of understanding concerning these problems. This exposure originally led to a redirection of my clinical work away from CBT toward a psychoanalytic-developmental perspective. This perspective is, however, in a state of reorganisation as a result of the literature review that comprises Chapter Two. It is clear that future models of treatment of BPD will draw from Paris’ (1999) concept of multidimensional risk factors for BPD, and

this is likely to result in integrationist models of therapy that draw from diverse evidence-based sources that include neurobiological, neuropsychological, psychoanalytic, and cognitive-behavioural traditions.

APPENDIX VII

DESCRIPTIONS OF EACH RESEARCH PARTICIPANT

DESCRIPTIONS OF PARTICIPANTS IN STUDY

Participant One

Research participant number one was a divorced woman in her mid 40's who was referred specifically because she had been diagnosed by the referring psychiatrist as meeting ICD-10 criterion for BPD. It should be noted that ICD-10 is the diagnostic system used within the Western Australian Health system. This participant had an extensive history of prior admissions to hospital in another state of Australia over a five-year period from her mid 30's. Originally, this participant had received the diagnosis of Major Depressive Disorder, but her condition continued to deteriorate to the point where she made a number of suicide attempts. The participant reported that these were motivated by the 'wish not to live'. At the time of recruitment into the study, the participant had recently returned to live in Perth because of the anticipated death of a parent and the desire to resolve difficulties with this parent. It was within the context of this return to Perth, and the disappointments associated with her attempts to restore her relationship with her parent that she was recruited into the study.

Historically, the participant is one of three children. She reported a series of childhood experiences of extrafamilial sexual abuse and serious episodes of sadistic physical and emotional abuse from one of her parents when the other parent was absent. The participant lives alone, and experiences a series of superficial and brief relationships, which are characterised by significant degrees of relational conflict. The participant sought assistance because her treating psychiatrist in the state she previously resided in insisted that she seek treatment upon her return to Western Australia.

Participant Two

Research participant number two was a divorced woman in her early 40's who was in a long-term de facto relationship. This participant had been referred because of long-standing difficulties in managing an intermittent dysthymic disorder. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The participant admitted to one psychiatric admission in her 20's for a major depressive episode. The participant attributed this admission to the interpersonal stress associated with emotional and physical abusiveness of her former husband. The participant also admitted to a long-standing history of injecting drug use, which was supported by sex work.

Historically, the participant is one of six children. She reported a history of intrafamilial sexual abuse with a maternal uncle, which continued until the death of the uncle when the participant was approximately 19 years of age. The participant has been in a second relationship for a number of years but reported bewilderment that someone could care about her. She admits to being terrified at the loss of the relationship. This participant sought assistance because she recognised that her anxiety regarding the loss of her relationship was unreasonable, and masked issues associated with her history of childhood sexual abuse.

Participant Three

Research participant number three was a single woman in her late 20's. She was also a single mother of a 10-year-old daughter. This participant was referred for psychotherapy for a mood disorder and also to assist her in addressing attachment issues with her daughter - she reported rejecting her daughter and

expressed concerns with regard to her capacity to emotionally care for her daughter. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The participant confirmed one brief psychiatric admission at age 16 for a situational crisis associated with her mother's demand that she leave home. This situation was provoked by the participant's disclosure of her sexual abuse by her step-father.

Historically, the participant is the youngest of three children. Her parents separated when she was approximately five years of age. Her mother refused her father access to the participant throughout her childhood, and the participant understood throughout her childhood that her father did not wish to see her. The participant found this account of events was found to be incorrect when reunification between the father and the participant when she was an adult.

After her parents separated, the participant's mother remarried, and from the ages of eight to approximately 15 years of age the participant was involved in a sexually abusive relationship with her stepfather. The participant initially sought assistance with regard to managing anger more effectively and in managing her daughter's behaviour more effectively.

Participant Four

Research participant number was a 30-year-old woman who was referred after attempting to kill herself through an episode of carbon monoxide poisoning. The participant had been found by a friend, and brought to the emergency department of Fremantle Hospital. At the time of the suicide attempt, she had recently experienced the termination of a relationship she had been in for a number of years. Psychiatric review at the time of admission determined that the participant

met criterion for BPD, and she was referred for psychotherapy. Psychological assessment conducted at the initial interview further confirmed that the participant met the study criteria for BPD.

The participant admitted to no other psychiatric admissions other than the one, which occurred approximately three weeks prior to the referral, described above. She was a middle level manager in a government business enterprise, and appeared committed to her career, but struggled with the human resource and customer liaison components of the role.

Historically, the participant was the only child of a couple whom separated when she was approximately five years of age. She reported a conflicted relationship with her mother and a distant relationship with her father. The participant attributes this in part to her mother's refusal to allow contact between the participant and her father during her childhood. The participant had sought psychological assistance for specific supportive work with regard to her depressive mentation and ongoing risk for suicide.

Participant Five

Research participant number five was a 24 year old single man who had been diagnosed Hepatitis C positive, and had relocated from another state to Western Australia in order to receive interferon therapy. At the time, Western Australia had the most liberal admission protocols for the accessing interferon therapy. The patient was aware of this, and had moved to Western Australia to increase the likelihood of receiving treatment. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The patient was referred for assessment to the clinical psychology service for psychological support whilst receiving interferon treatment. He had admitted to a

long-standing and untreated depressive disorder and as a result was referred for assessment. It was in the context of the initial interview and psychological assessment process that the participant was diagnosed as meeting the study criteria for BPD.

Historically, the participant is the youngest of three boys. His parents are reported as 'skid row' type alcoholics, and he reported an ambivalent relationship with both parents. He reported that his father was once a successful businessman, but he went bankrupt as a result of alcohol abuse. The participant further reported feeling trapped in the position of attempting to caretake both of his parents. The move to Perth was an attempt to resolve some of the difficulties he experienced in managing his parents, as well as addressing his medical issues regarding Hepatitis. He reported feeling very frustrated and angry with his father in particular, and this seemed to be associated with the participants feeling of disappointment in his father.

Participant Six

Research participant number six was a 33 year old married woman with three children. The participant was referred for assistance in addressing cleaning rituals (obsessive-compulsive disorder), and in the context of examining this, the participant was found to meet criterion for BPD.

Historically, the participant grew up in a regional centre of Western Australia, and came to Perth to live in early adolescence. The participant reported significant difficulties in the management of anger, and engages in obsessive-compulsive behaviour in large part as an affect regulation mechanism to manage anger. She reported conflictual relationships with her husband, mother, and eldest son.

The participant also admits to significantly impoverished memories of her childhood. She reported that she could not recall any memories of her life before the age of seven and clear memories of her life only began to emerge from around the time of her adolescence. She reported that she believed that she had been sexually abused as a child, but could not recall clear examples of this.

Participant Seven

Research participant seven was a 31-year-old single man referred from the sexual health service of the department of Infectious Diseases. He was referred after medical review because he reported signs of depression, conflict with his partner, and admitted to unresolved difficulties with his parents. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The participant was the youngest child of three in his family, and has an older brother and sister. He described his father as an alcoholic, and appeared to idealise his mother whom he described as a 'saint'. The participant reported that his early childhood was neglectful, and he recalled truanting from school regularly, and sneaking out at night to spend time at a local fairground. He reported that it was in this location that he began to be solicited by older men for sex. He described a self-experience whereby he believed that he was a 'sexually abused child'.

Clinically, the participant sought assistance for a number of co-related issues. Principally, the participant was aware of his difficulties with regard to regulating affects, and in particular, experienced significant difficulties managing anger and rage. In addition, he reported significant difficulties with regard to trusting people, and finally, the participant was generally dissatisfied with his life, and

wanted to examine other opportunities to redirect his life in a more satisfying manner.

Participant Eight

Research participant number was a 52-year-old divorced woman who had made a number of serious suicide attempts over a 10-year period. In addition, it appeared that she had developed a dependence on both benzodiazapines as well as to prescription painkillers. The Mental Health Directorate of Fremantle Hospital had referred the participant for supportive psychotherapy. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The participant reported that her difficulties had first emerged when her marriage ended approximately one year prior to her first psychiatric admission. She reported that the loss of her marriage had been devastating, and had resulted in a severe depressive episode. Thereafter, the participant felt that she never fully recovered and began using various psychiatric medications and began to abuse pain medication after she sustained an injury.

The participant was an only child who grew up in Great Britain in a single-parent household. She reported that she did not know her father, and described her mother as abusive. Although her memories of childhood are impoverished, she recalled being smacked regularly by her mother. She reported feeling little warmth from her mother, and recalls the experience in childhood of wanting to grow up so that she 'could be free'. Her marriage took place when she was 18 years of age, and it was her husband's decision to move to Australia. The marriage ended unexpectedly approximately 15 years later, and the participant was left to raise two children by herself. It appeared that the participant was unable to embargo her emotional difficulties from her children, and they often

directly witnessed her suicide attempts. This had in turn resulted in one of her children also making a series of suicide attempts.

Participant Nine

Research participant number nine was a 41 year old married woman with two daughters who had recently separated from her husband. She reported that she had been married for approximately 30 years to a man whom she felt extremely hostile towards. Some eight months prior to referral, the participant had attempted suicide by taking sleeping pills. She reported that she had felt increasingly trapped within the marriage, and a suicide attempt was enacted as a means for solving the interpersonal conflicts reported as central to her dilemma.

Historically, the participant is the oldest child of parents who moved to Australia in the years after WWII. She reported significant conflict with her parents, and her father in particular. He is described as dictatorial, rigid, and has a history of severe physical abuse of the participant. Despite this, the participant reported a desire to somehow resolve her issues with her father although she feels that this remains unlikely.

Participant Ten

Research participant 10 was a 42-year-old married woman referred on self-request from the Sexual Health Service of the Department of Infectious Diseases. The participant sought assistance with regard to addressing issues associated with what she described as ‘rage’ attacks. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The participant reported that she had grown up in the south-east region of Australia. She described her early history as unremarkable and claimed that her childhood was an essentially happy one. Despite this, the participant was unable

to recall a coherent narrative regarding her childhood, and only began to report a coherent 'life-story' from the age of approximately 16 onward.

The participant reported that she was in a stable marriage in the sense that she and her husband were committed to each other. She reported significant guilt with regard to her relationships with her husband and two, now adult, children. The basis of her guilt was associated with her manner of treating them. It appears that there was a long history verbally abusive and physically aggressive behaviour being directed to all members of her family. The participant was unable to account for this phenomenon, but indicated that she had consulted numerous psychiatrists about this matter over the years. Her last treating psychiatrist indicated that this behaviour was associated with alcohol misuse, but the participant vehemently denied abusing alcohol.

Participant Eleven

Research participant 11 was a 32-year-old single woman referred from the sexual health service of the department of Infectious Diseases. The participant had experienced a number of intermittent sexual relationships during the seven years prior to attendance at the clinic. Each relationship ended with the participant experiencing these terminations as surprising and reporting a sense of lack of awareness that the relationship was in jeopardy. Psychological assessment at initial interview confirmed the diagnosis of BPD.

The participant sought therapeutic assistance in the context of a recent termination of a long-standing relationship. She reported feeling devastated at the termination of this relationship, and experienced a sense of loss and emptiness afterward. Her presentation was driven by a desire to understand the nature of

these difficulties and to examine what role she might play in the development and continuance of these difficulties.

Historically, the participant was the oldest daughter from a family resident in suburban Perth. Her early life was spent in a third world, non-English speaking culture and the effect of this appears to be one where the participant reported an organising self experience as an ‘outsider’. Phenomenologically, this participant was able to articulate a sense of her self experience that was characterised by a sense of inner emptiness, a lack of felt self-coherence, and a sense of identity that was experienced by the participant as ‘vague’.

APPENDIX VIII

STUDY II INITIAL SCREENING INTERVIEWS

STUDY II INITIAL SCREENING INTERVIEW

NEUROLOGICAL HISTORY

Throughout the course of your life, have you:

1. Ever experienced some form of neurological illness, such as:

Meningitis? *Yes/No*

Encephalitis? **Yes/No**

Epilepsy? **Yes/No**

Have you experienced any other neurological illness?

_____

2. Ever suffered from 'seizures'? **Yes/No**

If so, what is the nature of these?

_____

3. Ever experienced some form of head trauma? **Yes/No**

Head injury? **Yes/No**

Loss of consciousness? **Yes/No**

(In excess of five minutes at any time in life)

4. Ever consulted a neurologist? **Yes/No**

If so, what were the reasons the consultation?

_____

Did the neurologist prescribe medication? **Yes/No**

Refer you to a neurosurgeon? **Yes/No**

5. Have you undergone neurosurgery? **Yes/No**

6. Please describe the nature of this procedure?

_____

7. Which hand do you write with?

Right/Left/Ambidextrous

8. Which hand do you throw with?

Right/Left/Ambidextrous

9. Which hand do you kick a ball with?

Right/Left/Ambidextrous

10. Is English your 'first' language? **Yes/No**

PSYCHIATRIC HISTORY

Throughout the course of your life, have you ever:

1. Received Electroconvulsive Therapy (ECT)? **Yes/No**

   Within the past 90 days? **Yes/No**

2. Experienced a history of psychotic illness? **Yes/No**

   (Psychotic illness was defined as Schizophrenia,

   Bipolar Affective Disorder, Schizoaffective

   Disorder, Organic Psychosis, or

   Psychosis Not Otherwise Specified.

3. Have you been 'using' street or illicit drugs in the

   past 90 days? **Yes/No**

   (Defined as Marijuana, Heroin, Ecstasy, Amphetamine,

   LSD or other known substances)

COLOUR SCREENING

Show Participant the Four Colour Swatches.

Could you tell me the colour of these swatches?

Red Swatch Administered

Correct/Incorrect

Green Swatch Administered

Correct/Incorrect

Blue Swatch Administered

Correct/Incorrect

Yellow Swatch Administered

Correct/Incorrect

APPENDIX IX.
STUDY II INFORMATION SHEET

INFORMATION SHEET

TO BE USED IN CONJUNCTION WITH THE CONSENT FORM

AFFECTIVE AND SEMANTIC REPRESENTATIONS IN BORDERLINE PERSONALITY DISORDER (BPD): A DISCOURSE ANALYSIS.

I invite you to participate in a clinical research study examining the ways in which people diagnosed with Borderline Personality Disorder (BPD) think and feel. This study has been approved by the Fremantle Hospital Ethics Committee.

If you decide to take part in the research study, it is important that you understand the purpose of the study and the procedures you will be asked to undergo. Please read the following pages which will provide you with information about the procedure involved, the potential benefits, discomforts, and precautions of the study.

For some of you, this will be the first time you have been told about Borderline Personality Disorder. There is an additional information sheet about Borderline Personality Disorder on the last page of this document which might be helpful.

NATURE AND PURPOSE OF THE STUDY

The study aims to categorise the types of emotion, and the ways in which people diagnosed with Borderline Personality Disorder (BPD) control their emotions. This will allow us to understand how people with this disorder make sense of the world in which they live. This information will then be used to develop new treatments for the condition.

WHAT THE STUDY WILL INVOLVE

I have asked you to participate in this study because the assessment you have already completed indicates you experience Borderline Personality Disorder. If you decide to participate in this study, you will be asked to undertake a maximum of four interviews. The interviews will examine how you feel about yourself, and the ways in which you relate to other people. Finally, the interviews will examine the sorts of difficulties you have encountered in your life. The interviews will be unstructured, and the interviewing process will conclude when it appears that no new information is forthcoming. Each of the four interviews will take one hour to complete.

The interviews will be audio-recorded, and their contents will then be transcribed into a computer in order to analyze the data. After the interview material has been transcribed, the original audio recording will be destroyed. The transcript of the interview will be securely stored on floppy disk for a period of five years, but it will be done in a way that the information obtained will not identify you.

FOLLOW-UP PROCEDURES

At the completion of the interviews, further assessment or psychological assistance will be offered. If in your opinion you do not require further assistance, you will be free to terminate contact.

BENEFITS

The principal benefit to you in being involved in the study is likely to be that the experience of what it is like to be you will be understood by another person, perhaps for the first time in your life. This in itself will be of benefit. The second major benefit will be that this information will assist in the development of more specific treatment options for managing the disorder. Whilst there is no guarantee that you will benefit, the knowledge gained from your participation may help others in the future.

DISCOMFORT AND RISKS

There are minimal risks involved in this study. The possible side-effects arising through involvement in the study are associated with a possible increase in levels of distress because of the personal nature of the material discussed. This information will however be collected within the context of a professional relationship, so there will be ample opportunity to alleviate this distress should it arise.

CONFIDENTIALITY OF RECORDS.

The data collected will be stored in a non-identifiable manner. Subject data will be coded with a non-identifying subject code. The storage of data will be subject to the Australian Psychological Society's Code of Conduct with regard to the storage of Scientific data. This requires data to be securely housed for a period of five years prior to it's destruction.

Information derived from the study which is subject to the likelihood of publication will be reported in such a way that the identity of subjects contributing to the study will be protected, and their confidentiality assured. Raw data will not be accessible by personnel external to the study.

VOLUNTARY PARTICIPATION AND WITHDRAWAL FROM STUDY.

Participating in this study is entirely voluntary. If you decide not to participate in this study, your condition will be treated according to routine guidelines.

You are free to withdraw from the study at any time without any prejudice to present or future management in this hospital.

You may withdraw from this study at any time, for whatever reason. Such withdrawal will not in anyway influence decisions regarding other treatments you may require.

If you withdraw from the study at any point, all audiotaped information will be immediately destroyed.

INFORMATION ABOUT BORDERLINE PERSONALITY DISORDER (BPD).

Borderline Personality Disorder (BPD) is a disorder which is estimated to affect between 1% and 5% of the general population. BPD is generally regarded as difficult to treat although most people who suffer from the condition receive a mixture of drug and counselling treatments.

BPD is characterised by instability of emotion, difficult interpersonal relationships, and an impaired self-image. BPD is generally seen as a disorder where the person feels that their emotions are unstable, and as a result their behaviour can become dramatic and/or erratic. People diagnosed with BPD often describe that they feel that they have a sense of self which feels in some way deficient or lacking.

People with BPD often have interpersonal relationships which fluctuate between the extremes of intense closeness and idealisation of other people, to deprecation and devaluation of the other people. In addition, fears concerning threatened abandonment (either imagined or real) often characterise the disorder. In addition, people with BPD often report an identity disturbance (not knowing who they really are), and either have attempted, or at least have thought about committing suicide at some point in their lives.

The key characteristics of the disorder are:

1. **Affect (Emotion) Dysregulation**: People with BPD often experience significant difficulties in controlling their emotions - their emotions easily get "out of control." In particular, the emotions of anger, sadness, and shame appear to be the most frequent emotions where loss of control of the emotion occurs.

2. **Identity Diffusion**: People with BPD often report experiencing a "defective" sense of self. Specifically, people with this condition often state that they feel that they do not "know" who they are, that they feel that the core of their self is "hollow," or "empty." They often report a sense of not knowing what is "real," and what is "not real."

3. **Cognitive Impairments**. People with BPD often report difficulties in understanding the actions of others, and make inferences about other peoples' behaviour which they know logically is probably not correct. In addition, they often report that they seem to make the same mistakes over and over and seem not to be able to learn from the experience of making those mistakes. Finally, people with BPD often report the belief that they do not think in the ways that "everybody else does."

4. **Interpersonal Difficulties**. People with BPD report difficulty in maintaining relationships with others. This particularly true for intimate relationships.

> Many people with this condition report that they feel a sense of aloneness - that they have a profound difficulty in “making contact,” or forming a relationship with another person.

Overall, Borderline Personality Disorder is a serious condition which is neither well recognized nor well understood. It causes significant suffering not only to the person with the condition, but also to the friends and loved ones of that person. For these reasons, it is an important condition to study. It is also important to study the condition in order to develop more effective treatment. This is really an area where the requirements for effective and economical treatment are desperately required.

APPENDIX X
STUDY II CONSENT FORM

CONSENT FORM

TO BE USED IN CONJUNCTION WITH THE INFORMATION SHEET

AFFECTIVE AND SEMANTIC REPRESENTATIONS IN
BORDERLINE PERSONALITY DISORDER (BPD):
A DISCOURSE ANALYSIS.

STATEMENT OF UNDERSTANDING

Patients Name:..... Date of Birth:.....

1. I agree entirely voluntarily to take part in Affective And Semantic Representations In Borderline Personality Disorder (BPD): A Discourse Analysis.

2. I am over 18 years of age.

3. I have been given a full explanation of the purpose of this study, of the procedures involved and of what will be expected of me. The possible problems which might arise as a result of my participation in the study have been explained to me.

4. I agree to inform the supervising doctor of any unexpected or unusual symptoms that I may experience as soon as possible.

5. I understand that I am entirely free to withdraw from the study at any time and that this withdrawal will not in any way affect my future standard or conventional treatment or medical management.

6. I understand that I will not be referred to by name in any report concerning this study. In turn, I cannot restrict in any way the use of the results which arise from this study.

7. I have been given and have read a copy of this consent form and information sheet.

If I have any further questions regarding the study I may contact Chris Theunissen on phone number 431 2149.

Signature by Patient	Signature by Researcher
Signed:.....	Signed:.....
Date:.....	Date:.....

APPENDIX XI

SAMPLE ITEMS FROM THE AFFECT CATEGORY JUDGEMENT TASK

14 January 1999.

Dear ,

Many thanks for your agreement to participate in my Doctoral research as an expert judge. Your task is to review a list of words and judge which, if any, affect categories these words fit. Find enclosed a copy of the booklet with the list of words in it. The directions for the task are included within the booklet.

Again, many thanks for your agreement to participate. If you have any further queries, please do not hesitate to contact me on 9431 2149.

Yours sincerely,

CHRIS THEUNISSEN
SENIOR CLINICAL PSYCHOLOGIST

DIRECTIONS

On the following pages are listed approximately 4,800 English language, affect related words. Examine each word, and judge if the word fits any of the affect categories of :

1. DISTRESS
2. ANGER;
3. SADNESS;
4. ANXIETY;
5. SHAME;
6. JOY
7. NEUTRAL (Not incorporated into the other five categories.)

If you judge a word to be associated with a particular category of affect, please tick the relevant affect desciptor box associated with the word. Please note that it is possible for a word to be listed in one or more of the first six categories. That is, for example, a word might be associated with the categories of Anger and Sadness. Therefore, it is permissible to more than 1 box for each word for the categories of Distress, Anger, Sadness, Anxiety, Shame, and Joy.

IT IS NOT HOWEVER, PERMISSIBLE TO TICK A BOX ASSOCIATED WITH AN AFFECT CATEGORY, AND THE CATEGORY MARKED "NEUTRAL." IF A WORD IS JUDGED BY YOURSELF TO BE NEUTRAL, NO OTHER BOXES FOR THAT WORD CAN BE TICKED.

In order to assist you in the judgement task, definitions of each of the affect categories are provided.

DISTRESS: An affect state characterised by a state of anguish often associated with the the experience of psychological or emotional pain.

ANGER: An affect state characterised by a state of "extreme displeasure."

SADNESS: An affect state characterised by a state of sorrow or mournfullness.

ANXIETY: An affect state characterised by a state of fearfullness, or concern for the future.

SHAME: An affect state characterised by a state of humiliation excited by consciousness of one's own guilt or shortcomings, or having been made to feel ridiculous.

JOY: An affect state characterised by a state of happiness, elation, or pleasure.

NEUTRAL: Not relevant to the above-mentioned categories.

	ANGER	**SADNESS**	**ANXIETY**	**SHAME**	**JOY**	**NEUTRAL**
Forbade	☐	☐	☐	☐	☐	☐
Invading	☐	☐	☐	☐	☐	☐
Paralysing	☐	☐	☐	☐	☐	☐
Tricks	☐	☐	☐	☐	☐	☐
Furies	☐	☐	☐	☐	☐	☐
Possessions	☐	☐	☐	☐	☐	☐
Truths	☐	☐	☐	☐	☐	☐
Rebellion	☐	☐	☐	☐	☐	☐
Forgive	☐	☐	☐	☐	☐	☐
Mutiny	☐	☐	☐	☐	☐	☐
Quarrelsome	☐	☐	☐	☐	☐	☐
Heartbreak	☐	☐	☐	☐	☐	☐
Suppression	☐	☐	☐	☐	☐	☐
Unliked	☐	☐	☐	☐	☐	☐
Victories	☐	☐	☐	☐	☐	☐
Shatters	☐	☐	☐	☐	☐	☐
Hatred	☐	☐	☐	☐	☐	☐
Fulfilled	☐	☐	☐	☐	☐	☐
Forces	☐	☐	☐	☐	☐	☐
Satan	☐	☐	☐	☐	☐	☐
Placid	☐	☐	☐	☐	☐	☐

APPENDIX XII

WORD LISTS FOR EACH AFFECT CATEGORY REVIEWED BY SPEECH PATHOLOGIST JUDGES

WORD JUDGEMENT TASK

Dear _____,

Many thanks for agreeing to be involved in this aspect of my research. The task, as an expert judge, is to review the words listed below. You will note that the words are listed in relation to six (6) categories of affect. The categories are Shame, Sadness, Anger, Anxiety, Joy and Neutral. Your task is to review each word contained in the respectively named category. If you judge that the word **corresponds only to the affect category in which it is listed,** please place a tick in the box that corresponds to this statement. If on the other hand you judge that the word does not fit that category, or that it fits with other listed affect categories, please place a tick in the box that corresponds to this statement.

Many thanks for agreeing to complete this task.

SHAME

	Corresponds to This Word	Does Not Correspond To This Word
BELITTLES	☐	☐
GUILTY	☐	☐
DISGUSTING	☐	☐
WICKEDNESS	☐	☐
PUNISHES	☐	☐
GEEKS	☐	☐
GUILT	☐	☐
GEEK	☐	☐
JAIL	☐	☐
BELITTLING	☐	☐
DISGRACING	☐	☐
FORBIDDING	☐	☐
SHOPLIFT	☐	☐
YUCK	☐	☐
DISGRACES	☐	☐
GROTESQUELY	☐	☐
ENVIOUS	☐	☐
DISGUST	☐	☐
GROTESQUE	☐	☐
REMORSEFUL	☐	☐

SADNESS

	Corresponds to This Word	Does Not Correspond To This Word
LONELINESS	☐	☐
EVILS	☐	☐
WRETCHEDLY	☐	☐
DESPAIR	☐	☐
EVILLY	☐	☐
MOROSENESS	☐	☐
BLEEDING	☐	☐
LETDOWN	☐	☐
DEPRESSING	☐	☐
SUFFERS	☐	☐
DESPAIRING	☐	☐
DROWN	☐	☐
BURNED	☐	☐
DISAPPOINTMENTS	☐	☐
DISILLUSIONED	☐	☐
BEREAVES	☐	☐
DROWNED	☐	☐
FUNERAL	☐	☐
DISAPPOINTMENT	☐	☐
SUFFER	☐	☐

ANGER

	Corresponds to This Word	Does Not Correspond To This Word
ALIENATES	☐	☐
BASTARD	☐	☐
ATTACKS	☐	☐
BELLIGERENT	☐	☐
AGGRAVATING	☐	☐
BITTERNESS	☐	☐
IRRITATES	☐	☐
QUARREL	☐	☐
ATTACK	☐	☐
HOSTILE	☐	☐
BITCHY	☐	☐
BOSSING	☐	☐
FIGHTING	☐	☐
HATED	☐	☐
HATEFUL	☐	☐
TREACHEROUS	☐	☐
FRUSTRATION	☐	☐
TORMENTING	☐	☐
ANGERING	☐	☐
ANNOYING	☐	☐

ANXIETY

	Corresponds to This Word	Does Not Correspond To This Word
TERRIFIES	☐	☐
STRESS	☐	☐
UNCOMFORTABLE	☐	☐
TERRIFYING	☐	☐
PHOBIA	☐	☐
SCARED	☐	☐
SCARY	☐	☐
CHAOS	☐	☐
CHAOTIC	☐	☐
DEATHLY	☐	☐
ANXIETY	☐	☐
EERINESS	☐	☐
ARMAMENT	☐	☐
ANXIOUS	☐	☐
BEWILDERED	☐	☐
BEWILDERING	☐	☐
FEARS	☐	☐

JOY

	Corresponds to This Word	Does Not Correspond To This Word
FUN	☐	☐
DELIGHTED	☐	☐
HAPPINESS	☐	☐
INSPIRE	☐	☐
LOVE	☐	☐
DELIGHTS	☐	☐
JOY	☐	☐
KIND	☐	☐
PLAY	☐	☐
EXCELLENT	☐	☐
LOVED	☐	☐
ENJOYMENT	☐	☐
KISS	☐	☐
KISSED	☐	☐
CHARM	☐	☐
FRIEND	☐	☐
FRIENDS	☐	☐
JOYFUL	☐	☐
EXCELLENT	☐	☐
PLAYING	☐	☐

NEUTRAL

	Corresponds to This Word	Does Not Correspond To This Word
WORDS	☐	☐
JOYSTICK	☐	☐
NUMBERS	☐	☐
JOBS	☐	☐
ON	☐	☐
MESSAGES	☐	☐
COPE	☐	☐
MEDICINE	☐	☐
MONOPOLY	☐	☐
SPSS	☐	☐
BEIGE	☐	☐
WHEEL	☐	☐
FOCUS	☐	☐
COURSE	☐	☐
CRACKED	☐	☐
AGENT	☐	☐
ATTENDING	☐	☐
DIPPED	☐	☐
COMPARING	☐	☐
CONQUER	☐	☐

	Corresponds to This Word	Does Not Correspond To This Word
MALIGNANCIES	☐	☐
CUNNINGLY	☐	☐
BEAMING	☐	☐
DEVOUTLY	☐	☐
TRANSFORMATIONS	☐	☐
REPUTATIONS	☐	☐

APPENDIX XIII
NEWSPAPER ARTICLE IN SUNDAY TIMES
18 SEPTEMBER 1999

A PERTH clinical psychologist has launched a world-first effort to get to the bottom of the “black hole” of psychiatric disorders.

Using a series of simple tests, Fremantle Hospital’s Chris Theunissen is running a clinical study on borderline personality disorder – a puzzling condition which may affect 15,000Perth people, or at least one per cent of the population.

People with Borderline Personality disorder often complain of experiencing an inner emptiness, a “black hole” in the centre of themselves. And their overwhelming sense of aloneness can often lead to suicide.

The problem is that not only is the disorder difficult to diagnose correctly, medical experts are uncertain of the cause.

But Mr Theunissen has received ethics committee approval to put two main theories to the test.

“People who exist with this disorder suffer enormously,” said Mr Theunissen this week.

“To use an engineering metaphor, if you thought of these people as structures they would collapse and fall apart quite easily.”

“The reason this disorder is not spoken of commonly is that people don’t present themselves to us saying they have this condition.”

“They say they are depressed of they feel worried of they have harmed themselves. They talk about their lives falling apart.”

The first theory behind the condition is that it is naturally associated with ADHD or an anti-social personality disorder, which triggers impulsive, destructive behaviour under stress.

The other is that it is a mood regulation problem, where people lack the ability to control their moods.

Mr Theunissen will use a series of information processing tasks – such as the stop/signal test and the emotional Stroop test – in a bid to determine the cause of the disorder.

- Volunteers are needed for the study. People aged 19-59 who have been diagnosed with borderline personality disorder or display the symptoms listed can register on 9431 2149.

SYMPTOMS of borderline personality disorder:

- Frantic efforts to avoid real or imagined abandonment.
- A pattern of unstable and intense interpersonal relationships that are idealised and then devalued
- Persistent, unstable self-image or sense of self
- Impulsive, potentially damaging behaviour such as spending, sex, substance abuse, reckless driving, binge eating.
- Recurrent suicidal behaviour, gestures, threats, or self-mutilating behaviour
- Instability due to intense mood swings.
- Chronic feelings of emptiness
- Frequent displays of temper, constant anger, recurrent physical fights.
- Transient stress-related paranoid ideation, or severe dissociative symptoms.

APPENDIX XIV
TELEPHONE SCREENING INTERVIEW TO EXCLUDE POTENTIALLY CONFOUNDED CASES

Good Morning/Afternoon, could I speak with (Name of Person) please? Hello (Name of Person), my name is Chris Theunissen. I am the Clinical Psychologist attached to the Department of Infectious Diseases at Fremantle Hospital. I am phoning you as a result of the message you left on my answering machine indicating that you were interested in participating in the study that was described in the 'Sunday Times' article'. Can I just check that you are still willing to be involved?

If the participant declined: That's fine. Thank you for interest. (Call was then terminated).

If the participant agreed: Good. Thank you for your interest. Before we go any further, I need to ask you a few screening questions in order to ensure that there are no factors that might exclude you from the study. Are you agreeable to this?

(If the participant refused to answer these questions, exclude from the study)

Firstly,

1. Have you ever sustained a head injury? (If yes, clarify severity and exclude if severe).
2. Have you ever suffered from a neurological illness? (If yes, clarify severity and exclude if severe).
3. Have you ever suffered from a psychotic illness such as schizophrenia of bipolar disorder? (If yes, exclude).
4. Could you tell me what hand you use to write and throw?
5. Do you suffer from colour-blindness?

If the participant confirmed any points 1-5 above they were excluded from the study. This was communicated to the participant as follows:

‘Unfortunately, it appears that you suffer from one of the conditions that specifically excludes you from continuing in the study. Nevertheless, thank you for being prepared to be involved in the study’.

If the participant denied all of the above they were included in the study. This was communicated to the participant as follows:

‘It appears that you meet the inclusion criteria for the study. Could we make an appointment to see me here at the hospital to commence the screening and assessment process’? (Appointment then made to commence assessment).

APPENDIX XV
TELEPHONE CONTACT TO RECRUIT BPD CASES THROUGH THE DEPARTMENT OF INFECTIOUS DISEASES

Good Morning/Afternoon, could I speak with (Name of Person) please? Hello (Name of Person), my name is Chris Theunissen. I am the Clinical Psychologist attached to the Department of Infectious Diseases at Fremantle Hospital. I am phoning you because I am attempting to recruit participants into a research study that I am conducting, and one of the methods that I am using to do this is to contact people coming to the department for assessment for treatment for Hepatitis C.

The study that I am conducting is examining neuropsychological functions in people diagnosed with a condition known as Borderline Personality Disorder. I am phoning to ascertain whether you might be prepared to be involved in the study.

If the participant declined: That's fine. Thank you for interest. (Call terminated).

If the participant wanted further information with regard to the diagnostic criteria for BPD, these were then provided. If the participant declined, they were advised: That's fine. Thank you for interest. (Call was then terminated).

If the participant agreed to further involvement, see next paragraph.

If the participant agreed: Good. Thank you for your interest. Before we go any further, I need to ask you a few screening questions in order to ensure that there are no factors that might exclude you from the study. Are you agreeable to this?

(If the participant refused to answer these questions, exclude from the study)

Firstly,

1. Have you ever sustained a head injury? (If yes, clarify severity and exclude if severe).
2. Have you ever suffered from a neurological illness? (If yes, clarify severity and exclude if severe).
3. Have you ever suffered from a psychotic illness such as schizophrenia of bipolar disorder? (If yes, exclude).
4. Could you tell me what hand you use to write and throw?
5. Do you suffer from colour-blindness?

If the participant confirmed any points 1-5 above they were excluded from the study. This was communicated to the participant as follows:

'Unfortunately, it appears that you suffer from one of the conditions that specifically excludes you from continuing in the study. Nevertheless, thank you for being prepared to be involved in the study'.

If the participant denied all of the above they were included in the study. This was communicated to the participant as follows:

'It appears that you meet the inclusion criteria for the study. Could we make an appointment to see me here at the hospital to commence the screening and assessment process'? (Appointment then made to commence assessment).

APPENDIX XVI
TELEPHONE RECRUITMENT PROCEDURE FOR MEDICAL CONTROL PARTICIPANTS

Good Morning/Afternoon, could I speak with (Name of Person) please? Hello (Name of Person), my name is Chris Theunissen. I am the Clinical Psychologist attached to the Department of Infectious Diseases at Fremantle Hospital. I am phoning you as a result of the conversation that you had with (Name of Staff Person) who is a nurse at this clinic.

As I understand it, (Name of Staff Person) briefly described a study that I am conducting, and from discussing this with her, she indicated that you might be prepared to act as a participant in this research. Can I just check that you are still willing to be involved?

If the participant declined: That's fine. Thank you for interest. (Call was then terminated).

If the participant agreed: Good. Thank you for your interest. Could we make an appointment to see me here at the hospital to commence the screening and assessment process? (Appointment then made to commence assessment).

APPENDIX XVII
INFORMATION SHEET FOR STUDY FOUR

INFORMATION SHEET

TO BE USED IN CONJUNCTION WITH THE CONSENT FORM

AFFECTIVE DYSREGULATION AND BEHAVIOURAL DISINHIBITION IN BORDERLINE PERSONALITY DISORDER (BPD): A COMPARISON OF THE "EMOTIONAL STROOP" AND THE STOP STOP-SIGNAL TASK.

You are invited to participate in a clinical research study examining emotional regulation and impulse control in Borderline Personality Disorder (BPD). This study has been approved by the Metropolitan Health Services Board Ethics Committee.

If you decide to take part in the research study, it is important that you understand the purpose of the study and the procedures you will be asked to undergo. Please read the following pages which will provide you with information about the procedure involved, the potential benefits, discomforts, and precautions of the study.

For some of you, this will be the first time you have been told about Borderline Personality Disorder. There is an additional information sheet about Borderline Personality Disorder on the last page of this document which might be helpful.

NATURE AND PURPOSE OF THE STUDY

The aim of the study is to determine whether the main difficulty for patients diagnosed with BPD is related to problems in controlling their behaviour (impulsivity) or difficulties with controlling emotion. In other words, do patients with BPD act first and think later, or do they, first and foremost. have problems in managing emotion.

WHAT THE STUDY WILL INVOLVE

You have been asked to participate in this study because the assessment you have already completed indicates you meet criterion for a condition referred to as Borderline Personality Disorder. Alternatively, you have been recrutied into this study to act as a control subject. If you decide to participate in this study, you will be asked to complete a number of assessment instruments. Initially, these will involve **brief** tests to assess your current mood state, memory, planning abilities, IQ, and word recognition capacities. At the completion of this, you will be asked to complete the main two tasks of the study. These principal tasks involve two computerised tasks - the Emotional Stroop, and the Stop-Signal Task.

THE EMOTIONAL STROOP TASK

The Emotional Stroop Task asks you to simply name the colour of 60 words which will be displayed on a computer screen. One word is presented at a time. Each word is presented eight times in one of four colours (Red, Blue, Green, and Yellow) and is displayed at both a 240 mSec, or at 2000 mSecs time duration. Your task is simply to name the colour of the word. The task **does not** require the word itself to be named. For the duration of the task, a microphone headset will be attached to you, and will record the speed of your response to the task. **The aim of the task is however to accurately and as quickly as possible name the colour of the word.**

REMEMBER: YOU ARE NOT REQUIRED TO NAME THE WORD.

THE STOP-SIGNAL TASK

The Stop-Signal Task consists of the presentation of the letters "X" or "O" on a computer screen. Attached to the computer is a junction box which has two keys. One is marked "X" and the other "O". Your task is to depress the "X" key when an "X" is presented on the screen, and to depress the "O" key when the "O" is presented on the screen. At unspecified times in the presentation of the task, the computer will randomly issue a "beep" when either he "X" or the "O" is displayed. **When this occurs, you are required to stop (inhibit) your intention to press the "X" or the "O" key.**

At the completion of the task, any additional questions you might have, or information you might require will be provided.

BENEFITS

DISCOMFORT AND RISKS

There are no known risks involved in this study. Both of these methods have been employed in hundreds of studies with no reported adverse outcomes. The only likely discomfort will be associated with persevering with a task for a period of approximately one hour.

CONFIDENTIALITY OF RECORDS.

The data collected will be stored in a non-identifiable manner. The data will be coded with a non-identifying subject code. The storage of data will be subject to the Australian Psychological Society's Code of Conduct with regard to the storage of Scientific data. This requires data to be securely housed for a period of five years prior to it's destruction.

Information derived from the study which is subject to the likelihood of publication will be reported in such a way that the identity of subjects contributing to the study will be protected, and their confidentiality assured. Raw data will not be accessible by personnel external to the study.

VOLUNTARY PARTICIPATION AND WITHDRAWAL FROM STUDY.

- Participating in this study is entirely voluntary. If you decide not to participate in this study, any treatment you might subsequently undertake will be treated according to routine guidelines.
- You are free to withdraw from the study at any time without any prejudice to present or future management in this hospital.
- You may withdraw from this study at any time, for whatever reason. Such withdrawal will not in anyway influence decisions regarding other treatments you may require.
- If you withdraw from the study at any point, all data will be immediately destroyed.

INFORMATION ABOUT BORDERLINE PERSONALITY DISORDER (BPD).

Borderline Personality Disorder (BPD) is a disorder, which is estimated to affect between 1% and 5% of the general population. BPD is generally regarded as difficult to treat although most people who suffer from the condition receive a mixture of drug and counselling treatments.

BPD is characterised by instability of emotion, difficult interpersonal relationships, and an impaired self-image. BPD is generally seen as a disorder where the person feels that their emotions are unstable, and as a result their behaviour can become dramatic and/or erratic. People diagnosed with BPD often describe that they feel that they have a sense of self which feels in some way deficient or lacking.

People with BPD often have interpersonal relationships which fluctuate between the extremes of intense closeness and idealisation of other people, to deprecation and devaluation of the other people. In addition, fears concerning threatened abandonment (either imagined or real) often characterise the disorder. In addition, people with BPD often report an identity disturbance (not knowing who they really are), and either have attempted, or at least have thought about committing suicide at some point in their lives.

DIAGNOSIS

The current, primary diagnostic system, the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-IV) lists the following criteria as indicative of BPD:

1. Frantic efforts to avoid real or imagined abandonment.

2. A pattern of unstable and intense interpersonal relationships characterised by alternating between extremes of idealisation and devaluation.

3. Identity disturbance: Markedly and persistently unstable self-image or sense of self.

4. Impulsivity in at least two areas that are potentially self-damaging (eg: spending, sex, substance abuse, reckless driving, binge eating).

5. Recurrent suicidal behaviour, gestures, or threats, or self-mutilating behaviour.

6. Affective instability due to a marked reactivity of mood (eg: intense episodic dysphoria, irritibility, or anxiety usually lasting a few hours and only rarely more than a few days).

7. Chronic feelings of emptiness.

8. Inappropriate, intense anger or difficulty controlling anger (eg: frequent displays of temper, constant anger, recurrent physical fights).

9. Transient, stress-related paranoid ideation or severe dissociative symptoms.

CHARACTERISTICS OF BORDERLINE PERSONALITY DISORDER

In summary, the key characteristics of the disorder are:

1. **Affect (Emotion) Dysregulation**: People with BPD often experience significant difficulties in controlling their emotions - their emotions easily get "out of control." In particular, the emotions of anger, anxiety, sadness, and shame appear to be the most frequent emotions where loss of control of the emotion occurs.

2. **Identity Diffusion**: People with BPD often report experiencing a "defective" sense of self. Specifically, people with this condition often state that they feel that they do not "know" who they are, that they feel that the core of their self is "hollow," or "empty." They often report a sense of not knowing what is "real," and what is "not real."

3. **Cognitive Impairments**. People with BPD often report difficulties in understanding the actions of others, and make inferences about other peoples' behaviour which they know logically is probably not correct. In addition, they often report that they seem to make the same mistakes over and over and seem not to be able to learn from the experience of making those mistakes. Finally, people with BPD often report the belief that they do not think in the ways that "everybody else does."

4. **Interpersonal Difficulties**. People with BPD report difficulty in maintaining relationships with others. This is particularly true for intimate relationships. Many people with this condition report that they feel a sense of aloneness - that they have a profound difficulty in "making contact," or forming a relationship with another person.

Overall, Borderline Personality Disorder is a serious condition which is neither well recognised nor well understood. It causes significant suffering not only to the person with the condition, but also to the friends and loved ones of that person. For these reasons, it is an important condition to study. It is also important to study the condition in order to develop more effective treatment.

This is really an area where the requirements for effective and economical treatment are desperately required.

APPENDIX XVIII
CONSENT FORM FOR STUDY FOUR

AFFECTIVE DYSREGULATION AND BEHAVIOURAL DISINHIBITION IN BORDERLINE PERSONALITY DISORDER (BPD): A COMPARISON OF THE "EMOTIONAL STROOP" AND THE STOP STOP-SIGNAL TASK.

PATIENT CONSENT FORM AND STATEMENT OF UNDERSTANDING

Patients Name:..... Date of Birth:.....

1. I am volunteering to take part in the study "Affective Dysregulation and Behavioural Disinhibition in Borderline Personality Disorder (BPD): A Comparison of the "Emotional Stroop" and the Stop-Signal Task.

2. I am over 18 years of age.

3. The study will be conducted in a manner conforming to the principles established by the National Health and Medical Research Council

4. I have been given a full explanation of the purpose of this study, of the procedures involved and of what will be expected of me. The possible problems which might arise as a result of my participation in the study have also been explained to me. I also agree to inform the supervising doctor of any unexpected or unusual symptoms that I may experience as soon as possible.

5. I understand that I am entirely free to withdraw from the study at any time and that this withdrawal will not in any way affect my future standard or conventional treatment or medical management.

6. I understand that I will not be referred to by name in any report concerning this study. In turn, I cannot restrict in any way the use of the results which arise from this study.

7. I have been given and have read a copy of this consent form and information sheet.

8. If I have any further questions regarding the study I may contact Chris Theunissen on phone number (08) 9431 2149.

Signature by Patient Signed:.....
Date:.....
Signature by Researcher Signed:.....
Date.....

Signature by Witness Signed:.....
Date.....

APPENDIX XIX
SCREENING INSTRUMENT FOR STUDY FOUR

STUDY IV INITIAL SCREENING INTERVIEW

NEUROLOGICAL HISTORY

Throughout the course of your life, have you:

1. Ever experienced some form of neurological illness, such as:

Meningitis? *Yes/No*

Encephalitis? **Yes/No**

Epilepsy? **Yes/No**

Have you experienced any other neurological illness?

_____

2. Ever suffered from 'seizures'? **Yes/No**

If so, what is the nature of these?

_____

3. Ever experienced some form of head trauma? **Yes/No**

Head injury? **Yes/No**

Loss of consciousness? **Yes/No**

(In excess of five minutes at any time in life)

4. Ever consulted a neurologist? **Yes/No**

If so, what were the reasons the consultation?

_____

Did the neurologist prescribe medication? **Yes/No**

Refer you to a neurosurgeon? **Yes/No**

4. Have you undergone neurosurgery? **Yes/No**

5. Please describe the nature of this procedure?

_____

6. Which hand do you write with?

Right/Left/Ambidextrous

7. Which hand do you throw with?

Right/Left/Ambidextrous

8. Which hand do you kick a ball with?

Right/Left/Ambidextrous

9. Is English your 'first' language? **Yes/No**

PSYCHIATRIC HISTORY

Throughout the course of your life, have you ever:

1. Received Electroconvulsive Therapy (ECT)? **Yes/No**

   Within the past 90 days? **Yes/No**

2. Experienced a history of psychotic illness? **Yes/No**

   (Psychotic illness was defined as Schizophrenia,

   Bipolar Affective Disorder, Schizoaffective

   Disorder, Organic Psychosis, or

   Psychosis Not Otherwise Specified.

3. Have you been 'using' street or illicit drugs in the

   past 90 days? **Yes/No**

   (Defined as Marijuana, Heroin, Ecstasy, Amphetamine,

   LSD or other known substances)

COLOUR SCREENING

Show Participant the Four Colour Swatches.

Could you tell me the colour of these swatches?

Red Swatch Administered

Correct/Incorrect

Green Swatch Administered

Correct/Incorrect

Blue Swatch Administered

Correct/Incorrect

Yellow Swatch Administered

Correct/Incorrect

APPENDIX XX

SUMMARY OF THE FINDINGS OF THE STUDY

Dear (Participant's Name),

Re: AFFECTIVE DYSREGULATION AND BEHAVIOURAL DISINHIBITION IN BORDERLINE PERSONALITY DISORDER (BPD): A COMPARISON OF THE "EMOTIONAL STROOP" AND THE STOP STOP-SIGNAL TASK.

You may recall being a participant in this research project. Thankyou for your involvement, and find enclosed a brief document that summarises the findings of the study.

Again thank you for your contribution and if you have any queries regarding the study, please do not hesitate to contact me.

Kind regards.

CHRIS THEUNISSEN
SPECIALIST CLINICAL PSYCHOLOGIST
DEPARTMENT OF INFECTIOUS DISEASES

SUMMARY OF THE FINDINGS OF THE STUDY: AFFECTIVE DYSREGULATION AND BEHAVIOURAL DISINHIBITION IN BORDERLINE PERSONALITY DISORDER (BPD): A COMPARISON OF THE "EMOTIONAL STROOP" AND THE STOP STOP-SIGNAL TASK.

Theoretical Model

The study was based on a theory suggesting that people with BPD experienced deficits in executive function. The model proposed a multidimensional developmental neuropsychobiological model of BPD. Table 1 attached outlines the theoretical model underpinning the study. The model suggested that BPD involves a number of impaired executive functions which include working memory, behavioural inhibition, affect attentional bias, and complex-problem solving ability. These impaired executive functions represent the cognitive manifestations of underlying deficits in orbitofrontal-subcortical pathways of the Central Nervous System (CNS). These deficits were thought occur as a result of the influence of a number of independent risk factors which included genetic, psychobiological, early loss and/or separation, parent and/or family psychopathology, impaired parental bonding and/or attachment pathology, and trauma usually in the form of child abuse and/or neglect. The interaction of these factors was hypothesised to contribute to the failure of an 'experience-dependent' maturation of orbitofrontal-subcortical networks in the brain. This was thought to result in impaired executive disorders in BPD.

The proposed model argued that the executive functions of working memory, behavioural inhibition, affective-attentional bias (affect regulation), and problem solving share interdependent relationships with each other, and act in a 'co-operative' or 'seamless' fashion in order to effectively regulate the transactions between the person and the environment. Impairment in one domain of executive functioning has the potential to contribute to impairment in other domains of executive functioning. For example, the inability to effectively regulate affective states is likely to result in episodes of affect dysregulation that can in turn provoke behavioural dysregulation which can in turn provide the basis for 'impulsive' acting out. Similarly, failure to successfully execute a problem solving sequence can lead to affective dysregulation that in turn can lead to 'impulsive' behavioural enactments as a means of restabilising a dysregulated affective-attentional system.

Executive Function

This study examined the hypothesis that Borderline Personality Disorder (BPD) is characterised by a series of neuropsychological deficits known as 'Executive Function'. Executive Functions refer to a variety of different cognitive functions that are essential for functioning in a competent and coherent way.

In this study, executive functions were defined as:

Working Memory. This is a form of brief memory lasting anywhere from two to 20 seconds duration which enables the person to 'hold in mind' material in order

to solve some other task. Working memory is seen as a basic executive function that subserves the execution of a variety of other executive tasks.

Response Inhibition. Response inhibition refers to the capacity to stop an act from commencing, or alternately, to stop an act from continuing once it has commenced. Response inhibition relies to some extent on working memory for successful execution. Response inhibition is an important executive function because it serves the function of assisting in the control of behaviour and further assists in enabling alterations in behavioural strategies. This executive function was assessed through the use of the Stop-Signal Task.

Attentional Bias. Attentional Bias refers to a form of perceptual bias that predisposes people to attend to particular forms of stimuli. This executive function also relies to some extent on the operation of working memory, and it is important in that it permits us to attend to particular stimuli that might be significant for operating successfully in specific environments. A particular form of attentional bias was examined in this study – the study focussed on examining whether people with BPD are attentionally biased toward emotionally laden material. This executive function was assessed through the use of the Stroop Task.

Problem-Solving. Problem-solving refers to a variety of different tasks which examine the capacity of people to address novelty, test hypothesis, and 'figure out' particular problems. Clearly, problem-solving is a complex set of tasks, and a series of tasks were employed in this study. A number of different forms of problem-solving were included which involved copying patterns, maze-learning, and use of planning strategies to solve problems.

Hypotheses

The study proposed that participants diagnosed with BPD would perform more poorly than control participants on measures of executive functioning. Two control groups were included in the design of the study: a depressed control group and a medical control group. The depressed control group was included in order to account for the co-morbid depressive phenomenon that typically occurs with people with BPD. Using a depressed control group, it was possible to control for the effects of mood disorder – mood disorder is known to affect performance on the tests used in the study. The medical control group was employed because they represented an essentially 'normal' group of people and therefore acted as a comparison with so-called 'normal' people.

Results

The study found no evidence of impairment on the measures of working memory, response inhibition, and problem-solving for people with BPD. In contrast, the study demonstrated that people with BPD were more attentionally biased than controls.
Therefore, the study provided limited support for original hypotheses. The findings suggest that people with BPD do not experience difficulties with most executive functions, but have difficulties with material that is more emotionally

laden, or involves responding to what might be called 'response-conflict' tasks. It also appears that people with BPD are more hypervigilant that others. Put simply, it seems that the Stroop findings might have detected difficulties that occur when people with BPD are placed in arousing or situations requiring them to execute a non-automatic response.

It also appears that this difficulty predates the establishment of mature, adult cognition. This is probably the case because all of the other tasks assessed in the study are known to consolidate in the late adolescence period. Because these tasks returned normal results, it suggests that the functions that underpin Stroop performance developed at a point in time *prior* to the establishment of other measured executive functions. The reasons for this are unclear, but the possibilities include a genetic predisposition, or the development of 'hard-wired' automatic modes of responding to novel stimuli that *serve some form of adaptive or protective response.*

Implications of the Findings

There are a number of implications of the findings for both further research and also for assisting people with BPD.

First, greater attention needs to be directed toward understanding the arousal, priming and hypervigilance mechanisms involved in BPD. These factors suggest that there are psychobiological mechanisms associated with BPD, and a better understanding of these is likely to lead to the development of new treatments for the disorder.

Second, the findings of the study emphasise the importance of emotional variables in BPD. This further suggests that new treatments should emphasise the management of both arousal and the affective features of the disorder. Assisting people with BPD to more capably regulate emotion is essential.

Finally, the results also point to the *developmental* nature of the disorder. The Stroop findings suggest that the attentional bias inherent in BPD probably develops in the preadolescent phase of development and may well be an adaptive psychobiological response to abusive or chaotic environments in which the borderline-to-be child often lives. Therefore, the results also point to an important role for prevention of BPD through a multi-tiered approach consisting of good post-natal aftercare, parent education regarding affective development in children, public health programmes to build resilient families, support for mothers, and last but not least, comprehensive child protection services.

It is still premature to suggest that BPD can be completely prevented, but the development of services and structures in the community that can support adequate parenting and prevent adverse treatment of children will greatly assist in reducing the risk factors known to contribute to the development of the disorder.

Figure 1: Multidimensional Developmental Neuropsychological Model of Impaired Executive Function in BPD

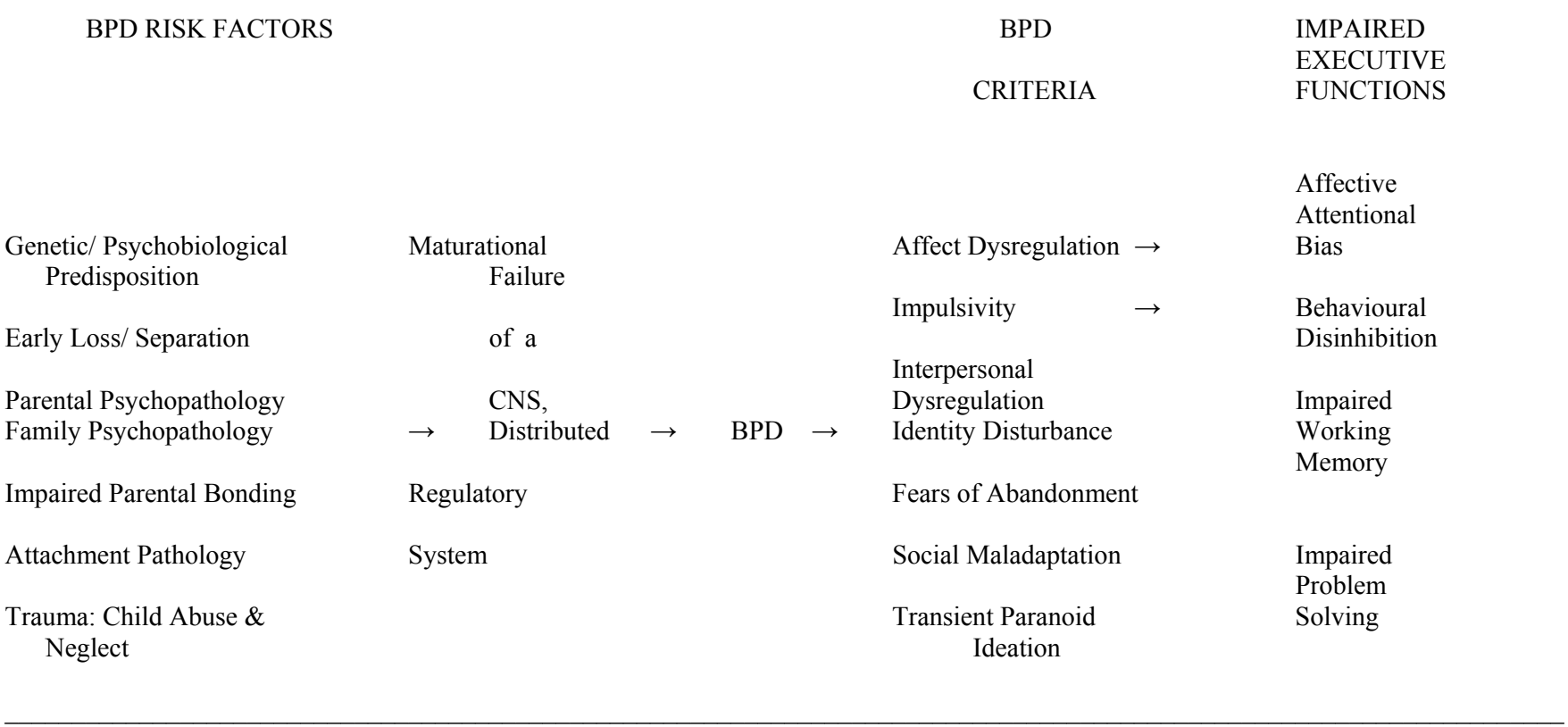

Figure 2: Modified Multidimensional Developmental Neuropsychological Model in BPD