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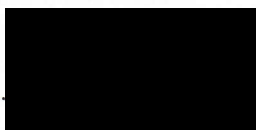
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**The Developmental Precursors of Borderline
Personality Disorder Symptoms at 11 years in a
British Cohort**

By

Catherine Winsper

Thesis submitted in fulfilment of the requirements for the degree of
Doctor of Philosophy in Psychology

University of Warwick, Department of Psychology

January 2012

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Acknowledgments

Thanks to Professor Dieter Wolke for his supervision and tireless input over the three years.

I would also like to thank my wonderful office mates, Mal and J who were willing to discuss ideas and concepts, and have been very supportive at the more stressful times. A special thanks to James Hall for his help and advice with the most challenging statistical concepts, and endless discussions regarding *MPlus*! Also, thanks to Linda Muthén for her kind support and prompt advice regarding the more technical aspects of *Mplus*.

An extra special thanks to my fiancé, Steve Martin (“snizzle”) who has been very supportive of my return to full time study and my regular absences over the last six years.

I would also like to thank the families taking part in the ALSPAC study, and midwives who recruited them. Additionally, a big thanks to all those involved with the ALSPAC cohort, including my “data buddy” Laura Miller.

I was funded by a scholarship from the University of Warwick.

Declaration

Chapters one, two, three, four, five and nine have been carried out entirely by the author. Contributions to the three research papers are as follows:

Study one (Chapter 6. In Press, Psychological Medicine):

- Idea: Winsper & Wolke following review of literature
- First draft: Winsper
- Statistical Analysis: Winsper
- Subsequent revisions: Winsper , Wolke & Zanarini*

Study two (Chapter 7. Published in the Journal of Child Psychology and Psychiatry, 2012, Early View - doi: 10.1111/j.1469-7610. 2012.02542. x):

- Idea: Wolke
- First draft: Wolke
- Initial analysis: Schreier
- Subsequent analysis: Winsper
- Subsequent revisions: Winsper & Wolke

Study three (Chapter 8):

- Idea: Winsper & Wolke following review of literature
- First draft: Winsper
- Analysis: Winsper – statistical advice from Hall
- Subsequent revisions: Winsper & Wolke

An ancillary paper has been published: Winsper, C., Lereya, T., Zanarini, M., & Wolke, D. (2012). Involvement in bullying and suicide related behaviour at 11 years: A prospective birth cohort study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51, 3, 271-282.

**Zanarini developed the original Childhood interview for DSM-IV BPD, which was then adapted for the UK.*

Catherine Winsper

Abstract

Borderline Personality Disorder (BPD) is a severe and chronic mental health disorder, affecting many areas of functioning including: affect regulation, impulse control, interpersonal relationships and self-image. Causal factors are only partly known due to a scarcity of prospective, longitudinal studies which enable one to delineate the time ordering of antecedents, and allow for tentative causal inferences. This thesis explored the developmental precursors of BPD symptoms at 11 years, using a British cohort sample, with assessments pertaining to the study child from pregnancy to 11 years of age.

Three studies were conducted. Firstly, the predictive relationship between exposure to maladaptive parenting and subsequent BPD symptoms was explored within a child population, using a clinically relevant assessment of BPD symptoms. This association has been previously shown in a range of retrospective studies. Secondly, the role of peer victimisation in the development of BPD was considered. This study was designed to extend current aetiological models, which focus on parental rather than peer relationships. It was based on the recognition of a strong interpersonal core in the BPD symptom constellation, and the role of trauma experiences in the development of BPD. Finally, the third study was designed to consider how these two experiential factors (maladaptive parenting and peer victimisation) might magnify a predisposition towards dysregulation, eventually culminating in BPD symptoms.

Data was obtained from the Avon Longitudinal Study of Parents and Children (ALSPAC), which studied 6,050 children (43.1% of the total sample population), using questionnaire and interview assessments.

Results revealed that, firstly, family adversity during pregnancy and suboptimal parenting, during early to middle childhood was predictive of BPD symptoms at 11 years. Secondly, peer victimisation during early to late childhood was predictive of BPD symptoms at 11 years. There was an especially strong dose response effect for severe, combined or chronic victimisation. Finally, those evincing stable dysregulated trait behaviour from 4 to 8 years were more likely to develop BPD symptoms, and this effect was especially strong for high levels of dysregulation. Consistent with the biosocial developmental model of BPD, the association was fully mediated by psychosocial risk factors (peer victimisation). Those with high levels of dysregulation were more likely to be victimised and, in turn, develop BPD symptoms. Further, the indirect associations were significantly stronger for BPD, compared to psychotic or depression outcomes.

The strengths and weaknesses, along with practical and theoretical implications, and future directions are discussed in the final chapter.

Chapter One: Development of the Borderline Personality Disorder (BPD) Construct: History, Diagnosis and Description

Overview: The following chapter will lay foundations for an understanding of the Borderline Personality Disorder (BPD) construct, by tracing the development of this concept as it has evolved through history to the present day. Further, there will be a discussion of current assessment and diagnostic techniques, and a consideration of the features, prevalence and life course of this disorder. The current diagnostic system will be evaluated with suggestions for future directions. Because many of the issues highlighted are relevant to personality disorders (PDs) generally, the discussion will be broadened in places, yet remain relevant to BPD.

1.1 The History of the BPD Construct

In the early 1900s, psychoanalysts identified a subset of patients inconsistent with any predefined diagnostic category, but situated on the border between neurosis and psychosis. These apparently depressed or anxious patients appeared to exhibit temporary psychotic phenomena under stressful conditions. In reflection of this observation, the nomenclature “borderline” was chosen (Rothschild, Cleland, Haslam, & Zimmerman, 2003).

Since this delineation, the grouping known as “borderline” has been subject to considerable controversy and alternative descriptions. A brief synopsis of the history of this diagnostic entity concludes with the current conceptualisation of

BPD, as it appears in the Diagnostic and Statistical Manual (DSM - IV- TR, 2000, APA).

The defining of borderline pathology in the early 1900s was indicative of a change in the psychoanalytic tradition. Originating from the study of the neurotic symptoms of hysteria, psychoanalysis began to incorporate the problem of character pathology (Gabbard, 2001). Initiated by Freud's seminal paper detailing anal eroticism and character (1908/1959), early psychoanalysts made the distinction between *ego dystonic* factors: likely to cause distress to the patient; and *ego syntonic* factors: likely to cause distress to others. The distinction remains today (Tyrer & Ferguson, 2000), though clinicians suggest this dichotomisation underplays the distress experienced by PD patients, who are classed as evincing ego syntonic factors (Gabbard, 2001).

The first systematic attempt to describe the borderline patient was undertaken by the psychoanalyst Stern in 1938. In an influential article, Stern used clinical observations to compile a list of ten symptoms characteristic of the "borderline group" (see **Box 1.1**). Considering the age of this article, the similarities between Stern's delineated symptoms (albeit in a different "psychoanalytic language") and the current DSM-IV-TR symptoms support the validity and longevity of the borderline diagnosis. Indeed, six of Stern's criteria bear close resemblance to the current symptom criteria of BPD in the DSM-IV-TR (see **Box 1.2**).

Firstly, *inordinate hypersensitivity* described patients who were consistently

“insulted and injured by trifling remarks” and occasionally developed “mildly paranoid ideas” (Stern, 1938. p.471). This symptom is reminiscent of *emotional instability* and *stress related paranoid ideation* as described in the *DSM*.

Secondly, *negative therapeutic reactions* describe the patient as experiencing: “readily aroused anger, discouragement and anxiousness as responses to any interpretation involving injury to self-esteem” (p. 473). Again, counterparts may be found in the *DSM* criteria in the form of *inappropriate anger* and *emotional instability*.

Box 1.1 Clinical Symptoms of the Borderline Group as devised by Stern (1938)

1. Narcissism
2. Psychic bleeding
3. Inordinate hypersensitivity
4. Psychic and body rigidity – “The rigid personality”
5. Negative therapeutic reactions
6. What looks like constitutionally rooted feelings of inferiority, deeply imbedded in the personality of the patient
7. Masochism
8. What can be described as a state of deep organic insecurity or anxiety
9. The use of projection mechanisms
10. Difficulties in reality testing, particularly in personal relationships

Thirdly, *masochism*, describes the features of: “self-pity and self-commiseration” and the tendency of borderline patients to: “hurt themselves in their business, professional, social, in fact all affective relationships” (p. 475). Such behaviour is referred to in the *DSM* under the rubric of *impulsive acts*, which are potentially

self-damaging, suicidal and self-mutilating. Fourthly, Stern's "*somatic*" *insecurity or anxiety* refers to the lack of "self-assurance and self-confidence" observed in borderline patients (p. 476). This feature may be likened to the *DSM* symptom of *identity disturbance*. Fifthly, Stern described the term *projection* as an attempt to: "explain his difficulties on the basis of a hostile attitude of the environment towards him and the inordinate difficulties that his conceptions of reality present" (p.478). Again this symptom parallels the *DSM* criterion of *paranoid ideation*. Finally, *difficulties in reality thinking* concerned mostly problems within the context of the therapeutic relationship, with the distorted attitudes of the patient swinging from idealisation to devaluation. This feature may be recognised in the *DSM* criterion of *unstable, intense interpersonal relationships*.

Subsequent to Stern's seminal work, four broad approaches to the borderline construct may be identified, developing from the 1950s onwards. Firstly, and congruent with the current BPD construct, Grinker (1979) and Gunderson (1979) viewed borderline pathology as a distinct, clinical disorder, distinguishable by certain behavioural characteristics. The conceptions of Grinker and Gunderson were largely compatible as illustrated in Table 1.1.

In the second approach, borderline pathology was categorised as a milder, genetically related form of schizophrenia, called *borderline or pseudo* schizophrenia (Hoch & Polatin, 1949; Hoch & Catell, 1959; Kety, Rosenthal, Wender, & Schulsinger, 1968).

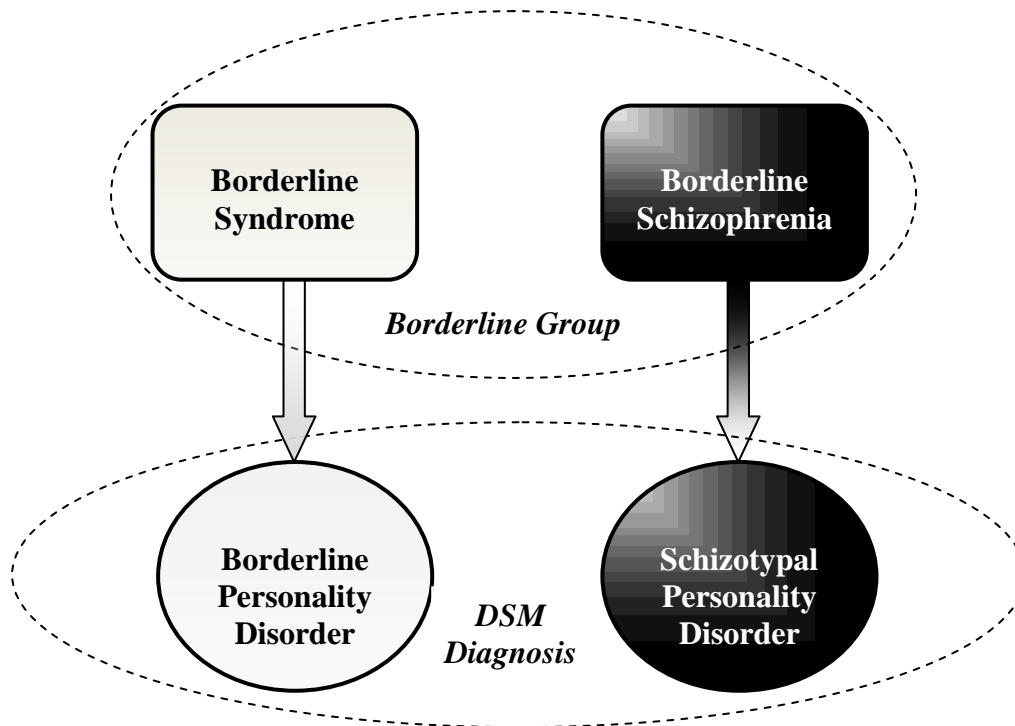
Table 1.1 *Borderline Pathology as Defined by Grinker and Gunderson*

Grinker (1979)	Gunderson (1979)
Anger as main or only affect	Intense affect, usually hostile/depressed
Analytic, dependent relationships	Vacillating relationships, often dependent
Lack of self-identity	Brief psychotic experiences
Depression, characterised by loneliness	History of impulsive behaviour
	Loose thinking in unstructured situations

Symptoms delineated were similar to those observed in schizophrenia, including: subtle disturbances in thought and associations, micro-psychosis, anhedonia (loss of pleasure) and problems with sexual relationships. Identified patients, however, did not manifest the full-blown hallucinations and delusions characteristic of traditional schizophrenic patients (Hoch & Pollatin, 1949). Nevertheless, Kety and colleagues (1976) presented genetic evidence for a link between borderline pathology and schizophrenia; finding a significant concentration of schizophrenic spectrum disorders in the biological relatives of borderline schizophrenics. This finding has been corroborated subsequently, following the discovery of a genetic link between schizotypal personality disorder (the current equivalent of pseudo schizophrenia, see **Figure 1.1**) and schizophrenia (Siever & Davis, 1991).

The competing ideas of Gunderson and Grinker versus Hoch, Pollatin and Kety initiated a splitting of the broad “borderline” grouping into two distinct personality disorders: BPD and Schizotypal Personality Disorder (SPD). Spitzer and Endicott (1979), using observations from both paradigms, compiled a list of eight criteria for both BPD and SPD, for inclusion in the DSM-III (1980, APA) (see **Figure 1.1**). These two categories (with small modifications) remain in the DSM-IV-TR today.

Figure 1.1 The Advent of BPD and SPD Diagnoses in the DSM



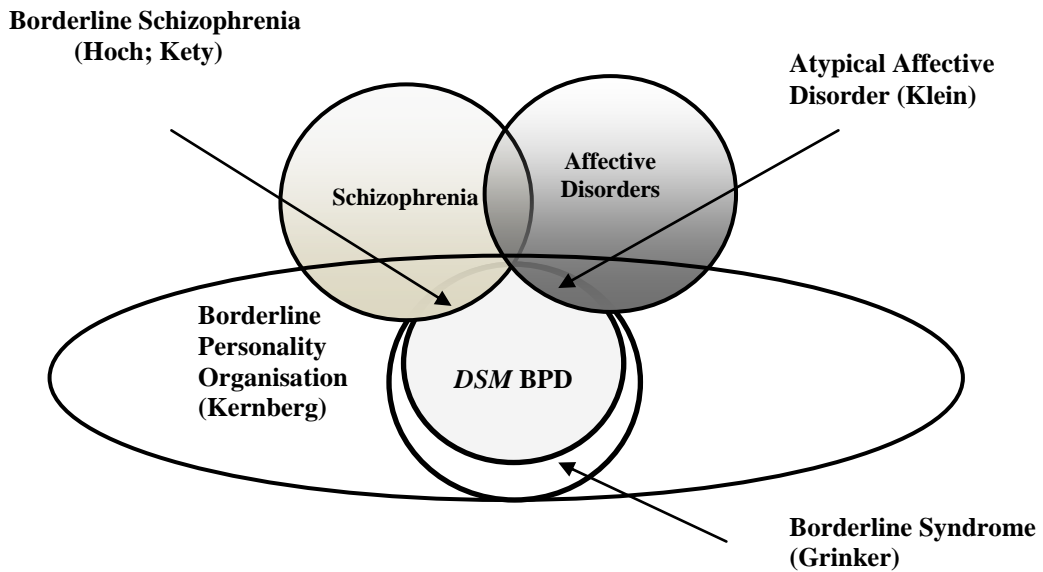
The third approach to the borderline construct marked a return to the psychoanalytic roots of the nomenclature. Kernberg's (1975) delineation of *Borderline Personality Organisation (BPO)* focused on the ego structural underpinnings of the borderline personality, rather than a list of characteristic symptoms. Kernberg identified three personality types: neurotic, borderline and psychotic, the borderline type being intermediate between the neurotic and psychotic. The BPO construct encompassed a much broader group of patients, incorporating a number of PDs, defined in terms of identity diffusion, primary defences, and reality testing. While researchers, including Gunderson and Grinker, classed "borderline" as a distinct entity with defined clinical symptoms,

Kernberg considered the common pathological ego structure of an eclectic group of borderline individuals.

In the fourth approach to the borderline construct, Klein (1977) suggested that borderline pathology was a type of atypical affective disorder, in which characterological symptoms were secondary to affective vulnerabilities. This viewpoint is still influential today, and affective features (e.g. suicidal attempts, feelings of emptiness) are included in the BPD criteria of the DSM-IV-TR (APA, 2000). Indeed, current researchers have suggested that the borderline category is better conceptualised as a state disorder, due to the overlap of symptom criteria and high levels of comorbid depression (Coid, 2003; Tyrer & Ferguson, 2000) [see section 1.4.2].

To a varying extent, all of the approaches outlined above are reflected in the DSM-IV-TR diagnostic criteria for BPD (see **Figure 1.2**). Since first appearing in the DSM-III (APA, 1980), BPD criteria have been altered somewhat in an attempt to improve the validity of the construct (Livesley, 2001). In the DSM-III-R, for example, “*frantic efforts to avoid abandonment*” was added to the symptom list (Livesley, 2001) [For a full description of criteria changes to BPD in successive *DSM* editions, the interested reader is directed towards Skodol et al., 2002]. Despite these efforts, problems with the categorisation of BPD remain, and will be outlined subsequently.

Figure 1.2 *Diagrammatical Representation of the Relationship between the current BPD construct and its Historical Forebearers (Gunderson, 1984)*



Beginning with the DSM-III in 1980 (APA, 1980), the *DSM* system has incorporated a multi-axial design, allowing for the diagnosis of multiple distinguishable disorders situated on different axes (Tyrer et al., 2007). Utilising this feature, the PDs were placed on a separate axis (Axis II) from Axis I mental disorders, thereby distinguishing mental illness from personality (Livesley, Schroeder, Jackson, & Jang, 1994; Tyrer et al., 2007). This segregation was not theoretically grounded so much as practical, for it was feared that PDs would be overlooked if in direct competition with more florid Axis I disorders (Tyrer et al., 2007). The rationale behind this decision has been questioned, however, as clinical and genetic evidence challenges the dichotomisation of mental illness and PD (Kendell, 2002; Lenzenweger, Lane, Loranger, & Kessler, 2007). The theoretical and clinical implications of this division will be discussed subsequently.

1.2 Diagnosis and Description

BPD is a severe and chronic psychiatric disorder, being one of ten PDs listed in the DSM-IV-TR (APA, 2000). A PD, as described by the DSM-IV-TR, is: “an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individuals’ culture” (p. 685). The *DSM* warns against the diagnosis of PD in individuals under the age of 18, citing that this diagnosis is “relatively unusual” in youth, and should only be confirmed if PD features are present for at least one year (p. 687). Historically, this opinion reflects a consensus between researchers that personality is dynamic in youth, rendering the diagnosis of PD in childhood or adolescence untenable (Meekings & O’ Brien, 2004). This view has changed in recent years, however, and the importance of recognising borderline features in youth has been acknowledged (Chanen, McCutcheon, Jovev, Jackson, & McGorry, 2007; Crick, Murray-Close, & Woods, 2005; De Clercq, De Fruyt, Van Leeuwen, & Mervielde, 2006) [For a full discussion see chapter three].

The DSM-IV-TR divides the ten PDs into three main clusters: *A or odd/eccentric*; *B or dramatic/ impulsive*; and *C or anxious/withdrawn*. This format was utilised to facilitate identification and memory, rather than reflect theoretical or research findings (Trull & Durrett, 2005). BPD is situated within dramatic cluster B. Reflecting the categorical approach of the *DSM*, BPD is diagnosed when at least five of nine behavioural criteria are identified (See **Box 1.2**). These criteria reflect disturbance in four key areas: poorly regulated

emotions; impulsivity; impaired perception and reasoning; and markedly disturbed relationships (Friedel, 2004).

Box 1.2 DSM-IV-TR Diagnostic criteria for Borderline Personality Disorder

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 behavior covered in Criterion 5
- (2) A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization 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 behavior covered in Criterion 5
- (5) Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior.
- (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

*Directly taken from manual; APA, 2000

An equivalent disorder to BPD is found in the *International Classification of Disease (ICD)*: The diagnostic manual of the World Health Organisation (WHO). Originally, the ICD-9 and the DSM-II shared the same nomenclature of “*explosive personality disorder*” for borderline pathology. Subsequently, the DSM-III adopted the “*borderline*” PD label, while the ICD-10 (WHO, 1992) adopted the “*emotionally unstable*” PD label.

Box 1.3 ICD-10 Diagnostic criteria for Emotionally Unstable Personality Disorder

Personality disorder characterised by a definite tendency to act impulsively and without consideration of the consequences; the mood is unpredictable and capricious. There is a liability to outbursts of emotion and an incapacity to control the behavioural explosions. There is a tendency to quarrelsome behaviour and to conflicts with others, especially when impulsive acts are thwarted or censored. Two types may be distinguished: the impulsive type, characterised predominantly by emotional instability and lack of impulse control, and the borderline type, characterised in addition by disturbances in self-image, aims and internal preferences, by chronic feelings of emptiness, by intense and unstable interpersonal relationships, and by a tendency to self-destructive behaviour, including suicide behaviour and attempts.

Personality (disorder):

Aggressive
Impulsive
Borderline

Excludes: dissocial personality disorder.

*Directly taken from manual; WHO, 1992

In a departure from the *DSM* model, the ICD-10 subdivides emotionally unstable personality disorder into impulsive and borderline types (see description in **Box 1.3**).

This subdivision was initiated following mediation by the *DSM* Task Force, to create a parallel *borderline* disorder in the *ICD* and *DSM*; increasing congruency between the two manuals (Widiger, 2001). A number of disparities remain, however, largely reflecting format differences between the *DSM* and *ICD*. For example, the *ICD* does not place PDs on a separate axis to mental disorders, and has separate manuals for clinical and research purposes (Saratorius et al., 1993). Further, despite efforts, there is no *directly* comparable borderline category in the *DSM* and *ICD*, leading to claims that the attempt to facilitate a convergence between manuals has been unsuccessful (Coid, 2003).

The comparative diagnosis of borderline pathology in the *DSM* and *ICD* has been explored. Perez *et al.* (2005) found that the DSM-IV yielded more positive diagnoses and comorbidity than the ICD-10. Further, Sara, Raven, and Mann (1996) found that only 29% of reviewed patients received the same primary diagnosis in each system. This lack of concordance likely results from different criteria formulations and arbitrary thresholds for diagnosis (Ottonsson, Ekselius, Grann, & Kullgren, 2002). Indeed, the observed diagnostic discrepancies between manuals have highlighted the need for a unification of PD categories and diagnostic criteria in subsequent *DSM* and *ICD* editions (Perez et al., 2005).

1.3 Prevalence and Life Course

1.3.1 Epidemiological / Quasi-Epidemiological Studies

“True” epidemiological studies of BPD, using formal epidemiological data, are scarce due to the resources required for such an endeavour (Mattia & Zimmerman, 2001). Subsequently, there has been a reliance on evidence derived from quasi-epidemiological studies based on experimental, family and survey designs (Black, Noyes, Pfohl, Goldstein, & Blum, 1993; Lenzenweger, Loranger, Korfine, & Neff 1997; Klein et al., 1995; Maier, Lichtermann, Klinger, Heun, & Hallmayer, 1992; Moldin, Rice, Erlenmeyer-Kimling, & Squires - Wheeler, 1994). Factors such as scarcity of adequate assessment instruments, successive changes in diagnostic criteria, and doubts over the current categorical system, further impinge upon inter-study agreement (Coid, Yang, Tyrer, Roberts & Ullrich, 2006).

A large disparity between prevalence figures for PDs generally, ranging from 2.1% to 18%, has been observed (Tyrer & Ferguson, 2000). For BPD specifically, a review of the more methodologically sound studies suggests a range in prevalence between 0.5% and 5.9 % (Coid et al., 2006; Grant et al., 2008; Torgersen, Kringlen, & Cramer, 2001; Samuels et al., 2002) [See **Table 1.2**]. Considering superior methodology, discrepancies here may be due to variations in assessment techniques. The especially high figure reported by Grant *et al.*, for example, is likely attributable to use of *lifetime prevalence* rates.

Table 1.2 The Prevalence of BPD in Non-Clinical Samples

Authors	Population	Assessment	Prevalence %	Comments
Swartz <i>et al.</i> (1990)	1,541 19 to 55yr olds from North Carolina	DIS ^a Borderline Index	1.8	DIS diagnostic standard discordant from DSM. Benefit of weighted data
Maier <i>et al.</i> (1992)	452 normal controls, their partners and relatives in Germany	SCID – II ^b DSM-III-R	1.1	Unrepresentative sample. Subjects without first degree relatives not included
Black <i>et al.</i> (1993)	247 relatives of obsessive compulsive and normal control probands in Iowa	SIDP ^c DSM-III	3.2	Small, unrepresentative sample
Moldin <i>et al.</i> (1994)	302 normal controls, parents and children in New York	PDE ^d DSM-III-R	2.0.	Small, unrepresentative sample
Klein <i>et al.</i> (1995)	229 relatives of normal controls in New York	PDE DSM-III-R	1.7	Small unrepresentative sample. Some interviews via telephone
Lenzenweger <i>et al.</i> (1997)	258 university students age 18/19 in New York	IPDE ^e DSM-III-R	0	Small specialist sample
Jackson & Burgess (2000)	10,641 individuals from a stratified random sample in Australia	IPDE ICD-10	0.96	Large representative sample. Borderline subcategory of ICD-10 not directly comparable with DSM

^a DIS, Diagnostic Interview Schedule; ^b SCID-II, Structured Clinical Interview for DSM Personality Disorders; ^c SIDP, Structured Interview for DSM-III-R Personality; ^d PDE, Personality Disorder Examination; ^e IPDE, International Personality Disorder Examination.

Table 1.2 continued The Prevalence of BPD in Non-Clinical Samples

Authors	Population	Assessment	Prevalence %	Comments
Torgersen <i>et al.</i> (2001)	2,053 individuals, 18 – 65 yrs in Oslo, Norway. Interviewed at home	SIDP-R ^a DSM-III-R	0.7	Large epidemiologically representative sample
Samuels <i>et al.</i> (2002)	742 individuals, 34 – 94 yrs re-interviewed from previous survey in Baltimore, USA	IPDE ^b DSM-IV	0.5	Epidemiologically representative sample. Restricted age range. Restricted to urban areas. Weighted data
Coid <i>et al.</i> (2006)	626 individuals 16-74 yrs from households in Great Britain	SCID-II ^c DSM-IV	0.7	Representative sample. Weighted prevalence. Not restricted to urban areas
Lenzenweger <i>et al.</i> (2007)	5,692 individuals taken from a household face-to-face survey in continental USA	IPDE DSM-IV	1.4	Use of multiple imputation. Only subsample received comprehensive assessment
Grant <i>et al.</i> (2008)	34, 653 Representative adult (18+) population from USA	AUDADIS-IV PD ^d	5.9	Large nationally representative sample. Based on lifetime prevalence
Huang <i>et al.</i> (2009)	21,162 individuals from 13 countries	IPDE DSM-IV	1.5 ^e	Use of multiple imputation. Did not distinguish BPD from other PDs within cluster B

^a SIDP-R Structured Interview for DSM-III-R; ^b IPDE International Personality Disorder Examination; ^c SCID-II, Structured Clinical Interview for DSM Personality Disorders; ^d Alcohol Used Disorder and Associated Disabilities Interview Schedule DSM-IV version; ^e Inclusive of all cluster B PDs (Borderline, Narcissistic and Antisocial PD)

1.3.2 Life Course of BPD

PDs tend to be chronic, emerging early in life and affecting functioning over many years. Indeed, it is often possible to identify childhood traits preceding adult PD, manifest as sub-clinical symptoms (Paris, 2003).

Although childhood precursors of PD are often present, it is argued that *diagnosable* symptoms appear for the first time during adolescence or early adulthood, in response to associated stressors (Paris, 2003; but see Kernberg, 1990, for a rebuttal). This view remains controversial, however, and will be discussed at length in chapter three.

Generally, the impulsive/dramatic (Cluster B) PDs, including BPD, tend to remit over time with symptoms lessening as middle age approaches (Paris & Zweig-Frank, 2001). Johnson *et al.* (2000), for example, found that PD traits declined 28% during adolescence and early adulthood. This trend is conjectured to result from social learning and other maturational processes, causing PD traits to diminish gradually over time. Additionally, impulsive disorders have been found to wax and wane over the life course (Grilo, McGlashan, & Skodol, 2000). In part, this may reflect reactions to stressful events, causing increases in symptoms during various life stages (Paris, 2003). Problems with *test-retest reliability*, however, may also cause fluctuations in diagnoses (Coid, 2003; Grilo *et al.*, 2004). Reporting at the *APA 1999 annual meeting*, Shea *et al.* (1999; cited in Coid, 2003) observed that 40% of BPD patients did not reach full criteria at six-month follow up. Conversely, Morey *et al.* (1999; cited in Coid, 2003) reported

during the same meeting that measures of personality traits revealed stability over time. As noted by Coid (2003), although the *DSM* states that PDs are “enduring patterns” of inner experience and behaviour, and “pervasive” and of “long duration,” research has consistently shown that they are subject to major fluctuations.

1.4 Issues in Description and Diagnosis of BPD

1.4.1 Heterogeneity

The *DSM* PD criteria are of a *polythetic* nature, meaning that there is not one single criterion necessary or essential for the diagnosis of a PD (Tyrer & Ferguson, 2000). This inevitably results in a heterogeneous group of patients, especially for BPD, which is characterised by a broad constellation of symptoms. There are 256 different ways to meet the *DSM* criteria for BPD, and two borderline patients may share only one common symptom (Trull, Distel, & Carpenter, 2011). This threatens the internal consistency of the BPD construct, challenging its validity as a single diagnostic entity (Livesley, Schroeder, Jackson, & Jang, 1994; Trull & Durrett, 2005), and leading to the label of “wastebasket diagnosis” (Tyrer & Ferguson, 2000).

1.4.2 Comorbidity

Comorbidity is ubiquitous in mental health, yet it is a term which causes confusion. The original construct of comorbidity, as coined by Feinstein (1970),

referred to instances when an individual with a disease under study suffered from an additional *distinct* disease. Since this conception, comorbidity has been used in reference to a variety of associations; ranging from disorders that are completely independent of one another to those which are so closely associated they may be thought of as identical. This latter phenomenon has been renamed *consanguinity*, reflecting instances when two seemingly co-occurring disorders are actually one inadequately defined disorder (Tyrer & Ferguson, 2000). Indeed, consanguinity may account for the observed overlap between various PDs, in some cases.

There is a wealth of research assessing comorbidity between BPD and both Axis I and Axis II disorders. Comorbidity between Axis II PDs occurs frequently (Lynam & Widiger, 2001; Torgersen et al., 2001; Zanarini et al., 1998a). Zanarini et al. (1998a) found that 72 % of 379 BPD patients also had an anxious cluster disorder; 40.1% had a dramatic cluster disorder; and 31.4% had an odd cluster disorder. Although comorbidity figures between PDs in the community are lower, ranging from 29% (Torgersen et al., 2001) to 46% (Coid et al., 2006), they are still considerable.

It is unsurprising that BPD, with its diverse range of symptom criteria, co-occurs with other PDs. For example, unstable relationships are characteristic of both borderline and antisocial PD, while paranoid ideation is associated with borderline, paranoid and schizotypal PDs. These observations are confirmed by empirical research, revealing comorbidity between BPD and both antisocial (McGlashan et al., 2000) and paranoid (Oldham et al., 1992) PDs. As noted, observed comorbidity may be an artefact of incorrect classification, preventing

sufficient distinctions among individual PDs (Brieger, Ehrt, & Maneros, 2003; Coid et al., 2006); thus challenging the utility of the current categorical system (a point returned to shortly).

The diversity of BPD criteria may also eventuate in Axis I comorbidity.

Comorbidity between BPD and a variety of Axis I disorders has been reported including: depression, bipolar disorder, anxiety, post-traumatic stress disorder (PTSD) and substance abuse.

The association between BPD and depression is well established (Klein, 1977), due to marked similarities between a number of symptom criteria, including suicide ideation and chronic feelings of emptiness. Subsequently, there is a substantial body of research assessing BPD in individuals with depression, and depression in individuals with BPD (see **Tables 1.3** and **1.4**).

Unfortunately, these studies are marred by similar problems to those associated with prevalence studies, i.e. inconsistent assessment and sampling; therefore, comorbidity figures tend to widely vary. Nevertheless, the observed association between BPD and depression appears robust. Researchers note, however, that the relationship between BPD and depression is non-specific (Gunderson & Phillips, 1991), as it appears that *all* PDs are related to an elevated incidence of depression (Skodol et al., 1999; Zanarini et al., 1998b).

Similarities between the symptoms of bipolar disorder and BPD have also been noted, specifically *impulsivity* and *emotional dysregulation*. When

Table 1.3 Prevalence of BPD in Mood Disordered Patients

Author	Comments	Unipolar	Bipolar
Ucok <i>et al.</i> (1998)	90 bipolar outpatients		10% (Bipolar I ^a)
Rossi <i>et al.</i> (2001)	Patients recovered from depressive (117) and bipolar (71) disorders requiring hospitalisation	30.8%	29.6%
Brieger <i>et al.</i> (2003)	117 patients with unipolar and 60 with bipolar disorders	12%	6.7%
Smith <i>et al.</i> (2005)	Young adults 46 major depressive disorder; 14 with bipolar affective disorder; 27 with bipolar spectrum disorder	None fulfilled diagnostic criteria for BPD, although there were higher reported borderline characteristics	
Zimmerman <i>et al.</i> (2005)	859 psychiatric patients with major depression	12.2%	

^a Bipolar I disorder includes psychotic symptoms, i.e. delusions and hallucinations

Table 1.4 Prevalence of Mood Disorders in BPD patients

Author	Comments	Unipolar	Bipolar
Swartz <i>et al.</i> (1990)	Community Sample of 1,541 19-55 yr olds	40.7%	14.1% (Bipolar Disorder)
Zanarini <i>et al.</i> (1998b)	379 borderline patients.	83%	10% (Bipolar II ^b)
Skodol <i>et al.</i> (1999)	571 subjects. Measured % co-occurrence (single figure representing the number of subjects with a mood disorder who have a PD and vice versa).	31%	9% (Bipolar I ^a) 4% (Bipolar II)

^b Bipolar II disorder involves hypomanic but not manic episodes and no psychotic features

individual criteria are examined closely, however, subtle differences become apparent. For example, the emotional dysregulation observed in BPD is often in response to external events, while emotional changes in bipolar disorder tend to emerge from internal biological shifts (Dolan-Sewell, Krueger, & Shea, 2001). Similar to unipolar depression, relationships appear non-specific. Obsessive compulsive, histrionic, and paranoid PDs occur as commonly as BPD in individuals with bipolar disorder (Ucok, Karaveli, Kundakci, & Yazici, 1998).

Post-Traumatic Stress Disorder (PTSD) is also commonly associated with BPD, and may be considered in light of the established relationship between BPD and trauma. Studies reveal that between 50% and 70% of BPD patients have suffered from childhood abuse (Golier et al., 2003; Herman, Perry, & van der Kolk, 1989; Ogata et al., 1990; Weaver & Clum, 1993; Westen Ludolph, Misle, Ruffins, & Block 1990; Zanarini et al., 1997; 2002), suggesting that, for a substantial sub-set of individuals, BPD is a form of complex PTSD (Hodges, 2003) [See chapter two].

BPD and PTSD, though commonly co morbid (Hyer, Woods, Boudewyns, & Harrison, 1990; Southwick, Yehuda, & Giller, 1993), are not ubiquitously so. Zanarini *et al.* (1998b) found that only 56% of BPD patients met criteria for PTSD. Further, PTSD is associated with other PDs including: avoidant, paranoid, obsessive compulsive and schizoid (Bollinger, Riggs, Blake, & Ruzek, 2000; Gomez Salazar - Fraile, & Gonzalez - Lujan, 2006; Hyer et al., 1990; Southwick et al., 1993).

Finally, BPD has been associated with substance abuse (Rounsaville et al., 1998; Zanarini et al., 1998b). Prior to the *DSM* axial separation of personality from substance use (Axis I) disorder, researchers considered the classification of an “addictive personality” (Ball, 2005). A revisiting of this idea can be recognised in the identification of an *impulsivity dimension*, thought to account for the association between substance use disorders and cluster B PDs (Paris, 2003; Siever & Davis, 1991).

Comorbidity between substance use disorders and cluster B PDs is commonplace, and associations with BPD appear particularly common. Rounsaville *et al.* (1998) found that 30% of drug users met criteria for BPD, while Zanarini *et al.* (1998b) found very pronounced levels of overall substance abuse disorder in borderline patients (82%). These extremely high figures, however, partly reflect the assessment of lifetime rather than point prevalence.

The above reported associations may be accounted for by the *psychobiological model* (Siever & Davis, 1991), which posits that Axis I and Axis II disorders are situated on the same four dimensions: *cognitive/perceptual*; *affect regulation*; *impulse control*; and *anxiety modulation*. Therefore, Axis I and Axis II disorders are quantitatively, but *not* qualitatively distinct. Extreme, acute, maladaptive combinations of these four dimensions are thought to lead to Axis I disorders, while less extreme, more persistent disturbances are thought to lead to PDs (Dolan-Sewell et al., 2001).

1.4.3 The Categorical versus Dimensional Approach

The current categorical approach to PDs is shrouded in controversy due to problems including: heterogeneity of group, consanguinity (co-occurrence of disorders) and instability of diagnosis (Clark, 2007; Dahl, 2008; Tyrer et al., 2007). In a damning indictment, it has been suggested that: “*categorical diagnosis represents instead a simplistic and presumptive understanding of personality disorder pathology that is a hindrance to empirical research and clinical practice*” (Widiger, 1993, p.1). In response to the above concerns, there has been a burgeoning of research assessing the dimensional approach to the classification of PDs (Clarkin, Hull, Cantor, & Sanderson 1993; Krueger, 2005; Skodol et al., 2005; Sprock, 2003; Trull & Durrett, 2005).

1.4.3.1 Structural Analysis of the BPD Construct

Researchers have utilised *taxometric*, *factor* and *latent class* analyses to ascertain whether BPD is better represented as a dimensional construct, and if so, the number of dimensions manifest. Taxometric analysis addresses the first question, by indicating whether BPD is better represented as a latent class (taxon) to which individuals may or may not belong, or a dimension of psychopathology, represented by a continuum of severity (Trull et al., 2011). Of the taxometric studies published, there appears to be agreement that BPD, as defined by the *DSM* and *Personality Assessment Inventory (PAI)*, is better conceptualised as a dimensional construct (Edens, Marcus, & Ruiz, 2008; Rothschild et al., 2003; Trull, Widiger, & Guthrie, 1990).

Factor analysis may be utilised to ascertain how many *dimensions* of psychopathology underpin the BPD construct. To date, results are mixed, and support the utility of both unitary and multiple latent factors (Sanislow, Grilo, & McGlashan, 2000; Giesen-Bloo, Wachters, Schouten, & Arntz, 2010). Sanislow *et al.* (2000) found support for a three (disturbed relatedness, behavioural dysregulation and affective dysregulation) and a one-factor solution, with comparable fit indices for both models. The utility of the three factor model, however, may be questioned, as the three factors were very highly correlated ($r = 0.84$ to 0.90). Giesen-Bloo *et al.* (2010) using confirmatory factor analysis, found support for both a nine and a one dimensional model, using the BPD Severity Index-IV (BPDSI-IV) based on the DSM-IV criteria. The manifestation of nine dimensions was unsurprising, however, as the BPDSI-IV scale was devised to reflect the nine criteria of the *DSM*, and this factor structure was validated when devising the BPDSI (Trull *et al.*, 2011).

It is pertinent to note that although the above studies claim to offer support for the dimensional approach, one factor models were also supported. Therefore, further research is required before the dimensional approach is declared superior. The present lack of consensus is likely due to discrepancies in measurement and sample usage (Trull *et al.*, 2011).

Latent class analysis (LCA) is used to group individuals into *latent classes*, according to their scores on a range of categorical indicators (Trull *et al.*, 2011) [see chapter eight]. Fossati, Maffei, and Bagnato (1999) found that 564 patients, interviewed with the DSM-IV, were optimally represented by 3 distinct groups:

1) *asymptomatic*; 2) *high endorsement of all BPD symptoms*; 3) *low endorsement of all BPD symptoms*, excepting impulsivity and inappropriate anger. Clifton and Pilkonis (2007) using the DSM-III-R, found that 2 classes best represented their sample of clinical and non-clinical adults. One class represented those with *high to moderate endorsement of BPD criteria*, while the other represented individuals with *low rates of endorsement*, thereby supporting an underlying single dimension of BPD pathology.

1.4.3.2 “Normal” Personality Correlates of BPD

The association between established “normal” traits and PDs has been extensively assessed (Trull, 1992; Soldz, Budman, Denby, & Merry, 1993; Hyler, Woods, Boudewyns, & Harrison, 1990; Duijsens & Diekstra, 1996; Benjamin, 1994; Ball, Tennen, Poling, Kranzler, & Rounsaville, 1997; Lynam & Widiger, 2001; Miller, Reynolds, & Pilkonis, 2004; Miller, Bagby, Pilkonis, Reynolds, & Lynam, 2005; Samuel & Widiger, 2006; Saulsman & Page, 2004; Wiggins & Pincus, 1989). Existing models of trait theory including: the interpersonal circumplex (Gifford & O’Connor, 1987), Eysenck’s (1978) three-factor model, and the five factor model (FFM) (Costa & McCrae, 1990) have been utilised.

Empirical studies have mainly centred on the FFM, which identifies the traits of openness, conscientiousness, extraversion, agreeableness and neuroticism, each of which is sub-divided into a further six factors (see Samuel & Widiger, 2008 for more details). Recent meta-analysis examining the relationship between FFM

traits and BPD (Samuel & Widiger, 2008) reveals a moderate positive correlation with neuroticism ($r = 0.54$), and negative correlations with agreeableness ($r = -0.24$) and conscientiousness ($r = -0.29$). Further, correlations were found for all six sub-facets of neuroticism, including: anxiousness, angry hostility, depressiveness, self-consciousness, impulsiveness and vulnerability. Similarly, negative correlations were found for a range of extraversion, agreeableness and conscientious facets, including: warmth, positive emotions, trust, straightforwardness, compliance, competence, dutifulness, self-discipline and deliberation. These findings are consistent with current *DSM* criteria, confirming that BPD is associated with emotional instability and antagonistic attitudes (Trull, 1992), but offer increased precision through the inclusion of sub-facets (Wiggins & Pincus, 1989).

While numerous studies support the conceptualisation and assessment of PDs in terms of trait dimensions, the clinical application of this approach remains unresolved. Although the dimensional approach accommodates problems inherent in the categorical system, including comorbidity and illusionary boundaries, using this approach *exclusively*, for the assessment of PDs may be problematic. PDs involve more than a straightforward collection of maladaptive traits (Livesley & Jang, 2000), and the application of “normal” (though negative) traits may trivialise PDs, which could become applicable to a substantial proportion of the population (Benjamin, 1993).

Though there are disadvantages associated with the categorical approach, attendant advantages include: parsimonious conveyance of clinical information;

guidance in approach and treatment (Benjamin, 1993); and an informational format congruent with human information processing (Blashfield & Livesley, 1999). Further, the categorical paradigm is completely embedded within the current mental health system. As clearly illustrated by Sprock (2003); in terms of clinical utility, clinicians rated categorical models more favourably than dimensional, despite evidence of lower inter-rater reliability.

1.4.4 A New Hybrid Approach?

Due to the taxonomy issues discussed above, researchers have suggested a “hybrid” model for the classification of PDs, combining both categorical and dimensional approaches (Cloninger, 2000; Livesley, 1998; 2001). In this vein, the DSM-5 Personality Disorders Workgroup has proposed a 3 stage process for the diagnosis of BPD (<http://www.dsm5.org>). Firstly, clinicians are asked to give a “type rating” indicating the degree to which the patients’ presentation matches the narrative description (on a scale of 1 to 5) of the “borderline type.” Secondly, a set of 10 traits are rated on a scale of 0 to 3. These traits are organised into 4 higher order domains: *negative emotionality* (emotional lability, self-harm, separation insecurity, anxiousness, low self-esteem, and depressivity); *antagonism* (hostility and aggression); *disinhibition* (impulsivity) and *schizotypy* (dissociation proneness). Thirdly, the individual level of self (identity integration, integrity of self-concept, and self-directedness) and interpersonal (empathy, intimacy and cooperativeness, and integration of representation of others) functioning is rated on a 5-point scale (Trull et al., 2011).

There has been a flurry of criticisms in response to this proposal (Gunderson, 2010; Shedler et al., 2010; Trull et al., 2011). Namely, it is classed as too complicated and lacking in parsimony, leading to fears that busy clinicians will not use, or understand, the largely psychodynamic formulations (Shedler et al., 2010; Trull et al., 2011). Further, the choice of traits is based on inadequate clinical rationale (Shedler et al., 2010), and each trait is given equal weighting, despite the fact that BPD evinces core traits, such as affective instability (Trull et al., 2011). Finally, the prototype approach, while proven as a clinical diagnostic tool (Kim & Ahn, 2002), is hindered by the use of many features, which would broaden the BPD construct and increase heterogeneity (Trull et al. 2011), contra to what is required (as discussed in previous sections).

Inevitably, this has led to counter proposals. Gunderson (2010) posits a less radical approach, in which the existing BPD criteria are retained, but organised into 4 domains: *interpersonal hypersensitivity*, *emotional dysregulation*, *behavioural dyscontrol* and *disturbed self*. Each of these domains would represent an *endophenotype* (collection of related symptoms with genetic underpinnings). BPD criteria would be rated on a dimensional scale and diagnosis would require that at least 5 symptoms are identified (as in the DSM-IV-TR), with at least one symptom from 3 of the 4 domains. Importantly, by focusing on the number of phenotypes, rather than solely symptoms, heterogeneity within the BPD construct would be reduced (Gunderson, 2010). These changes, being incremental, would prevent major disruptions caused by a radical change in format, and sustain research and clinical development (Gunderson, 2010). The controversy and disagreement pertaining to the new

BPD construct, however, are likely to continue until the launch of the DSM-5 in 2013.

1.5 The Consequences of BPD

The potential consequences of BPD are stark. Studies suggest that patients with BPD are more likely to die prematurely, with approximately 10 % of individuals with BPD eventually committing suicide (Paris, 2002). Further, premature death generally appears higher in this group. In a 27-year follow up study, Paris and Zweig-Frank (2001) reported that 18.2% (10.3% accounted for by suicides) of 64 BPD patients were deceased before the age of 50. Increased risk of death may result from impulsive and affective instability traits, which predispose individuals to health problems and reckless behaviour (Paris & Zweig-Frank, 2001).

The financial costs associated with BPD are high. Individuals with BPD are common treatment seekers and the most prevalent consumers of mental health services (Coid, 2003; Coid et al., 2006; Gross et al., 2002). Further, BPD is widespread within the prison service. Twenty-three per cent of male prisoners on remand, 14% of sentenced male prisoners and 20% of female prisoners are diagnosed with BPD (NICE, * 2007).

* National Institute for Clinical Excellence

Individuals with any PD are more likely to be separated or divorced, and unemployed or economically inactive (Coid et al., 2006). Those with BPD, however, are especially likely to demonstrate significant impairment at work, in social relationships and leisure pursuits; faring much worse than patients with obsessive compulsive PD and major depressive disorder (Skodol et al., 2002). Family life is often affected, and less than half of BPD patients get married, with even fewer having children (Paris, 2003). All evidence suggests that individuals suffering from BPD have a severely reduced quality of life.

1.6 Conclusions

Chapter one outlined the history of the borderline construct; the classification, description, prevalence, life-course, and long-term sequelae of BPD, in addition to problematic issues concerning definition and assessment. From this review it is evident that the BPD construct has longevity, remaining remarkably similar from conception in 1938 to *DSM* diagnosis today. It is also clear that BPD affects a substantial number of people within the general population and is associated with premature death and reduced quality of life. Additionally, BPD places a substantial burden on the mental health and prison services. Debate continues regarding the classification of BPD, and a hybrid (combined categorical and dimensional approach) system has been proposed for the DSM-5 in 2013. A categorical approach to BPD was utilised for all analyses following in chapters 6, 7 and 8 in reflection of the dominant paradigm currently embedded within research and clinical fields.

Chapter Two: Existing Theories and Associated Research Findings for the Aetiology of BPD

Overview: The following chapter outlines classic theories pertaining to the aetiology of BPD, and empirical research supporting these models, which fall within a stress-diathesis framework. Particular attention is paid to the Biosocial Developmental Model (BDM), which is a comprehensive developmental theory, incorporating many aspects from earlier theories. The concluding section points towards future directions for expanding our understanding of the aetiology of BPD.

2.1 Classic Aetiological Theories

There are numerous theories, from various paradigms, pertaining to the development of BPD, which will be outlined briefly below and in Table 2.1. A detailed description of each theory, however, is beyond the scope of this thesis.

2.1.1 Cognitive Theories

Cognitive theories draw heavily on the concept of *schemata*; a set of generalisations about the self, world and others (Beck, Freeman, & associates 1990; Ryle, 1997; Young, Klosko, & Weishaar, 2003; Young, 1999). Once these schemata are formed, they heavily guide subsequent information processing, leading to further cognitive distortions, including: dichotomous or black and white thinking and a weak sense of identity (Beck et al., 1990). Maladaptive schemata, characteristic of the borderline individual, are believed to be the

consequence of family dysfunction or trauma (Janoff-Bulman, 1989), and an overly emotional temperament, and once formed become increasingly resistant to change.

2.1.2 Attachment Theories

Common with cognitive theories, attachment models centre on the notion of schemata or modes, though development is specifically linked to early interactions with caregivers (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004; Bateman & Fonagy, 2004; Fonagy, Target, Gergely, Allen & Bateman, 2003; Fonagy et al., 1996; Levy, 2005). Despite a focus on early interactions, attachment theory may be framed within a developmental paradigm, in which BPD is viewed as resulting from a series of successive interactional processes along the developmental trajectory. While new experiences influence the individual, they are not independent of pre-existing schemata, and should be understood within the context of these models, which are believed to interact with constitutional predispositions (Levy, 2005).

2.1.3 Emotional Dysregulation Theories

Some theorists suggest that emotional dysregulation is the core feature and primary cause of BPD (Crowell, Beauchaine, & Linehan 2009; Fruzzetti, Shenk, & Hoffman 2005; Linehan & Koerner, 1993; Linehan, 1993; Putnam & Silk, 2005). The *biosocial* model (Linehan, 1993) is the most established and influential of these theories. Essentially, BPD is believed to emerge from

transactions between biological vulnerability (an overly emotional temperament) and an “invalidating environment” (ranging from relatively moderate invalidation to extreme abuse). During development, the child’s inborn pronounced emotional response is not adequately responded to by caregivers; therefore, the ability to self-soothe or inhibit inappropriate emotional responses is never learnt, leading to vacillation between emotional suppression and extreme over emotionality.

2.1.4 Trauma Theories

Due to the reported association between childhood trauma and abuse and BPD (e.g. Waller, 1994; Weaver & Clum, 1993; Yen et al., 2002), historically, trauma theories have been popular (Herman, 2001; Zanarini & Frankenberg, 1997). Trauma is believed to exacerbate, or trigger, existing temperamental predisposition (Zanarini & Frankenberg, 1997), or set in motion a form of Complex Post-Traumatic Stress Disorder, leading to BPD typical symptoms (Hodges, 2003).

2.1.5 Socio-Cultural Theories

The social learning theory (SLT) of BPD (Millon, 1993) takes into consideration the wider social context, and suggests that societal changes, such as the collapse of the traditional family structure, have exacerbated risk factors implicated in the development of BPD. In summary, the SLT of BPD purports that individuals with a constitutional predisposition, exposed to early maladaptive experiences,

within a culture of reduced stability and guidance, are at increased risk of developing BPD.

2.2 Comment on Classic Aetiological Theories.

As outlined above, and in summary Table 2.1 (see below), classic aetiological theories share a number of commonalities, and all posit a stress-diathesis model, in which diatheses, internal to the individual, interact with external stressors, leading to the development of BPD (Ingram & Price, 2001). Each theory, though divergent in detail, tends to posit the same diatheses and stressors; diatheses in terms of an overly emotional temperament, and stressors in terms of psychosocial risk factors. Recently, the integrative Biosocial Developmental Model (BDM) (Crowell et al., 2009) has been developed, which incorporates aspects of the theories outlined above.

2.3 The Biosocial Developmental Model (Crowell et al., 2009)

Although the biosocial model (Linehan, 1993) has been influential, new insights pertaining to the biological correlates of BPD, and a focus on the developmental precursors of the disorder, have facilitated an extension and refinement of this theory (Crowell et al., 2009). Specifically, Crowell and colleagues identify impulsivity as the earliest emerging trait among those who later develop a diagnosis of BPD; and distinguish between impulsivity and emotional dysregulation, which are physiologically distinct before the canalisation of BPD.

Table 2.1 Summary of Classic Theories for the Aetiology of BPD

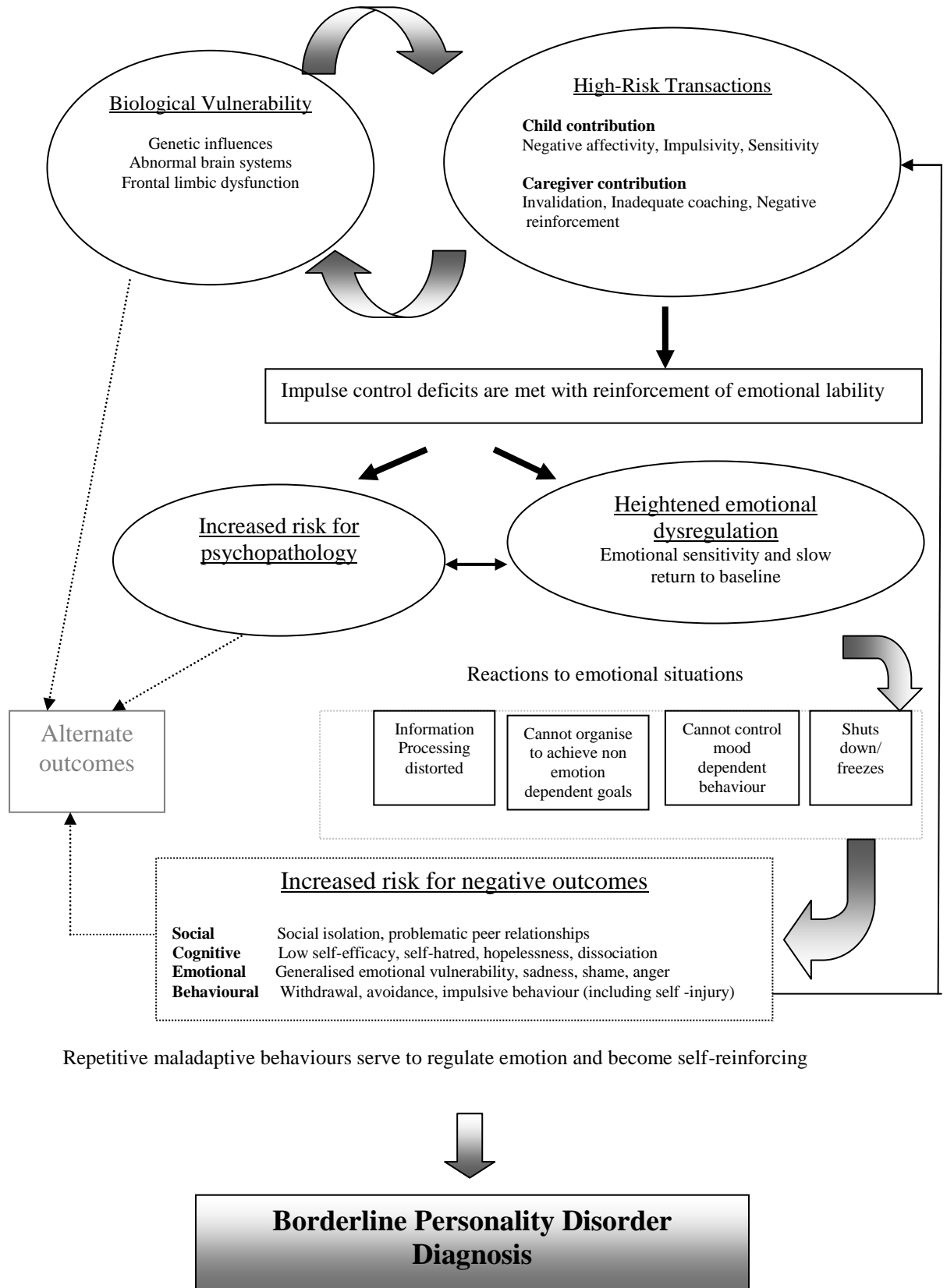
Paradigm <i>Examples</i>	Diathesis	Stress	Diathesis x Stress
Cognitive			
<i>Cognitive Distortion Theory (Beck, 1990)</i> <i>Schema Mode Theory (Young, 2003)</i>	Overly emotional temperament	Childhood experiences, possibly including abuse and neglect	-Maladaptive environment leads to the formation of cognitive distortions, e.g. <i>maladaptive schema</i> , <i>dichotomous thinking</i>
Attachment			
<i>Mentalisation Theory (Bateman & Fonagy, 2004)</i>	Genetic predisposition	Attachment trauma	-Inadequate attachment experiences lead to a deficit in mentalisation ability due to the formation of maladaptive relationship schema
Emotional			
<i>Biosocial Model (Linehan, 1993)</i>	Inborn tendency towards over-emotionality	“Invalidating environment” (from disqualifying of private experience to abuse and neglect)	-Due to the invalidating environment, the child is not taught how to regulate a tendency towards over-emotionality
Trauma			
<i>Tripartite Theory (Zanarini, Frankenberg, 1997)</i>	Hyperbolic temperament	Triggering event in addition to a range of traumatic childhood experiences	-A triggering event reminds the individual of earlier adversity and frustration leading to clinical BPD
<i>C-PTSD Theory (Herman, 2003)</i>	Vulnerable temperament	Prolonged childhood abuse	-Prolonged and extreme exposure to trauma leads to a re-programming of the neuro-endocrine system
Socio-cultural Theory			
<i>The Social Learning Theory (Millon, 1993)</i>	Constitutional predisposition (unspecified)	Problematic parent-child relationships plus sociological influences	-Biological predisposition and parental problems interact and are exacerbated by the social climate

The BDM states that complex transactions between biological vulnerability and psychosocial risk factors (family psychopathology, disturbed attachment, invalidating environment, child maltreatment, and socio-cultural correlates) influence emotional and behavioural development from conception onwards (Crowell et al., 2009). One probable developmental trajectory has been identified (see **Figure 2.1**, overleaf). An early vulnerability to impulsivity becomes heightened emotional sensitivity, which is then potentiated across development by environmental risk factors, leading to more extreme emotional, behavioural and cognitive dysregulation.

As shown in Figure 2.1, when extreme emotional reactions occur repeatedly over months and years, emotional dysregulation becomes trait like, and outcomes such as social isolation and problematic peer relationships become canalised. These traits and behaviours, although maladaptive, become increasingly frequent and reinforcing, due to their emotional regulation or avoidance functions. In this way, early vulnerability interacts with learning history to shape and maintain dysregulated emotional, behavioural, interpersonal and cognitive aspects of the self, thus creating the “borderline” personality.

Figure 2.1 The Biosocial Developmental Model.

(Taken from Crowell et al., 2009)



2.4 Empirical Evidence Supporting Stress-Diathesis Factors

2.4.1 Stressors

As the above overview reveals, most aetiological theories pertaining to BPD identify maladaptive childhood experiences as the predominant stressor leading to the development of borderline pathology. These experiences, mostly familial in origin, range from profound abuse to bonding problems and emotional neglect (Zanarini & Frankenberg, 1997). Each of these factors has received ample empirical attention, though mainly through retrospective methodology, the shortcomings of which will be discussed subsequently.

2.4.1.1 Parent - Child Interactions.

“Looking Back” (Retrospective Studies)

Abuse

Many retrospective studies have revealed a strong association between childhood sexual abuse (CSA) and BPD in adulthood (Bandelow et al., 2005; Herman, Perry, & van der Kolk 1989; Ludolph et al., 1990; Ogata et al., 1990; Paris, Zweig-Frank, & Guzder, 1994a, b; Waller, 1994; Weaver & Clum., 1993; Westen, Ludolph, Mistle, Ruffins, & Block, 1990; Yen et al., 2002; Zanarini et al., 1997; Zerkowitz, Paris, Guzder, & Feldman, 2001). Nevertheless, the true nature of these findings remains unqualified. In a meta-analytic review, Fossati

Madeddu and Maffei (1999) revealed a moderate correlation ($r = 0.279$) between childhood sexual abuse and BPD, challenging that CSA is the main causal antecedent in the development of this disorder.

Similarly, a robust association between childhood physical abuse and BPD has been observed (Herman et al., 1989; Ludolph et al., 1990; Ogata et al., 1990). Reported findings, however, are non-specific. Strong relationships between physical abuse and other personality disorders have also been noted (Battle et al., 2004).

Neglect and Parental Bonding

Retrospective studies reveal that borderline patients tend to report more emotional neglect from parents compared to controls (Links, Steiner, & Huxley, 1988; Zanarini, Gunderson, Marino, Schwartz, & Frankenberg, 1989; Zweig-Frank & Paris, 1991). Further, neglect often tends to be bi-parental (Frank & Hoffman, 1986; Gunderson, Kerr, & Englund, 1980; Links et al., 1988), and in some cases the result of parental psychopathology (Links et al., 1988).

Zweig-Frank and Paris (1991) compared Parental Bonding Inventory [PBI] (Parker, 1990) scores in two large, diverse samples. Compared to controls, BPD patients remembered their parents as less caring and more overprotective, consistent with the model of “affectionless control” (Parker, 1983). Affectionless control, however, has also been linked to the development of depression (Parker, 1983), suggesting that emotional neglect and biparental failure can have various psychopathological consequences (Paris & Zweig-Frank, 1993).

“Looking Forward” (Prospective Studies)

Abuse, Neglect and Maladaptive Parenting

Longitudinal, prospective studies assessing the association between maladaptive childhood experience and subsequent BPD are sparse at present. There is one developing body of research, however, which draws on the “Children in the Community (CIC)” longitudinal resource. The CIC study is an on-going investigation based in two upstate counties in New York, tracing developmental trajectories towards various psychiatric disorders over 20 years. Using prospective data pertaining to approximately 800 youths (initial sample), the team have explored risk factors (both environmental and early characteristics) for personality disorders and symptoms. There have been several waves of data collection: early childhood (1-10 years); early adolescence (mean age 14 years); mid adolescence (mean age 16 years); early adulthood (mean age 22 years), and at 33 years of age (Cohen, Crawford, Johnson, & Kasen 2005).

In the first CIC study, Johnson, Cohen, Brown, Smailes, and Bernstein (1999) found that sexual abuse and neglect (as ascertained from the New York State Central Registry for Child Abuse and Neglect) were each predictive of *elevated symptom levels* of BPD, after age and parental psychiatric disorders were controlled for. These associations were non-specific, however. Sexual abuse was also predictive of elevated histrionic, depressive and total PD symptom levels; and neglect of antisocial, avoidant, dependent, narcissistic, paranoid, passive-aggressive, schizotypal and total PD symptom levels. Similarly, physical abuse

was predictive of borderline, antisocial, dependent, depressive, passive aggressive, schizoid and total PDs.

In a subsequent study, Johnson, Cohen, Chen, Kasen, and Brook (2006) found that low parental nurturance and aversive parental behaviour in childhood were predictive of elevated borderline symptom levels at 22 and 33 years of age, after controlling for covariates. The *aversive parental behaviours* composite comprised measures including: harsh punishment, inconsistent maternal enforcement of rules, frequent loud arguments between parents, difficulty controlling anger towards the child, possessiveness, use of guilt to control the child, and verbal abuse. The *low parental nurturance* composite comprised measures including: low parental time spent with child, poor parental communication with child, poor home maintenance, low educational aspirations for the child, poor parental supervision, low paternal assistance to the child's mother, and poor paternal role fulfilment. These results confirm that parenting problems, other than serious on-going abuse, can potentiate in the development of borderline pathology.

Due to the relatively small sample (approximately 600 following attrition) size in the above studies, results pertain to *symptom scales* of BPD rather than a clinically relevant collection of symptoms (i.e. 5 or more). Because of the low base rate of BPD in the community (Paris, 2003), clinical diagnosis according to the *DSM* would have yielded groups lacking in statistical power. Therefore, the direct clinical relevance of the findings reported here is unclear.

In a pseudo-prospective study, Rogosch and Cicchetti (2005) compared the *BPD precursor composite* scores of maltreated (as derived from the Department of Human Services records) and non-maltreated children between 6 and 12 years of age. The precursor composite (full description: chapter three) comprises developmentally appropriate measures of: personality features, interpersonal relationship characteristics, representations of self and others, and self-harming behaviour or suicidal ideation; all thought to presage the development of BPD. Maltreated children evinced higher mean scores on the BPD precursor composite, supporting that childhood maltreatment, in terms of childhood abuse and/or neglect, is predictive of BPD related features.

Further prospective support is provided by Widom, Czaja, and Paris (2009) who compared 500 individuals with documented cases of abuse and neglect to 396 demographically-matched controls. Significantly more abused and/or neglected children met criteria for BPD as adults. This relationship was mediated by parental drug or alcohol problems, having a diagnosis of drug abuse, major depressive disorder or post-traumatic stress disorder, and being unemployed or dropping out of high school. Therefore, childhood abuse and neglect appear to confer heightened risk for BPD, rather than solely determine the disorder.

2.4.1.2 Parental Psychopathology

Familial studies reveal that BPD is significantly more common among first-degree relatives of borderline individuals compared to controls (for review see: White, Gunderson, Zanarini, & Hudson 2003). A review of existing studies

suggests a 4-20 fold increase in the occurrence of BPD in relatives of BPD probands compared to the general population (White et al., 2003). This relationship appears non-specific, however. Studies have also consistently reported a high incidence of affective disorders, particularly unipolar depression, and impulsive spectrum disorders, such as substance abuse and antisocial personality disorder, in the relatives of BPD probands (Zanarini & Frankenberg, 1997).

Increased inter-generational incidence of BPD may be attributed to two factors. Firstly, parental psychopathology may negatively impact on parental ability, exposing the child to a maladaptive environment (as described above). Secondly, a predisposition towards the development of BPD may be conferred via genetic transmission.

In familial studies, it is impossible to disentangle the effects of genes versus the environment. Research, however, suggests a robust relationship between parental psychopathology and poor parenting behaviour and abuse (Kandel, 1990; Lovejoy, Graczyk, O'Hare, & Newman 2000; Magura & Laudet, 1996; Wolock & Magura, 1996). A meta-analytic review by Lovejoy *et al.* (2000) revealed a strong association between parental depression and negative maternal behaviour. Similarly, an association between parental substance abuse and child maltreatment has been observed (Magura & Laudet, 1996; Wolock & Magura, 1996). Additionally, parental psychopathology is thought to negatively impact on attachment relationships (Gerhardt, 2004; Lovejoy et al., 2000). The psychiatrically unwell mother, preoccupied with her own feelings, may have

scant resources remaining to appropriately respond to her child. In extreme cases, there may be a trans-generational transmission of disorganised attachment patterns, predisposing offspring to borderline pathology (Gerhardt, 2004).

Evaluation of Empirical Research Regarding Child Maltreatment

Currently, the majority of research pertaining to childhood maltreatment is retrospective in nature, due to the temporal and financial investment required for prospective, longitudinal designs (Paris, 2003). Subsequently, most existing research is limited in what it can reveal about the causative nature of aetiological factors identified. There are a number of reasons for this.

Firstly, retrospective designs are vulnerable to the vagaries of memory (Fiske & Taylor, 1991). Compounding this problem is the tendency of borderline patients to misinterpret or misremember childhood events (Bailey & Shriver, 1999). Researchers suggest that borderline individuals, particularly those prone to cognitive distortions, e.g. splitting, are likely to demonise family members as a way of avoiding ambivalent feelings (Paris, 1995). Thus, adult reports of childhood experiences should be considered with caution. Indeed, in a meta-analytic review, the study with *externally validated* childhood sexual abuse (CSA) had the lowest (non-significant) reported association with BPD (Fossati, Madeddu, & Maffei, 1999). This finding is congruent with an over reporting of abuse in non-validated studies.

Secondly, due to the lack of temporal ordering in retrospective studies, the *correlational* relationship between abuse and BPD precludes verification of

causality (Paris, 1995). While the observed relationship may reveal the aetiological nature of trauma, it could conversely reflect that those suffering from BPD are more likely to encounter abuse. Further, other unidentified variables may be implicated in the observed relationship. For example, those with BPD may have “difficult” temperamental traits, making them more likely to encounter trauma (Paris, 1998a).

Thirdly, abuse does not occur in a vacuum and various environmental factors may co-exist, confounding results. These factors include: physical and emotional neglect, separations from caretakers, verbal abuse, temperamental predisposition, parental psychopathology, chaotic home environment, and grossly inappropriate parental behaviour (Paris & Zweig-Frank, 1993). It is common for many forms of maladaptive experience to co-occur (Zanarini & Frankenberg, 1997). For example, childhood sexual abuse almost always occurs in the context of bi-parental abuse and neglect (Battle et al., 2004; Johnson et al., 1999; Paris, 1998a; Zanarini et al., 2002). Retrospective designs make it very difficult to disentangle the relative contributions of these various aetiological factors.

Finally, the studies reported above fail to incorporate the full stress-diathesis model. Without measures of temperament, for example, it is impossible to ascertain the contributory role of biological predisposition in the development of psychopathology. In contrast, prospective, longitudinal designs offer more promise of elucidating stress-diathesis interactions, and the relative contribution of biological risk factors in the development of BPD.

2.4.2 Diatheses

2.4.2.1 Genes

Studies addressing the role of genes in the development of BPD are scarce. Two twin studies investigating the genetic underpinnings of BPD have been conducted (Torgersen, 1984; Torgersen et al., 2000). While the earlier study did not support that BPD is genetically transmitted, the latter reported a higher concordance rate between monozygotic (38%) compared to dizygotic (11%) twins. The discrepancy between the two studies may be attributable to the small number of monozygotic twins available in the first study, possibly occluding any effect.

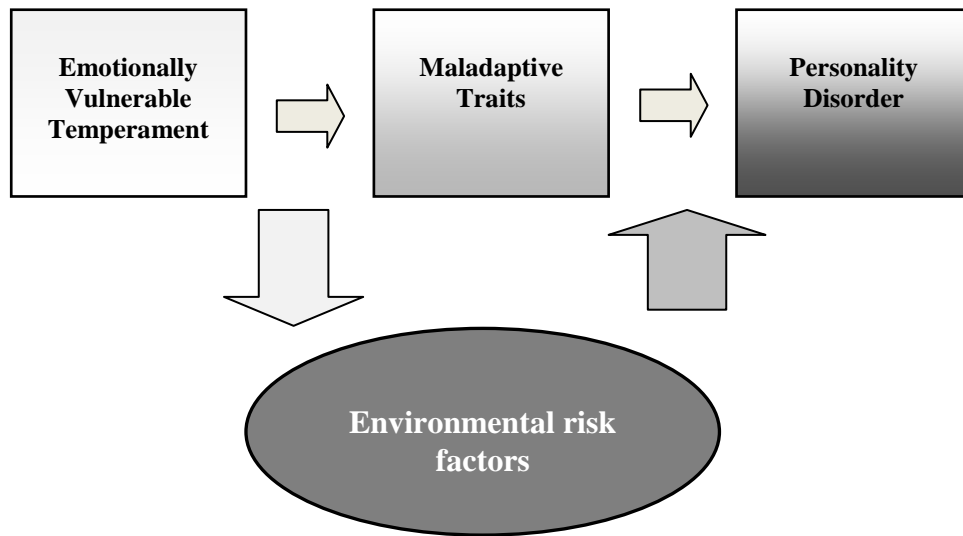
Due to the complexity of BPD, which is associated with multiple comorbid conditions; it may be more tenable to uncover genetic correlations with related personality dimensions (Emmelkamp & Kamphuis, 2007; Lis, Greenfield, Henry, Guile, & Dougherty, 2007; Siever & Davis, 1991). Congruent with the biosocial developmental model of BPD, researchers have focused on *impulsivity* and *emotional dysregulation* as core trait dimensions (Coccaro, Bergeman, Kavoussi, & Seroczynski, 1997; Livesley, Jang, & Vernon, 1998; Paris, 2003; Siever & Davis, 1991; Trull et al., 2000). Familial studies reveal strong family aggregation of both impulsive and mood disorders, among those diagnosed with BPD (White et al., 2003). Further, literature pertaining to externalising psychopathology points towards strong heritability of impulsivity and aggression (Coccaro et al., 1997; Hinshaw, 2002; Krueger et al., 2002), though expression appears strongly

linked to environmental opportunity (Jaffe et al., 2005). Similarly, research suggests that emotional dysregulation is also heritable (Livesley et al., 1998; Torgersen, 1984), though the process of development appears complex. Theorists suggest that, given repeated negative reinforcement, emotional dysregulation may overlay temperamental impulsivity, heightening sensitivity to environmental stress (Crowell et al., 2009).

2.4.2.2 *Temperament*

As discussed, the majority of aetiological models identify an *emotionally vulnerable temperament* as an important risk factor in the development of BPD. Temperament refers to an *inherited* profile, marked by distinctive behavioural and affective styles (Kagan, 1994). Theorists suggest that emotional vulnerability is characterised by: emotional sensitivity, emotional reactivity, and slow return to baseline arousal (Fruzzetti et al., 2005; Linehan, 1993; Putnam & Silk, 2005). Innate temperament is believed to interact with future environmental perturbations creating exaggerated traits, which may become PDs if significant dysfunction ensues (see **Figure 2.2**). Importantly, it is temperamental variation which may explain why a wide range of PDs are associated with similar adversities (Paris, 2003). A “difficult” temperament leads to greater sensitivity to environmental risk factors, more traumatic events, and more negative interactions with others (Paris, 2003); all of which may create negative feedback loops, potentially spiralling out of control.

Figure 2.2 *The Development from Temperament to Personality Disorder*



While researchers have not investigated the predictive role of temperament in the development of BPD specifically, longitudinal designs suggest that temperament in childhood may predict adult psychopathology (Caspi, Moffitt, Newman, & Silva, 1996; Stevensen & Goodman, 2001; Tremblay, Pihl, Vitaro, & Dobkin, 1994). For example, Caspi and colleagues (1996) found that individuals classified as *under controlled* or *uninhibited* at 3 years of age were more likely to meet diagnostic criteria for antisocial personality disorder (ASPD) at 21 years. These children were described as irritable, impulsive and labile in their emotional responses.

Although not specific to BPD, the above studies provide a useful model for exploring the role of temperament in the aetiology of borderline pathology. Indeed, Paris (1997) suggests that BPD and ASPD have a common base in impulsive personality traits, but that gender leads to differential expression of

these traits (Paris, 2003). While males are more likely to turn their aggression outwards, resulting in antisocial behaviour, females are more likely to turn aggression inwards, resulting in the self-harm behaviours observed in BPD.

2.5 Conclusions and Future Directions

2.5.1 Summary

As the above overview reveals, existing research and theory converge on a stress-diathesis model for the aetiology of BPD. Stressors are identified as childhood maltreatment, parental psychopathology, attachment disturbances, invalidating environment and socio-cultural correlates. Diathesis has been recognised as temperamental predisposition, which is underpinned by genetic substrates, and subject to experiential influences. In summary, temperamental traits are magnified by environmental events, leading to a collection of maladaptive traits, and potentially a clinically diagnosed personality disorder (Paris, 2003).

While empirical support for identified environmental stressors is abundant, conceptual and methodological issues cast doubt over conclusions drawn from these studies. The relationship between childhood abuse and BPD is not straightforward. Lack of specificity, and the confounding of abuse with other correlated factors, e.g. family adversity, precludes delineation of direct causal relationships. Further, an over-reliance on retrospective designs has potentially led to an inflation of the supposed aetiological role of trauma and abuse.

2.5.2 A Refined Approach to Aetiological Theory and Research

Due to methodological weaknesses and outdated theoretical notions, our understanding of the development of BPD has met an impasse. This necessitates a modified approach, in which individual development is considered from the earliest years of life (conception), taking into account key developmental milestones along the trajectory towards adulthood. This approach underpins the biosocial developmental model (BDM) of BPD, which extends existing stress-diathesis theories by incorporating a consideration of risk *across* development (Ingram & Price, 2001).

According to the BDM, BPD is the outcome of multiple interacting factors, causal events and dynamic processes; which by adolescence, have become a constellation of identifiable features and maladaptive coping strategies, indicating heightened risk for later BPD. Although the BDM appears a promising theoretical approach, it has yet to be tested longitudinally (Crowell et al., 2009). Therefore, prospective studies are required to assess the associations between psychosocial risk factors and a BPD phenotype along the developmental trajectory. The rationale behind this approach will be explored in the following chapter.

Chapter Three: A Developmental Approach to Aetiological Theory and Research

Overview: As the preceding chapter has reviewed, there is a large body of research assessing potential risk factors implicated in the development of BPD. A number of outstanding questions remain, however. Uncertainty pertaining to issues of cause and effect, and a frustrating lack of specificity, surround current research findings. This signals the need for a new developmental approach, both theoretically and methodologically, in which the unfolding life trajectory of the individual is considered. The following chapter will discuss how this approach may be operationalised through the identification of an intermediate BPD phenotype. Further, unexplored risk factors rendered apparent by the developmental paradigm will be discussed.

3.1 An Intermediate BPD Phenotype

3.1.1 Why Identify an Intermediate Phenotype?

Research to date has mainly focused on adult populations with BPD, providing retrospective reports of childhood experiences. Although this has provided a basis on which to theorise about the potential antecedents of BPD, there are a number of problems inherent in this approach (detailed discussion: chapter two). Consequently, there is an impetus towards new methodology in which populations are followed prospectively along the life trajectory.

To facilitate this approach, the identification of a *BPD phenotype*, i.e. a collection of sub-clinical symptoms, is required, which manifests years before

the onset of the clinically diagnosed disorder. This will make possible repeated assessments of the associations between borderline features and identified risk factors, along the developmental trajectory (Zammit et al., 2008). Further, by identifying sub-clinical symptoms, mental illnesses with relatively low prevalence rates will be identifiable in a reasonable number of cases within *community* populations. By utilising community populations, problems associated with the use of clinical samples, including lack of generalisability, may be avoided (Paris, 2003). There is an existing, small body of work in this vein (see: chapter two, section 2.4.1.1).

3.1.2 Evidence Supporting the Presence of an Intermediate Phenotype

There are various threads of evidence supporting the feasibility of an intermediate BPD phenotype. Firstly, there are existing models of intermediate phenotypes for schizophrenia, antisocial personality disorder (ASPD), and psychopathology generally, i.e. the “dysregulation phenotype.” Secondly, there is a growing body of research supporting the existence of borderline pathology in youth (Chanen, Jovev, McCutcheon., Jackson, & McGorry, 2008; Kernberg, Weiner, & Bardenstein, 2000; Lofgren, Bemporad, King, Lindem, & O’ Driscoll, 1991; Stepp, Pilkonis, Hipwell, Loebar, & Stouthamer-Loeber, 2010).

3.1.2.1 *Intermediate Phenotypes for Other Psychopathologies*

Mental disorders rarely appear in adulthood without warning. There are often signs, sometimes subtle, of dysfunction in childhood or adolescence (Paris, 2003). Existing phenotype models (described below), and associated research

findings, support that sub-clinical features in childhood and adolescence may directly precede specific mental disorders in adulthood. Further, Axis I disorders in youth have been found to predict a range of personality disorders, while a combination of internalising and externalising problems, i.e. a *dysregulation phenotype*, especially portends future mental health problems (Althoff, Verhulst, Rettew, Hudziak & van der Ende, 2010; Meyer et al., 2008).

3.1.2.1. a *The Psychosis Continuum*

The recognition that psychosis exists along a continuum, with a significant proportion of the population experiencing sub-clinical psychotic symptoms (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994; Crow, 1986; Meehl, 1962), has led to the identification of a *psychosis phenotype*. Converging findings, as clearly delineated in a review by van Os, Linscott, Myin-Germeys, Delespaul, and Krabbendam (2009), support this contention.

Firstly, statistical simulations support that the most likely distribution for multi-factorial, psychiatric disorders, including psychosis, is “half normal” (van Os et al., 2009). While diseases caused by a single dominant gene defect may exist as truly dichotomous disorders with bimodal distributions, disorders subject to various environmental influences are likely continuous in distribution. The majority of the population will have very low values, and a significant proportion will display progressively higher values.

Secondly, the psychosis phenotype appears to display a degree of “psychopathological validity” (van Os et al., 2009). Sub-clinical psychosis symptoms have similar comorbidity patterns to those observed in clinical psychosis (van Os, Hanssen, Bijl, & Ravelli, 2000). Additionally, sub- and clinical psychosis tend to display a similar pattern of highly correlated positive and negative symptoms (van Os et al., 2000).

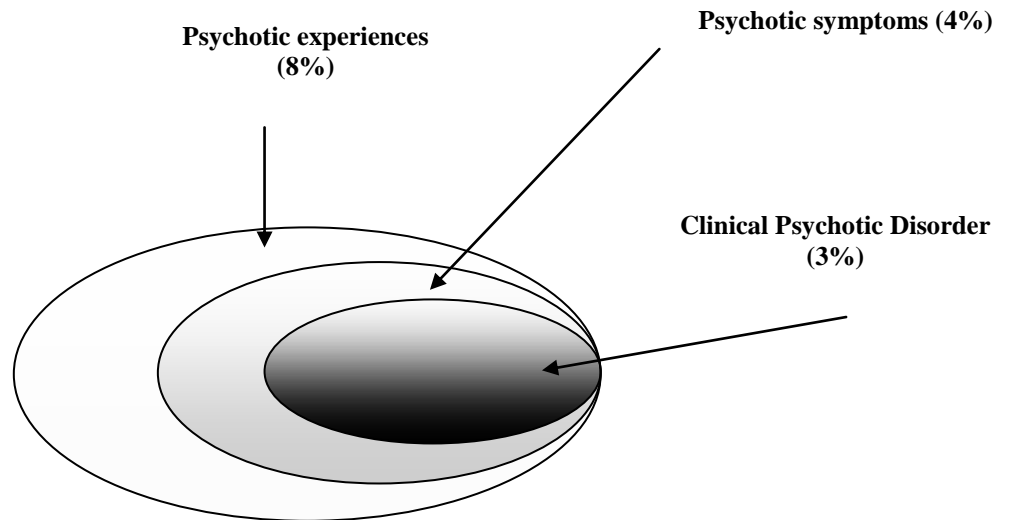
Thirdly, there is evidence that sub-clinical psychosis demonstrates a degree of “epidemiological validity” (van Os et al., 2009). In a meta-analysis of 47 studies, van Os and colleagues (2009) found that the prevalence, and annual incidence rates, of sub-clinical psychosis were much higher than those of the clinical phenotype. This is congruent with a continuum model (as discussed), which accommodates high prevalence rates of psychosis-like symptoms.

Fourthly, individuals with psychosis symptoms and psychotic disorders appear to share similar demographic correlates. van Os and colleagues (2000) found that sub-clinical and clinical psychosis are more common amongst males, migrants, ethnic minorities and the unemployed. Congruent with these observations, sub-clinical and clinical psychosis appear to share associated risk factors, including: psychoactive drug use, traumatic experiences and urbanicity; suggesting an aetiological continuity between sub-clinical and clinical psychosis phenotypes (van Os et al., 2009). It should be noted, however, that these observations have not been supported by all studies; Zammit and colleagues (2008) failed to find evidence for similar patterns of risk factors between psychosis like symptoms observed in children (PLIKS) and schizophrenia.

Finally, sub-clinical psychosis appears to demonstrate “predictive validity” over time, with transitions from sub-clinical to clinical psychosis observed in a reasonable percentage of cases. For example, Chapman *et al.* (1994) found that youngsters who rated high on scales of magical ideation (i.e. delusions) and perceptual aberration (i.e. hallucinations) manifested high rates of psychotic outcomes 10 years later. Similarly, 25 % of children with psychotic experiences at age 11 had developed schizophreniform disorder by age 26 (Poulton *et al.*, 2000). Further, Hanssen, Bak, Bijl, Vollebergh, and Van Os, (2005) found that the 2 year transition rate from sub-clinical to clinical psychotic disorder was 8%, representing a 60-fold increase in risk for those evincing sub-clinical features.

The evidence outlined above has led to the developmental *psychosis proneness-persistence-impairment model* (van Os *et al.*, 2009), which describes the transition from psychotic symptoms to full-blown psychotic disorder. The model posits that most sub-clinical psychotic experiences (75-90%) are transitory; however, for a substantial percentage of individuals psychotic symptoms will persist, and become clinically relevant (representing approximately 4% of the population). Within this 4%, who may experience a degree of distress and help seeking behaviour, a subset, of approximately 3% will eventually develop a true psychotic disorder (see **Figure 3.1**). In summary, there will be a significant proportion of the population genetically vulnerable to psychotic experiences, yet only a percentage of these individuals will subsequently develop clinical psychosis, contingent on exposure to environmental risk.

Figure 3.1 *The Continuum of Psychosis (van Os et al., 2009)*



Developmental researchers have studied psychosis symptoms in child populations (Poulton et al., 2000; Schreier et al., 2009; Zammit et al., 2008), in an attempt to elucidate the aetiological mechanisms underlying schizophrenia. By repeatedly assessing symptoms over time, the developmental trajectories leading to schizophrenia may be traced, facilitating the identification of developmentally relevant risk and protective factors (Zammit et al., 2008).

3.1.2.1. b *Life-Course-Persistent Antisocial Behaviour*

The Diagnostic Statistical Manual (DSM) presents strict criteria (i.e. symptoms must be present for over one year), discouraging the diagnoses of personality disorders (PDs) before the age of 18. This reflects a general reluctance to diagnose PDs in childhood or adolescence, due to associated stigma and the assumed malleability of personality in youth (Meekings and O'Brien, 2004).

Antisocial personality disorder (ASPD) is unique as the only PD with an officially recognised direct childhood precursor. Conduct Disorder (CD) is situated on Axis I of the DSM, thereby defining it as a state, rather than trait, disorder. The CD-ASPD model is underpinned by the methodologically sound, seminal research of Robins (1966, 1978). In a series of studies, an association between conduct problems in youth and the development of ASPD in adulthood was revealed. More importantly, antisocial adults *always* had prior history of CD, making CD in childhood a *necessary* criterion for a diagnosis of ASPD within the DSM.

While a diagnosis of CD is required for the subsequent diagnosis of ASPD, not all children manifesting conduct problems will later develop ASPD. Therefore, a sub grouping of *life-course-persistent antisocial behaviour* has been delineated. This grouping refers to individuals who engage in some form of antisocial behaviour at every stage of the lifespan (Moffitt & Caspi, 2001; Raine et al., 2005), and explains the observed continuity between CD and ASPD.

In common with the psychosis continuum model, while a substantial proportion of those with relevant symptoms will eventually develop the associated adult disorder, the majority will not. In the case of CD, it appears that temperamental predisposition determines the later manifestation of ASPD. Those exhibiting symptoms strongly rooted in temperament (Paris, 2003), and exposed to an environment fostering under control, are liable to manifest antisocial features across all developmental stages (Moffit, 1993).

The CD-ASPD model may be especially relevant when considering the development of BPD. Researchers have suggested that BPD is the female “mirror image” of ASPD. While both disorders share a common impulsive dimension, they are characterised by different behavioural manifestations, possibly due to gender typical development (Paris, 1997). As BPD and ASPD share a number of commonalities, including associated risk factors and reported family histories, they may also share common precursors, albeit gender biased (Paris, 2003).

3.1.2.1. c Axis I Disorders (Externalising and Internalising) as Intermediate Phenotypes of Personality Disorder

A large body of research confirms the association between Axis I disorders in childhood and the development of personality disorders in adulthood.

Externalising disorders, often considered as a singular group of *disruptive disorders* [Conduct Disorder (CD); Attention Deficit Hyperactivity Disorder (ADHD); Oppositional Defiant Disorder (ODD)], appear strongly linked to adult

PDs in both clinical and community samples. Internalising disorders (anxiety and depression) are also predictive of adult PDs, though to a lesser extent.

Clinical Samples

Externalising and Internalising Disorders

Rey, Morris-Yates, Singh, Andrews, and Stewart (1995) interviewed 145 young adults (mean age 19.6 years), who were diagnosed with a variety of emotional and disruptive disorders during adolescence (mean age 13.7 years). Using the Personality Disorder Examination (PDE), they found that 40% of subjects with disruptive disorders, and 12% of subjects with emotional disorders in adolescence had personality disorders in young adulthood. Of note, a specific association between ADHD and subsequent BPD was found (see later discussion).

Helgeland, Kjelsberg, and Torgersen (2005) used a quasi-prospective study to investigate the continuity between emotional and disruptive disorders in adolescence and personality disorders in adulthood. One hundred and thirty participants diagnosed with emotional or disruptive disorders in adolescence (mean age 14.6 years), were interviewed 28 years later (mean age 43.2 years). They found that 64.7% of adolescents with disruptive disorders, and 57.8% of adolescents with emotional disorders had PDs at follow up.

Attention Deficit Hyperactivity Disorder (ADHD)

PDs and ADHD tend to share deviant patterns of behaviour in cognition, affectivity, interpersonal functioning and impulse control. BPD and ADHD, for example, share features of impulsivity, emotional dysregulation and cognitive impairment (Philipsen, 2006). Subsequently, ADHD has been considered specifically, as a childhood precursor to later PD (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998).

Mannuzza and colleagues found that boys clinically diagnosed with ADHD in childhood (mean age 7.3 years) were more likely to be diagnosed with ASPD in adulthood (mean age 24.1 years). Similarly, compared to 71 community control children, 147 hyperactive children (aged 4-12 years) evinced higher levels of histrionic, passive aggressive, antisocial and borderline PDs when re-assessed in adulthood (mean age 20-21 years) (Fischer, Barkley, Lori Smallish, & Fletcher, 2002).

More specifically, Fossati, Novella, Donati, Donini, and Maffei, (2002) assessed the association between a childhood history of ADHD and BPD in adulthood. Four groups (BPD patients; any cluster B* PD controls; cluster A* or C* PD controls; and non-clinical controls) were compared according to responses on the Wender Utah Rating Scale (WURS): a 25-item, self-report instrument designed to retrospectively assess childhood ADHD symptoms. The BPD group had significantly higher mean WURS total scores than all other control groups.

*Cluster A: Paranoid, Schizotypal, Schizoid; Cluster B: Antisocial, Histrionic, Paranoid; Cluster C: Obsessive-Compulsive, Dependent, Avoidant.

Further, when the WURS score was dichotomised, approximately 60% of the BPD patients scored above the cut-off point, suggestive of a probable ADHD diagnosis in childhood. This percentage was significantly larger than all other control groups, supporting that ADHD in childhood is especially predictive of BPD in adulthood. These results should be considered with caution, however, due to the retrospective nature of the study design.

Community Samples

In a prospective, longitudinal study, Lewinsohn, Rohde, Seeley and Klein, (1997) screened for Axis I disorders in 299 adolescents at two time points (14 through 18 years of age). The sample was assessed again at age 24, for the presence of Axis I and Axis II psychopathology. The occurrence of all four Axis I disorders (major depression, anxiety, disruptive behaviour disorders, and substance use disorders) in adolescence were associated with elevated PD *dimensional* scores; the association for disruptive behaviour disorders being particularly strong.

In another community study, Kasen, Cohen, Skodol, Johnson, and Brook (1999) obtained prospective assessments from 551 youths and their mothers, at three time points (mean ages: 12.7; 15.2; and 21.1 years). They found that disruptive disorders increased the odds of adult cluster A, B and C PDs fourfold, while depression increased the odds of cluster B PDs six fold, and cluster C PDs eight fold. The latter finding is consistent with meta-analyses revealing that childhood

depression is associated with a number of negative sequelae, including conduct problems and personality pathology (Birmaher et al., 1996).

Temporal Ordering of Axis I and Personality Disorders?

The above studies suggest that Axis I disorders precede PDs. Crawford, Cohen, and Brook (2001a; 2001b), however, have tested the predictive association between personality pathology and subsequent Axis I disorders.

Using a prospective, cross-lagged, longitudinal design, with a sample of 407 community adolescents, mental disorder was assessed at three time points: 1983 (10-14 years), 1985-6 (12-17 years), and 1991-3 (17-24 years). Cluster B personality disorder symptoms were determined using symptom scales for histrionic, borderline and narcissistic PDs (Bernstein et al., 1993). Internalising disorders were assessed using the *Diagnostic Interview Schedule for Children (DISC)* depression and overanxious scales (Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982), and the *Symptom Check List (SCL)* depression scale (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). Externalising disorders were assessed using the DISC oppositional defiant, conduct disorder, and attention deficit hyperactivity scales (Costello et al., 1982).

While girls evinced strong associations between Cluster B and both externalising and internalising symptoms; strong associations between Cluster B and externalising symptoms, only, were observed in boys. The authors hypothesise that girls may be prone to suffer concurrent internalising disorders, as they are

more likely distressed by PD-related disruptions in interpersonal relationships (Crawford et al., 2001a).

The temporal ordering of the observed associations was assessed using a cross-lagged experimental design (Crawford et al., 2001b). The comorbidity of Axis I and Axis II disorders was tracked over time to ascertain whether Axis I disorders lead to personality pathology (*complication model*) (Dolan-Sewell, Krueger, & Shea, 2001), or Cluster B symptoms predict Axis I disorders (*predisposition model*) (Dolan-Sewell et al., 2001).

Again gender differences emerged. For girls, consistent with the complication theory, model fit significantly improved with the addition of lagged paths from both (internalising and externalising) Axis I symptom clusters to subsequent Cluster B PD symptoms. While internalising symptoms at 10 to 14 years predicted Cluster B PD symptoms in mid adolescence, internalising symptoms at 12 to 17 years did not predict cluster B PD symptoms at age 17 to 24. Therefore, the disruptive effects of internalising symptoms may be limited to “local” experiences, occurring during the sensitive period of transition into adolescence (Crawford et al., 2001b).

Girls’ externalising symptoms during mid-adolescence (12 to 17) predicted Cluster B disturbances at 17-24. Of note, antisocial personality disorder symptoms were not included in the cluster B assessment, so this association cannot be attributed to a continuance from externalising symptoms, i.e. conduct problems, to antisocial PD in early adulthood. Interestingly, an association

between antisocial behaviour in female adolescents and subsequent BPD in adulthood has previously been reported (Goodman, Hull, Clarkin, & Yeomans, 1999). This may reflect gender typical development, in which antisocial behaviour in females becomes less common during adulthood, and is directed inwards, manifesting as borderline or histrionic symptoms.

For girls and boys, consistent with the predisposition model, a pathway from Cluster B symptoms at ages 10 to 14 to externalising symptoms two years later was observed. If Cluster B symptoms are organised around a novelty seeking temperament, then disruptive behaviour may be a maladaptive expression of this personality style. Conversely, internalising symptoms were independent of the course of Cluster B symptoms over the same time period, possibly due to the fact that mood and anxiety states are not ubiquitously associated with novelty seeking temperament (Mulder, Joyce, & Cloninger, 1994).

While these results confirm associations between externalising and internalising disorders and cluster B PDs, the temporal ordering of these disorders requires further clarification. Both the predisposition and complication models were partially supported, and there were gender-specific developmental effects. Due to the small sample size, however, three PDs were conflated. Although these PDs belong to the same *DSM* cluster group (but see section 1.2), they represent separate diagnostic entities, with possible gender biases and differential developmental trajectories. Therefore, the true associations between Axis I disorders and specific PDs remain unclear.

Considering the above findings, it seems likely that Axis I disorders act as a behavioural marker or intermediate phenotype for ensuing PDs, and/or contribute towards the shaping of these maladaptive personality traits (Dolan-Sewell et al., 2001). Further exploration of these relationships may help clarify the gender typical patterning of ASPD and BPD, and further specify the pathways towards BPD.

3.1.2.1. d A “Dysregulation” Phenotype

While Axis I disorders appear to act as behavioural markers for burgeoning personality pathology, associations lack specificity. The observed multifinality of outcome may be explained by the existence of a “*dysregulation phenotype*,” which has been operationalised as a combination of externalising (attention and conduct) and internalising (emotionality) symptoms (Althoff et al., 2010). This dysregulation constellation is predictive of a number of negative outcomes, including PDs (Althoff et al, 2010; Biederman et al., 2009; Brotman et al., 2006; Meyer et al., 2008), and appears to be relatively stable over time (Ayer et al., 2009; Biederman et al., 2009). Therefore, it seems likely that behavioural dysregulation is a marker for an underlying dysregulated trait, implicated in the development of BPD (Crowell et al., 2009; Linehan, 1993).

To improve the predictive specificity of the dysregulation phenotype, future studies should assess the developmental trajectory of dysregulation, along with pertinent environmental correlates, *prior* to adult diagnoses. This will facilitate discrimination between various psychopathologies before they become fully

canalised (Crowell et al., 2009; Geiger & Crick, 2001), and antecedents become difficult to disentangle (Bradley et al., 2005).

3.1.2.2 Support for an Intermediate Borderline Phenotype

There is growing support for the presence of an intermediate borderline phenotype, identifiable in youth before the age of 18 (Bemporad, Smith, Hanson, & Cicchetti, 1982; Chanen et al., 2008; Guzder, Paris, Zelkowitz, & Marchessault, 1996; Lofgren, Bemporad, King, Lindem & O' Driscoll, 1991; Paris, Zelkowitz, & Feldman, 1999; Stepp et al., 2010). The early conceptualisations of “borderline” pathology in youth were overly broad. In a follow-up study, “borderline” children (see Bemporad et al., 1982) were found to develop a variety of PDs in later years (Lofgren et al., 1991). Subsequently, the original borderline nomenclature was reconceptualised into the broader category of “multiple complex developmental disorder” (Ad-Dab'bagh & Greenfield, 2001), and a refined BPD diagnostic tool for children: *the Child Version of the Retrospective Diagnostic Interview for Borderlines (C-DIB-R)*, was developed (see Greenman, Gunderson, Cane, & Saltzman, 1986).

3.1.2.2. a Studies Assessing Borderline Pathology in Childhood

A small body of research has utilised the *C-DIB-R* (Guzder et al., 1996; 1999; Paris et al., 1999) to assess which risk factors are associated with BPD diagnosed in childhood. In the first study, a clinical group of 98 children, aged 7 to 12 years (79 boys and 19 girls), were divided into borderline (41) and non-borderline (57).

While both groups demonstrated severe functional impairment, risk factors differentiating the borderline group were: sexual abuse, physical abuse, severe neglect, and parental substance abuse and criminality; all of which have been commonly reported by adults with BPD (Guzder et al., 1996).

In the second study, a clinical group of 94 children (81 boys and 13 girls) aged 7 to 12 years, were assessed using a cross-sectional version of the *C-DIB-R*.

Similar risk factors: physical abuse, sexual abuse, severe neglect, and parental criminality, distinguished the borderline from non-borderline groups (Guzder et al., 1999).

In the third study, a clinical grouping of 89 children (76 boys, 13 girls, aged 7 to 12) was divided into borderline (38) and non-borderline (51) using the *C-DIB-R*, and compared on neuropsychological performance (Paris et al., 1999). The borderline children evinced problems with executive functioning, comparable to the neurological deficits observed in borderline adults.

The above findings that similar risk factors are associated with borderline pathology in child and adult populations, lend “aetiological validity” (van Os et al., 2009) to the borderline construct, as measured by the *C-DIB-R*. The samples utilised were gender biased, however, casting doubt over the generalisability of these findings. While there is a predominance of females with BPD in adult clinical populations (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004) the samples in these studies were male biased.

Additional support for borderline pathology in childhood is provided by a recent study carried out by Stepp and colleagues (2010). Using factor analysis, constructs underlying the development of BPD in girls aged five to eight were assessed. The authors concluded that the underlying features of BPD in adults, conceptualised as impulsivity, negative affectivity and interpersonal aggression (Gunderson, 2007; Skodol et al., 2002) can be reliably measured in 6-12 year old girls, and that each underlying feature demonstrates a degree of stability over time.

Finally, research suggests that adolescents with BPD features may evince measurable physiological abnormalities. For example, teenage girls (age 14 to 19 years) with BPD features exhibited abnormal brain maturation, as reflected in P300 amplitude measures (for more details see Houston, Ceballos, Hesselbrock, & Bauer, 2005). Physiological studies in this vein may offer future promise in resolving clinical controversies (Houston et al., 2005) by providing converging evidence for the presence of BPD pathology in adolescence.

3.1.2.2. b Existing BPD Symptom Scales

Progress in understanding the precipitants of BPD has been delayed by a lack of validated assessment tools for children and adolescents. In recognition of the importance, for both theoretical and intervention purposes, of defining BPD in youth, new assessment tools have been developed (Crick et al., 2005; Rogosch & Cicchetti, 2005).

The Borderline Personality Features Scale for Children (BPFS-C) (Crick et al., 2005)

Crick and colleagues (2005) have devised a dimensional scale for the identification of borderline features in non-clinical children. The scale is a modified version of the borderline (BOR) section of the Personality Assessment Inventory (PAI) (see Morey 1991), which was designed to assess borderline personality features in adults. Modification was based on a consideration of age appropriate features in four areas: affective instability, interpersonal problems, suicide/ self-harm and identity problems (see **Table 3.1** below for sample items).

The construct validity of the *BPFS-C* scale was tested in a short term, longitudinal study over one year. Borderline features were assessed at three time points: spring year 1, autumn year 1, and autumn year 2, using a sample of 400 children (54% female) aged 10 to 12 years.

Three findings supported the construct validity of the *BPFS-C* scale. Firstly, *BPFS-C* scores significantly “tracked” with theoretically driven childhood indicators of borderline features over time, including: cognitive sensitivity, emotional sensitivity, friend exclusivity and aggression (see Geiger and Crick, 2001). Secondly, *BPFS-C* scores remained moderately stable across the three time-points, suggesting that, although BPD assessed during late childhood is not entirely rigid, some degree of crystallisation appears to occur prior to adulthood. Finally, borderline personality features remained associated with childhood indicators of BPD after controlling for depressive symptoms, supporting that the

BPFS-C is predictive of borderline indicators specifically, rather than psychopathology generally.

Although this study offers tentative support for the use of *dimensional* measures in the assessment of borderline features during childhood, further investigations over longer time spans and across broader age ranges are required. Crucially, prospective, longitudinal studies are necessary to assess the specificity of childhood borderline features in predicting adult BPD verses other types of psychopathology (Crick et al., 2005). The use of dimensional scales here was necessary because of the limited sample size. Due to the low base rate of BPD in normative samples, categorical assessment would render a very small number of borderline cases, precluding statistical analysis.

The Borderline Precursors Composite (Rogosch & Cicchetti, 2005)

The borderline precursors composite (Rogosch & Cicchetti, 2005) comprises assessments from a number of areas including: personality features, representational models of self, parent and peers, interpersonal relationship difficulties and suicidal/self-harm behaviour (see **Table 3.1** below for details).

Rogosch and Cicchetti collated a variety of assessments from self, peer and counsellor report. The precursor scores of 185 maltreated and 175 non-maltreated children (51.4% boys, ages 6 to 12) were compared during a week-long summer camp research programme. Maltreated children were chosen for two reasons. Firstly, a link between childhood maltreatment and BPD is

established (Battle et al., 2004; Zanarini et al., 1997), and these two groups share similar deficits in functioning, including increased suicidality and relationship disturbance (Rogosch & Cicchetti, 2005). Secondly, due to the low prevalence of BPD in community populations, use of high-risk populations ensures the identification of an adequate number of BPD cases.

The majority of indicators comprising the borderline precursors composite distinguished maltreated from non-maltreated children, including: personality features (lability, reversed conscientiousness), interpersonal problems (conflicted relationships, upsets other, disliked, relational aggression), and self-harm. Further, maltreated children evinced significantly higher *total borderline precursors composite scores*, and significantly more children in the maltreated group were categorised into the high BPD precursors (had scores greater than 1 SD above the mean) group. Thus, the borderline precursors composite proved successful in distinguishing maltreated from non-maltreated children, supporting the discriminative validity of this scale.

These results provide tentative support for a prospective pathway from childhood maltreatment to borderline features in childhood, suggesting that a subgroup of maltreated children is at risk for the later emergence of BPD features.

Nevertheless, BPD is not always synonymous with childhood abuse (see chapter two), and emotional abuse singularly did not distinguish maltreated from non-maltreated children.

Table 3.1 A Comparison of the BPFS-C and the Borderline Precursors Composite

Features	Scale	Assessment	Respondent	Sample Item
<u>Affective Instability</u>	BPFS-C	BPFS-C	Child	“My feelings are very strong. When I get mad, I get very mad”
	Borderline Precursors Composite	-Emotion regulation checklist (ERC) (Shields & Cicchetti, 1997) -California Child Q Set (CCQ) (John et al., 1994)	Counsellor Counsellor	“Exhibits wide mood swings” Conscientious sub section
<u>Interpersonal Problems</u>	BPFS-C	BPFS-C	Child	“I have picked friends who have treated me badly”
	Borderline Precursors Composite	-Student-Teacher relationship scale (STRS) (Pianta & Steinberg, 1992) -Peer sociometric ratings	Counsellor Peers	“This child and I always seem to be struggling with one another” “Child upsets everyone, wants everyone to do things his\her way”
		-Relationship Stance Questionnaire (RSQ)	Child	Vignettes of stressful situations are presented to the child: test for preoccupied or non-preoccupied response
<u>Suicidal/ Self Harm</u>	BPFS-C	BPFS-C	Child	“I get into trouble because I do things without thinking”
	Borderline Precursors Composite	-Teacher Report Form (TRF) (Achenbach, 1991) -Children’s Depression Inventory (CDI) (Kovacs, 1992)	Counsellor Child	“Deliberately harms self or commits suicide” Suicidal Ideation
<u>Identity problems</u>	BPFS-C	BPFS-C	Child	“I feel that something important is missing about me but I don’t know what it is”
	Borderline Precursors Composite	Not included		
<u>Representational models</u>	BPFS-C	Not included		
	Borderline Precursors Composite	-Perceptions of peers and self (POPS) (Rudolph et al. 1995)	Child	“Other Kids will try and tease you or put you down if they have the chance”

This suggests that severity and cumulative effects are more important than presence of abuse per se. Further, the cross sectional design of this study precludes confirmation of the reliability and predictive qualities of the precursor composite.

3.2 Conjectured Developmental Risk Factors for the Aetiology of BPD

Chapter two described risk factors integrated into current theories for the aetiology of BPD. While many of these risk factors have empirical support, a lack of specificity suggests that additional antecedents could be involved in the development of BPD. Potential antecedents receiving scant attention to date include peer relationships, in particular peer victimisation, and exposure to domestic violence. The following discussion will briefly outline existing evidence linking these factors to BPD or other psychopathologies.

3.2.1 Peer relationships

There are very few existing studies considering the role of peer relationships in the development of BPD (Crick et al., 2005; Geiger & Crick, 2001; Rogosch & Cicchetti, 2005). With a shift towards the developmental paradigm, however, the role of peer relationships should be considered. While parental relationships are most influential early in development (Sroufe et al., 2010), peer relationships become increasingly salient as the child approaches adolescence (Elicker & Englund, 1992).

Peer relationships likely operate as both markers, e.g. *relational aggression/ overly close relationships*, and risk factors, e.g. *bully victimisation*, for borderline pathology. Relational aggression may develop from a tendency towards overly close relationships and attachment issues (e.g. hypermentalisation), in individuals with borderline features (Werner & Crick, 1999). Individuals with this profile are especially sensitive to perceived slights, reacting with intense emotions, subsequently harming idealised relationships.

Crick *et al.* (2005) in a short-term, longitudinal study assessed overly close relationships in terms of *friend exclusivity* and *relational aggression*, ascertained by questions such as: “It bothers me if my friend hangs out with other kids even if I’m busy.” Self-reported friend exclusivity and teacher-reported relational aggression were both associated with borderline features. This is congruent with previous research revealing similarities between relational aggression and borderline pathology, including: jealousy, enmeshment, and manipulation for control (Grotmeter & Crick, 1996).

The consequences of bully victimisation have been considered in reference to a variety of mental health outcomes (Arsenault *et al.*, 2006; Kaminski & Fang, 2009; Schreier *et al.*, 2009). Bullying has been found to predict suicide ideation (Kaminski & Fang, 2009), psychotic symptoms (Arseneault *et al.*, 2011; Schreier *et al.*, 2009) and neurobiological changes in the brain (Teicher *et al.*, 2010a, b), all of which are common features of BPD. Nevertheless, the association between peer victimisation and BPD has yet to be tested. This is surprising considering the core relationship disturbances associated with BPD, and the predominance of

reported trauma experiences in the histories of borderline patients (Zanarini et al., 1997).

3.2.2 Domestic Violence/ Parental Conflict

Exposure to domestic violence may be one deleterious factor encountered, especially when growing up within an adverse family environment. Retrospectively, borderline patients tend to report higher levels of exposure to domestic violence compared to non-borderline groups (Herman et al., 1989; Weaver & Clum, 1993). While studies prospectively considering the association between exposure to domestic violence and subsequent BPD are non-existent, meta-analyses reveal links between domestic violence and internalising/ externalising disorders and Post-Traumatic Stress Disorder (PTSD) (Evans, Davies, & DiLillo, 2008; Kitzmann, Holt, & Kenney, 2003; Wolfe Crooks, Lee, McIntyre-Smith, & Jaffe, 2003). This suggests a possible aetiological association with BPD related symptoms.

Exposure to domestic violence may be viewed as one of many associated risk factors for BPD (Bradley et al., 2005), especially in instances of extreme, profound violence, which could lead to trauma response in the developing child.

3.3 Conclusions

As summarised (see **Table 3.2** below), there are a number of risk factors implicated in the development of BPD, identified either through specific links

with BPD, or through prospective links with related psychopathologies. In order to advance current knowledge, prospective, longitudinal designs are required; which consider a broad range of factors across development. Consideration of these factors, within a developmental framework, will facilitate the identification of antecedents when they are aetiologically active; thereby aiding differentiation of psychopathologies as they develop (Geiger & Crick, 2001).

Table 3.2 Empirical Evidence for Established and Conjectured Antecedents in the Development of BPD

Factors across lifespan	Sub factors	Retrospective studies of BPD	Pseudo\ Prospective studies of BPD	Prospective links to other psychopathologies
Temperament	<u>“Under controlled”</u>	N/A	N/A	<u>Antisocial PD:</u> Caspi et al. 1996 <u>Behavioural/Externalising Problems:</u> Stevensen & Goodman 2001
*See chapter eight	<u>“Behavioural Dysregulation”</u>	N/A	N/A	<u>Bipolar Disorder:</u> Meyer et al. 2009; Bierderman et al. 2009; <u>Anxiety/Depression:</u> Meyer et al. 2009; Althoff et al. 2010; Bierderman et al. 2009 <u>Disruptive Disorders:</u> Althoff et al. 2010; Bierderman et al. 2009; Meyer et al. 2009
Axis I Disorders	<u>Externalising/internalising disorders</u>	Fossati et al 2002	Lewinsohn et al., 1997	<u>Cluster B PDs:</u> Crawford et al 2001a; 2001b <u>Cluster A,B,C PDs:</u> Kasen et al. 1999; Lewinsohn et al. 1997 <u>Cluster B PDs:</u> Meyer et al. 2009
*See chapter six				
Family environment	<u>Sexual/Physical Abuse</u>	Paris et al. 1994a, b; Waller, 1994; Yen et al. 2002; Zanarini et al. 1997; Zerkowitz et al. 2001; Bandelow et al. 2005	Johnson et al. 1999 Widom et al 2009 Rogosch & Cicchetti 2005	
	<u>Neglect</u>	Zanarini et al.1997; Links et al.1988, Zweig – Frank & Paris, 1991; Frank & Hoffman, 1986; Gunderson et al. 1980	Johnson et al. 2000 Widom et al 2009	
*See chapter six	<u>Harsh parenting</u>	Zweig-Frank & Paris, 1991	Johnson et al. 2006	
*See chapter six	<u>Exposure to parental conflict/ domestic violence</u>	Weaver & Clum, 1993; Herman et al. 1989		<u>Externalising:</u> Evans et al., 2008 ; Wolfe et al., 2003 <u>Internalising:</u> Evans et al., 2008 ; Wolfe et al., 2003 <u>Trauma symptoms:</u> Evans et al., 2008; Wolfe et al., 2003

Table 3.2 Continued...Empirical Evidence for Established and Conjectured Antecedents in the Development of BPD

Factors across lifespan	Sub factors	Retrospective studies of BPD	Pseudo\ Prospective studies of BPD	<u>Prospective links to other psychopathologies</u>
Peer relationships				
<i>*see chapter seven</i>	<u>Peer bully-victimisation</u> - <i>bully</i> - <i>victim</i> - <i>bully/victim</i>	N/A		<u>Psychosis</u> : Schreier et al. 2009; Arseneault et al. 2011 <u>Suicide ideation</u> : Kaminski & Fang, 2009; Kim et al. 2009 <u>Behavioural problems</u> : Sourander et al. 2007 <u>Anxiety/Depression</u> : Bond et al. 2001

Chapter Four: Research Questions

Overview: The thesis comprises three research studies, following in chapters six, seven and eight. This chapter outlines the rationale behind each of these studies, and summarises the specific research questions to draw together information from the preceding chapters, and present an introduction to the following methodology chapter. It concludes with a brief outline of the main features of the three studies.

The main aim of the thesis was to explore the developmental precursors of Borderline Personality Disorder symptoms, using a prospective, longitudinal cohort design (described in chapter 5). Specific aims of each of the research studies are described below.

4.1 Study one: Prospective study of family adversity and maladaptive parenting in childhood and borderline personality disorder symptoms in a non-clinical population at 11 years

Maladaptive parenting has been consistently reported by adults with BPD in retrospective studies (Links, Steiner, & Huxley, 1988; Paris & Zweig-Frank, 1993; Zanarini, Gunderson, Marino, Schwartz, & Frankenberg, 1989; Zweig-Frank & Paris, 1991). Further, prospective, longitudinal studies have begun to reveal links between exposure to maladaptive parenting in childhood and subsequent BPD symptoms in early adulthood (Johnson et al., 2006). To date, however, prospective studies have not confirmed these links with a collection of clinically relevant BPD symptoms in late childhood/early adolescence.

Research Questions

- Is exposure to maladaptive parenting (harsh parenting and parental conflict), during the preschool and school periods, predictive of BPD symptoms (5 or more) in late childhood/early adolescence?
- Is this association mediated by potential markers: Axis I DSM-IV diagnoses and IQ, at age 7 to 8 years?

4.2 Study Two: Bullied by Peers in Childhood and Borderline Personality Symptoms at 11 Years of Age: A Prospective Study

Despite established links between exposure to bullying and various psychopathologies, there are no studies to date that considered the prospective link between peer victimisation and BPD. Peer victimisation has been linked with BPD-typical features including: suicide ideation (Kaminski & Fang, 2009); psychotic symptoms (Schreier et al., 2009); and neurobiological changes in the brain (Teicher, Samson, Sheu, Polcari & McGreenery, 2010). This suggests that exposure to bullying may be predictive of BPD specifically, which encompasses cognitive, emotional, behavioural, and relational symptoms.

Research Questions

- Is exposure to peer victimisation, in the form of bullying during elementary school, predictive of clinically relevant (5 or more) BPD symptoms in late childhood/early adolescence?

- Is there a dose-response relationship between severe (frequency), combined (overt and relational) and chronic victimisation, and the risk of BPD symptoms?
- Are these associations independent of confounding variables including: IQ; Axis I disorders; maladaptive parenting; and sexual abuse?

4.3 Study Three: Dysregulated Behaviour in Early and Middle Childhood and Borderline Personality Disorder Symptoms at 11 years: A Test of the Biosocial Developmental Model

The third study was designed to test the biosocial developmental model (BDM) of BPD (Crowell et al., 2009) by considering whether a predisposition towards dysregulation in early to middle childhood is potentiated across development by exposure to psychosocial risk factors (harsh parenting or peer victimisation). Further, the specificity of the BDM for predicting BPD symptoms was tested by comparing direct and indirect associations to those observed for psychotic and depression outcomes.

Research Questions

- Do children evince stable dysregulated behaviour between four and eight years of age, thus indicating the presence of an underlying dysregulated behaviour trait?
- Are dysregulated behaviour and psychosocial risk factors (harsh parenting and peer victimisation) predictive of BPD symptoms at 11 years?
- Do psychosocial risk factors add to, or potentiate, dysregulated trait behaviour?

- Are the associations from dysregulated behaviour classes, via environmental risk factors, to BPD outcome stronger than associations to psychotic or depression outcomes?

4.4 Brief Description of Main Features of the Studies

A summary of predictor, control and outcome variables and the statistical methodology used in the three studies is shown in Table 4.1. A more detailed description of the study variables follows in chapter five.

Table 4.1 Summary of the Main Features of the Three Studies

	Study one	Study two	Study three
Outcome variable	BPD symptoms (5+) at age 11.7 years	BPD symptoms (5+) at age 11.7 years	BPD symptoms (5+) at age 11.7 years
Predictor variables	Maladaptive parenting: Harsh parenting and parental conflict	Peer victimisation: Overt and relational; severe, combined and chronic	Dysregulated behaviour trait Harsh parenting Peer victimisation
Statistical analysis	Logistic regression Path analysis	Logistic regression	Latent class growth analysis Path analysis Configural Frequency Analysis
Software	<i>SPSS version 18</i> <i>Mplus version 6</i>	<i>SPSS version 18</i>	<i>SPSS version 18</i> <i>Mplus version 6</i>
Control variables	Family adversity IQ Axis I disorder Gender	Family adversity IQ Axis I disorder Gender Sexual/physical abuse Maladaptive parenting	Family adversity Gender

Chapter Five: Methodology

***Overview:** The purpose of this chapter is to give a broad overview of the Avon Longitudinal Study of Parents and Children (ALSPAC) data resource, in terms of sample characteristics and cohort design, and the thesis design in terms of predictor and outcome variables. The outcome variable is described fully, as it is used in all three subsequent studies. A brief overview of the predictor variables is presented; however, these will be described in more detail in the separate studies.*

5.1 Design of the ALSPAC Cohort

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a geographically defined population study devised to produce detailed data pertaining to the environmental, genetic and psychosocial influences encountered by an individual during development. The geographical study area is 120 miles west of London and borders on the Severn Estuary (see **Figure 5.1**).

Figure 5.1 Map of the Avon area



Data collection, from the mother and father of the study child, began during early pregnancy, producing detailed information from before the child was born. The study child has taken part in a series of clinic days, incorporating physiological and psychological assessments, and has additionally responded to a number of questionnaires during development. Further, questionnaire data from teachers of the study child has been collected, regarding the child and the school environment generally (see **Table 5.1**).

Table 5.1 *Summary of Assessment Tools Used in the ALSPAC Cohort Study*

Respondent	Type of Assessment	Time-point
Mother	Child-based Questionnaires	4 weeks, 6, 15, 18, 24, 30, 38, 42, 54, 57, 65, 69, 78, 81, 91, 103 months, 9, 10, 11, 13 years
	Mother-based Questionnaires	8, 12, 18, 32 weeks (prenatal); 8 weeks, 8, 21, 33, 47, 61, 73, 85, 97, 110 months, 10, 11, 12 years
Partner	Child and Partner-based Questionnaires	12, 18 weeks (prenatal), 8 weeks, 8, 21, 33, 47, 61, 73, 85, 97, 108 months, 10, 11, 12 years
Teacher	Child and School-based Questionnaires	7, 8 & 10 years
Child	Child-based Questionnaires	65, 69, 73, 77, 81, 85, 91, 97, 103, 110, 116, 122, 128 months, 11, 12, 13, 14 years
	Clinic Assessments: Physical & Mental (direct assessments and interviews)	7, 8, 9, 10, 11*, 12, 13 years

*BPD interview took place during this focus clinic

5.1.1 Advantages of the ALSPAC Cohort

As the study is based in one geographical area, linkage to medical and educational records is relatively straightforward, and high quality control is maintained with hands-on assessments of children and parents using local facilities (Golding, Pembrey, Jones, & the ALSPAC Study Team, 2001). Using a total population sample unselected by disease status produces an unbiased sample for data analysis, while frequent contact with mothers and their partners is maintained by sending postal questionnaires at regular intervals (usually bi-yearly).

The cohort design facilitates prospective studies, in which developmental trajectories may be assessed, while taking into account the time ordering of events and outcomes (as discussed in chapter three). The questionnaires and assessments cover a wide variety of physical, behavioural and psychological aspects, and therefore can be used to test a range of specific hypotheses in terms of causative and preventative factors. Further, the wide variety of measures and informants utilised (see **Table 5.1**) provide converging data for variables of interest.

5.2 The Sample

5.2.1 Initial Sample

The cohort comprises mothers who were pregnant with an expected delivery date of between 1st April 1991 and 31st December 1992. The disparity between the number of

mothers and children is explained by the fact that 199 of the pregnancies were known to be multiple: 195 twins, 3 triplets and 1 set of quadruplets (see **Figure 5.2**). The total number of pregnant women enrolled was 14, 541. Of these, 551 pregnancies had ended before 20 weeks gestation, and 69 had an unknown outcome. 13, 921 had given birth on or after 20 weeks. Prior to commencement of the study, data from 13,135 children in the Child Health and Education Study was analysed to assess whether the ALSPAC sample was representative of the total UK population. It was concluded that the Avon population was likely to be fairly similar to that of the UK overall (Golding et al., 2001).

5.2.2 The Target Sample

The target sample comprises the study children who completed the face-to-face Borderline Personality Disorder interview: The *UK Childhood Interview for DSM-IV Borderline Personality Disorder [UK-CI-BPD]*; during the *focus 11+* assessment clinic at 11 years of age. During the focus clinic day, each child took part in assessments over approximately three hours, including: vision and hearing tests, blood tests and psychological interviews. The UK-CI-BPD was part of the *Friends and You* session, which took approximately 40 minutes and comprised various tasks and measures. The child attended this session unaccompanied by parents, due to the confidential nature of the interviews. If, however, the parents objected to this arrangement, they could accompany their child, and certain parts of the session, e.g. the *UK-CI-BPD*, were not carried out.

5.2.2.1 Dropout

Families were eligible to be invited to the focus clinic at age 11 if they satisfied the following conditions (See **Figure 5.2** overleaf):

- 1) Child is alive
- 2) Address not recorded as unknown
- 3) Participating in the study (have not refused the whole study, may have refused certain questionnaires)

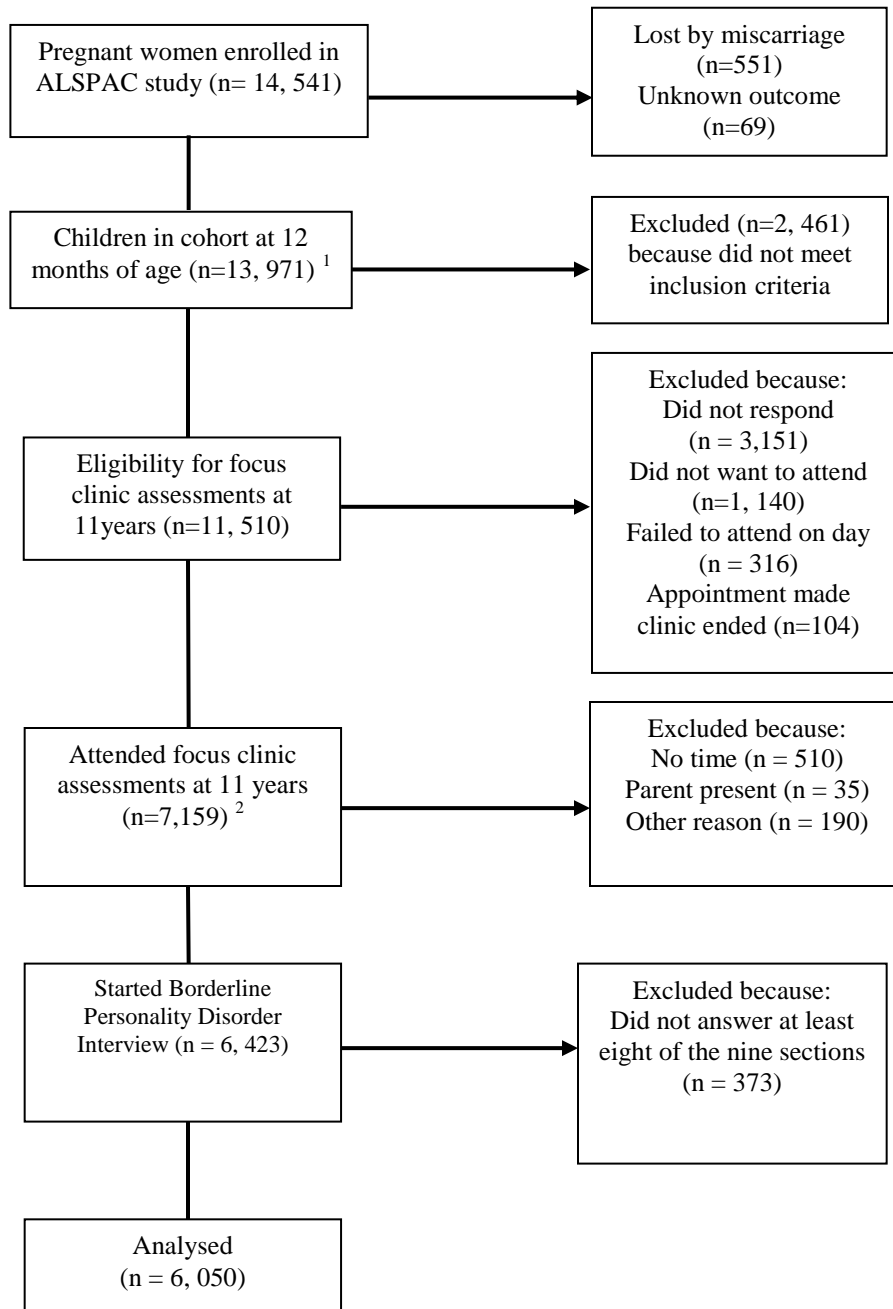
According to the above guidelines, of the original 13, 971 children, 11, 510 (82.4%) were eligible for the focus clinic. Three thousand, one hundred and fifty one (22.6%) did not respond to the initial letter and 1140 (8.2%) refused attendance, and a further 419 (3%) did not attend for unspecified reasons. A total of 7,159 (51.2% of the original sample) children attended the focus clinic; however, 359 of these were new cases,* thus not appropriate for inclusion in analyses.

5.2.2.2 Final Sample

Of the 7,159 children attending the clinic, 6,423 (89.7%) started the BPD interview. The final sample consisted of 6,050 (43.3% of the original cohort; 52.6% of the eligible sample) children, who completed at least eight of the nine sections (each pertaining to one BPD symptom, as defined by the DSM-IV) of the interview (see **Figure 5.2**).

* These were children eligible for the study but not recruited from birth for one reason or another

Figure 5.2 Attrition of participants from the ALSPAC cohort study



¹ Includes multiple births (accounting for 50 additional children); ² Includes new cases not assessed previously (accounting for additional 359 children)

The focus at age 11 clinics ran from January 2003 to January 2005, and the mean age of the child was 140.97 months (11 years and 9 months) with a standard deviation of 2.86 months.

The final sample used in study three (see chapter eight) was 5,711 children, who fulfilled all necessary criteria for inclusion in the study (see description in chapter 8). Despite drop out and exclusions, a very large sample was available for all three studies; therefore facilitating the use of a clinically relevant, categorical assessment of BPD.

5.3 Instrumentation: Outcome and Predictors

5.3.1 Outcome: The UK Childhood Interview for DSM-IV Borderline Personality Disorder (UK-CI-BPD)

The borderline interview was adapted from the *Childhood Interview for DSM-IV Borderline Personality Disorder [CI-BPD]* (Zanarini, Horwood, Waylen, & Wolke, 2004): the first semi- structured interview designed for use on latency (5/6 years to puberty) aged children. This face-to-face interview, originally developed by Mary Zanarini, was adapted for use with children in the UK by Dieter Wolke, Andrea Waylen, Mary Zanarini and Jeremy Horwood (See **Table 5.2** overleaf).

The *CI-BPD* was designed to indicate the prevalence of identified behaviours and emotions, rather than diagnose the child as having BPD. Children were told that the

Table 5.2 Summary of Questions Asked During the BPD Interview

BPD Symptoms	Questions asked
Anger* (1 behaviour 25% of time)	<ul style="list-style-type: none"> -Have you felt angry a lot of the time? -Have you felt angry but managed to hide it from other people? -Have you been angry and shown it? -Have you been so angry that you have got into a physical fight with someone you are close to?
Affective Instability* (1 behaviour 25% of time)	<ul style="list-style-type: none"> -Have you found that your mood has changed suddenly from feeling ok to feeling very sad, or cross, or extremely nervous or scared? -How about changing from feeling ok to feeling very angry, panicked or totally hopeless? -Have you been told that you are a moody person?
Emptiness* (1 behaviour 25% of time)	<ul style="list-style-type: none"> -During the past two years have you felt empty a lot of the time? -How about that you have no feelings inside
Identity disturbance* (1 behaviour 25% of time)	<ul style="list-style-type: none"> -Have you often been unsure of what kind of person you are? -Frequently gone from feeling ok about yourself to feeling you're a bad person (or even evil)? -Often felt that you have no consistent or steady idea of who you are? (Like you had no identity?) -That you had no idea of who you are or what you believe in? (That you don't even exist?)
Paranoid* (1 behaviour linked to stress, not present all of the time)	<ul style="list-style-type: none"> -Have you often felt very suspicious of other people? (did not trust them) -How about believe that they were taking advantage of you or blaming you for things that weren't your fault? -Were staring at you, talking about you behind your back, or laughing at you? -Frequently felt that you were physically separated from your feelings or as though you were viewing yourself from a distance? -You felt like you had no emotions, you felt emotionally dead? (Had times when you felt spaced out or numb?)
Stormy Relationships* (1 behaviour 25% of time)	<ul style="list-style-type: none"> -Have you often gone from loving and admiring someone to feeling that you can't stand him or her? -Had any stormy relationships or friendships with a lot of ups and downs? -Any relationships or friendships with a lot of very intense arguments? -How about times when you stopped talking to someone or stopped seeing them?

*Criterion for scoring [2]

Table 5.2 Continued..... Summary of Questions Asked During the BPD Interview

BPD Symptoms	Questions asked
Abandonment (1 behaviour 25% of time)*	<ul style="list-style-type: none"> -Have you frequently tried to avoid feeling completely alone e.g. phoned someone you're close to because you were feeling totally alone? -Tried to avoid being left alone or abandoned e.g. pleaded with people not to leave you, clung to them physically, refused to leave their home?
Suicidal Behaviour (1 behaviour present 2 or more times)*	<ul style="list-style-type: none"> -Have you ever hurt yourself on purpose? -Have you ever told someone that you are going to kill yourself to let them know that you are in pain? To see if they care? -Have you thought about killing yourself? -Have you made plans to kill yourself? -Have you actually tried to kill yourself?
Impulsivity (Two behaviours must have been present five times or more)*	<ul style="list-style-type: none"> -Have you got really drunk on alcohol? -Have you used prescription or illegal drugs to get high? -Had times when you have eaten so much food you have been in a lot of pain or had to force yourself to throw up? -Spent all of your money as soon as you got it? -Lost your temper and really shouted, yelled or screamed at anyone? -Threatened to physically harm anyone? -Shoved, slapped, kicked or punched someone? -Been in any fistfights? -Deliberately damaged property? -Done anything against the law?

*Criterion for scoring [2]

interview referred to the past two years of their life. The interview incorporated assessment of nine symptoms (consistent with *DSM* criteria) as described in Table 5.2 above. Further, probing questions were used, when necessary, to clarify whether the symptom was present, and that the child had understood the question correctly. Symptoms were presented both as dichotomous (yes/no) and quantitative (how often) questions. For example, for the symptom of anger, the dichotomous question was: *During the past two years have you felt angry a lot of the time?* The corresponding quantitative question being: *If yes, how often has this happened?*

Once all of the questions had been asked, a decision was made as to whether the symptom was present. If present, behaviour was graded as: [1] *probably present* and [2] *definitely present and significant* (see **Table 5.2**). The probably present criterion was used, as the focus was on BPD *precursor symptoms* rather than the clinically diagnosed disorder. For subsequent analyses, an overall BPD dichotomous outcome variable was constructed, with BPD symptoms classed as present if the child reported 5 or more symptoms (as consistent with BPD diagnosis in the *DSM*).

5.3.2 Brief Description of Predictor Variables

Independent and control variables were chosen according to existing theory, discussed in chapters two and three (see summary **Table 3.2**). Below is an outline of predictor and control variables used in studies one to three (see **Table 5.3**). A full description of variables, including details of construction, will be presented in the following chapters.

Table 5.3 Summary of Independent and Control Variables Used in Subsequent Studies

Study	Variable	Time-points	Respondent details
Factors			
Study 1: Maladaptive Parenting			
Mother/child relationship	<i>Child shouted at</i>	24, 42, 77 months	Mother postal questionnaire
	<i>Child hit</i>	24, 42, 77 months	Mother postal questionnaire
	<i>Maternal hostility</i>	21, 47, 85 months	Mother postal questionnaire
	<i>Maternal Resentment</i>	21, 33 & 47 months	Mother postal questionnaire
	<i>Harsh parenting composite (sum of above)</i>	See above	See above
Parental conflict	<i>Physical domestic violence</i>	8, 21, 33 & 47 months	Mother postal questionnaire
	<i>Partner broken/thrown things</i>	21 & 33 months	Mother postal questionnaire
	<i>Emotional domestic violence</i>	8, 21, 33 & 47 months	Mother postal questionnaire
	<i>Conflicting partnership</i>	33, 73 months	Mother postal questionnaire
	<i>Parent conflict composite (sum of above)</i>	See above	See above
Study 2: Peer Victimization			
Mother reported victimisation	<i>Picked on or bullied by other children</i>	47, 81 months 9, years	Postal questionnaire
Teacher reported victimisation	<i>Picked on or bullied by other children</i>	7, 10 years	Postal questionnaire
Self-reported victimisation	<i>Overt/ Relational</i>	8, 10 years	Child interviewed during focus clinics
	<u><i>Chronicity composite</i></u>		
	<i>Child report</i>	8 & 10 years	Child interview
	<i>Mother report</i>	47, 81 mths & 9 yrs	Mother questionnaire
	<i>Teacher report</i>	7 & 10 years	Teacher questionnaire
	<u><i>Combined composite</i></u>	10 years	Child interview
	<u><i>Severity composite</i></u>	8 & 10 years	Child interview

Table 5.3 Continued.... Summary of Independent and Control Variables Used in Subsequent Studies

Study	Variable	Time-points	Respondent details
Factors			
Study 3: Biosocial Developmental Model			
Dysregulated behaviour trait	<i>Emotionality, inattention and conduct problems</i> <i>Total dysregulated behaviour (sum of above)</i>	4,7 & 8 years	Mother postal questionnaire
Social environmental risk factors	<i>Harsh parenting</i>	9 years	Mother postal questionnaire
	<i>Peer victimisation</i>	8, 9, 10 years	Mother postal questionnaire/child interview
Control variables			
Social deprivation	<i>Family Adversity Index^a</i>	8, 12, 18 & 32 weeks gestation	Mother postal questionnaire
Intelligence	<i>IQ score^b</i>	Approx. 8 years	Child assessment at focus clinic
Axis I disorders	<i>DAWBA diagnosis^c</i>	Approx. 8 years	Mother and teacher report
Abuse	<i>Sexual/Physical^d</i>	18, 30, 42, 57, 69, 81 & 103 months	Mother postal questionnaire
Parenting	<i>Harsh Parenting composite^e (sum of hitting and hostility)</i>	21, 24, 42 & 47 months	Mother postal questionnaire

^a Studies 1, 2 & 3; ^b Study 1 & 2; ^c Studies 1 & 2; ^d Study 2; ^e Study 2

5.3.3 Approach to Missing Data

Missing data substitutions were not applied for repeat measures of the various exposures. When attrition occurred, no exposure, e.g. no hitting, was assumed for any missing data, therefore, scores were conservative. The implications of this approach are discussed in detail in the following relevant chapters.

5.4 Summary

This chapter provided an overview of the ALSPAC data resource. Assessment tools, sources of information and sample characteristics have been described to furnish the reader with an introduction to the ALSPAC cohort for the following three studies.

Chapter Six: Prospective study of family adversity and maladaptive parenting in childhood and borderline personality disorder symptoms in a non-clinical population at 11 years

Background. *Retrospective studies have consistently indicated an association between maladaptive parenting and borderline personality disorder (BPD). This requires corroboration with prospective, longitudinal designs. We investigated the association between sub-optimal parenting and parent conflict in childhood and BPD symptoms in late childhood using a prospective sample.*

Method. *A community sample of 6,050 mothers and their children (born between April 1991 and December 1992) were assessed. Mothers' family adversity was assessed during pregnancy and parenting behaviours such as hitting, shouting, hostility and parent conflict across childhood. IQ and DSM-IV axis I diagnoses were assessed at 7 to 8 years. Trained psychologists interviewed children at 11 years (mean age 11.74 years) to ascertain BPD symptoms.*

Results. *After adjustment for confounders, family adversity in pregnancy predicted BPD probable [OR (95% CIs)]: 1 to 2 adversities: 1.34 (1.01-1.77); > 2 adversities: 1.99 (1.34-2.94) and definite: 1 to 2 adversities: 2.48 (1.01-6.08) symptoms. Each point increase in the sub-optimal parenting index predicted BPD probable: 1.13 (1.05-1.23) and definite: 1.28 (1.03-1.60) symptoms. Parent conflict predicted BPD probable: 1.19 (1.06-1.34) and definite: 1.42 (1.06-1.91) symptoms. The associations between suboptimal parenting and BPD at 11 years were not significantly mediated by IQ or DSM-IV diagnosis at 8 years.*

Conclusions. *Children from adverse family backgrounds who experience sub-optimal parenting, more conflict between parents, and have poor cognitive abilities, are at increased risk of BPD symptoms at 11 years.*

Winsper, Zanarini, & Wolke

In Press: Psychological Medicine

6.1 Introduction

Maladaptive experiences during childhood have been consistently linked with borderline personality disorder (BPD) including: abuse and neglect (Guzder, Paris, Zelkowitz, & Feldman, 1999; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006), parent hostility and resentment (Hooley & Hoffman, 1999; Johnson, Cohen, Chen, Kasen, & Brook, 2006) and exposure to domestic violence and parent conflict (Herman, Perry, & van der Kolk, 1989; Weaver & Clum, 1993). Most studies have been retrospective, however, with concomitant methodological issues, such as the tendency of patients with BPD to misinterpret or misreport past experiences with family members (Bailey & Shriver, 1999). Further, domestic conflict and child maltreatment usually occur in family environments characterised by multiple risk factors (Fergusson, Boden & Horwood, 2006), which are difficult to disentangle with retrospective designs.

A series of prospective, longitudinal studies revealed an association between abuse, neglect, parenting and BPD features (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Johnson et al., 2000; Johnson et al., 2001; Johnson et al. 2006). However, associations were focused on scales of personality disorder symptoms, assessed in early adulthood, rather than symptoms in the clinical range. Subsequently, large prospective, longitudinal studies are now necessary to identify younger individuals with clinically relevant levels of BPD symptoms. These studies are challenging due to the low base rate of BPD and protracted duration before formal diagnosis, typically during early adulthood, is made. However, BPD is unlikely to suddenly appear in early adulthood,

rather it may be considered within a developmental trajectory as the end point following the appearance of BPD symptoms during childhood or adolescence.

The importance of the early identification of BPD symptoms, manifest as a childhood phenotype, has been highlighted, both for the facilitation of intervention programmes (Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008) and delineation of aetiological factors (Geiger & Crick, 2001). Further, BPD assessments for children have been developed (Crick, Murray-Close, & Woods 2005; Rogosch & Cicchetti, 2005), and it appears that BPD related features may be identified as early as six years of age, and remain relatively stable over time (Stepp, Pilkonis, Hipwell, Loebar, & Stouthamer-Loeber, 2010). Nevertheless, it has not been ascertained whether risk factors associated with BPD in adulthood are also associated with BPD symptoms during late childhood/early adolescence.

In the current study we investigated whether exposure to family adversity and maladaptive parent behaviour, during preschool and school periods, was predictive of BPD probable and definite symptoms (5 or more) in late childhood. Additionally, the developmental pathways through which this association manifests were explored by considering the mediating effects of potential markers: axis I DSM-IV diagnoses and IQ at age 7 to 8 years.

6.2 Method

6.2.1 Participants

The ALSPAC (Avon Longitudinal Study of Parents and Children) birth cohort (www.alspac.bris.ac.uk) includes children from the South West of England who had an expected delivery date between April 1, 1991 and December 31, 1992. The children are considered broadly representative of children in the United Kingdom (Golding et al., 2001). Starting from the first trimester of pregnancy, parents completed regular postal questionnaires about their family circumstances, their health and the study child's health and development from birth onwards. The study children have attended annual assessment clinics since the age of 7.5 years comprising of face-to-face interviews, psychological and physical tests. This study is based on 6050 children (age range: 10.4 to 13.6 years; mean age: 11.74 years) who completed the BPD interview as part of a series of clinic assessments running over half a day.

6.2.2 Ethical approval

Ethical approval was obtained from the ALSPAC Law and Ethics committee and the local research ethics committees.

6.2.3 Measures

Borderline personality disorder features interview

Borderline features were assessed using a face-to-face semi-structured interview: the *UK Childhood Interview for DSM-IV Borderline Personality Disorder (UK-CI-BPD)*

(Zanarini *et al.* 2004), based on the borderline module of the Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV) (Zanarini *et al.* 1996), which is a widely used semi-structured interview for all DSM-IV axis II disorders. The inter-rater and test-retest reliability of the DSM-III, DSM-III-R and DSM-IV versions of this measure have all proven to be good to excellent (Zanarini *et al.* 2000; Zanarini & Frankenberg, 2001). The UK-CI-BPD was adapted from the CI-BPD (US version), with small changes in wording making it appropriate for a UK sample, e.g. “being angry” was changed to “being cross.” The convergent validity of the CI-BPD was investigated using 171 adolescents (boys and girls) 13-17 years of age. 111 met criteria for BPD and 60 were normal comparison subjects. A Spearman's rho of 0.89 was obtained when comparing a dimensional score for BPD on the CI-BPD and the total score on the Revised Diagnostic Interview for Borderlines (DIB-R).

The UK-CI-BPD differs from the adult interview in three ways: The language is simpler. Two forms of impulsivity are omitted (reckless driving and promiscuity) due to lack of developmental appropriateness. Finally, the childhood interview is more structured than the adult version with the answer to each question, and not just the rating for each of the nine criteria, entered into the data set (Zanarini *et al.*, 2011).

The inter-rater reliability (Kappa) of the UK-CI-BPD, assessed from taped interviews of 30 children, ranged from 0.36 to 1.0 (median value 0.88), and 86% of the kappa values were within the excellent range of > 0.75 (Zanarini *et al.*, 2011).

The UK-CI-BPD is the first semi-structured interview designed to assess DSM-IV BPD in latency aged children. Similar to DSM-IV criteria, the interview consists of nine sections: intense inappropriate anger; affective instability; emptiness; identity disturbance; paranoid ideation; abandonment; suicidal or self-mutilating behaviours; impulsivity and intense unstable relationships. Once a trained assessor had explored each section, a judgment was made as to whether each symptom was definitely present, probably present or absent. A symptom was classed as definitely present if it occurred daily or approximately 25 % of the time, and probably present if it had occurred repeatedly, but did not meet criterion for definitely present.

Two outcome variables were constructed for use in the logistic regression analyses: BPD symptoms probably present (symptoms present *less* than daily or 25% of the time) and BPD symptoms definitely present, both of which were based on the presence of five or more symptoms. Diagnosis of BPD according to the DSM-IV is based on the presence of five or more definite features, thus the probable BPD outcome represents a sub-syndromal assessment of BPD.

Exposure Variables: Family Adversity, Suboptimal Parenting and Parent Conflict

Family Adversity during Pregnancy

Multiple family risk factors were indicated using the Family Adversity Index (FAI) (Bowen et al., 2005), which consists of 18 items taken from questionnaires administered throughout pregnancy (8, 12, 18 and 32 weeks gestation). The FAI index comprised of

items pertaining to young maternal age at first pregnancy (<17 years) or birth of study child (<20 years); housing (e.g. inadequacy: overcrowding or periods of homelessness); financial difficulties; problematic partner relationship; maternal affective disorder (depression, anxiety, suicidality); substance abuse (drugs or alcohol); or involvement in crime (i.e. in trouble with police or convictions). For the current analysis the item reflecting partner cruelty (emotional or physical) was removed from the FAI to prevent confounding with the domestic violence predictor variables. The remaining adversity items were summed and trichotomised into: none (no adversity); mild (1 or 2 adversities); and severe (>2 adversities).

Sub-optimal Parenting Index

Selection of the sub-optimal parenting predictors was based on a previous study (Waylen *et al.* 2008), which factor analysed questions pertaining to maternal attitudes, behaviours and feelings within the ALSPAC cohort. Three factors (hostility, resentment and hitting/shouting) were evidenced, which were found to be predictive of a variety of negative health outcomes during mid-childhood. These factors have been prospectively linked to personality disorders (and BPD features) within the literature (Johnson *et al.*, 2006), thus were combined to create a sub-optimal parenting index.

Scales assessing parent behaviour, as reported by the mother, were dichotomised, indicating whether the maladaptive behaviour was present or absent. Where available, variables were constructed for the preschool (birth to up to 5 years) and school (5 to 8 years) periods. The sub-optimal parenting index was constructed by summing 7 items across the preschool and school periods to create an index of increasing exposure to sub-

optimal parenting on a scale of 0-7. The items were: hitting (preschool, school); shouting (preschool, school); hostility (preschool, school) and resentment (preschool).

Hitting and shouting

Maternal hitting and shouting were indicated by the following two items: ‘When you are at home with your child how often do you slap him?’ and ‘When you are at home with your child how often do you shout at him?’ (Waylen, Stallard, & Stewart-Brown, 2008) For the preschool period (24 & 42 months), hitting was coded as present if it occurred daily or every week at either time point, and shouting if it occurred daily at either time point. For the school period (77 months), hitting was recorded as present if reported often or sometimes. We used less stringent criteria for the school period to reflect the observed reduction in hitting and shouting, as the child grows older (Hyman, 1997).

Hostility and resentment

Hostility and resentment were constructed from a number of items loading on two distinct factors (Waylen et al., 2008). Preschool hostility items included: ‘mum feels that whining makes her want to hit child’ (21 months); ‘mum often irritated by child’ (47 months); ‘mum has battle of wills with child’ (47 months); and ‘child gets on mum’s nerves’ (47 months). Preschool hostility was classed as present if at least 3 items were reported. Preschool resentment items included: ‘mum dislikes mess from child’ (47 months); ‘mum feels unbearable when child cries’ (21 months); ‘mum feels child’s desires cause anger’ (21 months); and ‘mum feels has no time alone’ (33 months). Preschool resentment was classed as present if at least 2 items were reported. For the

school period, only hostility items were available: ‘mum often irritated by child’ (85 months); ‘mum has battle of wills with child’ (85 months); ‘child gets on mum’s nerves’ (85 months). School hostility was considered present if all 3 items were reported.

Conflicting Partnership Index

Domestic violence and conflicting partnership measures were chosen according to reported prospective associations with negative child outcomes generally (Kitzman et al., 2003), and BPD specifically in retrospective studies (Herman et al., 1989; Weaver & Clum, 1993). The parent conflict index was constructed across the preschool and school periods from 5 items, on a scale of 0-5, reflecting increasing exposure to conflict between primary caregivers. The items were: conflicting partnership (preschool, school); partner broken or thrown things (preschool); physically hurt by partner (preschool) and emotional domestic violence (preschool).

Physical and emotional domestic violence

Physical and emotional domestic violence variables (Bowen, Heron, Waylen, Wolke, & the ALSPAC study team, 2005) were available for the preschool period only. Two physical domestic violence variables were constructed: physically hurt by partner and partner broken or thrown things. The variable: physically hurt by partner was constructed from the two items: ‘physically hurt by partner’ (8, 21, 33, 47 months) and ‘slapped or hit by partner’ (21 & 33 months), and was coded as present if the mother responded yes to one or more of the six items. The variable ‘partner broken or thrown things’ (21 & 33 months) was considered present if reported at either time point. An emotional domestic violence variable was constructed from the item ‘your partner was

emotionally cruel to you' (8, 21, 33, 47 months) (Bowen et al., 2005), and considered present if reported at one or more time points.

Conflicting partnership

A conflicting partnership variable was derived for the preschool (33 months, or 22 months if the 33 month response was missing) and school (73 months) periods. It was constructed from the following items: 'mum and partner argued'; 'not speaking to partner for more than 30 minutes'; 'one of you walking out of the house'; and 'shouting or calling partner names'. For the preschool and school periods, each of these items was dichotomised; if either the mother, her partner, or both parties had engaged in the behaviour the item was coded as present. Conflicting partnership was considered positive if reported in three or all four items.

Sociodemographic and birth variables

Mother-reported sociodemographic information during the antenatal period included marital status (married versus single); home ownership (home owner versus rented); parent social class (based on the highest of the mother's or partner's occupational social class: dichotomised into non-manual versus manual); and maternal education (dichotomised into below O-level versus O-level or above. O-levels being the standard school leaving qualifications at age 16 in the United Kingdom until recently). The ethnic origin of the child (white versus black or minority ethnic) and birth weight were obtained from birth records. Birth weight was dichotomised into below or equal to 2499 grams (low birth weight) and above or equal to 2500 grams.

Potential confounders

Study child IQ was assessed with the Wechsler Intelligence Scale for Children III (UK version) (Wechsler, Golombok, & Rust, 1992) during the focus at 8 years clinic. DSM–IV psychiatric diagnoses were derived at 91 months using the Development and Well Being Assessment (DAWBA) (Goodman, Ford, Richards, Gatward, & Meltzer 2000), completed by parents and teachers. A dichotomous variable, indicating the presence of any major Axis I disorder (attention-deficit hyperactivity disorder; conduct disorder; oppositional defiant disorder; depression or anxiety) was constructed.

6.2.4 Statistical analysis

Initial analyses were carried out with *SPSS version 18* statistical software. Selective dropout was determined by comparing those who completed the borderline interview to those lost to follow up (**Table 6.1**). Odds ratios (OR) and 95% confidence intervals (95% CI) were computed to test for gender differences in parenting variables and BPD symptoms (**Table 6.2**). Crude associations between the maladaptive parenting measures and BPD symptoms were computed. Associations were then adjusted for age and gender, and additionally for age, gender, IQ and DSM-IV diagnosis. Odds ratios with 95% confidence intervals are reported for the preschool and school periods respectively (**Tables 6.3 and 6.4**).

Path analysis was conducted using *Mplus version six* to elucidate the direct and indirect relationships between exposure to family adversity, suboptimal parenting and parent conflict, manifestation of DSM-IV axis I diagnoses, IQ and the borderline symptoms outcome (**Figure 6.1; Table 6.6**). A categorical, ordinal BPD outcome was utilised in the path analysis, reflecting increasing severity of BPD (less than 5 symptoms; 5 or more probable symptoms; 5 or more definite symptoms). *Mplus version six* software is suitable for the analysis of categorical outcomes producing estimates in the form of probit coefficients. Probit coefficients indicate the strength of relationship between predictor variables and probability of group membership, representing the difference that a one-unit change in the predictor variable makes in the cumulative normal probability of the outcome variable (Lee, Uken, & Sebold, 2007). For ordinal outcomes one co-efficient per predictor is produced. This may be interpreted in the same way as a continuous dependent variable, as an ordinal dependent variable is comparable to a continuous latent response variable, which exceeds thresholds to give various outcome categories (Muthén, 1998-2004).

6.3 Results

6.3.1 Differences between participants with and without the completed borderline interview

The frequencies of sociodemographic factors, psychiatric diagnoses and IQ are shown for ALSPAC participants with and without BPD interviews in Table 6.1. Those lost to

follow up were more often boys, ethnic minority children, of low birth weight, born to single mothers of lower education level, and from rented properties with parents in manual jobs. They were more likely to have been born into family adversity, and have had psychiatric diagnoses at 91 months. Children who dropped out had a lower IQ at 8 years. In sum, participants remaining in the analysis were less severely disadvantaged than those who dropped out.

6.3.2 Frequency of borderline personality disorder and maladaptive parenting variables

Table 6.2 reports the frequencies of BPD probably and definitely present and parenting variables by total and gender. 6.4% of the ALSPAC cohort had 5 or more probable symptoms, and 0.9% had 5 or more definite symptoms at 11 years. These findings are largely concordant with a previous community study, which reported that 7.8% of 9-19 year olds had moderate BPD, and 3 % had severe BPD (Bernstein et al., 1993), with the lower figures here possibly attributable to the younger age of the cohort.

Hitting and shouting were common during the preschool period, becoming rarer during the school period (Table 6.2). Significantly more boys than girls were hit during both periods and shouted at during the preschool period. Hostility and resentment did not differ according to gender of the study child. Domestic violence was reported for the preschool period only, with emotional domestic violence more common than physically hurt by partner and partner broken or thrown things. There were no gender differences

for living in a household with domestic violence. Conflicting partnerships, during both periods, did not differ according to gender of child.

Table 6.1 Dropout analysis with regard to availability of BPD interview at 11 years

Characteristic	BPD not available N (%)	BPD available N (%)	BPD available vs. not available OR (95% CI) ^a
Gender			
Male	4328 (59.6)	2938 (40.4)	[reference]
Female	3669 (54.1)	3112 (45.9)	1.25 (1.17 – 1.34)
Ethnicity			
White	5967 (51.9)	5541 (48.1)	[reference]
Black and minority ethnic	395 (64.6)	216 (35.4)	0.59 (0.50 – 0.70)
Birth weight			
≤2499 grams	7370 (56.4)	5707 (43.6)	[reference]
≥2500 grams	517 (65.4)	273 (34.6)	0.68 (0.59 – 0.79)
Marital Status			
Single	2206 (66.8)	1095 (33.2)	[reference]
Married	5031 (51.1)	4821 (48.9)	1.93 (1.78 – 2.10)
Home ownership			
Mortgage	4701 (49.0)	4901 (51.0)	[reference]
Rented	2532 (72.6)	958 (27.4)	0.36 (0.33 – 0.40)
Education of mother			
Below O – level	2476 (66.2)	1262 (33.8)	[reference]
O – level or above	4142 (47.5)	4577 (52.5)	2.17 (2.00 – 2.35)
Social class			
Non-manual	2729 (46.4)	3152 (53.6)	[reference]
Manual	3210 (56.9)	2430 (43.1)	0.66 (0.61 – 0.71)
FAI			
None	2565 (47.9)	2791 (52.1)	[reference]
Moderate; 1 to 2 adversities	3125 (56.0)	2454 (44.0)	0.72 (0.67-0.78)
Severe: >2 adversities	1577 (68.7)	717 (31.3)	0.42 (0.38-0.46)
DSM-IV Axis I diagnoses (DAWBA)			
None	2791 (36.6)	4839 (63.4)	[reference]
At least one diagnosis	257 (45.5)	308 (54.5)	0.69 (0.58 – 0.82)
IQ			
mean (S.D) ^b	100.6 (17.2)	105.8 (15.8)	1.02 (1.02-1.02)

Abbreviations: CI: Confidence Interval; DAWBA: Development and Well-being Assessment; FAI: Family Adversity Index; OR: odds ratio; BPD: Borderline Personality Disorder Symptoms Interview. ^a Bold indicates that the 95% CI does not include 1.00. ^b for BPD interview not available, n = 1669; for BPD interview available, n = 4787.

Table 6.2 *Frequencies of borderline personality disorder diagnosis (probably/definitely) and maladaptive parenting variables shown for the total sample and by gender*

Borderline diagnosis or parenting variables	Total N (%)	Girls	Boys	Girls vs. Boys, OR (95% CI) ^a
Borderline Diagnosis:				
Probable				
0 No	5606 (93.5)	2882 (93.5)	2724 (93.5)	
1 Yes	389 (6.4)	200 (6.4)	189 (6.4)	1.00 (0.81-1.23)
Borderline Diagnosis: Definitely				
0 No	5995 (99.1)	3082 (99.0)	2913 (99.1)	1.13 (0.67-1.93)
1 Yes	55 (0.9)	30 (1.0)	25 (0.9)	
Hitting and shouting				
Preschool hitting				
0 No	3235 (56.6)	1783 (60.9)	1452 (52.1)	0.7 (0.63-.78)
1 Present	2479 (43.4)	1145 (39.1)	1334 (47.9)	
Preschool shouting				
0 No	3125 (54.7)	1703 (58.1)	1422 (51)	0.75 (0.68-.83)
1 Present	2593 (45.3)	1227 (41.9)	1366 (49)	
School hitting				
0 No	1868 (61.6)	999 (64.3)	869 (58.8)	0.79 (0.83-0.92)
1 Present	1163 (38.4)	544 (35.7)	609 (41.2)	
School shouting				
0 No	2644 (84.7)	1349 (84.9)	1295 (84.5)	0.97 (0.8-1.81)
1 Present	477 (15.3)	240 (15.1)	237 (15.5)	
Parental attitudes				
Preschool hostility				
0 No	4918 (85.8)	2526 (85.9)	2392 (85.8)	0.99 (0.86- 1.15)
1 Present	812 (14.2)	416 (14.1)	396 (14.2)	
School hostility				
0 No	4595 (89.5)	2341 (89.2)	2254 (89.8)	1.06 (0.89-1.27)
1 Present	539 (10.5)	283 (10.8)	256 (10.2)	
Preschool resentment				
0 No	5011 (86.6)	2597 (87.3)	2414 (85.9)	0.89 (0.76-1.03)
1 Present	776 (13.4)	379 (12.7)	397 (14.1)	
Domestic violence				
Physically hurt				
0 No	5527 (93.7)	2849 (94)	2678 (93.5)	0.93 (0.75-1.14)
1 Present	369 (6.3)	183 (6.0)	186 (6.5)	
Broken or Thrown				
0 No	5283 (95.7)	2724 (95.8)	2559 (95.5)	0.92 (0.71-1.2)
1 Present	240 (4.3)	119 (4.2)	121 (4.5)	
Emotional				
0 No	4828 (81.9)	2471 (81.5)	2357 (82.3)	1.05 (0.92-1.20)
1 Present	1067 (18.1)	560 (18.5)	507 (17.7)	
Conflicting partnership				
Preschool				
0 No	4265 (77.2)	2181 (76.7)	2084 (77.7)	1.06 (0.93-1.2)
1 Present	1262 (22.8)	663 (23.3)	599 (22.3)	
School				
0 No	3890 (80.7)	1976 (80.5)	1914 (81.0)	1.04 (0.90-1.2)
1 Present	929 (19.3)	480 (19.5)	449 (19.0)	

6.3.3 Associations between maladaptive parenting and borderline personality disorder symptoms

Table 6.3 shows the associations between maladaptive parenting and borderline personality disorder probable symptoms. Column A shows the crude associations, Column B shows associations after controlling for age and gender; and Column C shows associations controlling for age, gender, DSM-IV diagnoses and IQ.

Family adversity (1 to 2 items, > 2 items); hitting (preschool), hostility (school), partner breaking or throwing things, emotional domestic violence and conflicting partnership (preschool and school) were all significantly associated with BPD probable symptoms. After controlling for confounders, conflicting partnership (preschool and school) was no longer predictive of BPD probable symptoms, and the association between family adversity (>2 items) and BPD was reduced to a certain extent, but remained significant. Sub-optimal parenting and parent conflict led to higher odds of BPD probable symptoms, after adjusting for confounders.

Table 6.4 shows the associations between family adversity, maladaptive parenting and BPD definite symptoms. Hitting (preschool), resentment, hostility (preschool and school), emotional domestic violence, physically hurt by partner and conflicting partnership (school) were predictive of BPD definite symptoms. After controlling for confounders, hostility (school), emotional domestic violence, physically hurt by partner and conflicting partnership (school) remained significantly predictive of BPD definite

Table 6.3 Associations between maladaptive parenting and BPD probable symptoms (showing crude associations [A], adjustment for age and gender [B] and additionally DSM-IV diagnosis and IQ [C])

Exposure	Subgroup	BPD Status N (%)	A OR (95% CI) ¹	B OR (95% CI) ²	C OR (95% CI) ³	
Family Adversity	0 items	141 (5.1)				
	1to2 items	163 (6.7)	1.35 (1.07-1.70)	1.35 (1.07-1.71)	1.34 (1.01-1.77)	
	>2 items	77 (11.0)	2.30 (1.72-3.08)	2.32 (1.74-3.11)	1.99 (1.34-2.94)	
Hitting and shouting						
Preschool	Hitting	No (3235)	185 (5.8)			
		Yes (2479)	181 (7.4)	1.31 (1.06-1.62)	1.31 (1.06-1.62)	1.43 (1.10-1.86)
	Shouting	No (3125)	185 (6.0)	1.20 (0.97-1.48)	1.20 (0.97-1.49)	
		Yes (2593)	182 (7.1)	1.20 (0.97-1.48)	1.20 (0.97-1.49)	1.22 (0.94-1.58)
School	Hitting	No (1868)	118 (6.4)			
		Yes (1163)	88 (7.6)	1.22 (0.91-1.62)	1.21 (0.91-1.62)	1.24 (0.88-1.74)
	Shouting	No (2644)	181 (6.9)	1.04 (0.71-1.53)	1.05 (0.72-1.53)	
		Yes (477)	34 (7.2)	1.04 (0.71-1.53)	1.05 (0.72-1.53)	1.11 (0.71-1.74)
Parental Attitude						
Preschool	Hostility	No (4918)	298 (6.1)			
		Yes (812)	63 (7.9)	1.32 (0.98-1.75)	1.32 (0.99-1.75)	1.49 (1.07-2.08)
	Resentment	No (5011)	314 (6.3)	1.11(0.82-1.50)	1.11 (0.82-1.50)	
		Yes (776)	53 (6.9)	1.11(0.82-1.50)	1.11 (0.82-1.50)	1.17 (0.81-1.67)
School	Hostility	No (4595)	268 (5.9)			
		Yes (539)	47 (9.0)	1.58 (1.14-2.18)	1.58 (1.14-2.19)	1.56 (1.06-2.29)
	Sub-optimal parenting Index⁴			1.12 (1.04-1.20)	1.12 (1.04-1.19)	1.13 (1.05-1.23)
Conflict						
Domestic Violence	Emotional	No (4828)	287 (6.0)			
		Yes (1067)	87 (8.3)	1.42 (1.11-1.83)	1.43 (1.11-1.83)	1.52 (1.12-2.06)
	Physically hurt by partner	No (5527)	345 (6.3)	1.31 (0.89-1.95)	1.33 (0.89-1.97)	1.58 (0.99-2.53)
	Broken or thrown things	Yes (369)	29 (8.1)	1.31 (0.89-1.95)	1.33 (0.89-1.97)	1.58 (0.99-2.53)
Conflicting partnership	Preschool	No (5283)	318 (6.1)			
		Yes (240)	23 (9.7)	1.66 (1.06-2.58)	1.68 (1.08-2.63)	1.92 (1.14-3.23)
	School	No (4265)	249 (5.8)			
		Yes (1262)	93 (7.4)	1.28 (1.00-1.64)	1.28 (1.00-1.64)	1.23 (0.90-1.67)
	School	No (3890)	253 (5.8)	1.42 (1.09-1.86)	1.43 (1.09-1.86)	
		Yes (929)	77 (8.1)	1.42 (1.09-1.86)	1.43 (1.09-1.86)	1.32 (0.96-1.82)
	Parent conflict index⁵			1.17 (1.06-1.28)	1.17 (1.07-1.29)	1.19 (1.06-1.34)

¹ Bold indicates that the 95% CI does not include 1.00; ² Controls include gender and age; ³ Controls include gender, age, DSM-IV diagnosis and IQ; ⁴ Sub-optimal parenting index on scale of 1-7; ⁵ Parent conflict index on scale of 1-5.

Table 6.4 Associations between maladaptive parenting and BPD definite symptoms (showing crude associations [A], adjustment for age and gender [B] and additionally DSM-IV diagnosis and IQ [C])

Exposure	Subgroup	BPD Status N (%)	A OR (95% CI) ¹	B OR (95% CI) ²	C OR (5% CI) ³	
Family Adversity	0 items					
	1-2 items		2.12 (1.11-4.08)	2.13 (1.11-4.09)	2.48 (1.01-6.08)	
	>2 items		3.95 (1.88-8.32)	3.98 (1.89-8.39)	2.53 (0.78-8.18)	
Hitting and shouting						
Preschool	Hitting	No (3235)	22 (0.7)	1.79 (1.03-3.11)	1.84 (1.06-3.21)	1.05 (0.50-2.25)
		Yes (2479)	30 (1.2)			
	Shouting	No (3125)	27 (0.9)	1.12 (0.65-1.93)	1.14 (0.66-1.97)	1.17 (0.55-2.48)
		Yes (2593)	25 (1.0)			
School	Hitting	No (1868)	18 (1.0)	1.07 (0.51-2.23)	1.06 (0.51-2.21)	1.26 (0.54-2.95)
		Yes (1163)	12 (1.0)			
	Shouting	No (2644)	26 (1.0)	0.85 (0.30-2.45)	0.85 (0.30-2.45)	0.82 (0.24-2.80)
		Yes (477)	4 (0.8)			
Parental Attitude						
Preschool	Hostility	No (4918)	36 (0.7)	2.38 (1.28-4.43)	2.38 (1.28-4.43)	1.93 (0.81-4.64)
		Yes (812)	14 (1.7)			
	Resentment	No (5011)	38 (0.8)	2.06 (1.07-3.95)	2.07 (1.08-3.98)	2.21 (0.92-5.28)
		Yes (776)	12 (1.5)			
School	Hostility	No (4595)	30 (0.7)	4.36 (2.33-8.15)	4.34 (2.32-8.12)	3.85 (1.69-8.78)
		Yes (539)	15 (2.8)			
	Sub-optimal parenting Index⁴			1.29 (1.10-1.51)	1.30 (1.11-1.52)	1.28 (1.03-1.60)
Conflict						
Domestic Violence	Emotional	No (4828)	34 (0.7)	2.56 (1.45-4.50)	1.96 (1.08-3.57)	3.63 (1.71-7.73)
		Yes (1067)	19 (1.8)			
	Physically hurt by partner	No (5527)	42 (0.8)	4.01 (2.05-7.86)	3.02 (1.49-6.12)	3.08 (1.17-8.10)
Yes (369)		11 (3.0)				
	Broken or thrown things	No (5283)	42 (0.8)	1.05 (0.25-4.36)	0.89 (0.21-3.74)	0.82 (0.11-6.21)
		Yes (240)	2 (0.8)			
Conflicting partnership	Preschool	No (4265)	38 (0.9)	0.53 (0.22-1.26)	0.43 (0.18-1.03)	0.74 (0.27-2.04)
		Yes (1262)	6 (0.5)			
	School	No (3890)	26 (0.6)	2.81 (1.50-5.26)	2.38 (1.25-4.51)	2.89 (1.29-6.47)
		Yes (929)	16 (1.7)			
	Parent conflict index⁵			1.33 (1.07-1.65)	1.20 (0.95-1.51)	1.42 (1.06-1.91)

¹Bold indicates that the 95% CI does not include 1.00; ²Controls include gender and age; ³Controls include gender, age, DSM-IV diagnosis and IQ; ⁴Sub-optimal parenting index on a scale of 1-7; ⁵Parent conflict index on a scale of 1-5.

symptoms, though the association between physically harmed by partner and BPD became noticeably weaker. Sub-optimal parenting and parent conflict remained predictive of BPD definite symptoms after controlling for confounders.

6.3.4 Predictive associations between the FAI, parenting variables, potential mediators and borderline personality disorder symptoms

The predictive associations between family adversity, parenting variables, mediators and BPD probable and definite symptoms are shown in Tables 6.5 (a, b & c). These associations were tested according to time ordering; therefore, family adversity was considered a predictor, while Axis I DSM-IV diagnoses (DAWBA), IQ and BPD were considered outcomes of family adversity and the maladaptive parenting indices.

Univariate analysis indicated that family adversity was predictive of sub-optimal parenting, parent conflict, DSM-IV diagnosis, IQ and BPD symptoms probable and definite (Table 6.5a). Sub-optimal parenting and parent conflict were predictive of DSM-IV diagnoses, IQ and BPD probable and definite symptoms (Table 6.5b). DSM-IV diagnoses were predictive of BPD probable symptoms and IQ was predictive of BPD definite symptoms (Table 6.5c). These findings are consistent with a pathway model in which family adversity is associated with sub-optimal parenting and parent conflict, which in turn, are associated with DSM-IV diagnoses and lower IQ (child markers), which are both related to BPD symptoms.

Table 6.5.a FAI as an exposure; maladaptive parenting, DAWBA diagnosis, IQ and BPD as outcomes

Exposure	Outcomes					
	Sub-optimal parenting ¹	Parent conflict ¹	DAWBA ²	IQ ¹	BPD Probable ²	BPD Definite ²
Mild	1.29 vs. 1.42 (p=0.00)	0.40 vs. 0.73 (p=0.00)	1.60 (1.31-1.96)	-3.43 (-4.28 to -2.59)(p=0.00)	1.34 (1.06-1.69)	2.12 (1.11 -4.10)
Severe	1.29 vs. 1.49 (p=0.00)	0.40 vs. 1.18 (p=0.00)	3.11 (2.46-3.93)	-8.97 (-10.25 to -7.68) (p=0.00)	2.26 (1.69-3.02)	3.95 (1.88-8.32)

FAI: Family Adversity Index; Significant associations in bold; ¹ Comparison of mean scores; ² Odds Ratio (CIs).

Table 6.5.b Maladaptive parenting as an exposure; DAWBA, IQ and BPD as outcomes

Exposure	Outcomes			
	DAWBA ¹	IQ ²	BPD Probable ¹	BPD Definite ¹
Sub-optimal	1.38 (1.31-1.46)	- 0.47 (-0.74 to- 0.19)	1.11 (1.01-1.19)	1.29 (1.10 -1.50)
Conflict	1.33 (1.24-1.42)	- 0.96 (-1.35 to -0.57)	1.16 (1.06-1.28)	1.33 (1.07-1.65)

Significant associations in bold; ¹ Odds Ratios (CIs); ² Beta co-efficients (CIs) both significant at p=0.001.

Table 6.5.c DAWBA and IQ as exposures; BPD as an outcome

Exposure	Outcomes	
	BPD Probable ¹	BPD Definite ¹
DAWBA	1.57 (1.05-2.36)	2.33 (0.91 -6.00)
IQ	0.99 (0.99-1.00)	0.95 (0.93-0.97)

Significant associations in bold; ¹Odds Ratio (CIs)

6.3.5 Path analysis

The path model incorporated the family adversity, sub-optimal parenting, and parent conflict indices as predictors. IQ and DSM-IV diagnoses were entered as potential mediators, while gender was entered as a control. Model fit indices indicated good fit: $\chi^2 = 11.58$, $P = 0.00$, $RMSEA = 0.02$, $CFI = 0.99$. **Fig. 6.1** shows the unstandardised and

standardised (in brackets) estimates of the direct path coefficients between the various predictor and mediating variables. Non-significant paths ($p > 0.05$: one tailed) are not shown.

The direct relationships between family adversity (1 to 2 & >2 adversities), sub-optimal parenting, IQ and BPD outcome at 11 years were significant. Direct and indirect path coefficients to the BPD outcome are shown in **Table 6.6**.

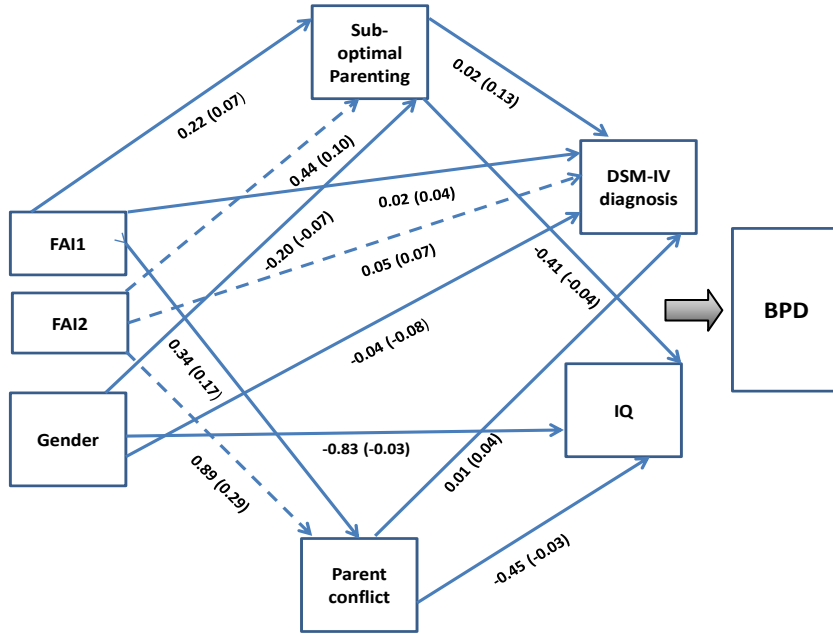
Table 6.6 *Unstandardised probit coefficients (B) for the direct and indirect paths between FAI, harsh parenting, parental conflict, IQ and subsequent borderline personality disorder symptoms at 11 years*

	Direct to BPD outcome			Indirect to BPD outcome					
	B	S.E	P value	Via DSM -IV diagnosis			Via IQ		
B				S.E	P value	B	S.E	P value	
FAI (1) ²	0.13 ¹	0.05	0.02	0.03	0.002	0.14	0.01	0.01	0.02
FAI (2) ³	0.35	0.07	0.00	0.01	0.01	0.09	0.02	0.01	0.02
Suboptimal Parenting	0.053	0.02	0.00	0.004	0.002	0.08	0.002	0.001	0.07
Parent Conflict	0.04	0.02	0.09	0.002	0.001	0.13	0.002	0.001	0.13
IQ	-0.01	0.002	0.01	-	-	-	-	-	-
DSM-IV	0.18	0.10	0.07						

¹ A probit coefficient of 0.13 indicates that for each unit increase in FAI there is an increase of 0.13 standard deviations in the predicted Z score of the cumulative normal distribution of BPD symptoms;

² The FAI1 category denotes 1 - 2 items ³ The FAI2 category denotes >2 items. The BPD outcome is an ordinal categorical outcome: none; probable: 5 or more symptoms; definite: 5 or more symptoms.

Figure 6.1 Final model showing unstandardised and standardised (in brackets) coefficients for the direct effects of FAI, harsh parenting, parent conflict and child IQ



Direct and indirect predictors of BPD are shown in Table 6 →

Significant coefficients for FAI2 - - - - - →

Significant Coefficients for other predictors ———— →

$\chi^2 = 11.58$, $P = 0.00$, $RMSEA = 0.02$, $CFI = 0.99$; Values are given in unstandardised probit coefficients. Standardised coefficients are in parentheses. Non-significant paths at the 0.05 level are not shown. The FAI (family adversity index) is coded into 3 categories: none, moderate and severe. **FAI1** (1 or 2 items) and **FAI2** (>2 items) are dummy variables with FAI (0 items) used as the reference group. Relationships for the FAI2 are shown in broken lines. **Gender** is a nominal variable: the negative relationship represents that male sex is a significant predictor of parenting problems. Correlation between parenting and conflict = 0.20 (0.02), $p=0.00$, not shown in diagram for clarity.

6.4 Discussion

6.4.1 Comment

Congruent with previous research, we found that sub-optimal parenting and parent conflict were more likely within families experiencing adversities, ranging from poverty and overcrowding to mental health problems (Fergusson et al., 2006). Family adversity was assessed during pregnancy, thereby excluding reverse causality effects of parenting, or a challenging child, on adversity.

Family adversity had a direct impact on BPD symptoms at 11 years of age, and indirect effects via harsh parenting and parent conflict and poorer cognitive functioning of the child. Further, there was a dose response effect with increasing family adversity (i.e. 3+ items) leading to increased odds of BPD symptoms. This indicates that children exposed to higher levels of family adversity and maladaptive parenting were at heightened risk of developing BPD symptoms. The direct impact of family adversity during pregnancy may be due to continued adversity throughout childhood, such as social deprivation, leading to increased BPD symptoms. Alternatively, adversity during early pregnancy may lead to increased stress for the foetus, and early programming alterations of the HPA (Hypothalamic - Pituitary - Adrenal) axis (Entringer, Kumsta, Hellhammer, Wadhwa, & Wüst, 2009), increasing the risk of BPD symptoms.

Despite controlling for other adversities, we found that sub-optimal parenting had significant direct associations with BPD symptoms, adding to the current research

literature by providing prospective evidence for a link between suboptimal parenting and subsequent BPD symptoms in late childhood/ early adolescence. When considering individual parenting items, resentment was not predictive of probable or definite BPD symptoms after controlling for confounders. Although this construct was chosen according to previous research literature (Waylen et al., 2008), it may be that some of the composite items, e.g. 'mum feels has no time alone' did not sufficiently tap into the resentment feelings of the mother.

IQ at 8 years was directly predictive of BPD symptoms, and in turn was significantly predicted by sub-optimal parenting. The indirect association between sub-optimal parenting and BPD via IQ was approaching significance; however, the effect was very weak. Of the total sub-optimal parenting to BPD relationship, only 3.3% was mediated by IQ. There is ample evidence that lower IQ is often indicative of a deleterious home environment, lacking in resources and academic encouragement (Brody & Flor, 1998; Van Ijzendoorn, Juffer, & Klein-Poelhuis, 2005). Therefore, maladaptive parenting is likely to contribute to poorer cognitive ability and increased BPD symptoms. Though a robust link between maladaptive parenting and lower offspring IQ has been reported, we cannot be sure that exposure to maladaptive parenting led to lower IQ in this study. As there were no baseline measures of IQ, it is possible that differences in IQ were present prior to exposure to maladaptive parenting.

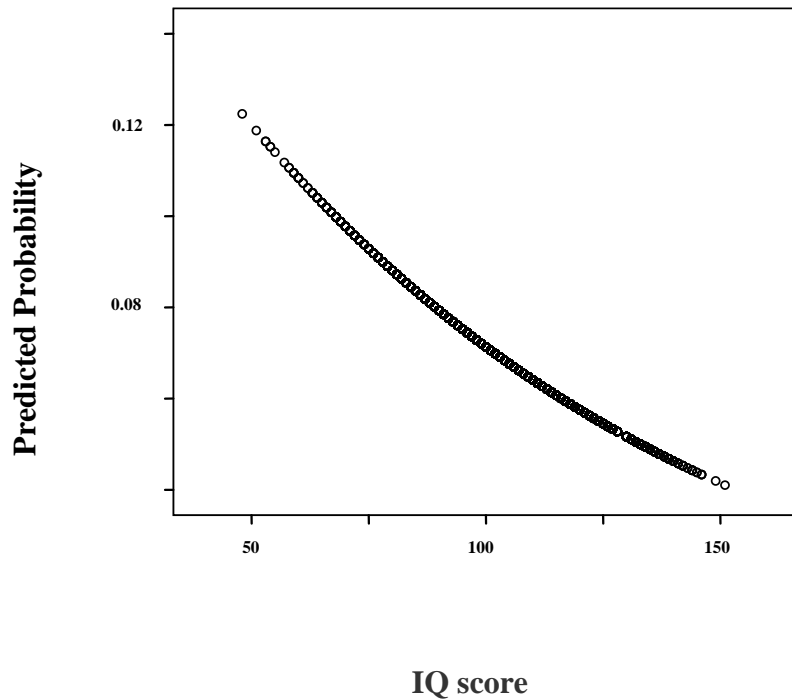
Considering the complexity of personality pathology (Tyrer et al., 2007) it is likely that BPD has a variety of aetiological pathways working in conjunction. A family

environment characterised by conflict, aggression, and anger directed towards the child may impact upon the child by: altering internal schemata of behaviour and relationships (Sharp et al., 2011; Westen Nakash, Thomas, & Bradley, 2006); altering stress responses, such as the HPA axis (Gunnar, 1998); or via interaction with genes (Belsky & Beaver, 2011). All of which may compromise cognitive and emotional regulation (Posner et al., 2003); indeed, individuals with BPD tend to display disturbances in cognitive control processes (Posner et al., 2003; Rogosch & Cicchetti, 2005).

Similarly, an association between IQ and increased psychotic symptoms during adolescence has been observed; curvilinear in nature with both low and high (to a lesser extent) IQ increasing risk (Horwood et al., 2008). Results here, however, suggest a more straightforward linear relationship between IQ and BPD symptoms (**Figure 6.2**), with high IQ possibly acting as a protective factor across the population (Batty, Mortensen, & Osler, 2005). It is likely that higher IQ facilitates the consideration and execution of more varied actions in difficult situations, and the mobilisation of more resources.

Axis I (DSM-IV) diagnoses at 91 months were not associated with BPD symptoms at 11 years, once family adversity, sub-optimal parenting, parent conflict and IQ were considered. While the “complication model” posits a predictive association between Axis I disorders and subsequent personality pathology (Philipsen et al., 2008), the “predisposition model” suggests that Axis I disorders develop subsequent to personality disorders (Sansone & Sansone, 2010).

Figure 6.2 The relationship between IQ score and predicted probability of BPD



Our findings indicate that a diagnosis of anxiety, depression, ADHD or externalising disorder (conduct disorder, oppositional defiant disorder) was not a direct precursor of BPD symptoms, consistent with the “predisposition model.” However, DSM-IV diagnoses here did not include post-traumatic stress disorder (PTSD) or continuous measures of emotional dysregulation. Research suggests that PTSD and emotional dysregulation may be precursors, and features, of BPD (Fruzzetti, Shenk, & Hoffman 2005; Mackinnon & Pies, 2006). PTSD is an acute symptomatic reaction to trauma; emotional dysregulation may develop following prolonged exposure to trauma and abuse (Gunderson & Sabo, 1993). Further, both disorders may result from a disturbance of the HPA axis (Shea, Walsh, MacMillan, & Steiner, 2005; Thomas et al., 2003), with prenatal exposure to maternal stress potentially affecting emotional regulation from

infancy (de Weerth, van Hees, & Buitelaar, 2003; Talge, Neal, & Glover, 2007). Thus, there are various potential routes leading to different stress responses, and PTSD or emotional dysregulation (see chapter 8) could be markers or precursors on one trajectory towards eventual BPD.

6.4.2 Strengths and limitations

Study strengths include the large sample size and the assessment of family adversity before the birth of the child, precluding any reverse causality. The *UK Childhood Interview for DSM-IV Borderline Personality Disorder (UK-CI-BPD)* was adapted from a well-validated instrument, piloted, administered by trained psychologists, and showed high inter-rater reliability. The findings support the presence of a late childhood phenotype for BPD, and buttress current literature (Chanen, Jovev, & Jackson, 2007; Cohen, Crawford, Johnson, & Kasen, 2005), by demonstrating that borderline personality symptoms, identified in late childhood, are associated with similar risk factors to BPD diagnosed in adulthood. However, before firm conclusions can be drawn, it needs to be ascertained whether these BPD symptoms demonstrate predictive validity (Crick, Murray-Close, & Woods 2005), and are related to BPD clinically diagnosed in adulthood.

There was substantial and selective attrition in this study. Those with more family adversity were more likely to have been lost from follow-up. Thus, the study is likely to underestimate the prevalence of BPD symptoms in late childhood (Bernstein et al.,

1993). Despite selective dropout we found strong and hypothesised associations between family adversity, harsh parenting and parent conflict, and BPD symptoms among the remaining, less severely disadvantaged individuals. Wolke *et al.* (2009) demonstrated in simulations that even when dropout is correlated to the predictor/confounder variables, the relationship between predictors and outcome is unlikely to be substantially altered by selective dropout processes. However, it cannot be precluded that selective dropout had an influence on the predictive relationships reported.

Due to the very low prevalence of reported sexual abuse in this sample (0.05%) it was excluded as a predictor, potentially omitting an important experiential factor (Zanarini *et al.*, 2006). Existing research, however, suggests that sexual abuse is not linked to the whole spectrum of BPD diagnoses, and milder forms of BPD (traits or symptoms, as assessed here) may be associated with forms of maladaptive parenting other than sexual abuse (Salzman *et al.*, 1993).

6.4.3 Implications and future directions

Our results suggest that cognitive mechanisms play a direct and weak mediational role in the development of BPD symptoms. Assessing cognition via IQ supports that general cognitive ability relates to psychopathology (Batty *et al.*, 2005). However, given the proposed centrality of cognitive-emotional dysregulation within the BPD construct (LeGris & van Reekum, 2006), it would be prudent for future developmental studies to

tap into the domains of cognitive-emotional dysregulation more directly (see chapter 8). In addition, results here concur with previous studies that exposure to family adversity, harsh parenting and parent conflict may have numerous negative outcomes for children, including lower academic achievement and axis I disorders. Further, we expand current literature by providing prospective evidence of a link between maladaptive parenting and subsequent BPD symptoms at age 11, suggesting that interventions focused on improving parenting may produce wide ranging positive effects (Johnson et al., 2006).

We tentatively speculate that harsh parenting may be a marker for maternal irritable temperament (Siever & Davies, 1991), potentially exposing children to the double jeopardy of an inherited irritable temperament and harsh parenting, which may manifest in subsequent borderline personality symptoms, including affective instability and intense inappropriate anger. Therefore, it would be desirable for future studies to ascertain whether there are prospective links between emotional/irritable temperament and later BPD symptoms. Assessing BPD symptoms in late childhood appears to be a promising avenue for understanding the development of borderline personality disorder.

Chapter Seven: Bullied by Peers in Childhood and Borderline

Personality Symptoms at 11 Years of Age: A Prospective Study

Background: Abuse by adults has been reported as a potent predictor of Borderline Personality Disorder (BPD). Unclear is whether victimisation by peers increases the risk of borderline personality symptoms.

Method: The Avon Longitudinal Study of Parents and Children (ALSPAC) prospective, longitudinal observation study of 6050 mothers and their children. Child bullying was measured by self-report and mother and teacher report between 4 and 10 years. Family adversity was assessed from pregnancy to 4 years, parenting behaviours from 2 to 7 years, sexual abuse from 1.5 to 9 years, and IQ and DSM-IV axis I diagnoses at 7 to 8 years. Trained psychologists interviewed children at 11.7 years to ascertain DSM-IV borderline personality disorder symptoms (5 or more).

Results: Accounting for known confounders, victims of peer bullying had an increased risk of BPD symptoms according to self-report (OR, 2.82; 95% CI, 2.13-3.72); mother report (OR, 2.43; 95% CI, 1.86-3.16); and teacher report (OR, 1.95; 95% CI, 1.34-2.83). Children who were chronically bullied (OR, 5.44; 95% CI, 3.86 - 7.66) or experienced combined victimisation (OR, 7.10; 95% CI, 4.79-10.51) according to child report, had highly increased odds of developing BPD symptoms. Children exposed to chronic victimisation according to mother report were also at heightened risk of developing BPD symptoms (OR, 3.24; 95% CI, 2.24 - 4.68).

Conclusions: Intentional harm inflicted by peers is a precursor or marker on the trajectory towards the development of BPD symptoms in childhood. Clinicians should be adequately trained to deal with, and ask users of mental health services routinely about, adverse experiences with peers.

Wolke, Schreier, Zanarini & Winsper (2012)

Journal of Child Psychology and Psychiatry, early view, doi: 10.1111/j.1469-7610.2012.02542. X

7.1 Introduction

Borderline Personality Disorder (BPD) is a serious and persistent mental illness (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004), affecting between 0.7 and 5.9% of the adult population (Lenzenweger, Lane, Loranger, & Kessler, 2007). It is characterised by persistent instability in affect regulation, impulse control, interpersonal relationships, and self-image (Lieb et al., 2004). Adverse childhood experiences in combination with biological vulnerability and heightened emotional dysregulation are thought to be pertinent in the aetiology of BPD (Crowell, Beauchaine, & Linehan, 2009). Specifically, physical and sexual abuse and neglect (Schmahl, Vermetten, Elzinga, & Bremner, 2004), parental hostility and resentment (Johnson, Cohen, Chen, Kasen, & Brook, 2006) and exposure to domestic violence (Herman, Perry, & van der Kolk, 1989) have been identified as precursors to BPD.

Peer victimisation (bullying) in childhood is a form of systematic abuse of power, and links with suicide ideation (Kaminski & Fang, 2009); psychotic symptoms (Schreier et al., 2009); and neurobiological changes in the brain (Teicher, Samson, Sheu, Polcari, & McGreenery, 2010) have been reported. It is therefore surprising that it has not been investigated in relation to BPD, which encompasses cognitive, emotional, behavioural, and relational symptoms.

There are various mechanisms via which peer victimisation could lead to BPD symptoms. Firstly, physiological responses to peer-related trauma may lead to altered

stress responses (Ouellet-Morin et al., 2011) and exacerbate regulatory problems (Rudolph, Troop-Gordon, & Flynn, 2009), manifesting as the core impulsive and affective instability symptoms of BPD. Secondly, negative peer interactions could impact upon the relational schemata of the child (Salmivalli & Isaacs, 2005), leading to BPD-typical responses, as observed in the relationship difficulties associated with this disorder. Finally, genetic vulnerability related to emotional regulation (Crowell, Beauchaine, & Linehan, 2009) may moderate the impact of exposure to peer victimisation on BPD symptoms, as has been previously demonstrated in relation to depression symptoms (Sugden et al., 2010) .

Large prospective, longitudinal studies pertaining to the developmental precursors of BPD are now necessary to advance aetiological knowledge (Crick, Murray-Close, & Woods, 2005). Indeed, BPD symptoms are unlikely to suddenly appear in adulthood, but may be identified in childhood or adolescence as potential precursors, i.e. a BPD phenotype, on the pathway towards BPD (Reich & Zanarini, 2001; Zanarini et al., 2011).

The current study investigated whether exposure to peer victimisation, in the form of bullying during elementary school, was predictive of clinically relevant (5 or more) BPD symptoms in late childhood. This threshold was chosen, as we were interested in identifying children evincing a BPD phenotype, consistent with BPD diagnosis according to the Diagnostic Statistical Manual. A well tested clinical interview was adapted for the UK, facilitating comparison with adult studies (Zanarini et al., 2011),

and the only extant community-based study of prevalence in children and adolescents (Bernstein et al., 1993). Further, we investigated whether there was a dose-response relationship between combined, severe and chronic victimisation, and the risk of BPD symptoms. Confounders were incorporated into the analysis according to reported prospective associations with personality disorders, including: IQ (Belsky et al., in press; Moran, Klinteberg, Batty, & Vagero, 2009); Axis I disorders (Kasen, Cohen, Skodol, Johnson, & Brook, 1999); maladaptive parenting (Johnson et al., 2006); and sexual abuse (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999).

7.2 Methods

7.2.1 Participants

The Avon Longitudinal Study of Parents and Children (ALSPAC) consists of children from the South West of England who had an expected delivery date between April 1, 1991 and December 31, 1992. The children are considered broadly representative of children in the United Kingdom (Golding, Pembrey, Jones, & Team, 2001). Starting from the first trimester of pregnancy, parents completed regular postal questionnaires regarding family circumstances and the study child's health and development from birth onwards. The study children attended annual face-to-face assessments from 7.5 years of age. This study is based on 6050 children who took part in the Childhood Interview for DSM-IV Borderline Personality Disorder: UK Version (CI-BPD-UK) (Zanarini, Horwood, Waylen, & Wolke, 2004) at 11.7 years of age.

7.2.2 Differences between participants with and without the completed borderline interview

Sample characteristics are shown in Table 7.1. Those lost to follow up were more often boys, minority children, of low birth weight, born to single mothers of lower education level from rented properties, with parents engaged in manual jobs. They were more likely to be born into family adversity, and to have had a psychiatric diagnosis at 7.5 years and a lower IQ at 8 years. Frequency of sexual abuse did not differ between those with or without BPD interviews. Those retained in the study experienced higher mean levels of maternal hitting and hostility.

7.2.3 Ethical approval

Ethical approval for the study was obtained from the ALSPAC Law and Ethics Committee and the Local Research Ethics Committees. Informed consent was obtained from the parents of the children, following explanation of the nature of the study.

7.2.4 Measures

Borderline Personality Disorder Features Interview

Borderline Features were assessed using a face to face semi-structured interview: the *Childhood Interview for DSM-IV Borderline Personality Disorder: UK Version (CI-*

Table 7.1 Dropout Analysis with regard to availability of BPD interview

Characteristic	BPD interview not available	BPD interview Available	Available vs. not available
	N (%)	N (%)	OR (95% CI)
Gender			
Male	4328 (59.6)	2938 (40.4)	[reference]
Female	3669 (54.1)	3112 (45.9)	1.25 (1.16 to 1.34)
Ethnicity			
White	5967 (51.9)	5541 (48.2)	[reference]
Black	395 (64.7)	216 (35.4)	0.59 (0.59 to 0.69)
Birth weight			
≤2499grms	7370 (56.4)	5707 (43.6)	[reference]
≥2500 grms	517 (65.4)	273 (34.6)	0.68 (0.58 to 0.78)
Marital Status			
Single	2206 (66.8)	1095 (33.2)	[reference]
Married	5031 (51.1)	4821 (48.9)	1.93 (1.77 to 2.10)
Home Ownership			
Mortgaged	4701 (49.0)	4901 (51.0)	[reference]
Rented	2532 (72.6)	958 (27.5)	0.36 (0.33 to 0.39)
Educational level of mother			
Below O-Level	2476 (66.2)	1262 (33.8)	[reference]
O-Level or above	4142 (47.5)	4577 (52.5)	2.17 (2.00 to 2.35)
Social Class			
Non-Manual	2729 (46.4)	3152 (53.6)	[reference]
Manual	3210 (56.9)	2430 (43.1)	0.66 (0.60 to 0.71)
DSM-IV Axis I diagnosis			
None	2791 (36.6)	4839 (63.4)	[reference]
At least 1 diagnoses	257 (45.5)	308 (54.5)	0.69 (0.58 to 0.83)
Peer victimisation status			
No victim	1186 (27.6)	3117 (72.4)	[reference]
Any victim	988 (27)	2674 (73)	1.03 (0.93 to 1.14)
	Mean (S.D)	Mean (S.D)	
Family Adversity index	4.32 (4.27)	3.76 (3.87)	0.97 (0.96 to 0.97)
Sexual abuse	0.01 (0.08)	0.01 (0.09)	1.10 (0.72 to 1.67)
Maternal hostility	1.42 (1.66)	1.83 (1.82)	1.15 (1.12 to 1.17)
Maternal hitting	2.64 (1.91)	2.96 (1.98)	1.09 (1.07 to 1.11)
IQ	100.6 (17.2)	105.8 (15.8)	1.02 (0.99 to 1.03)

Abbreviations: N = Number; OR = Odds Ratio; CI = Confidence Intervals; BPD = Borderline Personality Disorder

BPD-UK); based on the borderline module of the Diagnostic Interview for DSM-IV Personality Disorders [DIPD-IV] (Zanarini, Frakenburg, Sickel, & Yong, 1996). The inter-rater reliability (Kappa) of the UK-CI-BPD, assessed from taped interviews of 30 children, ranged from 0.36 to 1.0, (median value 0.88), with 86% of the kappa values in the excellent range (> 0.75) (Zanarini et al., 2011). The interview, carried out by trained psychologists, consisted of nine sections: intense inappropriate anger, affective instability, emptiness, identity disturbance, paranoid ideation/dissociation, frantic efforts to avoid abandonment, suicidal or self-mutilating behaviours, general impulsivity, and intense unstable relationships. A judgment was made as to whether each symptom was definitely present, probably present or absent. A symptom was classed as definitely present if it occurred daily or approximately 25 % of the time (Zanarini et al., 2011), and probable if it had occurred repeatedly but did not meet criterion for definitely present.

Peer victimisation

Peer victimisation was assessed via child report, at 8 and 10 years of age, with the Bullying and Friendship Interview Schedule (Hamburger, Basile, & Vivola, 2011; Wolke, Woods, Bloomfield, & Karstadt, 2000). Trained psychology graduates asked children about victimisation by peers during the previous six months. Five items pertained to overt and four to relational, victimisation (see **Table 7.2**). If children reported either form of bullying, they were asked how frequently it had occurred. Respondents could choose from: *Infrequently*: 1 to 3 times in past 6 months; *Frequently*: more than 4 times in the past 6 months, but less than once a week; and *Very Frequently*:

at least once per week. Overt and relational victims were defined as those experiencing victimisation frequently or very frequently, to capture repeated occasions of bullying rather than isolated events. The following child report victimisation variables were derived:

- 1) **Any peer victimisation** (overt and/or relational at 8 and/or 10 years of age)
- 2) **Chronicity of victimisation:** unstable (reported at one time point); stable (reported at both time points); and never victimised (no report of victimisation)
- 3) **Combined victimisation (i.e. multiple types of victimisation) at 10 years:** both (victim of relational and overt bullying); victim of relational bullying only; victim of overt bullying only; or never victimised. Children receiving both overt and relational victimisation have been previously reported to be more severely affected and to experience more behavioural, emotional or psychotic symptoms (Schreier et al., 2009; Wolke & Samara, 2004).
- 4) **Severity of overt, relational and total victimisation (number of items and frequency) at 8 and 10 years:** Relational (4 items on scale of 0 to 3) and overt (5 items on scale of 0 to 3) items (see **Table 7.2**) were summed to indicate increasing severity of overt and relational victimisation at 8 and 10 years. Thus, overt severity scores ranged from 0 to 15 and relational from 0 to 12. A total severity of victimisation score was derived from totalling the overt (at 8 and 10 years) and relational (at 8 and 10 years) scores across time points and dividing by 4, for ease of interpretation (i.e. producing an average of the four scores so all indexes were on the same scale).

Table 7.2. Peer Victimization variables according to child report at 8 and 10 years

Victimisation items	Derived victimisation variables			
	Any peer victimisation (8 and/or 10 years)	Severity (8 & 10 years)	Chronicity (8 & 10 years)	Combined victimisation (10 years)
Overt victimisation				
1. Having belongings stolen				
2. Having been threatened or blackmailed				
3. Having been hit or beaten up	0 None	Overt severity Sum of 5 items averaged	0 None	0 None
4. Having been called nasty names	1 Overt and/or relational ^a		1 Unstable ^b	1 Overt only ^d
5. Having nasty tricks played on Them		Relational severity Sum of 4 items averaged	2 Stable ^c	2 Relational only ^e
Relational victimisation				3 Both ^f
6. Other children not wanting to play with them				
7. Trying to get them to do something they didn't want to				
8. Spreading lies, rumours about child				
9. Spoiling games to upset child				

All answers are based on report of 4+ times in past 6 months (frequently) or weekly (very frequently); ^a Reported at least 1 from 9 items; ^b Reported at least 1 of 9 items at 8 or 10 years only; ^c Reported at least 1 of 9 items at 8 and 10 years; ^d Reported at least 1 overt item at 10 years; ^e Reported at least 1 relational item at 10 years; ^f Reported at least 1 relational and overt item at 10 years.

A single item included in the Strengths and Difficulties Questionnaire (Goodman, 1997): “Picked on or bullied by other children in the past 6 months” was used to assess peer victimisation according to parent and teacher report. This was rated on a scale from “not true” “somewhat true” to “certainly true” If the response was somewhat or certainly true, at any assessment point (parent: 4, 6.8 and 9 years; teacher: 7 and 10 years), the

child was considered a parent or teacher reported victim of bullying, respectively (Schreier et al., 2009).

A chronicity variable was also constructed for mother (none; unstable = 1 time point; stable = 2 or 3 time points) and teacher (none; unstable = 1 time point; stable = 2 time points) report.

Potential confounders

Sexual abuse was assessed using one item included in the *upsetting events questionnaire* completed by the mother (“He/she was sexually abused”) when the study child was 1.5, 2.5, 3.5, 4.8, 5.8, 6.8 and 8.6 years old. If any sexual abuse occurred across the 7 time points it was scored as present. These items were then summed for a total sexual abuse score across the 7 time points.

Maladaptive parenting was assessed using indicators of maternal hitting (2, 3.5 & 6.4 years) and hostility (2, 4 & 7 years) according to parental report. Hitting was coded at 2 and 3.5 years on a scale of 1-4 and at 6.4 years on a scale of 1-2, with higher scores representing increasing frequency of hitting. An overall hitting variable was constructed by summing these 3 scales to produce a score from 0 to 10. Hostility was indicated by 4 items, e.g. *mum feels that whining makes her want to hit child* (Waylen, Stallard, & Stewart-Brown, 2008) at 2 and 4 years and 3 items at 7 years. These items were summed to give a total maternal hostility score from 0 to 7.

Multiple family risk factors during pregnancy (long index); birth to 2 years (long index); and 2 to 4 years (short index), were assessed using the Family Adversity Index (FAI) (Bowen, Heron, Waylen, Wolke, & Team, 2005). The FAI long version consists of 18 items e.g. financial difficulties, maternal affective disorder; and the short index has 15 of the same items, with the following 3 items not incorporated: social, practical and financial support. If an adversity item was reported, it was recorded as 1 point, and the points were then summed to derive a total FAI index score for each time point. The three FAI indexes were summed and entered into the analysis as a continuous variable, in accordance with suggested use (Bowen et al., 2005).

An abbreviated form of the Wechsler Intelligence Scale for Children (WISC) -III (UK version) was administered during the assessment clinic (8 years) deriving an overall intelligence quotient (IQ) (Wechsler, Golombok, & Rust, 1992). DSM-IV psychiatric diagnoses according to parent and teacher reports were made at 7.5 years, using the Developmental and Wellbeing Assessment (DAWBA). The diagnoses were made using a DSM-IV-TR algorithm, and reviewed by two experienced child psychiatrists (Robert Goodman, Tamsin Ford). The DAWBA has been validated for axis I diagnoses and shown to have utility as a clinical assessment tool (Goodman, Ford, Richards, Gatward, & Meltzer, 2000) (for further information see [http:// www.dawba. com/](http://www.dawba.com/)). The presence of any Axis I diagnosis of attention deficit-hyperactivity disorder, conduct disorder, oppositional defiant disorder, depression or anxiety versus no diagnosis was recorded.

7.2.5 Statistical Analysis

All analyses were carried out using SPSS version 18. Logistic regression models were used to estimate odds ratios (OR) with 95% confidence intervals (CI). The outcome variable was borderline personality disorder (BPD) symptoms, which was based on the presence of 5 probable or definite symptoms (for more details see Zanarini et al., 2011). Gender differences were assessed for BPD symptoms and peer victimisation variables (**Table 7.3**). Crude associations between peer victimisation and presence of BPD symptoms were computed. The experience of being a victim of any type (child, parent and teacher report); chronicity (child, parent and teacher report); severity (number of items and frequency); and combined (relational and overt) victimisation were the independent variables (**Table 7.4**). The analyses were repeated controlling for potential confounders in multiple logistic regression analyses, using the forced entry method, i.e. all variables were entered together (**Table 7.5**). Model A is based on the full dataset of children who completed the BPD interview, adjusted for gender, and age at BPD assessment. Model B also controlled for gender and age only, but was conducted with the reduced data set, including only participants with information on all confounders used in model C. The analyses for model C are also based on this reduced dataset, controlling for age, gender, and additionally FAI, DSM-IV diagnoses, sexual abuse, maternal hitting and hostility and IQ.

7.3 Results

7.3.1 Frequency of BPD and peer victimisation

Overall, 7.3% of the sample had 5 or more probable/definite BPD symptoms, and the prevalence according to gender was remarkably similar (female 7.4%; male 7.3%). Prevalence rates for any peer victimisation at any time point were as follows: child report: 46.2%, mother report: 37.0% and teacher report: 14.1%. At one time point (e.g. at 10 years reported by children, **Table 7.3**) any victimization was 23.9%. This one time point prevalence is fairly similar to reported prevalence rates ranging from 15% to 30% (Analitis et al., 2009; Stassen Berger, 2007); and the relative prevalence according to informant is congruent with previous reports, suggesting that victimisation is not always recognised by teachers (Ronning et al., 2009). Any, overt or chronic peer victimisation was more frequent in boys than girls independent of informant (child, mother or teacher). In contrast, relational victimisation was more frequent in girls (**Table 7.3**).

Victimisation (child, parent and teacher report) was a significant predictor of BPD symptoms (**Table 7.4**). Significant crude associations included: child report (Odds Ratio: 3.14; 95% Confidence Intervals: 2.51 - 3.92); mother report (2.48; 2.03 - 3.04); and teacher report (2.05; 1.55 - 2.70). According to child report, both chronic (6.28; 4.67 - 8.43) and combined victimisation (7.19; 5.28 - 9.80) evinced especially strong associations with BPD symptoms compared to those not exposed.

Table 7.3 Frequency of BPD and peer victimisation variables by total and gender

	Total N (%)	Females N (%)	Males N (%)	Females vs. Males OR (95% CI)
BPD				
No	5606 (92.7)	2882 (92.6)	2724 (92.7)	
Yes	444 (7.3)	230 (7.4)	214 (7.3)	1.02 (0.83-1.24)
Peer victimisation				
<u>Any victim by informant</u>				
<u>Child^b</u>				
No	3117 (53.8)	1705 (57.1)	1412 (50.4)	1 [Reference]
Yes	2674 (46.2)	1282 (42.9)	1392 (49.6)	0.76 (0.68-0.84)
<u>Mother</u>				
No	3682 (63.0)	1987 (66.0)	1695 (59.8)	1 [Reference]
Yes	2167 (37.1)	1025 (34.0)	1142 (40.3)	0.77 (0.68-0.85)
<u>Teacher</u>				
No	3814 (85.9)	2062 (90.0)	1752 (81.5)	1 [Reference]
Yes	626 (14.1)	229 (10.0)	397 (18.5)	0.49 (0.41-0.58)
<u>Chronicity of peer victimisation</u> (Child Report)				
None	2457 (50.8)	1356 (54.0)	1101 (47.4)	1 [Reference]
Unstable ^b	1720 (35.6)	857 (34.1)	863 (37.1)	0.81 (0.71-0.91)
Stable ^c	660 (13.6)	300 (11.9)	360 (15.5)	0.68 (0.56-0.80)
<u>Combined victimisation^d</u>				
None	4117 (76.1)	2205 (78.8)	1912 (73.2)	1 [Reference]
Overt only	874 (16.2)	361 (12.9)	513 (19.6)	0.61 (0.52-0.71)
Relational only	151 (2.8)	98 (3.5)	53 (2.0)	1.60 (1.14-2.26)
Overt and relational	270 (5.0)	135 (4.8)	135 (5.2)	0.87 (0.67-1.11)

Abbreviations: N=number; OR=odds ratio; CI=confidence intervals; BPD=borderline personality disorder;

^aBoldface type indicates that the 95% CI does not include 1.00; ^bAny victimisation (overt or relational) at either 8 or 10 years; ^cvictimisation at both 8 and 10 years; ^dat age 10 years.

7.3.2 Crude associations between peer victimisation and borderline personality symptoms

Further, those exposed to chronic victimisation were (2.65; 2.03 - 3.46) times more likely to evince BPD symptoms than those exposed to unstable victimisation; and those exposed to both types of victimisation were (2.41; 1.71 - 3.38) times more likely than those exposed to overt, and (6.26; 2.91 - 13.41) times more likely than those exposed to

Table 7.4 Crude associations between peer victimisation and BPD status

Peer victimisation status	Probable/definite BPD status	
	N (%)	OR (95% CI) ^b
Any victim by informant		
(Total N in brackets)		
<u>Child</u>		
No (3117)	118 (3.8)	[reference] ^a
Yes (2674)	294 (11.0)	3.14 (2.51 to 3.92)
<u>Mother</u>		
No (3682)	179 (4.9)	[reference]
Yes (2167)	244 (11.3)	2.48 (2.03 to 3.04)
<u>Teacher</u>		
No (3814)	238 (6.2)	[reference]
Yes (626)	75 (12.0)	2.05 (1.55 to 2.70)
Chronicity of peer victimisation		
<u>Child</u>		
No (2457)	84 (3.4)	[reference] ^c
Unstable vs. none (1720)	133 (7.7)	2.37 (1.78 to 3.14)
Stable vs. none (660)	120 (18.2)	6.28 (4.67 to 8.43)
Stable vs. unstable		2.65 (2.03 to 3.46)
<u>Mother</u>		
No (2886)	143 (5.0)	[reference]
Unstable vs. none (911)	79 (8.7)	1.82 (1.37 to 2.42)
Stable vs. none (483)	68 (14.1)	3.14 (2.31 to 4.27)
Stable vs. unstable		1.73 (1.22 to 2.44)
<u>Teacher</u>		
No (3814)	238 (6.2)	[reference]
Unstable vs. none (565)	63 (11.2)	1.89(1.41 to 2.53)
Stable vs. none (61)	12 (19.7)	3.68 (1.93 to 7.01)
Stable vs. unstable		1.95 (0.99 to 3.87)
Combined victimisation		
None (4117)	191 (4.6)	[reference] ^c
Overt vs. none (874)	111 (12.7)	2.99 (2.33 to 3.83)
Relational vs. none (151)	8 (5.3)	1.15 (0.55 to 2.38)
Both vs. none (270)	70 (25.9)	7.19 (5.28 to 9.80)
Relational vs. overt		0.38 (0.18 to 0.80)
Both vs. overt		2.41 (1.71 to 3.38)
Both vs. relational		6.26 (2.91 to 13.41)

N = Number with BPD symptoms; OR = Odds Ratio; CI = Confidence Intervals; BPD = Borderline Personality Disorder; ^aReference group in all analyses consists of probands who haven't been victimised; ^bBold indicates that the 95% CI does not include 1.00; ^cReference group for all comparisons labelled vs. none.

relational victimisation to evince BPD symptoms (**Table 7.4**). Similarly, chronic exposure according to mother report was more strongly associated with BPD symptoms than intermittent (1.73; 1.22 – 2.44) and no exposure (3.14; 2.31-4.27). Chronic exposure according to teacher report was more strongly associated with BPD symptoms than no exposure (3.68; 1.93 - 7.01). Severity of exposure to both relational: 8 years

Table 7.5 *Associations between increasing severity of victimisation and BPD symptoms*

	Crude Associations OR (95% CIs)	Model A OR (95% CIs)	Model B OR (95% CIs)	Model C OR (95% CIs)
Overt severity at 8 years ^a	1.21 (1.17 to 1.26)	1.22 (1.17 to 1.26)	1.23 (1.18 to 1.28)	1.21 (1.16 to 1.26)
Relational severity at 8 years ^b	1.23 (1.17 to 1.29)	1.23 (1.18 to 1.29)	1.25 (1.19 to 1.32)	1.23 (1.17 to 1.30)
Overt severity at 10 years ^a	1.35 (1.30 to 1.41)	1.36 (1.30 to 1.42)	1.38 (1.31 to 1.45)	1.36 (1.29 to 1.43)
Relational severity at 10 years ^b	1.37 (1.30 to 1.45)	1.38 (1.30 to 1.45)	1.37 (1.29 to 1.46)	1.35 (1.26 to 1.44)
Total severity of victimisation ^c	1.57 (1.48 to 1.66)	1.58 (1.49 to 1.67)	1.62 (1.51 to 1.75)	1.59 (1.47 to 1.71)

^aSeverity index: 5 overt items summed (scale 0 - 15); ^bSeverity index: 4 relational items summed (scale 0 - 12); ^cSeverity index: overt and relational symptoms summed across both time-points and averaged, i.e. divided by four (scale 0 - 13); Abbreviations: OR: Odds Ratio, CI: Confidence Intervals. Model A presents logistic regression results for the full data set controlling for age and gender; Model B refers to the reduced data set controlling for age and gender; Model C refers to the reduced data set controlling for age, gender, total Family Adversity Index (FAI), maternal hitting and hostility, DSM-IV Axis I diagnosis, IQ and sexual abuse.

1.23 (1.17 to 1.29) and 10 years 1.37 (1.30 to 1.45) and overt: 8 years (1.21; 1.17 to 1.26) and 10 years (1.35; 1.30 to 1.41), victimisation was predictive of BPD symptoms. Total severity (combined and chronic) of victimisation was more strongly predictive of BPD symptoms: (1.57; 1.48 to 1.66) (**Table 7.5**).

7.3.3 Associations between peer victimisation and BPD symptoms controlling for possible confounders

Peer victimisation according to child report at age 8 or 10 years and BPD symptoms were associated with the following possible confounders: FAI, DSM-IV diagnoses (DAWBA), IQ, maternal hitting and hostility (**Table 7.7**) and gender (**Table 7.3**). Sexual abuse evinced a tendency towards increased BPD symptoms, but the association was not significant (**Table 7.7**). When controlling for age and gender, associations were very similar in the full (model A) and reduced (model B: only cases with information on all potential confounders) data sets (**Table 7.6**). Incorporating all known confounders into the analysis (Model C) led to minor changes in the observed associations, with the exception of teacher reported chronic victimisation, which was no longer predictive of BPD symptoms (OR; 95% CI: 1.97; 0.67 - 5.82). Any victimisation: child: 2.82 (2.13 - 3.72); mother: 2.43 (1.86 - 3.16); and teacher: 1.95 (1.34 - 2.83); child reported chronic victimisation: 5.54 (3.86 - 7.66); mother reported chronic victimisation: 3.24 (2.24 - 4.68); combined victimisation: 7.10 (4.79 - 10.51), and total severity of victimisation: 1.59 (1.47 to 1.71) all remained little changed with the addition of confounding variables.

7.4 Discussion

To our knowledge, this is the first study to explore the prospective association between peer victimisation and BPD symptoms. Any peer victimisation in primary school was a predictor of BPD symptoms at age 11.7 years. In particular, children who were exposed

Table 7.6 Associations between peer victimisation and BPD controlling for potentially confounding factors

	Model A OR (95% CI)	Model B OR (95% CI)	Model C OR (95% CI)
Peer victimisation status ^a			
Any victim by informant			
Child (N_A = 5791 N_{B/C}=4161)			
Yes vs. No	3.16 (2.54-3.94)	3.12 (2.37-4.10)	2.82 (2.13-3.72)
Mother (N_A = 5849 N_{B/C}=4161)			
Yes vs. No	2.50 (2.04-3.05)	2.82 (2.18-3.63)	2.43 (1.86-3.16)
Teacher (N_A = 4440 N_{B/C}=3073)			
Yes vs. No	2.09 (1.58-2.76)	2.25 (1.56-3.24)	1.95 (1.34-2.83)
Chronicity			
Child (N_A = 4837 N_{B/C}=3856)			
None			
Unstable vs. none	2.39 (1.81-3.17)	2.18 (1.58-3.00)	2.02 (1.46-2.79)
Stable vs. none	6.40 (4.77-8.61)	6.27 (4.48-8.77)	5.44 (3.86-7.66)
Stable vs. unstable	2.68 (2.05-3.49)	2.88 (2.11-3.93)	2.70 (1.97-3.69)
Mother (N_A = 4280 N_{B/C}=3457)			
None			
Unstable vs. none	1.84 (1.38-2.44)	2.06 (1.49-2.86)	1.85 (1.32-2.58)
Stable vs. none	3.20 (2.35-4.35)	3.94 (2.78-5.59)	3.24 (2.24-4.68)
Stable vs. unstable	1.74 (1.23-2.46)	1.91 (1.30-2.81)	1.75 (1.18-2.60)
Teacher (N_A = 4400 N_{B/C}=3073)			
None			
Unstable vs. none	1.93 (1.43-2.59)	2.24 (1.54-3.27)	1.95 (1.33-2.87)
Stable vs. none	3.84 (2.00-7.37)	2.34 (0.81-6.76)	1.97 (0.67-5.82)
Stable vs. unstable	2.00 (1.01-3.96)	1.04 (0.35-3.12)	1.01 (0.33-3.07)
Combined victimisation (N_A =5142 N_{B/C} = 3914)			
None			
Overt vs. None	3.03 (2.36-3.88)	2.96 (2.18-4.02)	2.68 (1.96-3.66)
Relational vs. None	1.15 (0.56-2.38)	1.07 (0.43-2.68)	0.99 (0.40-2.49)
Both vs. None	7.25 (5.32-9.87)	7.78 (5.28 -11.44)	7.10 (4.79-10.51)
Relational vs. Overt	0.38 (0.18-0.80)	0.36 (0.14-0.92)	0.37 (0.15-0.95)
Both vs. Overt	2.40 (1.71-3.35)	2.63 (1.72-4.02)	2.65 (1.72-4.08)
Both vs. Relational	6.31 (2.94-13.53)	7.26 (2.77-19.00)	7.15 (2.72-18.79)

^a NA refers to the total N in model A, N_{B/C} refers to the total N in models B and C. Model A presents logistic regression results for the full data set controlling for age and gender; Model B refers to the reduced data set controlling for age and gender; Model C refers to the reduced data set controlling for age, gender, total Family Adversity Index (FAI), maternal hitting and hostility, DSM-IV Axis I diagnosis, IQ and sexual abuse.

to combined victimisation (overt and relational) or experienced chronic victimisation (at 8 and 10 years) were at *highly* increased risk for developing BPD symptoms, indicating a dose-response relationship. We found comparable associations using mother and teacher report; therefore, the observed relationships between victimisation and BPD cannot be attributed solely to self-report bias, i.e. the tendency of individuals with BPD to misinterpret or misreport (Bailey & Shriver, 1999) peer victimisation experiences. Furthermore, the addition of all possible confounders into the model, led to negligible changes in the strength of associations. This supports that the observed associations were not due to confounding effects of the examined variables, and is suggestive of a causal relationship between peer victimisation and BPD symptoms. This interpretation is congruent with recent prospective studies revealing links between exposure to bullying and the development of psychopathology, including: internalising problems and psychotic symptoms (Arseneault et al., 2011; Arseneault et al., 2008).

A substantial dose-response relationship was found for peer-combined victimisation, increasing severity (i.e. exposed to more behaviours, more often) of both forms of victimisation, and chronicity of exposure. By using a dimensional measure of overt and relational victimisation, it became clear that both relational and overt forms of victimisation are predictive of BPD symptoms. According to child report, those who experienced both relational and overt peer victimisation had 7 times increased odds of BPD symptoms compared to those not exposed. Similarly, children who were victims of bullying at 8 and 10 years had 5.5 times increased odds of BPD symptoms compared to those never victimised.

Table 7.7 Associations between Potentially Confounding Factors and Peer Victimisation and BPD

	no victimisation	victimisation ^a	victimisation vs. no victimisation	no BPD	BPD	BPD vs. no BPD
	N (%)	N (%)	OR (95%)	N (%)	N (%)	OR (95%)
DAWBA ^b						
None	2593 (55)	2125 (45)	[reference]	4516 (93.3)	275 (89.3)	[reference]
1 +	121 (40)	181 (60)	1.83 (1.44 to 2.31)	323 (6.7)	33 (10.7)	1.69 (1.15 to 2.45)
Family Adversity	3.41 (3.55)	4.07 (4.10)	1.05 (1.03 to 1.06)	3.67 (3.79)	4.91 (4.64)	1.07 (1.05 to 1.10)
Maternal Hit	2.82 (1.93)	3.13 (2.02)	1.08 (1.06 to 1.11)	2.93 (1.98)	3.22 (1.96)	1.08 (1.02 to 1.13)
Maternal Hostility	1.68 (1.75)	2.03 (1.90)	1.11 (1.08 to 1.14)	1.79 (1.81)	2.31 (1.93)	1.15 (1.10 to 1.21)
Sexual Abuse	0.01 (0.09)	0.01 (0.09)	0.98 (0.54 to 1.76)	0.005 (0.08)	0.01 (0.14)	2.02 (0.96 to 4.23)
IQ	106.6 (15.7)	104.9 (15.9)	0.99 (0.99 to 0.995)	105.98 (15.8)	103.12 (16.2)	0.99 (0.98 to 0.99)

Abbreviations: N = Number; OR = Odds Ratio; CI = Confidence Intervals; BPD = Borderline Personality Disorder; Boldface indicates significant associations. ^a Victimisation relates to any reported victimisation by child at 8 or 10 years; ^b Refers to any DSM-IV diagnoses at 7.5 years

This pattern was also observed for mother reported chronic victimisation, though to a lesser extent of approximately 3.5 times increased odds of BPD symptoms. In terms of effect size, the reported odds ratios pertaining to chronicity, according to both child and mother report, may be interpreted as moderate to strong (Ferguson, 2009).

Although an increased dose-response relationship, in terms of frequency (Lataster et al., 2006), chronicity and combined victimisation, i.e. overt and relational victimisation (Schreier et al., 2009), has been reported previously for psychotic symptoms; the associations here are especially strong, and a pattern of increasing association dependent on chronicity, was observed according to both child and mother report. In contrast, the same dose-response relationship was not found for teacher reports.

Why does chronic, severe or combined victimisation have an especially strong impact on BPD symptoms? BPD is characterised by unstable and intense relationships, affective dysregulation, and a broad incapacity to trust the actions and motives of others (Crowell, Beauchaine, & Linehan, 2009). Research indicates that peer victimisation may work itself “under the skin” of victims, both psychologically and physiologically. Psychologically, victimisation may impact upon schemata or internal working models pertaining to relationships, disrupting the individual’s ability to appropriately trust and interact with others, leading to unstable relationships, biased perceptions, and emotional dysregulation (Staebler et al., 2011). Feeling betrayed by peers, loneliness, anger, and loss of trust are experiences consistently described by victims of bullying (Stassen Berger, 2007), and have recently been observed in adolescents with BPD symptoms

(Sharp et al., 2011). Further, individuals with BPD struggle to trust, or “maintain co-operation,” with others during experimental social trust games, and work from pathological norms or models when planning strategies (King-Casas et al., 2008).

Physiologically, victimisation is a trauma which works itself “under the skin” by altering stress response (Ouellet-Morin et al., 2011) and impacting upon brain structures (Teicher et al., 2010a), such as the anterior insula (Teicher et al., 2010b). Neuroimaging and neuropsychological studies suggest that the anterior insula plays an important role in social cognition and emotion. Specifically, empathy, compassion and interpersonal phenomena, such as fairness and co-operation, have all been linked to activity within the anterior insula (Lamm & Singer, 2010). Subsequently, individuals exposed to chronic victimisation may not experience a “gut feeling” in response to socially inappropriate behaviour (King-Casas et al., 2008), and thus, not act accordingly in order to maintain, or repair, relationships (Meyer-Lindenberg, 2008). Therefore, it is not surprising that the strongest effects of victimisation have been observed for symptom complexes with psychotic (Arseneault et al., 2011; Schreier et al., 2009) or BPD constellations, where social dysfunction plays an important role (Meyer-Lindenberg, 2008).

Alternatively, victims differ from children not involved in bullying in aspects other than those examined. They are often withdrawn, unassertive, physically weak, easily emotionally upset, angry, have poor social understanding, no or few friends, and are often bullied by their siblings (Monks et al., 2009). All of these features potentially make these individuals more likely targets of peers (Sapouna et al., 2011), and may lead

to the development of BPD, independently of victimisation experiences. Viewed from this perspective, victimisation may be a marker within a developmental ‘risk factor’ model of BPD, rather than a cause (Miller, Muehlenkamp, & Jacobson, 2008), possibly resulting from adverse family relationships (Barker et al., 2008) or genetic origins (Ball et al., 2008).

This study has a number of strengths. A longitudinal, prospective design was utilised with bullying assessments available during childhood and BPD symptoms at 11.7 years. Direct and detailed assessments of peer victimisation and BPD symptoms in childhood were used, and there were multiple informants of peer victimisation. Further, the BPD interview is well validated with high inter-rater reliability. Prevalence rates in this study are similar to those reported in other studies in the UK, and children were drawn from the general population; therefore, confounding effects of treatment seeking can be ruled out. Finally, information was available on a variety of possible confounding factors.

With respect to the limitations, although BPD symptoms were assessed approximately two to up to six years after the bullying assessment, it is not known at what age BPD symptoms were first manifest, and there is no measure of BPD symptoms prior to the bullying assessment. Therefore, it is possible that BPD symptoms were present before exposure to peer victimisation, and may have led to an increased risk of victimisation. Further, emotional instability or irritability may be potential precursors of both victimisation and BPD symptoms (Crowell, Beauchaine & Linehan, 2009). However,

the relationship between victimisation and BPD symptoms was not affected by general mental health problems assessed at age 7.

Furthermore, BPD symptoms were based on interviews with the children, and strongest relationships with victimisation, were found according to child reports. Although these were replicated with mother and teacher reports, relationships may be inflated due to use of the same informant for predictor and outcome.

The BPD interview was conducted on just less than half the total cohort. However, peer victimisation itself was not related to selective dropout. Under these circumstances the relationship between predictors and BPD symptoms is unlikely to be substantially altered by selective dropout processes as shown in simulations (Wolke et al., 2009), but it cannot be ruled out.

Finally, concern has been expressed regarding whether BPD symptoms can, or should, be diagnosed in adolescence (Goodman & Siever, 2011). The alternative would have been the use of a dimensional scale of BPD symptoms (Belsky et al., in press; Crick, Murray, Close, & Woods, 2005). However, there is growing evidence for the existence of adolescent-onset BPD, and recognition of its negative consequences for facets of adult functioning (Chen et al., 2006), and subsequently, the need for early treatment (Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008).

A major implication of our findings is that chronic or severe peer victimisation has non-trivial adverse long-term consequences, particularly for the development of BPD symptoms in a non-clinical population. Reducing peer victimisation, and the resulting stress caused to victims (Farrington & Ttofi, 2009), should be a target for prevention and intervention in child and adolescent services. Clinicians should be aware of the importance of adverse interpersonal experiences with peers in respect to BPD; and be adequately trained to deal with, and routinely ask users of mental health services about, such experiences.

Chapter Eight: Dysregulated Behaviour in Early and Middle Childhood and Borderline Personality Disorder Symptoms at 11 years: A Test of the Biosocial Developmental Model

Abstract

Context: The biosocial developmental model (BDM) of Borderline Personality Disorder (BPD) proposes that early vulnerability, indicated by behavioural and emotional dysregulation, is potentiated across development by environmental risk factors, culminating in BPD. However, empirical research pertaining to this hypothesis is lacking.

Objective: To determine whether dysregulated behaviour in childhood is predictive of BPD symptoms in early adolescence, and whether this association is mediated or potentiated by negative parent or peer interactions.

Design: Prospective cohort study.

Setting: Assessment clinic for 11 year-old members of the Avon Longitudinal Study of Parents and Children (ALSPAC) in Bristol.

Participants: A total of 5,711 children with BPD interviews and dysregulated behaviour measures.

Main Outcome Measure: The UK Childhood Interview for DSM-IV Borderline Personality Disorder (UK-CI-BPD) based on the borderline module of the Diagnostic Interview for DSM-IV Personality Disorders. Trained assessors judged whether five or more BPD probable/ definite symptoms were present.

Results: Stable dysregulated behaviour and experience of harsh parenting and peer victimisation during childhood, predicted BPD symptoms at 11 years. The association between dysregulated behaviour and BPD was strongly related to whether the child had experienced peer victimisation: as level of dysregulation increased, the strength of association between

dysregulation and BPD, via peer victimisation, significantly increased. The indirect effect from dysregulated behaviour via peer victimisation to BPD outcome was significantly stronger than for psychotic or depression outcomes. This difference became more pronounced as level of dysregulation increased.

Conclusions: *Consistent with biosocial developmental theory, exposure to an invalidating environment (peer victimisation) in dysregulated individuals increases the risk of BPD symptoms specifically. Interventions targeting early dysregulated behaviour or peer victimisation may reduce the development of BPD symptoms.*

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Under Review: American Journal of Psychiatry

8.1 Introduction

Borderline Personality Disorder (BPD) is characterised by emotional instability, a lack of impulse control, disturbances in self-image, chronic feelings of emptiness and intense unstable relationships (Lieb, Zanarini, Schmahl, Linehan, & Bohus 2004). Historically, research exploring the aetiology of BPD has focused on experiential factors including abuse (Zanarini, 2000), parenting (Johnson, Cohen, Chen, Kasen, & Brook 2006), and attachment relationships (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004). These factors, while associated with BPD, are also related to other psychopathologies, therefore demonstrate a lack of predictive specificity (Paris, Zweig-Frank, & Guzder, 1994a; Paris, Zweig-Frank, & Guzder, 1994b). It has been suggested, however, that by considering the reciprocal transactions between biological vulnerability and environmental risk factors, predictive specificity could be improved (De Clercq & De Fruyt, 2007; Rutter, 1987).

Temperament is a term commonly used to describe individual differences in emotions or attention, and the ability to regulate these (Caspi & Shiner, 2008), and while it has a genetic basis, indicating biological liability, it is also subject to maturation and experience. Representing the kernel features from which adult personality develops (Rothbart, Ahadi, & Evans 2000), temperament demonstrates a degree of continuity across time and is assessed as traits expressed in behaviour (Rothbart, 2007; Rutter, 1987).

Emotional and behavioural dysregulation are features of BPD. The nature of the relationship between biological vulnerability, indicated by a dysregulated behaviour trait, and BPD has been considered in the *biosocial theory* (Linehan, 1993), recently reconceptualised as the *biosocial developmental model (BDM)* (Crowell, Beauchaine & Linehan, 2009). The BDM posits that an overly emotional or dysregulated behaviour trait, in combination with exposure to environmental risk factors, leads to the development of BPD (Crowell et al., 2009; Linehan, 1993). Early dysregulation has been operationalised in different ways, but usually incorporates the summation of anxious/ depressed, impulsive aggressive and attentional problems (Althoff, Verhulst, Rettew, Hudziak, & van der Ende, 2010; Jucksch et al., 2011; Meyer et al., 2008).

Although the biosocial developmental model is clinically (Linehan et al., 2006) and theoretically (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008) popular, it has yet to be tested longitudinally (Crowell et al., 2009). Therefore, to test the BDM theory of BPD, the following steps of analysis were undertaken: Firstly, it was determined whether children, assessed in this study, evinced stable dysregulated behaviour between four and eight years of age, thus indicating the presence of an underlying dysregulated behaviour trait. Secondly, the independent associations between dysregulated behaviour, environmental risk factors (harsh parenting and peer victimisation), and subsequent BPD symptoms at 11 years were assessed. Thirdly, the combined effects (via moderation and/or mediation) of dysregulated behaviour and environmental risk factors, on the development of BPD symptoms, were assessed. This was to determine whether environmental risk factors add to, or potentiate dysregulated behaviour. Finally, to test

the specificity of the BDM, the associations from dysregulated behaviour classes via environmental risk factors to BPD outcome were compared to those of psychotic and depression outcomes.

8.2 Methods

8.2.1 Participants

The ALSPAC (Avon Longitudinal Study of Parents and Children) birth cohort enrolled 14, 541 women, resident in the English region of Avon, if they had an expected delivery date between 1st April 1991 and 31st December 1992. A total of 13,971 children formed the original cohort (Golding, Pembrey, Jones & the ALSPAC study team, 2001). Ethical approval for this investigation was obtained from the ALSPAC Law and Ethics committee and the local research ethics committee.

Inclusion in the final sample was dependent on two criteria: 1. A completed BPD interview at 11 years; 2. At least one dysregulated behaviour measure at either 4, 7 or 8 years of age (76.9% had full dysregulation scores). Five thousand, seven hundred and eleven cases met these criteria. Those excluded were more often male, exposed to family adversity, had significantly higher mean dysregulated behaviour scores at 4, 7 or 8 years, and were more often victims of bullying. Further, they had significantly higher mean depression scores between the age of 11 and 12 years, and were more likely to report 1 or more psychotic symptoms at 12 years (See **Table 8.1**).

Table 8.1 Comparison of Participants Retained and Excluded from the Analysis

Predictor or outcome measure	Retained in analysis		Excluded from analysis		P value (chi square/ t-test)
	N	% or mean±SD	N	% or mean±SD	
Family Adversity Index	5662		7567		
No adversity	2660	47%	2634	35.8%	Reference
1 to 2 adversities	2293	40.5%	3219	42.5%	<0.001
3+ adversities	709	12.5%	1714	22.7%	<0.001
Gender	5711		8336		
Male	2771	48.5%	4495	53.9%	Reference
Female	2940	51.5%	3841	46.1%	<0.001
Dysregulation 4 years (mother report)	5123	6.29 (3.96)	3260	6.85 (4.29)	<0.001
Dysregulation 7 years	5295	7.12 (3.79)	4163	7.72 (4.00)	<0.001
Dysregulation 8 years	4935	6.27 (4.17)	2853	6.98 (4.64)	<0.001
Peer victimisation at 8 years (child report)	4805		1858		
No victimisation	2945	61.3%	1051	56.6%	Reference
Victimisation	1860	38.7%	807	43.4%	<0.001
Peer victimisation at 9 years (mother report)	4916		2573		
No victimisation	3869	78.7%	1934	75.2%	Reference
Victimisation	1047	21.3%	639	24.8%	<0.01
Peer victimisation at 10 years (child report)	5176		1650		
No victimisation	3954	76.4%	1200	72.7%	Reference
Victimisation	1222	23.6%	450	27.3%	<0.01
Harsh parenting at 9 years (mother report)	5105		2707		
No harsh parenting	4837	94.8%	2546	94.1%	Reference
Harsh parenting	267	5.2%	161	5.9%	p=0.186
BPD symptoms at 11 years	5711		339		
less than 5 symptoms	5299	92.8%	307	90.6%	Reference
more than 5 symptoms	412	7.2%	32	9.4%	p = 0.127
Depression symptoms (mean score)					
Early (11 to 12 years)	4119	6.03 (4.95)	1641	6.66 (5.45)	<0.001
Late (13 to 14 years)	3920	7.04 (6.03)	1118	7.42 (6.06)	P=0.061
Psychosis - like symptoms	4826		1611		
No symptoms	4231	87.7%	1326	82.3%	
1 or more symptoms	595	12.3%	285	17.7%	< 0.001

8.2.2 Measures

Symptoms of Borderline Personality Disorder at 11 years were assessed using the *UK Childhood Interview for DSM-IV Borderline Personality Disorder [UK-CI-BPD]* (Zanarini, Horwood, Waylen, & Wolke, 2004), based on the borderline module of the Diagnostic Interview for DSM-IV Personality Disorders [DIPD-IV] (Zanarini, Frankenberg, Sickle & Young, 1996). The inter-rater and test re-test reliability of the DIPD-III, DIPD-III-R and DIPD-IV are good to excellent (Zanarini et al., 2000; Zanarini & Frankenberg, 2001). The inter-rater reliability (Kappa) of the *UK-CI-BPD*, assessed from taped interviews of 30 children ranged from 0.36 to 1.0 (median value 0.88) with 86% of the Kappa values within the excellent range of > 0.75 (Zanarini et al., 2011).

The interview consisted of nine sections: intense inappropriate anger; affective instability; emptiness; identity disturbance; paranoid ideation; abandonment; suicidal or self-mutilating behaviour; impulsivity and intense unstable relationships. Trained assessors made a judgment as to whether each symptom was definitely present, probably present or absent. The derived outcome variable was BPD symptoms probably/ definitely present, based on the probable/ definite presence of five or more symptoms. Diagnosis of BPD according to the Diagnostic Statistical Manual (DSM-IV-TR) is based on the presence of five or more *definite* features, making our assessment more sensitive to BPD *symptoms*; thereby representing a precursor rather than clinically diagnosed BPD. Nevertheless, we used a five-symptom criterion, as we wanted to

identify children manifesting a collection of BPD symptoms, comparable in composition to BPD in adulthood.

Alternative Psychopathologies: Depression and Psychotic symptoms

Depression symptoms were assessed using the *Short Moods and Feelings Questionnaire (SMFQ)*: a 13-item scale measuring non-clinical depression symptoms, and demonstrating high reliability and validity (Thapar, Collishaw, Potter, & Thapar, 2010). The child completed the SMFQ at 11 & 14 years during assessment clinics. The mother responded via postal questionnaire when the child was 12 & 13 years. Each item is rated on a 3-point scale in respect to events occurring in the past two weeks. Scores from each time-point were standardised, and depression symptoms were classed as present if the child was in the top 90th percentile during either the early (11-12 years) or late (13-14 years) assessment period; thereby producing a clinically relevant dichotomous depression outcome. 13.6% of the sample was classified as having clinically relevant depression symptoms.

Psychotic symptoms were assessed using the *Psychosis-like Symptoms Interview* (Horwood et al., 2008; Schreier et al., 2009), when the study child was 12 years of age. Using 12 stem questions, psychology graduates rated whether adolescents had experienced any hallucinations, delusions or thought disorders in the previous 6 months. Questions were derived from the National Institute of Mental Health Diagnostic Interview Schedule for Children-IV and the Schedules for Clinical Assessment in Neuropsychiatry. The average inter-rater reliability was Kappa=0.72 (Schreier et al.,

2009). A dichotomous psychotic outcome variable was derived according to the definite or suspected presence of 1 or more psychotic symptoms. 12.3% of the sample had at least 1 psychotic symptom.

Dysregulated behaviour. Three measures of dysregulated behaviour were combined as suggested in recent research (Meyer et al., 2008; Stringaris, Maughan, & Goodman, 2010). Mothers completed the *Strengths and Difficulties Questionnaire [SDQ]* (Goodman, 1997; Goodman, Ford, Richards, Gatward, & Meltzer, 2000) over three time points; when the child was 4, 7 and 8 years old. Responses from three subscales: *negative emotionality*, *conduct disorder* and *hyperactivity*, were summed (in accordance with how the measure is used in its entirety), to derive a total dysregulated behaviour score (scale of 0 to 30) for each child at each time point. Item response was scaled from 0 to 2, with 0 corresponding to “not true”; 1 corresponding to “somewhat true” and 2 corresponding to “certainly true.” Individual items are: negative emotionality: *child complains of aches; child has many worries; child is often unhappy; child is nervous in new situations; child has many fears*; conduct problems: *child has temper tantrums; child is obedient (reverse scored); child often fights with others; child often cheats/lies; child steals from home*; hyperactivity: *child is easily distracted; child is fidgety; child is restless; child thinks before acting (reverse scored); child has good attention (reverse scored)*.

Social environmental risk factors. Peer victimisation and harsh parenting were used as indicators of social environmental risk factors. Peer victimisation was assessed at 8, 9 and 10 years. Child report was derived from the *Bullying and Friendship Interview*

Schedule (Schreier et al., 2009) at 8 and 10 years, while mother report was via postal questionnaire at 9 years. Peer victimisation was considered present if the child reported being overtly or relationally bullied repeatedly (4 or more times during the past 6 months) or very frequently (at least once per week), and if the mother reported her child being bullied as being “somewhat or certainly true”. The following frequencies were observed: no victimisation: 45.6%; victimisation at 1 time point: 33.3%; victimisation at 2 time points: 16.1%; and victimisation at 3 time points: 5%.

A peer victimisation variable was specified as a latent factor to represent repeated occasions of peer victimisation over the three time points (8, 9 and 10 years) within the Structural Equation Model (SEM). The factor scores derived represent a latent continuous measure of chronic victimisation for each participant, based on information from the whole SEM (Muthen, 1996). The mean factor score was 1.29 (S.D = 0.95), with increasing positive loading representing increasing level of victimisation.

Harsh parenting was assessed via maternal report of hitting when the child was 9 years of age, and was coded as present if the mother reported hitting her child: “every day, several times a week, once or twice a week, or once or twice a month.” A conservative cut-off point was chosen to reflect a general reduction in hitting, as the child grows older (Hyman, 1997). 5.2 % of children were exposed to harsh parenting.

Potential Confounders. The Family Adversity Index (FAI) (Bowen, Heron, Waylen, Wolke, & the ALSPAC study team, 2005) was included as a potential confounder because there is evidence of an association between adverse family background and offspring self-control (Evans, Kim, Ting, Tesher, & Shannis, 2007). Gender (51.5%

girls) was included due to a reported male bias in self-control problems (LaGrange & Silverman, 1999). The FAI, assessed during the prenatal period, comprises of 18 items pertaining to: young maternal age at first pregnancy (<17 years) or birth of study child (<20 years); housing (e.g. inadequacy: overcrowding or periods of homelessness); financial difficulties; problematic partner relationship; maternal affective disorder (depression, anxiety, suicidality); substance abuse (drugs or alcohol); or involvement in crime (i.e. in trouble with police or convictions). Adversity items were summed and trichotomised into: none (no adversity, 47% of the population); mild (1 or 2 adversities, 40.5% of the sample) and severe (>2 adversities, 12.5% of the population).

8.2.3 Statistical Analysis

Statistical analysis was conducted in the following stages:

1) Latent Class Growth Analyses (LCGA) were conducted, using *Mplus version six* (Muthen & Muthen, 2010), to ascertain whether dysregulated behaviour was stable across the three time points from four to eight years, i.e. the behaviour had trait qualities. LCGA is a second-generation Structural Equation Model (SEM), combining person and variable centred approaches to longitudinal data (Jung & Wickrama, 2009). FAI and gender were incorporated as confounders into the LCGA, and the presence of non-normality was controlled for through Robust Maximum Likelihood estimation procedures. Missing data was imputed using the reliable Full Information Maximum Likelihood method (Wiggins & Sacker, 2002).

- 2) Logistic regressions were conducted to ascertain whether BPD symptoms increased as a function of increasing dysregulated behaviour class, and exposure to social environmental risk factors (harsh parenting and peer victimisation).
- 3) Using path analysis, we examined how dysregulated behaviour and environmental risk factors act together to predict subsequent BPD symptoms. Path analysis was carried out to assess the direct, mediated and/or moderated associations between classes of dysregulated behaviour, social environmental risk factors (harsh parenting and chronic peer victimisation) and BPD symptoms at 11 years.
- 4) Prediction Configural Frequency Analysis (P-CFA) was conducted, using the *CFA 2002 program*, (von Eye, 2001) to identify individuals who according to dysregulated behaviour (*low, moderate, high, very high*) and experience of peer victimisation (*victimised versus not victimised*), have higher or lower than expected frequency of BPD symptoms. P-CFA analysis determines whether a cell constitutes a *type*, i.e. contains more cases than expected, or an *antitype*, i.e. contains fewer cases than expected, when compared to the base model (von Eye, 2010). The binomial test with z approximation was selected, which offers a relatively conservative estimation of discrimination types; and the alpha probability was Bonferroni-adjusted to account for multiple testing of cells (von Eye, 2002).
- 5) Using post hoc reruns of the path analysis, we tested the specificity of the BDM for BPD compared to other psychopathologies, by comparing the indirect associations between dysregulation and peer victimisation to BPD symptoms versus psychotic and depression symptoms. Harsh parenting was not included in this analysis, as it was not significantly predictive of BPD in the path model.

8.3 Results

8.3.1 Stage one: Latent classes of dysregulated behaviour

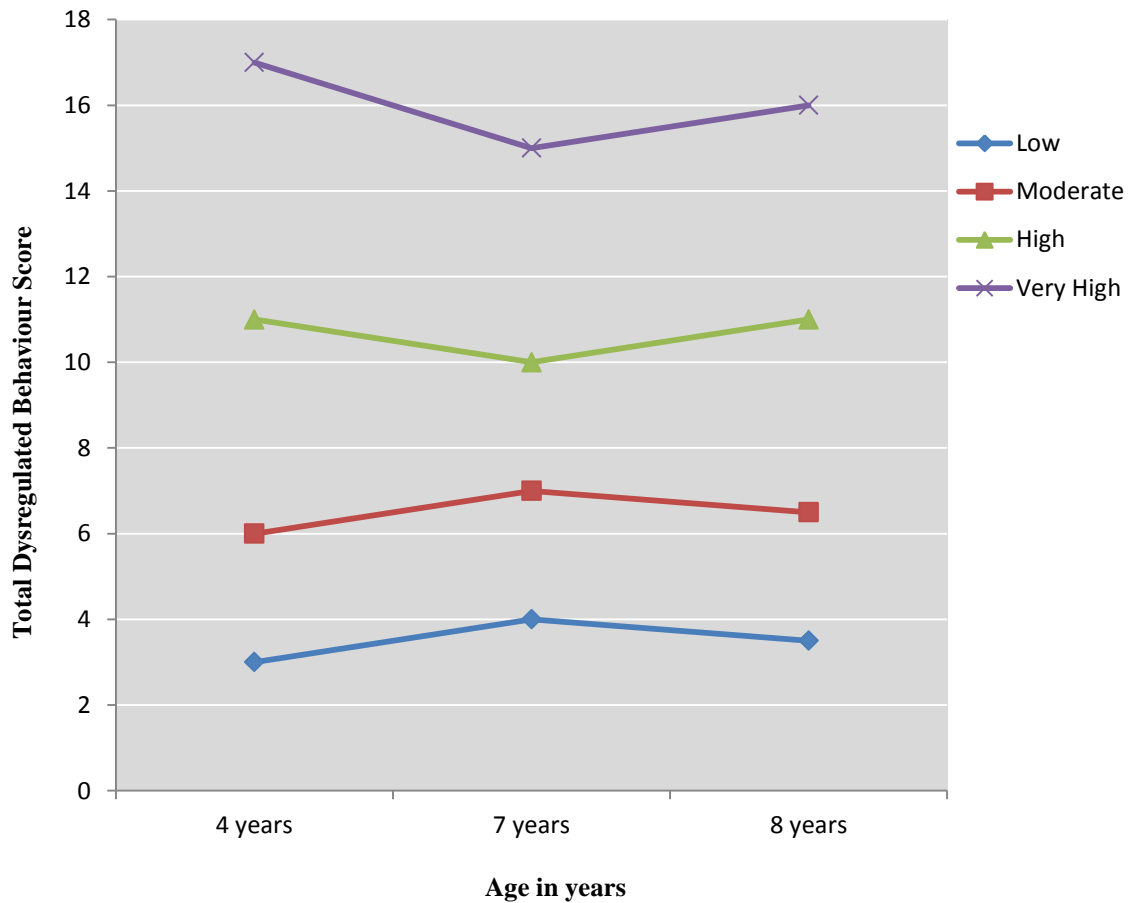
Results of the LCGA indicated that a four-class solution was optimum, with the likelihood ratio tests (Lo-Mendell-Rubin [LMR] and Vuong-Lo-Mendell-Ratio [VLMR]) suggesting that a four-class model was significantly better than a three-class alternative (**Table 8.2**). Low Bayesian Information Criteria (BIC) and Akaike Information Criteria (AIC) values were consistent with this finding. Congruent with previous findings (Ayer et al., 2009), the four latent classes (**Figure 8.1**), representing children with increasing levels of dysregulated behaviour, were relatively stable in trajectory over time. They represented: low dysregulated behaviour (class 1; 38.3% of children); moderate dysregulated behaviour (class 2; 40%); high dysregulated behaviour (class 3; 18%); and very high dysregulated behaviour (class 4; 3.7%).

Table 8.2 *Summary of the Log Likelihood and Goodness of Fit Criteria for Three possible models*

	Two-class model	Three-class model	Four-class model
AIC^A	101512.387	100391.230	99935.999
BIC^B	101638.740	100550.834	100128.853
LMR adjusted p value^C	0.0000	0.0000	0.0000
VLMR ratio test^D	0.0000	0.0000	0.0000

^A AIC: Akaike information criteria; ^B BIC: Bayesian information criteria: a smaller value suggests better fit; ^C LMR: Lo-Mendell-Rubin adjusted likelihood ratio test: significant value suggests improvement on model; ^D VLMR: Vuong-Lo-Mendell Rubin likelihood ratio test: significant value suggests improvement on model; Five class model rejected as yielded very small class precluding further analysis

Figure 8.1 *Dysregulated Behaviour [on scale of 0 to 30] Latent Classes from 4 to 8 years controlled for Gender and Prenatal Family Adversity Index*



The FAI index was significantly predictive of higher levels of dysregulated behaviour; when using the lowest dysregulation class as the reference group (Odds Ratio; 95% confidence intervals). *Class two:* 1 to 2 FAI adversities (0.96; 0.72 to 1.26); > 2 adversities (1.07; 0.72 to 1.59); *Class three:* 1 to 2 adversities (OR: 1.28; 0.94 to 1.74); > 2 adversities (OR: 2.28; 1.49 to 3.50); and *Class four:* 1 to 2 adversities (OR: 1.81;

1.08 to 3.05); > 2 adversities (OR: 4.78; 2.66 to 8.59). Gender was not significantly related to latent classes within the LCGA.

8.3.2 Stage two: Associations between predictors and subsequent BPD symptoms

Associations between predictors and BPD symptoms are reported in Table 8.3. Logistic regression analyses were conducted using latent dysregulated behaviour classes saved from the LCGA analysis (1 to 4 scale, representing increasing dysregulation).

Classes of dysregulation were linearly associated with BPD symptoms at age 11 years, i.e. higher dysregulation between 4 and 8 years increased the odds of having 5 or more BPD symptoms. Repeated peer victimisation between 8 and 10 years (represented by continuous factor scores representing increasing chronicity) was strongly associated with BPD symptoms. Those exposed to harsh parenting were also more likely to experience BPD symptoms at 11 years.

8.3.3 Stage three: Direct and indirect associations between dysregulated behaviour class, social environmental risks and BPD symptoms

A path model was specified to ascertain whether the association between dysregulated class (using low dysregulated class as the reference group) and BPD was moderated and/ or mediated by social environmental risk factors (chronic peer victimisation and harsh parenting).

Table 8.3 Predictive Associations Between Latent Dysregulated Classes, Social Risk Factors and Subsequent BPD Symptoms

Predictor	ORs (95% CI)
Dysregulated Behaviour^a	
Class 1 (low)	Reference
Class 2 (moderate)	1.54 (1.20 to 1.97)
Class 3 (high)	2.41 (1.82 to 3.18)
Class 4 (very high)	3.51 (2.28 to 5.42)
Social Risk Factors	
Harsh Parenting ^b	2.07 (1.42 to 3.02)
Peer Victimisation ^c	6.15 (5.34 to 7.10)

^aIncreasing dysregulated behaviour: moderate, high, very high; ^bMaternal hitting (reference group 0 = no hitting; 1 = hitting every day, several times a week, once/twice week, once/twice month); ^cPeer victimisation variable constructed from confirmatory factor analysis in path analysis across 8 to 10 years: Continuous factor scores saved from Confirmatory Factor analysis representing increasing chronicity

Fit indices indicated acceptable model fit: Chi-square = 82.737; p=0.00; CFI= 0.95; RMSEA = 0.036. (see **Figure 8.2**) The following relationships were revealed. Firstly, children with higher dysregulated behaviour between 4 and 8 years were significantly more likely to experience harsh parenting and chronic peer victimisation between 8 to 10 years, compared to those with low dysregulated behaviour (see **Table 8.4**). Secondly, controlling for other factors, chronic peer victimisation between 8 and 10 years was significantly predictive of BPD symptoms at age 11 years (probit coefficient = 0.67; p=0.00). Conversely, harsh parenting did not remain an independent predictor of BPD symptoms (0.09; p=0.15). Thirdly, the association between dysregulated behaviour and BPD symptoms was significantly mediated via peer victimisation at all levels of dysregulation. Further, this mediation was moderated (Preacher, Rucker, & Hayes,

2007) by level of dysregulated behaviour. Difference tests (Lau & Cheung, 2010) revealed that with increasing levels of dysregulated behaviour, the strength of relationship between dysregulation and BPD, via peer victimisation, significantly increased (see **Table 8.4**). Harsh parenting did not moderate or mediate the association between dysregulation and BPD.

Table 8.4 *Direct and Indirect Associations Between Dysregulated Trait Behaviour, Harsh Parenting, Peer Victimization and BPD Symptoms*

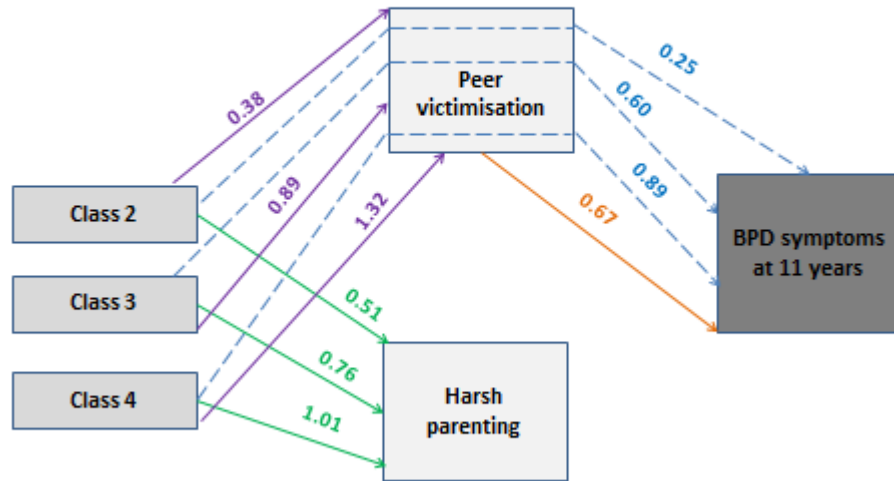
Class predictor (reference) ^A	Harsh Parenting (Direct associations)	Peer Victimization (Direct associations)	Mediation via Peer Victimization ^B	Difference Test ^C
Class 2	0.51**	0.38**	0.25**	<u>Class 3-2</u> ^D 0.34**
Class 3	0.76**	0.89**	0.6**	<u>Class 4-3</u> ^E 0.29**
Class 4	1.01**	1.32**	0.89**	<u>Class 4-2</u> ^F 0.63**

^A Class 1 reference group; ^B Mediation via parenting not included as insignificant; ^C See Lau et al. 2010 for details; ^D Class 3 - class 2 mediation; ^E Class 4 - Class 3 mediation; ^F Class 4 - Class 2 mediation; ** Significant at 0.00. Path associations reported as probit co-efficients.

8.3.4 Stage four: Configurations of dysregulated behaviour (4 categories) by peer victimisation (2 categories) according to the presence or absence of BPD symptoms

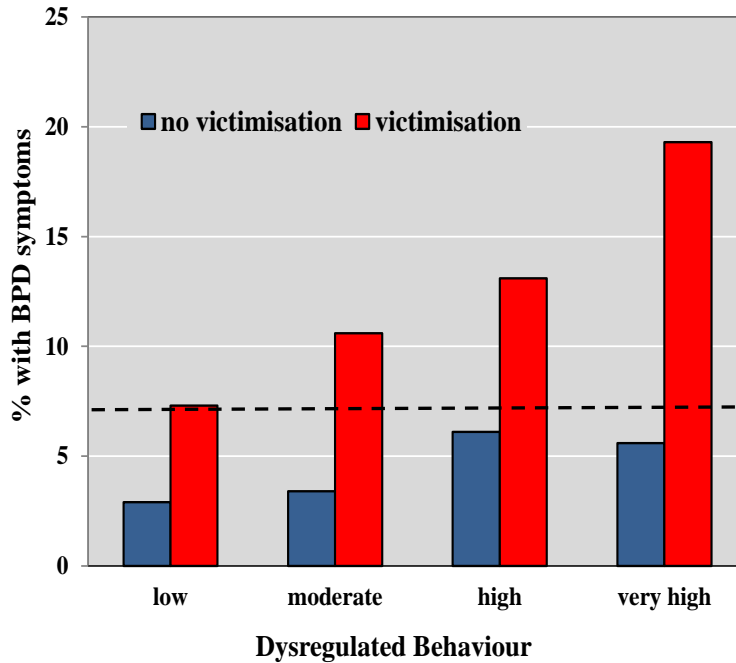
Figure 8.2 shows the observed frequencies of BPD symptoms, according to the degree of dysregulated behaviour, and whether children were victimised (at any time point) by peers. BPD symptoms were most likely found in children who were moderately to very

Figure 8.2 Final model showing the significant direct and indirect associations between dysregulated behaviour, harsh parenting, peer victimisation and BPD outcome.



Relevant coefficients reported above the arrow line. $\chi^2 = 84.61$, $P = 0.00$, $CFI = 0.95$, $RMSEA = 0.03$. Values are given in unstandardised probit coefficients. Non-significant paths at the 0.05 level are not shown. Dysregulated class is coded into 4 categories: low (class 1); moderate (class 2); high (class 3); and very high (class 4). Low dysregulation (class 1) is used as the reference group. Direct associations between dysregulation and peer victimisation are shown in purple. Direct associations between dysregulation and harsh parenting are shown in green. The direct association between peer victimisation and BPD is shown in orange. Indirect associations between dysregulation and BPD via peer victimisation are shown in broken blue lines.

Figure 8.3 *Relative Frequency of BPD Symptoms According to Dysregulated Behaviour and Peer Victimisation Experience*



highly dysregulated in their behaviour and additionally exposed to peer victimisation (Figure 8.2). Table 8.5 shows that for all three levels of increased dysregulation (moderate, high and very high), only those exposed to peer victimisation developed BPD symptoms more often (types) than expected at the $p < 0.05$ level.

Conversely, individuals in the two configurations: *low dysregulated behaviour, no peer victimisation* and *moderate dysregulated behaviour, no peer victimisation*, were observed to have BPD symptoms less often than expected (anti-types). Thus, the

combination of exposure to peer victimisation and higher levels of dysregulation appear to heighten the risk of BPD symptoms.

Table 8.5 Prediction Configural Frequency Analysis with Dysregulation and Victimization as Predictors and BPD as Outcome

Victimization?	Configuration ^A (Dysreg/ victim/ BPD)	Frequency ^B (BPD vs. no BPD)	Statistic (González-Debén's π)	P value (Bonferroni adjusted = 0.0062)	Type? (BPD vs. No BPD)
Victim (7.31%) ^C	111	69			
	<u>110</u>	<u>874</u>	<u>0.2244</u>	<u>0.411</u>	
No Victim (2.9%)	101	36			
	<u>100</u>	<u>1206</u>	<u>-6.585</u>	<u>0.0000</u>	<u>Anti- Type</u>
Victim (10.64%)	211	126			
	<u>210</u>	<u>1058</u>	<u>5.258</u>	<u>0.0000</u>	<u>Type</u>
No Victim (3.39%)	201	37			
	<u>200</u>	<u>1054</u>	<u>-5.363</u>	<u>0.0000</u>	<u>Anti- Type</u>
Victim (13.13%)	311	83			
	<u>310</u>	<u>549</u>	<u>6.204</u>	<u>0.0000</u>	<u>Type</u>
No Victim (6.1%)	301	20			
	<u>300</u>	<u>306</u>	<u>-0.73</u>	<u>0.233</u>	
Victim (19.3%)	411	27			
	<u>410</u>	<u>113</u>	<u>5.648</u>	<u>0.0000</u>	<u>Type</u>
No Victim (5.6%)	401	3			
	<u>400</u>	<u>51</u>	<u>-0.456</u>	<u>0.324</u>	

^A The three digits refer to respectively: Dysregulation (1-4 increasing dysregulation), victimisation (at 8, 9 or 10 years) and BPD status (0=No, 1=yes) ^B Number of individuals in each cell. Configurations increasing the risk of BPD symptoms in bold. ^C Refers to % with 5 or more BPD symptoms

8.3.5 Stage five: Comparison of indirect associations between dysregulated behaviour, peer victimisation, and psychopathological outcome

Post-hoc path analyses were carried out to ascertain the discriminative validity of the relationship between dysregulated behaviour, peer or parent behaviour and BPD symptoms. We added any psychotic symptoms at age 12 and depression symptoms between the age of 11 and 14, as dichotomous outcome variables. Results revealed that the indirect relationship between dysregulated behaviour, peer victimisation and psychopathological outcome was larger for BPD than for psychotic or depression symptoms at all levels of dysregulation (using low dysregulated behaviour as the reference group).

Further, this difference became more pronounced as level of dysregulation increased (see **Table 8.6**). Within the path model, BPD symptoms were significantly associated with psychotic symptoms (path coefficient = 0.25; $p=0.00$), and psychotic symptoms were significantly associated with depression symptoms (path coefficient=0.11; $p=0.01$). A difference test (Lau & Cheung, 2010) was utilised to test the statistical significance of these contrasts. The indirect relationship between dysregulation, peer victimisation and BPD symptoms was significantly greater than the equivalent path to either psychotic or depression symptoms at all levels of dysregulated behaviour (**Table 8.6**).

Table 8.6 Comparison of Indirect Associations of Dysregulation via Peer Victimization to BPD, Depression and Psychotic Symptoms

Class	BPD	PLIKS	Depression	Diff Test ^A BPD – PLIKS	Diff Test BPD - Depression
Class 2					
MED 1	0.28	0.17	0.20	0.11**	0.09**
Class 3					
MED 2	0.66	0.40	0.45	0.26**	0.20*
Class 4					
MED 3	1.00	0.61	0.69	0.39**	0.31*

Path associations reported as probit co-efficients; * significant at 0.05; ** significant at < 0.01; ^A See Lau & Cheung. (2010) for details. Analysis based on reduced number: 4826 to include those with information on depression, PLIKS and BPD.

8.4 Discussion

To our knowledge, this is the first study to assess the predictive association between dysregulated behaviour in early to middle childhood and BPD symptoms in late childhood/early adolescence.

Furthermore, we believe this to be the first investigation to test the biosocial developmental model of BPD. Consistent with the BDM (Crowell et al., 2009), we found that the effect of early vulnerability to dysregulation, on the development of BPD symptoms, was exacerbated by exposure to repeated peer victimisation between 8 and 10 years of age. Further, for those with extreme levels of dysregulation, the association between dysregulation and BPD, via peer victimisation, was especially strong.

Using Latent Class Growth Analyses we identified four trajectories (classes) of dysregulated behaviour between 4 and 8 years that were remarkably stable over time, and thus indicative of a dysregulated trait. Further, our analyses revealed that as dysregulation increased, the odds of evincing 5 or more BPD symptoms increased; illustrating the predictive validity of the derived dysregulation trajectories. The observed link between dysregulated behaviour and BPD is consistent with research literature revealing a robust relationship between Axis I (internalising and externalising) disorders and subsequent Personality Disorders (Fischer, Barkley, Lori Smallish, & Fletcher, 2002; Fossati, Novella, Donati, Donini, & Maffei, 2002). Further, dysregulated behaviour trajectories were significantly associated with family adversity assessed during pregnancy, congruent with previous research revealing a link between environmental adversity and subsequent dysregulation (Jucksch et al., 2011; Evans et al., 2007). Ultimately, these findings support the validity of a *dysregulation syndrome*, as assessed by the Strengths and Difficulties Questionnaire, and expand on previous findings by supporting the longitudinal stability of this marker (Holtmann, Becker, Banaschewski, Rothenberger, & Roessner, 2010).

A direct significant association between consistent peer victimisation from 8 to 10 years and BPD at 11 years was observed. While peer victimisation has been linked to a multitude of negative mental health outcomes (Schafer, Korn, Brodbeck, Wolke, & Schulz, 2005; Schreier et al., 2009; Sapouna et al., 2011), this is the first study to reveal a prospective link between peer victimisation and BPD specifically. This adds to the

literature by highlighting the importance of *peer*, in addition to parental, relationships in the development of BPD.

Both peer victimisation and harsh parenting were predicted by early dysregulated behaviour. These findings are intuitive; children evincing dysregulated behaviour are more likely to attract negative attention from peers and parents. Indeed, studies have shown that dysregulated behaviour is related to parental stress (Williford, Calkins & Keane, 2007), and maladaptive parenting practices (Calkins, Hungerford & Dedmon, 2004; Morrell & Murray, 2003). Similarly, the association between dysregulated behaviour and peer victimisation is well documented, with higher levels of dysregulation causing children to become more likely targets of victimisation (Shields & Cicchetti, 2001). Dysregulated children, sometimes labelled *provocative victims*, tend to have an “abrasive personality;” (Levinson, 1978) characterised by short-temper, restlessness, anxiety and a tendency to retaliate when attacked (Batsche & Knoff, 1994). Further, they are prone to low self-esteem and social competence, coupled with high levels of aggression (Matthiesen & Einarsen, 2007). Consequently, once victimised, this pattern tends to persist for months or years, even once the child changes school (Sapouna et al., 2011; Schafer et al., 2005).

When considered together, peer victimisation rather than harsh parenting mediated the relationship between dysregulated behaviour and BPD symptoms, suggesting that traumatic or stressful peer relationships in middle childhood are especially pertinent in the development of BPD. As revealed in the person-oriented P- CFA, peer victimisation

increased the risk of BPD symptoms, especially for individuals evincing high to very high levels of dysregulated behaviour, as also demonstrated by the *moderated mediation* (Preacher et al., 2007) effect within the path analysis. Thus, congruent with the BDM, it appears that dysregulation, especially in the extreme range, is fostered and maintained within an invalidating developmental context; eventually potentiating as BPD symptoms (Crowell et al., 2009).

In this instance, there may be two possible mechanisms via which this combined effect occurs, likely working in conjunction. Firstly, peer victimisation may lead to the formation or exacerbation of negative relational schemata (Salmivalli & Isaacs, 2005), altered social cognition (Gianluca, 2006), and a tendency to *hypermentalise* (Sharp et al., 2011). Hypermentalisation - a propensity to over attribute other's intentions - has been commonly observed in individuals with BPD; and may interact with dysregulated behaviour, preventing the development of healthy mentalising strategies (Sharp et al., 2011). So while negative biases and attributions are also found in association with psychosis and depression, they appear to be especially severe for BPD, following further dysregulation in response to exclusion and bullying (King-Casaas et al., 2008). In this way, an individual evincing dysregulated behaviour, upon encountering repeated negative interactions with others, may develop maladaptive interactional strategies, or "emotionally labile patterns of interaction" (Crowell et al., 2009), manifest as the core relational symptoms of BPD.

Secondly, for individuals evincing dysregulation, increased social stress due to victimisation, may physiologically "work itself under the skin," altering an already

vulnerable stress response (Teicher et al., 2010b ; Young, 2009); potentiating emotional dysregulation and leading to further behavioural dyscontrol, manifest as the core impulsive symptoms of BPD (Crowell et al., 2009). A vicious cycle may develop in which dysregulation is heightened, attracting more negative interactions and feedback, increasing dysregulation further, until trait dysregulation crystallizes; and following further perturbations, eventually culminates in a “borderline” personality (Crowell et al., 2009).

Harsh parenting did not mediate the association between dysregulation and BPD symptoms. In comparison to peer victimisation, parenting was considered at only one time point, possibly accounting for the lack of an indirect relationship within the path model. However, other available measures of harsh parenting (parental hostility), within the 8 to 10 year time frame, were not predictive of BPD symptoms singularly, precluding their use in further analysis. Therefore, it appears that the etiological effects of parenting, excepting profound continuous abuse, are most influential early on in childhood, through the initiation of a chain of events or developmental cascade (Hay et al., 2011; Bornstein et al., 2006); and are mediated by other factors later in the developmental trajectory (Belsky et al., in Press; Sroufe, Coffino, & Carlson, 2010) Conversely, peer relationships become especially salient as the child approaches adolescence (Elicker, & Englund, 1992), with children spending increasingly more time with peers than parents (Larson, 2001; Rutter, 1979). Thus, while harsh parenting was predictive of BPD symptoms singularly, the relationship became insignificant within the

path model, suggesting that the very strong effects of peer victimisation may have occluded any relationship.

As peer victimisation adversely affects cognition, emotions and stress regulation, it is not surprising that depression and psychotic symptoms were also associated with dysregulated behaviour via peer victimisation, though to a significantly lesser extent than BPD symptoms. This may partly reflect symptom overlap and co-morbidity between disorders (Black et al., 2007; Skodol et al., 2002); as observed here, between BPD and psychotic symptoms, and psychotic and depression symptoms. Nevertheless, the observed relationships were particularly strong, especially for individuals with high levels of dysregulation, for the unique constellation of BPD symptoms, which are underpinned by impulsive (Crowell et al., 2009), stress related and relational (Kernberg & Michels, 2009) core features.

There are strengths and limitations of the study. By utilising a person-centred approach, children could be grouped according to their individual growth trajectories (Jung & Wickrama, 2009) indicating a relatively stable marker of trait dysregulation. Using multiple analytical approaches, including path analyses (variable based) and CFA (person centred), we could show that moderate to very high dysregulation increases risk for BPD, especially in combination with exposure to chronic peer victimisation.

Although we used a reliable assessment of BPD for children, with comparable criteria to those used in adulthood (5 or more symptoms), it remains to be clarified whether the children evincing BPD symptoms at 11 years will develop clinically diagnosed BPD in

adulthood. Taking the Conduct Disorder to Antisocial Personality Disorder model (Raine et al., 2005) as a guide, it may be likely that a substantial proportion of these children will develop BPD in adulthood. Future work should reassess these individuals to ascertain continuity of BPD symptoms across development (Crick et al., 2005).

There was substantial, selective attrition in this study. Despite selective dropout we found strong and hypothesised associations between family adversity, dysregulated behaviour, harsh parenting, peer victimisation and BPD symptoms among the remaining, less severely disadvantaged individuals. Previous simulations (Wolke et al., 2009) demonstrated that even when dropout is correlated to the predictor/confounder variables, the relationship between predictors and outcome is unlikely to be substantially altered by selective dropout processes. However, it cannot be precluded that selective dropout had some influence on the predictive relationships reported.

8.4.1 Conclusions

Congruent with the BDM of BPD, children demonstrating dysregulated behaviour, especially high to very high dysregulation, in early to mid-childhood, are prone to the development of BPD symptoms when exposed to social risk factors. This underlines the importance of providing effective and readily available interventions for dysregulated behaviour (Keenan & Shaw, 1994), identifiable as early as infancy or preschool age (Hay et al., 2011; Hemmi, Wolke, & Schneider, 2011), to prevent the canalization of mental disorder (Crowell et al., 2009).

In addition, our results expand existing literature, by revealing the importance of peer relationships in the development of BPD symptoms, supporting that provocative victims are especially at-risk of negative sequelae (Arseneault et al., 2006). Pathways to BPD symptoms, for those with moderate to high dysregulation, may be altered by interventions reducing peer victimisation (Farrington & Ttofi, 2009).

Chapter Nine: Final Discussion

***Overview:** The final chapter presents a summary of the main findings reported pertaining to the developmental antecedents of BPD symptoms at 11 years. This is followed by a discussion of the strengths and weaknesses apparent in the research undertaken. Additionally, there is an outline of the implications, both theoretical and practical, of reported findings, and finally suggestions for future work.*

9.1 Summary of Results

Research pertaining to the causal risk factors for the development of BPD, using prospective assessments derived before the advent of the disorder, is scarce (but see: Johnson et al., 1999; 2006). This is the first programme of research, which prospectively considered the antecedents of BPD symptoms assessed in late childhood/early adolescence, using a clinically relevant, adapted assessment; based on the Diagnostic Interview for DSM-IV Personality Disorders in adults. The most salient findings include:

- Exposure to family adversity during pregnancy, and harsh parenting and parent conflict during early to middle childhood, was predictive of BPD symptoms at 11 years of age. The association between maladaptive parenting and BPD was partially mediated by IQ, but not Axis I psychopathology, at 8 years of age.
- Peer victimisation between 4 and 10 years of age was predictive of BPD symptoms at 11 years of age, according to child, mother and teacher report, with

a dose response effect observed for severe, combined and chronic victimisation. Observed associations remained virtually unchanged when family adversity, IQ, Axis I psychopathology, maladaptive parenting (hitting and hostility) and sexual abuse were controlled for.

- Dysregulated trait behaviour, between 4 and 8 years of age, was predictive of BPD symptoms at 11 years, and the higher the dysregulated behaviour, the stronger the relationship. This association was strongly mediated by chronic peer victimisation between 8 and 10 years of age; and the higher the level of dysregulation the stronger the mediation effect.
- The prevalence of BPD in this community population was 7.3%, somewhat higher than the reported prevalence in adult community populations (see chapter one: 1.3.1). This finding is congruent with a developmental psychopathology model (see section 3.1.2.1), in which only a proportion of individuals evincing precursor symptoms in childhood will develop the clinically diagnosed disorder in adulthood (van Os et al., 2009).
- The prevalence of BPD in males (7.3%) and females (7.4%) was almost identical in the ALSPAC population of 11 year olds. These findings contrast with the observation that approximately 70% of adults meeting criteria for BPD are women (Swartz et al., 1990; Torgersen et al., 2001). Further, the one community study (Bernstein et al., 1993) assessing the female/male prevalence of borderline features in childhood and adolescence, reported a female bias at both 11 to 14 (11.5% vs. 8.3%) and 18 to 21 (8.1 vs. 5.8%) years. Findings here may reflect

the particular developmental stage, i.e. cusp of adolescence, during which the BPD assessment was made. It may be that gender differences become more pronounced as the child matures, with girls turning impulsive predispositions inwards, as manifest in BPD typical features; and boys outwards, as manifest in antisocial features (Crawford et al., 2001b; Paris, 2003).

Collectively, the above findings reveal that BPD symptoms, assessed at 11 years of age, are associated with similar risk factors (maladaptive parenting, traumatic interpersonal experiences and dysregulation) to those reported by, or observed in, adults with BPD. Therefore, sub-clinical BPD symptoms, assessed in childhood, may exist on a continuum with clinically diagnosed BPD, supporting that childhood symptoms likely represent a direct precursor to BPD in adulthood (Guzder et al., 1999; Paris et al., 1999). Nevertheless, future research is required to determine whether childhood symptoms increase the odds of adult BPD.

It was observed that maladaptive parenting, in the form of maternal hitting and hostility and parental conflict, was predictive of BPD symptoms at 11 years of age. These results buttress existing research findings by providing *prospective* evidence for a link between exposure to maladaptive parenting during early to mid-childhood, and a clinically relevant constellation of BPD symptoms at 11 years of age. This supports that the observed relationship between sub-optimal parenting and BPD is not solely attributable to reporting or memory bias; and congruent with a recent birth cohort study (Belsky et

al., in press), that less profound (i.e. other than sexual, severe physical) forms of abuse may also contribute to the development of BPD symptoms.

Novel is the finding that peer victimisation, from middle to late childhood, strongly predicted BPD symptoms at 11 years of age. Most studies to date, largely retrospective in nature, have focused on traumatic experiences with parents rather than peers (e.g. Bandelow et al., 2005). Results here, however, suggest that peer victimisation is an important aetiological factor in the development of BPD. Congruent with developmental theory, peer factors appear especially salient as the child approaches late childhood/early adolescence (Elicker & England, 1992), as demonstrated by the strong mediational role of peer victimisation, in comparison to harsh parenting, between 8 and 10 years of age. Indeed, trauma inflicted by peers may add to previous negative events, e.g. harsh parenting, in a cumulative fashion in the developmental pathway towards BPD (Sroufe et al., 2010).

Finally, results suggest that psychosocial risk factors (peer victimisation and harsh parenting) are especially harmful for individuals evincing high dysregulated trait behaviour from early childhood onwards. In study three, it was observed that the negative consequences of peer victimisation are powerful, but that individuals with low levels of dysregulated behaviour were not at increased risk of BPD symptoms, following exposure to peer victimisation (see **Table 8. 5**). This supports the biosocial developmental model of BPD, which states that trait dysregulation in combination with

psychosocial risk factors increases the risk of BPD (Crowell et al., 2009; Linehan et al., 1993).

9.2 Limitations of the study

Despite the utility of the ALSPAC cohort resource for the study of developmental psychopathology, there are limitations:

- The cohort had a high rate of children lost to follow-up. Of the children first enrolled in the study, less than 50% were retained in the final analyses. Due to selective dropout, it is likely that prevalence figures, reported here, are an underestimation of the true prevalence rates (Costello et al., 2003). A publication by Wolke and colleagues (2009) confirmed that dropout in the ASLPAC study was systematic, and that children with teacher-reported disruptive behaviour disorders were more frequently lost to follow-up. However, the Wolke et al. study also revealed that predictive factors and their strength were the same for those who dropped out. Despite retaining the less severely disadvantaged participants, hypothesised associations were observed here, suggesting that findings pertaining to the precursors of BPD are valid and robust notwithstanding the selective dropout. Thus, dropout biases prevalence, but may have little biasing impact on longitudinal relationships between variables.
- Although assessments were well planned, controlled and validated, official records of reported sexual abuse indicated minute rates (Sidebotham, Heron,

Golding, & ALSAPC Team, 2002), and were not available into childhood.

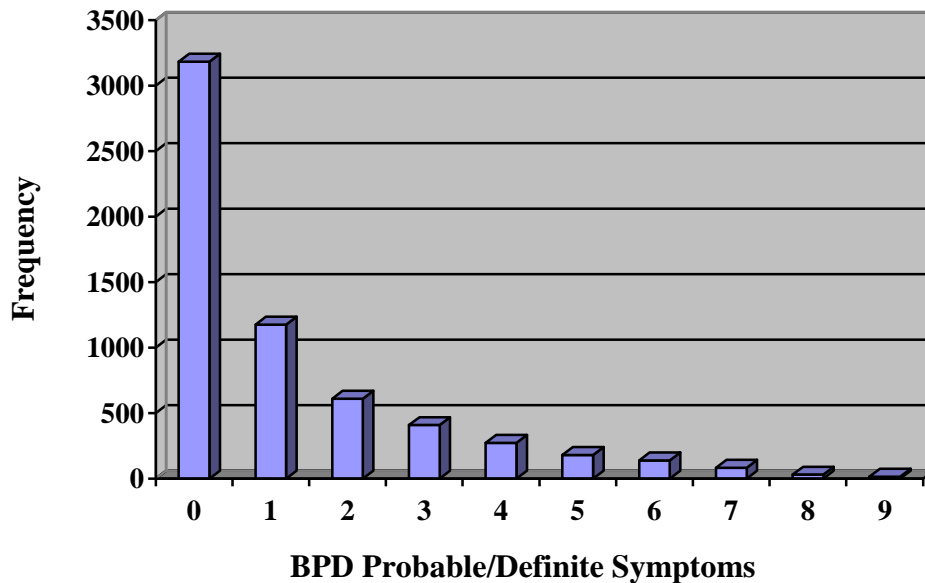
Mother reported prevalence of child sexual abuse in the ALSPAC population was 0.05%, well below reported figures for community populations (MacMillan et al., 1997), precluding the inclusion of this risk factor (Zanarini et al., 2006) in the statistical analysis. However, while historically sexual abuse was thought to be the prominent experiential risk factor in the development of BPD, existing research suggests that sexual abuse is not linked to the whole spectrum of BPD diagnoses. As observed here, milder forms of BPD (traits or symptoms) may be associated with other experiential exposures such as maladaptive parenting (Salzman et al., 1993).

- BPD symptoms were assessed at one time point only, thus a baseline measure against which to compare the development of BPD symptoms over time was not available. Research to date, however, suggests that the full constellation of BPD symptoms becomes manifest in early adolescence (Chanen et al., 2008), and is presaged by Axis I psychopathology, i.e. internalising and externalising disorders (Kasen et al., 1999; Lewinsohn et al., 1997). Therefore, Axis I disorders, assessed at 8 years, and dysregulated behaviours between 4 and 8 years, were incorporated into the analysis.
- The ALSPAC study data was purely observational; therefore, *strong* claims pertaining to causation were not possible (see Wu & Zumbo, 2008). However, the study population can be considered uncontaminated by treatment interventions, thereby increasing the generalisability of the findings compared to clinical studies.

- It is likely that there was residual confounding in this study. For example, the BPD status of the mother was not assessed at any point during data collection. Therefore, it was not possible to ascertain whether this important confounder had any impact on the development of BPD, either through the direct effects of genetic transmission, or via indirect effects of harsh parenting, for example.
- Follow up data, pertaining to the current BPD status of the study child, is not available. Therefore, it was not possible to determine whether the BPD phenotype, assessed in early adolescence, is predictive of a BPD diagnosis in adulthood. There are several reasons to believe that this would be the case, however. Firstly, the BPD precursor symptoms were associated with similar risk factors as those associated with clinical BPD. Secondly, a validated interview for BPD was used, based on the DSM symptom set used to diagnose BPD in adulthood (Zanarini et al., 1996). Thirdly, due to the large sample size, it was possible to construct a BPD outcome variable according to the presence of 5 or more BPD symptoms. Previous, smaller scale, community studies have been restricted to a dimensional approach (Crick et al., 2005; Johnson et al., 1999; 2006), making parallels with clinical BPD in adulthood difficult. A categorical approach was utilised in the analysis, as the UK-CI-BPD interview could not be easily converted into a dimensional scale, due to a highly skewed inverted j-shape distribution, which would not be suitable for linear modelling (see **Figure 9.1** below). The categorical approach reflects a measurement philosophy typically found in psychiatry, making this approach more compatible with the

current clinical climate, pending potential changes in the DSM-V in 2013 (see chapter one).

Figure 9.1 Distribution of the UK-CI-BPD Interview



9.3 Strengths of the study

A data resource of the magnitude of the ALSPAC cohort provides a number of attendant advantages when considering the developmental antecedents of BPD. These include:

- Previously, there has been an over reliance on the use of clinical populations in both child (e.g. Guzder et al., 1999; Paris et al., 1999) and adult (e.g. Weaver & Clum, 1993; Yen et al., 2002) studies. A large, community sample was used

here, found to be fairly representative of the UK population generally (Golding et al., 2001). By utilising a cohort population, the under-representativeness and sampling bias inherent in clinical populations was reduced, avoiding the confounding effects of treatment exposure.

- The ALSPAC longitudinal, birth cohort provides *prospective* data, facilitating the study of antecedents and risk factors as they occur along the developmental trajectory. This allows a level of control, in terms of *precedence* (Wu & Zumbo, 2008); thereby improving causal inferences pertaining to observed associations. For example, the reported association between family adversity, and subsequent parenting and BPD symptoms, is highly suggestive of a causal relationship, as family adversity was assessed during pregnancy, precluding any reverse causality effects.
- The ALSPAC cohort study provides a rich source of data, encompassing a variety of psychosocial risk factors, allowing for the control of a number of potential confounders. Individuals at risk of BPD are likely to be exposed to a number of *related* risk factors (Bradley et al., 2005); therefore, controlling for these allows for determination of the relative contributions from each.
- Assessments, both questionnaires and interviews, were available from a variety of sources, including: mothers, partners, teachers and the study child. Thus, converging data was available, aiding interpretation of results. The observed association between peer victimisation and BPD, for example, remained robust when utilising mother and teacher reports of victimisation. Therefore, observed

associations could not be attributed to self-report bias, resulting from BPD characteristics already manifest.

- Statistical techniques were carefully chosen to fully utilise the positive features of the ALSPAC cohort study. By utilising structural equation approaches, it was possible to model relationships among variables, taking into account the temporal ordering of factors. Further, SEM models allow for the specification of correlations among exogenous (predictor) variables, more closely representing real world situations in which risk factors tend to co-occur (Bradley et al., 2005). Finally, by incorporating *moderation* and *mediation* terms into the path model, one can decipher whether a third variable links a cause and effect (mediation) or modifies it (moderation). Therefore, an understanding of *potential* casual relationships within this non-experimental context (Wu & Zumbo, 2008) was possible.

9.4 Implications

Findings here support that a collection of BPD symptoms (BPD phenotype), similar in composition to the adult disorder, may be identified in late childhood/early adolescence, and are associated with similar risk factors. The identification of a BPD phenotype offers promise for the development of aetiological theory, by allowing for the repeated assessment of BPD pathology, and associated risk factors, during the developmental trajectory (Zammit et al., 2008). This will facilitate the assessment of risk factors when they are “aetiologically active” (Ingram & Price, 2001), rather than relying on

retrospective reports or assessments, which make it impossible to disentangle cause from effect.

The reported results support that a BPD *phenotype* is identifiable in a substantial proportion of the population during late childhood/early adolescence; thus confirming that a diagnosis of BPD in this age group is tenable (Chanen, Jovev, & Jackson, 2007). As BPD traits in young people have been found to be malleable (Lezenweger & Castro, 2005), this is a key period in which to intervene (Chanen, McCutcheon, Jovev, Jackson, & McGorry, 2007) to prevent the canalisation of BPD symptoms into clinical disorder. *Indicated prevention* may include targeting groups with precursor BPD symptoms (Chanen, McCutcheon, Jovev, Jackson, & McGorry, 2007). As mentioned, however, there is still uncertainty pertaining to the specificity of developmental trajectories (Price & Lento, 2001); therefore such programmes should focus on a collection of psychopathological outcomes (Chanen, McCutcheon, Jovev, Jackson, & McGorry, 2007).

Results here reveal that exposure to environmental *risk* factors including family adversity; maladaptive parenting and peer victimisation increase the risk of BPD symptoms at 11 years of age. This supports that *universal prevention* schemes (Mrazek & Haggerty, 1994) may also be useful in preventing the development of BPD, in addition to other psychopathology.

Further, this is the first study to empirically support the biosocial developmental theory of BPD, by revealing that dysregulated trait behaviour in early to mid-childhood increased the risk of BPD symptoms in those exposed to peer victimisation and harsh parenting. This supports that dysregulated trait behaviour is a *vulnerability* factor (Ingram & Price, 2010) in the development of BPD, which renders individuals especially susceptible to environmental perturbations along the developmental trajectory. Therefore, while researchers have suggested that universal prevention schemes (Mrazek & Haggerty, 1994) are the only viable prevention strategy, results here suggest that programmes targeted towards dysregulation, which may be identified as early as infancy (Hemmi et al., 2011), may be a cost effective and efficient option. Such interventions are likely to reduce human suffering and long term health costs, and provide a safer environment for children to grow up in. The behavioural profile of dysregulated children suggests that they are at risk of becoming *provocative* - or *bully* - *victims*; therefore interventions targeting bullying, especially within this subgroup, may be beneficial (Farrington & Ttofi, 2009).

9.5 Future Directions

Currently, the causal factors for BPD are only partly known. The following endeavours may prove fruitful in extending our understanding of the aetiology, and treatment, of this complex disorder:

- It is important that the individuals identified here are followed up and re-assessed for BPD symptoms as they enter adulthood. This would provide strong support for the *predictive validity* of the BPD childhood phenotype (Crick et al., 2005); and facilitate, for the first time, the identification of predictive associations between risk factors, experienced from early childhood onwards, and BPD in adulthood.
- Genetic studies should be carried out to ascertain the biological underpinnings of the dysregulated behaviour trait implicated in the development of BPD. Both *impulsivity* and affect *dysregulation endophenotypes* have been linked to the 5-HT system (Crowell et al., 2009) and are core features of BPD; however, the specific trajectories leading to BPD in adulthood (as opposed to other impulsive disorders) require clarification (Crowell et al., 2009). One key differential is likely to be the unique dynamics of the interpersonal vulnerabilities observed in individuals with BPD (as discussed below).
- As temperamental sensitivity appears to be an important component in the development of BPD, future work should consider *early* psychosocial risk factors, which likely play a key role in the development of temperamental vulnerability. Exposure to stress or toxins, prenatally, may predispose the individual to temperamental sensitivity, as key brain systems, including the HPA Axis, are extremely susceptible during this stage of development (Cozolino et al., 2002). Incorporating prenatal factors into the longitudinal study of BPD, would allow for the assessment of psychosocial risk factors, uncontaminated by the temperament of the child; thereby reducing interpretative concerns regarding

reverse causality. Ultimately, such studies (in addition to genetic endeavours) could help researchers ascertain whether the neurobiological dysfunctions observed in BPD patients are pre-existing, i.e. due to genetic or prenatal factors, or a consequence of the disorder itself (Lieb et al., 2004).

- Interpersonal dysfunction is a core symptom domain of BPD (Franzen et al., 2011). Experimental studies reveal that individuals with BPD display cognitive biases during social interactions with others (King-Casas et al., 2008; Sharp et al., 2011). Future studies should explore the physiological underpinnings of the affiliative system, specifically the role of neuropeptides, e.g. oxytocin and vasopressin, which may then be altered in BPD patients (Stanley & Siever, 2010). Similarly, further work pertaining to the specific psychological underpinnings, e.g. mentalisation strategies, of impaired social interactions in patients with BPD could inform therapy based treatments (Meyer-Lindenberg, 2008).
- This thesis has focused on the *risk factors* and *vulnerabilities* implicated in the development of BPD. Due to the complexity of PDs, future studies should also consider potential *protective* or *resilience* factors (Masten, 2001), which may prevent the canalisation of BPD psychopathology. Vulnerabilities may be exacerbated by certain factors, but may also be attenuated. For example, positive sibling relationships may nullify the negative effects of maladaptive parenting or peer victimisation (Bowes, Maughan, Caspi, Moffitt & Arseneault, 2010). An awareness of these protective factors could prove useful in informing targeted intervention strategies.

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