The theoretical relationship between stress and negative symptoms in first-onset pscyhosis

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Abstract

First-onset psychosis offers a unique window in which to observe the longitudinal interaction of the many semi-independent factors associated with the evolution of severe psychopathology. Research into these factors has been stimulated by the emergence of several vulnerability-stress models of psychosis. These models highlight interactions between biological and psychosocial precipitants, acute illness, and long-term outcome, and suggests that even if a biological vulnerability is present, this may not inevitably result in a functional 'disease'. It also has the potential to account for the significant heterogeneity and non-specificity of symptomology in severe mental disorder. However, a comprehensive theory of the many co-occurring and multiplicative factors that mediate dysfunction has so far eluded the literature, with segregated theories that lacking in detailed observable and subjective factors typically being presented. In this study, an integrative approach is taken to research associated to stress and psychosis, and it is argued that models of trauma and posttraumatic stress disorder may provide a valuable framework for explaining a range of psychotic phenomena. Specifically, the theoretical overlap between the phenomenology of stress and the negative symptoms of psychosis is developed as a contribution to better understanding deficit psychopathologies. Case studies from clients in a first-onset psychosis intervention programme will be presented to illustrate a number of associations between adverse life events and negative symptoms. Implications are discussed for integrating multiple factors related to mental illness, the subjective experience, and the socio-cultural context, into theories of psychosis. The clinical implications of regarding components of negative symptomology as potentially modifiable are also discussed.

Introduction

Over the relatively short history of psychology as a general science there has been a significant growth in research efforts aimed at the many domains of human functioning. This has resulted in a wide array of models and theories, often reflecting the particular paradigmatic orientation of the researchers, as well as features unique to the focal data set. As theories are vehicles for conveying the meaning components of relationships between events, or explaining the occurrence of patterns of events, theories can be ranked hierarchically (Ward & Hudson, 1998) with upper level theories providing increasingly inclusive amounts of explanatory information. Thus philosophers of science have promoted theory as a guiding process to empirical observation of the world, as well as offering building blocks to new levels of understanding via the development of comprehensive theories. However, with the developing sophistication of the human sciences, research efforts tend to be increasingly circumscribed, with methods that promote reliability over theoretical validity (Foullette & Houts, 1996). Also, research outcomes are often presented as single-factor associations, with potential overlapping factors between research models remaining unexplored – resulting in a wide array of segregated theories (Kalmar & Sternberg, 1988).

These issues apply equally to the multidisciplinary field of psychopathology, in which well constructed theories are the means by which assumptions and predictions related to mental illness are validated. Part one presents a range of issues related to the development of theory in psychopathology. Alternative approaches to existing research directives in the psychoses are argued for, highlighting the potential for building more valid and comprehensive models of complex biopsychosocial dysfunction. These issues provide the basis for the arguments made later for reconceptualising the psychoses, integrating theories from other areas into existing theories of psychosis, and for taking a more phenomenological approach to understanding the full gamut of thoughts, feelings, and life-experiences that constitute psychotic decompensation.

Part two extends this discussion in a critique of the schizophrenia literature. It is argued that while existing diagnostic categories facilitate research and communication, they also have a tendency to lend an air of validity to otherwise tentative agreements regarding diagnostic specificity, as well as allowing observers to disregard potentially critical data because it does

not fit the diagnostic category. The result is a self-supporting cycle of believing that diagnostic abstractions are more valid and real than the idiosyncratic features of the client's presentation, when it is the person that needs to be understood before the 'disease' (Holzman, 1996). It is further argued that these problems are highlighted in the psychoses, where a number of divergent precipitating factors and symptom clusterings result in heterogeneous research findings and a high co-occurrence of non-psychotic disorders. In chapter four, issues from crosscultural psychology are used to illustrate the potential benefit of taking a symptom-based approach to producing more meaningful conceptualisations and descriptions of complex psychopathology, and overcoming some of the problems associated with the current diagnosis of syndromes. The application of this approach is then presented in relation to a biopsychosocial vulnerability-stress model of psychosis.

In parts three and four, the schizophrenia construct is decomposed into two distinct syndromes, one characterised by positive or florid symptoms, and the other by negative or deficit symptoms. The importance of the negative symptoms in predicting the course and outcome of schizophrenic illness is highlighted. However, like the parent diagnosis (schizophrenia) the negative symptom construct is found to be characterised by variability in presentation, outcome, and causality. In terms of the multi-causality of this construct, a primary negative dimension and a secondary negative dimension are taken from the literature, and it is argued that secondary negative symptoms overlap or reflect reactive processes that are contingent on the person's response to adverse life events, including the primary psychosis. Further, it is argued that due to methodological and conceptual difficulties associated with complex systems, validation of negative symptoms – getting at the 'real' events – will require significant theoretical development at the levels of phenomenology, single factor associations, and multi-factor integration. This discussion is used to set the scene for exploring an association between psychosis and specific negative symptoms and known stress response processes.

Part five serves as an introduction to the concept of stress, highlighting the fact that stress, like psychosis, is not a unitary concept, but is comprised of a range of factors. These include the stressor or events, and the emotional and / or instrumental responses that relate to appraisals of the meaning of the stressor. Thus indicating stress as a conditional process that involves a transaction between the person and the environment. In chapter 13 the diagnostic category of posttraumatic stress disorder (PTSD) is introduced as a correlate of severe stress

response that has cognitive, emotional, and physiological components. In particular, it is argued that the sequelae of stress offers a vivid description of how psychological factors can elicit changes in the brain, and which can confer long-term vulnerability for complex psychopathological disturbance. It is suggested that trauma models may provide a meta-framework for organising and explaining subjective complexity and longitudinal processes in severe psychopathology.

The relationship between stress and psychotic phenomena is discussed in part six. In chapter 16 research from the field of health psychology is presented to describe an association between stress and general illness, highlighting the subjective issues of loss, uncertainty, identity, and adaptation that confront people who experience chronic or severe illness. These processes are then used to develop a theoretical relationship between psychosis and stress. It is argued that mental faculties represent an organisation of complex biopsychosocial regulatory systems that can 'accumulate' distress from internal and external sources over long periods of time, and that the processing of this distress can be both an outcome (illness), as well as a vulnerability for future disorder. In chapter 15 these interactive longitudinal factors, along with the potential benefits of taking an integrative phenomenological approach, are then applied to negative symptoms in both PTSD and psychosis. Similarities in phenomenology are highlighted, and it is argued that stress response process may have considerable explanatory power for the cognitive, affective, and social deficits found in some cases of psychosis, particularly when there is significant stress factors in the client's history, and / or reactive processes are implicated in the presentation.

In part seven it is argued that persons suffering their first episode of psychosis may experience significant personal and social disruption due to the schematic novelty of general mental deterioration, psychotic phenomena, and hospitalisation, in the psychosocial context of disrupted developmental targets. Totara House, a first-onset psychosis intervention service in Christchurch, New Zealand, is presented as a case study of the comprehensive management strategies which can be employed in this population. Part seven is concluded with six case histories from Totara House that illustrate the proposed theoretical associations between stress / trauma, illness, developmental stage, and concomitant negative symptoms.

Part eight provides an overview to the preceding chapters, reviewing the theoretical assumptions that are proposed, and highlighting the implications they hold for developing

multi-factorial explanatory models of psychosis, and potential methods for evaluation of the models. A number of clinical implications are also presented, in particular how trauma therapy may be integrated with existing interventions for the management of psychosis.

Part One

Theory Development and the Psychoses

Definitions I Clinical terms related to psychosis

This chapter will provide definitions for the main terms clinical terms referenced in this part of present thesis.

Mental Disorder

A clinically significant behavioural or psychological syndrome or pattern (American Psychiatric Association, 1995).

Schizophrenia

This is a general label for a number of psychotic disorders with various cognitive, emotional and behavioural manifestations. However, the borderline that distinguishes schizophrenia from other disorders is not obvious, and differential diagnosis can be problematic (Reber, 1985).

Schizophrenia is a disturbance that lasts for at least six months and includes at least one month of active-phase symptoms, the symptoms being delusions, hallucinations, disorganised speech, grossly disorganised or catatonic behaviour, or negative symptoms. (American Psychiatric Association, 1995).

Positive Symptoms and Negative Symptoms of Schizophrenia

Terms first coined by Hughlings-Jackson in an attempt to define the two major schizophrenic syndromes. At a clinical level these two subtypes represent distortions and/or excesses (positive symptoms), or reductions/losses (negative symptoms) in the individual's normal functioning (Andreason & Olsen, 1982).

Psychosis / Psychotic Disorder

A general term for a number of severe mental disorders of organic or emotional origin (Reber, 1985).

A dysjunction of thinking from reality - a severe mental disturbance in which thinking is disconnected from external reality be way of unreal perceptions (e.g. hallucinations) and unreal beliefs (e.g. delusions) (Trzepacz & Baker, 1993).

The narrowest definition of *psychotic* is restricted to delusions or prominent hallucinations, a more broader definition would include other positive symptoms of schizophrenia. Defined conceptually as a loss of ego boundaries or a gross impairment of reality testing. The different psychotic disorders emphasise different aspects of the various definitions of *psychotic.* (American Psychiatric Association, 1995).

Its like losing the boundary, or how to know the difference, between what you think, imagine or fantasise, and what's really happening in the external world. (Client's subjective account of their psychosis).

For differentiating between the psychoses, generally classification rests on:

A. Factors believed to have caused the psychosis (substance, mood, etc.).
B. Specific patterns of symptoms that occur during the psychotic episode.
C. The duration and outcome of the psychosis - chronicity (greater than six months) indicates a confirmation of the diagnosis of schizophrenia.

First-Onset Psychosis

Individuals presenting to treatment facilities with psychosis for the first time. The early course of psychosis has three phases: premorbid, prodromal, and acute (Larsen, McGlashan & Moe, 1996). The first-onset population is currently receiving considerable attention because, a) shifts toward a vulnerability – stress model of psychosis have highlighted potential areas of secondary prevention in psychotic disorder; b) identification of prodromal indicators is less confounded by chronic psychopathology in the early part of the illness course, thereby making the first contact a crucial point of research into psychotic processes and prevention; and c) early intervention is associated with improved outcome, reduces secondary morbidity, and long term cortical damage (Birchwood & Tarrier, 1992; Birchwood, Todd & Jackson, 1998).

Neuroleptic Medication

A range of drugs used in the treatment of the psychoses due to their 'normalising' effect on positive symptoms, and to a lesser extent negative symptoms (Adams & McKergow, 1993).

Chapter 1 The role of theory in scientific enterprise

This chapter introduces the role of theory in the general sciences. It is argued that theory has a pivotal scientific function in that it guides new research, and provides organisational structures that represent the relationships between events that are observed by researchers. A caution is made in terms of making theory explicit to promote ongoing progress in validation and comprehensiveness. Problems with the DSM classification system are discussed, highlighting a potential scientific conflict between the enormous amount of research generated and guided by this systems, while the system itself operates from an implicit and weak theoretical stance that makes re-conceptualisations of mental disorder potentially difficult. This discussion in this chapter sets the scene for the alternative approaches to methods of theory construction presented in the following chapter.

"A central feature of the scientific enterprise for psychologists involves the process of theory development: creating new theories, evaluating old theories, and resolving the resulting discrepancies between both competing theories and the data to which they apply" (p.153).

As Kalmar & Sternberg (1988) have so aptly announced, the business of getting involved in theories of human behaviour is a critical part of the psychologist's vocation. Whether it be in the clinic, business, or research laboratory, theories provide structure to the data we observe and manipulate, and provide guidance and focus to future empirical endeavour. Thakkar (1997) states that this interaction of existing theories with new theories and new data gives rise to a "dynamic process wherein the advancement of knowledge involves repeated modifications to the status quo" (p.30). The interaction between theory and empirical data in the general sciences was thoroughly discussed by Hempel (1965) following a request from the American Psychiatric Association to apply the tenets of logical empiricism to the taxonomy of mental disorders. Hempel (1965) asserted that both theory and observable data are interdependent domains that are mutually responsive within the scientific process, and are expressed through scientific terms. Extrapolating from logical empiricism in the field of psychopathology, the scientific terms are diagnostic categories which are comprised of lists of the signs and symptoms which specify particular conditions, the role of data is in describing

the symptomology, and the role of theory is in the postulation of etiological mechanisms (Hempel, 1965; Thakker, 1997) via theory formulation, theory testing, and theory development (Kalmar & Sternberg, 1988).

This method of science provides meta-level guidance, and is arguably critical to the field of psychology, because 1) psychology is a historically short lived discipline, 2) there are few metatheories in psychology (Holzman, 1996), possibly reflecting problems in integrating observed data with existing philosophies of human behaivour, 3) the enormous growth and productivity in the field of neurophysiology, which applies 'reductionist' approaches to data gathering, and 4) the great diversity of interest spawned by the various psychologies. However, psychology identifies itself generally as the science of the mind or mental life, and behaviour (Reber, 1985), albeit with different areas of the field highlighting particular aspects as suits their endeavour. Therefore psychologists need to abide wholeheartedly to a method of scientific progress, such as that elucidated by Hempel. Neither theory nor empirical research can be excluded from this process.

The same caveats apply to the domain of multi-disciplinary field of psychopathology – the science of mental disorders (Reber, 1985). In its essence, the science of psychopathology is founded on the classification of human behaviour that somehow deviates from contextual expectations, and as classification is at the heart of all scientific disciplines, progress needs to be governed by explicit methods (Barlow, 1991). It is in this vein that Hempel briefed the American Psychiatric Association on an appropriate scientific basis to its classification system the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (American Psychiatric Association, 1995). However, the business of doing science in psychopathology has been questioned due to mental disorders being hypothetical constructs that are abstract representations of peoples thoughts, feelings, and behaviour (Widiger & Sankis, 2000), and therefore do not readily lend themselves to reliable empirical observation. This problem was discussed by Hempel (1965) and Thakkar (1997) in that the feedback from data to theory ('empirical import') requires the matching of terms (categories of disorder) with their empirical correlates (specific signs or symptoms). Hempel states that this is problematic in the field of psychopathology as mental states are not conducive to the delineation of exact boundaries and readily observable mechanisms, and therefore operational definitions are less precise in the general sciences than in the physical sciences.

In response to this position, it would appear that the study of mental disorder by any other means than the physical sciences, such as neurology or biological psychiatry, would clearly have trouble exacting Hempel's method of empirical import. However, as science can be conceived of as a method rather than an outcome, it remains that psychosocial research cannot be disregarded as non-scientific, or considered as a distraction from scientific progress (Widiger & Sankis, 2000). Arguably, it is the application of scientific methods in all knowledge-based enquiries that should predominate, particularly for the general sciences where multiple interactions and outcomes between factors are common. For psychopathology, the scientific goal may be a robust and coherent approximation to what the 'real' data might be, suggesting the importance of sound methods to reduce the 'noise' in the data set, rather than potentially unattainable critical indicators with absolute predictive power and reliability. For example, the process of empirical import is only one of two aspects of logical empiricism according to Hempel (1965). A second function is 'systematic import' which represents the way theory controls the empirical data gathering process, this being the converse but interdependent aspect of scientific method. Therefore, scientifically valid progress is premised on the balancing of these two processes, theory development and testing data, not either or exclusively. Further, this balancing needs to be *explicit* to avoid situations in which researchers from either approach make implicit assumptions, either about the reliability of the data used to support their models, or the validity of the models being proposed as a guide to empirical work. Contemporarily, the argument for explicit statements of paradigmatic objectives has been applied to the atheorietical stance of the DSM-IV (Follette & Houts, 1996). The DSM system has succeeded in abandoning the largely untestable psychoanalytically based taxonomy, and increasing reliability in classification. However, Follette & Houts (1996) argue that the DSM system itself may be unable to be evaluated, as its 'weak' atheoretical position, which they argue is a façade to the underlying medical model upon which classification of syndromes is premised, cannot be robustly tested against competing models.

To highlight the potential difficulties in adhering only to empirical import and considering this to be a self-fulfilling science of human behaviour, the field of neurophysiology offers some points of discussion. Brain research has provided great advances in knowledge of how the brain operates, and allowed revolutionary ideas to explode into fields such as biomedics, psychopharmacology, education, ergonomic design, information technology, and computer science. The 1990's have been dubbed the 'decade of the brain'. However, this approach to

human functioning has also been castigated for its reductionist approach, and resulting inability to account for the wide spectrum of interrelated processes involved in human functioning (Widiger & Sankis, 2000). For example, the indications that there is a single gene for schizophrenia is confounded by the variability found in studies of cohorts with this illness, indicating that other factors are significant in this disorder (see Part 2 for further discussion). Therefore, a model that posits that there are biological contingencies for all human functioning, is potentially theoretically inadequate, and may result in many invalid propositions. Perhaps the reductionist may pose the biological factor as a vulnerability that is potentiated by the environment. However, the lack of theoretical advancement in the reductionist approach potentially gives pre-eminence to biological determinants, and leaves critical environmental factors unaccounted for. In support Holzman (1996) states "experimentation and measurement go on regardless of any overarching theory. Yet, in our more reflective moments, some of us feel a need for guidance by a view that helps us to know what is required to complete a proper study of a subject matter, lest we lose sight of what is important to include in its study....Accumulation of sheer numbers of facts does not make a science....It is the *ordering* of those facts and numbers and their *relation* to each other that give them meaning. Such is the guidance provided by grand theory...." (p.595, italics added).

A reconceptualisation of the problem of 'doing science' with abstract constructs, such as mental disorder, may be to make explicit the hypothetical nature of the focus of enquiry, and use this to develop encompassing and testable theories of psychopathology. Two factors may aid this process at a operational level: 1) explicitly accepting the conceptual nature of mental states, thereby recognising the theoretical malleability of data, and 2) supporting ongoing theoretical development as a means of enhancing the scientific validity of proposed categories and related etiological mechanisms. In support of this proposition, Dar, Serlin, & Omar (1994) argue that "we strongly believe in the importance of theory in guiding research....the task of the researcher becomes much clearer when studies are derived from theory and specific predictions are made" (p.80). Dar et al. add that such an approach can make research methodologies stronger, such as statistical testing, as *a priori* predictions increase statistical power via planned procedures and the use of one-tailed vs. two tailed tests. Similarly Kulka (1989) asserts that "when theory does not play a selective role, our data gathering activities belong to the realm of journalism rather than science"(p.794).

Further, it could be argued that the binding of concepts of mental disorder to the predominant disease-orientated classification system (DSM) may dissuade some researchers from going beyond separate diagnostic categories to developing alternative or integrative theories. Follete & Houts (1996) argue "the DSM has become the de facto standard for defining what to study and how to report data. Of course, one can choose to ignore these categories, but review boards are rarely sympathetic to such attempts. One can study members of diagnostic categories who exhibit comorbidity with other diagnostic categories, but even this will often draw concerns that one is now confounding different syndromes and will not know how to generalise the results. This implies that syndromes have not just reliability but now validity as well and that it is an a priori assumption that it is important to keep research on clients with different syndromes separate" (p.1129). Given these problems, the next chapter considers alternative approaches to psychopathology research.

Chapter 2 Alternative approaches to building theories of psychopathology

Kalmar & Sternberg (1988) propose that much of the theoretical development in psychology has been in the form of 'segregated' theories. The central feature of this process, they suggest, has been the juxtaposition of theories to highlight their divergent predictions, rather than describing commonalities and associations. This approach is predicated on scientific progress being the product of the accumulation of empirical data from different sources, with the disadvantage of potentially promoting fragmented perspectives. Kalmar & Sternberg argue that an alternative and potentially better method is not to reconcile competing theories, but to "grasp in enough sophistication the guiding assumptions underlying competing theories" (p.165). This strategy, which they dub as 'theory knitting', requires the integration of the best aspects of competing theories for a given phenomena. Thereby it is expected that emerging theories will handle greater volumes of information while avoiding problems of misleading research directions as the explication of guiding assumptions is the critical feature in the theory building process. However, two interrelated factors need to be kept in mind, 1) care needs to be taken in 'knitting' theories together to avoid the 'crazy-quilt phenomena', the result theories with little predictive power, and 2) theory knitting requires the presence of already developed theoretical structures, and therefore is inappropriate in the early stages of research. The value of this approach appears to be in providing an overview of theory building that can potentially mine the most important aspects from existing theories. This information can then be subsumed into more powerful theoretical accounts of the targeted phenomena, that potentially have a greater level of conceptual redundancy.

Arguably, the field of theory development may be the best arena in which the incumbent limitations could be modified; Follette & Houts (1996) suggest the use of theory-based research programs that make strong explicit statements regarding the guiding paradigm. For instance, developmental psychopathology is one area in which theories of biopsychosocial development have been melded data on functional and dysfunctional behaviour (Cicchetti & Cohen, 1995). One example has been this model's ability to provide coherence between categories of antisocial behaviour, such as convergent pathways between Oppostional Defiant Disorder, Conduct Disorder, and Antisoical Personality, as well as protective factors for

deviancy (Paterson, 1982; Moffitt, 1993). However, in regard to the DSM system, it is not to say that the effort to delineate categories of disorder itself is problematic (Sommers, 1985), rather that the relationships between symptoms and syndromes, as well as coexisting theories of etiology, need not be constrained by the classification process. And, as Follette and Houts (1996) pointed out, avoiding biased accounts of mental disorders means that issues of validity and reliability can be separated and addressed independently.

One alternative approach may be to explicate different levels of hypothesised relationships between observed events, as has been done by Ward & Hudson (1998) within the area of sexual offending, an area, they argue, that has an absence of integrated theory building. Ward & Hudson have proposed three structural levels of theory, level referring to the degree of abstractness or comprehensiveness. Level I represents comprehensive or multi-factorial theories that describe relationships between causal factors, such as poor social skills, and focuses on distal factors as well as referring to proximal triggers. Level I theories are not to be confused with global theory which, they argue, represents integration across all three levels, with each level providing unique and valid information. Level II represents middle or single-factor theories that explain specific phenomena, centring around a core construct such as maladaptive beliefs, and provide the conceptual building blocks for comprehensive theories, again focusing more on distal factors. Level III represents micro-theories or models of actual cognitive, behavioural, motivational, and contextual factors for specific events, the more fine grained nature readily lending itself to single case enquiries such as found in unique behavioural chains. Ward & Hudson (1998) argue that it is this descriptive level that is most overlooked, yet is crucial to theory development as it provides the 'touchstone' for the more general theories. Put plainly, the upper level theories need to be able to account for actual behaviour in individuals to be valid. Overall, the explicit nature of this structure could drive more valid descriptions of mental disorder as theories at each level are independently tested and then matched against each other, as well as subjective and objective descriptions of actual client behaviour. Thus pulling a range of segregated theories together into potentially more valid accounts (categories) of specific pathological states. In chapter four symptom-based research is presented as one possible approach to operationalising this method.

The arguments raised above about moving toward comprehensiveness in theory development generally, are equally able to be applied to the psychoses. This is particularly so given that a review of the schizophrenia literature indicates the high degree of between-subject variability

in presentation and illness course. For each factor associated with the psychoses that has been studied – genetic, biological, and psychosocial, research outcomes have only applied to a proportion of the research subjects, even when statistically significant. Therefore, to both describe and potentially explain the heterogeneity within the schizophrenia syndrome, the theories used would need to contain a variety of interdependent and nonexclusive factors. Further, it could be argued that this approach is critical given the current dissecting of schizophrenia into separable dimensions of florid / excess symptoms and deficit symptoms, with individual presentations being characterised by a mixture of both symptom types (see chapter 7). Potentially testing such a model may come from looking for consistent and plausible patterns in the relationships between particular factors, as replicated again and again by single subjects, or larger samples that are grouped according to their presenting symptoms rather than by potentially non-differentiating meta-categories used in psychiatric diagnosis. This would be more in keeping with the structure suggested by Ward & Hudson (1998). Further, given the enormous amount of research reported in the area of schizophrenia, the process of theory knitting proposed by Kalmar & Sternberg (1988) could readily be extract and integrate the most useful of this information.

For example, while a fuller discussion of the values and limitations of diagnostic classification appears in Part 2, for present purposes, Person's (1986) states "the focus on the diagnostic category of schizophrenia inhibits the development of adequate theories of the psychological processes underlying the symptoms of schizophrenia in several ways.... if they ignore symptoms, investigators risk committing themselves to theories of schizophrenia that do not account for any of the overt phenomena actually observed in schizophrenic [*sic*] patients" (p.1254). This situation is what Ward & Hudson (1998) predicted would occur if micro-theories were not used to test out more comprehensive theories. In a similar vein, Strauss (1992) has proposed a descriptive approach that takes into account the client's subjective account of their schizophrenia to better understand the factors that most concern them about their illness, and the factors that help them cope, thereby enabling a greater appreciation for the continuum between normality and dysfunction, as well as increasing the power of subsequent interventions. Reflecting on the need for valid approaches in psychopathology, Mirowsky & Ross (1989) state "no one should forget that we are talking about the disturbing or disruptive thoughts, feelings, and behaviours of people, and not about unseen entities that are somehow the cause of it all" (p23).

Overall, in the present thesis Kalmar & Sternberg's (1989) integrative approach to theory building is taken as an overriding motivation and justification for the present theoretical exploration of the psychoses, and Person's (1986) and Strauss's (1992) proposals for taking a more phenomenological and person – centred approach to clinical research as the 'method' by which a valid future integrative model may be reached, and tested (Ward & Hudson, 1998). However, to argue that the diagnostic category of schizophrenia is theoretically underdeveloped, and therefore also the associated negative symptom construct that is the focus of enquiry in the present thesis, first requires evidence. Therefore, a critique of the literature and arguments for alternative models and methodologies are presented next.

Part Two

Exploring Schizophrenia

Chapter 3 Introduction to Schizophrenia: psychiatry, diagnosis, and research

This chapter provides an orientation to schizophrenia, briefly surveying the significant impact of this illness on individuals and society. Then the dominant biological "disease" model of schizophrenia is reviewed and critiqued in relation to the interaction between the assumptions of mental disorder embedded in this model, the diagnostic system used in the representation of schizophrenia, politico-professional issues, and the diagnosis of schizophrenia. It is argued that the incumbent model is unable to fully account for the well documented heterogeneity found in samples of schizophrenic illness, and that this has impacted on the model's ability to present a coherent theory of schizophrenia and guide unbiased research.

Many people who experience the symptoms associated with schizophrenia either continue to live full and meaningful lives, or have gone on to once their acute illness is managed. For others, schizophrenia can have a devastating effect. The level of capable functioning that most non-schizophrenic individuals take for granted is drastically altered for such individuals. Emotional well-being, social interaction, occupational pursuits, creating and maintaining close relationships, living as part of a family, and exploring personal enjoyment/recreational options are all abilities detrimentally affected by the presence of mental illness. This loss of functioning can be exacerbated by losses of self-confidence and self-esteem as the individual reacts to a world that can be often radically distorted by uncontrollable cognitions, and the traumatic experiences that can both precede and accompany these disturbing changes. This response is further reinforced by self and social stigmatisation (Byrne, 1999), loss of social stability and social withdrawal (Halford, Schweitzer & Varghese, 1991). Included in this rubric is the potent effect severe mental illness can have on close interpersonal relationships, as: 1) implicit rules that govern relationships are contravened as a consequence of the unwell individual's variable cognition's and behaviour (Bellack, 1997); 2) families and close social contacts react aversively to the presence and presentation of schizophrenia (Carling, 1995). Additional to this impact, the National Foundation of Brain Research (1992) has estimated that nationally (U.S.) the financial costs of schizophrenia are greater than that spent on the treatment of cancer. Contemporarily, social and political interest in the psychopathological

nature of schizophrenia has become pronounced due to the popular media's exposure of cases of singularly violent behaviour perpetrated by individuals labeled 'schizophrenic', even though the literature suggests that individuals with schizophrenia do not commit serious crime at rates significantly greater than the general population (Read & Law, 1999; Rice & Harris, 1995).

Given the considerable impact of this illness on individuals and society, as well as its heterogeneity of severity and clinical presentation, many models of etiology and illness course have been developed. Broadly speaking, most mental health professionals approach the study and conceptualisation of the schizophrenia spectrum disorders via a biological 'disease' model (Boyle, 1990), or accept at least a biological component in these disorders. As well as the professional frameworks and inherent biases that clinician's have for regarding psychiatric illness, three major historical events may account for the general acceptance of the disease model. Most significant in the history of psychiatric care was the perceived futility of trying to rehabilitate such cases. This was due to the apparent refractory nature of the illness to existing forms of intervention, which included psychotherapy / analysis, insulin coma therapy, psychosurgery, and electroconvulsive therapy (Davison & Neale, 1996). This led to the incarceration of large numbers of these patients in asylums and mental hospitals throughout the developing world. Secondly, the discovery of medical interventions such as the phenothiazines, between the 1940's and 1950's, was hugely significant, both in terms of the impact these had on previously intractable symptoms, and through their stimulation of a 'new wave' of research into the neurophysiological nature of schizophrenia. Of these interventions, the use of neuroleptic medications, such as clorpromazine, thioridazine, and haloperidol, and new generation neuroleptics, such as risperidone and clozapine, have been used as exemplars of the biochemical underpinnings of the 'schizophrenic disease'. These biochemical interventions are now treatment of choice in most medical settings and have facilitated the deinstitutionalisation culture.

Within psychiatry, the prolific acceptance of biological explanations of schizophrenia can be arguably seen as due to two factors. Firstly, the historical taking over of the management of the mentally unwell by physicians has seen this domain of social care become increasingly medicalised, with mental illness conceptualised and discussed around themes analogous to physically diseases. Of these, schizophrenia has become central to this process as it represents a disturbing and costly (Durand & Barlow, 1997), and therefore politically noticeable, mental

deviance. At a professional level, schizophrenia could be seen as a medico-psychiatric 'straw-man', not, it seems, to be challenged, but as a prototype of how mental illness can be reified along biological lines. Bentall, Jackson & Pilgrim (1988) state "the continued dominant role of the medical profession in the management of psychosis depends upon the continued application of a biological approach" (p.315), with similar sentiments having been aired by writers such as Boyle (1990), Bentall (1990), Mirowsky & Ross (1989a).

Secondly, the classification system that is increasingly used by western psychiatry, the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM), implicitly promotes the identification of disease entities via a medical model (Follette & Houts, 1996), albeit a weak biological model. In his book review of the DSM (4th edition), Guze (1995) states "The emphasis and attention to psychiatric [*sic*] diagnosis reflects a broad redirection toward the goal of reintegrating psychiatry into medicine generally. Diagnosis has been the foundation of medicine for centuries, and its renewed emphasis in psychiatry expresses the movement toward the medical model for psychiatric [*sic*] disorders" (p.1228). That is not to say that the categorical approach of the DSM is untenable as categories significantly enhance people's social perceptions (Macrae & Bodenhausen,2000). Some of the problems associated with applying categories to syndromal entities has been addressed in the DSM-IV by making categories polythetic prototypes. Although, this has produced its own problems, particularly with differentiating between categories when there is a high overlap of symptoms, such as between schizophrenia and bipolar affective disorder (Moskowitz,1993), and heterogeniety within categories to the extent that two individuals with the same diagnosis can share few if any symptoms, such as obsessive – compulsive disorder (Frances et al.,1995).

The use of the DSM by mental health practitioners has the two fold advantage of providing recognisable categories of disorders that can stimulate reliable communication between professionals, and provide a more uniform platform for psychopathology research. Clinically, the DSM system provides good "rules of thumb" indicators for different types of disorder, and helps predict which treatment will be effective depending on the clients symptom cluster. However, the limitations of the DSM must be kept in mind, especially by those who are new to the incumbent system (Flaum, 1995). In practice the DSM cannot be atheoretical, particularly in terms of guiding research proposing common etiology for the DSM syndromal disorders, as the DSM "specifies those things we agree to study and thereby accept as propositions of a theories' underlying ontology" (Follette & Houts, 1996, p.1127). Therefore,

the increasing reliance on standardised psychiatric diagnostic manuals does run the risk of providing a veneer of validity and utility to the psychiatric disease paradigm. In recent years this trend has been further systematised through the emergence of clinician rated diagnostic scales that have provided clinicians and researchers with standardised diagnostic tools with quantifiable reliability. However, validity for the entities measured is premised on the underlying diagnostic system, which as discussed, is biased and potentially flawed. Again, particularly for professionals new to the incumbent system, there is a danger that the arguable 'face-validity' of disorders presented in the DSM will be confused for construct validity, and problems associated heterogeneous constructs such as schizophrenia will be potentially overlooked.

The centrality of the biological – disease paradigm in psychiatry and the use of a standardised diagnostic system are both mutually supportive processes in the medicalisation of the mentally ill, an approach premised on social and economic power (Follette & Houts,1996). Further, the proliferation of new diagnoses with every edition of the DSM, which in itself has been argued as highlighting the untenability of the DSM system in its current state (Follette & Houts,1996), is suggested by Ross & Pam (1995) as a reflection of the "moral authority of psychiatry" (p.217) in society. In this respect, the DSM could be considered to be a systemisation and operationalisation of the 'protector-of-society' role dubbed by psychiatry, and which has historically allowed psychiatry to take control over what behaviour is acceptable and not acceptable. In terms of the promulgation of schizophrenia as a psychiatric disorder, it has been argued that this construct is predominately based on the selective attention to conflicting bio-genetic data; data presented with supportive historical data taken from populations quite different from contemporary diagnostic populations; misrepresentation of data suggesting the construct is invalid (Boyle,1990; Mirowsky & Ross,1989a). The potential benefits of having a standardised approach to schizophrenia diagnosis, namely reliability, may therefore be diluted by the disregard then given to alternative explanations for the observed disordered behaviour, and by data that is lost from clinical assessments because it was extraneous to the dictates of the model.

The flow-on from the enormous attention schizophrenia has received politically, socially, and medically, has been the generation of a large body of research over the last 40 years, particularly aimed at discovering the etiology of the schizophrenic 'disease'. A number of plausible casual pathways have been implicated and received support; brain biochemistry,

genetic endowment, neurological factors, diet, season of birth, viral agents, social stress, life events, and family structure (Bentall, Jackson & Pilgrim, 1988). Based on statistical analyses, there is good evidence that schizophrenia is a brain disease (Johnson,1989). Even so, a closer review of methodologies used, and claims derived, indicate that despite the huge effort that this research represents, equivocal or spurious relationships have been the outcome (for comprehensive reviews see Bentall, 1990; Boyle, 1990; Harrop, Trower & Mitchell, 1996; Ross & Pam, 1995). For example, genetic endowment has been accepted as a significant schizophrenic mechanism, making some individuals vulnerable to schizophrenia (Durand & Barlow, 1997). Gottesman's (1991) research indicates that monozygotic twins have around a 50% risk of developing schizophrenia, as opposed to 17% in their dizygotic cohorts, thus suggesting a genetic anomalie, as the twins who share 100% of their genes have a significantly higher concordance rate. However, while a genetic component may be present in some cases, research to date has been unable to clearly disentangle genetic from environmental influence; while 85% of susceptibility to schizophrenia appears to be genetic, the susceptibility is polygenetic and dependent the interaction between physical and environmental factors (Widiger & Sankis, 2000). For example, the "Genain" quadruplets all developed schizophrenia, shared the same predisposition for schizophrenia, and were brought up in the same dysfunctional household, yet their time of onset, symptoms, diagnoses, illness course, and outcome were different (Durand & Barlow, 1997). Durand and Barlow (1997) suggest that while their environments appeared to be the same, children are not brought up in a parallel manner by their parents, further confounding the environment – gene debate.

Methodological problems are also present in well cited genetic studies, especially those in which retrospective family data, and shifting diagnostic criteria was used, often in the absence of blind face to face assessments (Marshall, 1990). Finally, the circularity of the disease-genetic model has played an important role in determining the biological account and management of schizophrenia (Marshall, 1990). However, even if a 'hard' biological marker, such as a single gene, is discovered, this does not predict the use of purely biological interventions, nor preclude non-biological interventions (Widiger & Sankis, 2000). That is not to say that medical interventions are unwarranted, as they play a significant role in the management of the psychoses. However, psychosocial factors, and some psychosocial expressions of biological dysregulation suggest that psychological interventions would be indicated given the environmental mediation of a possible genetic vulnerability.

Similar problems of unequivocal findings plague the other areas of research into the physical signs of schizophrenia. Even so, some research reports appear to highlight statistically significant findings, and provide qualitative descriptions of observed events that are selectively biased toward the underlying causal assumptions. This is at the expense of speaking to the heterogeneity in their data and negative findings, and providing propositions for integrating different models into a more powerful explanation of causal processes. For example, Weinberger et al.'s (1983) selective review of neuropathological studies, they state in their conclusion "several nonspecific CT abnormalities have been found among schizophrenic [*sic*] patients. No one finding is characteristic of all patients, and the majority of patients probably display no CT findings" (p.208). However, they then appear to 'loose' this theoretically important information and state "increased ventricular size, cortical atrophy, or both are the most prevalent findings" (p.208), but these are measured by CT for which the majority of subjects have no findings. They go on, "These abnormalities are not explained by past treatment, and they appear to predate the onset of the illness...as they occur in some first-break schizophrenic [*sic*] patients" (p.208), but 'some' is by no means even a majority of the subgroup of subjects categorised as first break patients, and this group typically has a prodromal phase that can last up to two years, as well as a history of co-occuring morbidity (Kendall & Brockington, 1980), either of which may be influencing brain morphology rather than the index illness. To finish, they claim "taken together, the studies reviewed support the hypothesis that subtle structural pathology of the brain, particularly the limbic system, is a factor in the pathogenesis of the schizophrenic syndrome" (p.209). However, much of their evidence for a specific limbic system anomalie is predicated on CT measured brain morphology, again for which "the majority of patients have no findings" (p.208). Speaking to this type of misinformation, Jackson (1990) states "simply finding differences between schizophrenics [*sic*] and non-schizophrenics [*sic*] on any measure, in the absence of an understanding of how those differences influence the expression of the disorder or the functional significance of the biological agents involved, is clearly insufficient. This interface between biology and psychology is extremely difficult to make and yet is most important for a full understanding of both the phenomena of schizophrenia and the processes from which they arise" (p.119).

Symptom-focused analyses have similarly described little in the way of what is 'clearly' schizophrenic. To note the potentially spurious nature of findings in studies of 'schizophrenia symptoms', Slade & Cooper (1979, in Boyle, 1990) found in their critical analysis of factor

analytic studies that statistically significant (+.67) inter-correlations between a group of 'schizophrenic' symptoms could be replicated (+.70, +.71, +.66) by assigning symptoms to 150 hypothetical cases using a set of bingo numbers. They suggested that rather than representing actual relationships amongst phenomena, these inter-correlations are more an artifact of the use of more severely disordered subjects in schizophrenia research. The point here is that at least with severe or complicated manifestations of 'schizophrenia' the clinical picture is far from characterised by simple patterns of cause and effect. This argument is in line with the observation that the more severe cases of schizophrenia are characterised by "generalised deficits"(Chapman, 1973, cited in Persons, 1986), thus equaling a greater diversity of symptoms at any assessment point. Further, when diagnostic status is linked with proposed psychogenic factors, the majority of research findings are essentially correlational, and therefore even when an apparently observable biological substrate is identified it provides little causal information as biological changes can be readily produced by environmental factors (Rose, 1984; Widiger & Sankis, 2000). Tsuang et al. (1990), following Lewis et al. (1987) and Chapman and Chapman (1977), notes that "the sophistication of new research tools has lead to the neglect of plausible hypotheses. They point out the temptation of using measures as an end in themselves rather than employing them in the context of testing hypotheses. Merely demonstrating differences between schizophrenics [*sic*] on a single measure is unlikely to advance our understanding of schizophrenia" (p.18).

Not only does the current literature lack in causal outcome, the findings presented are further limited by methodological difficulties. At the level of research method, these causal investigations are seriously hampered by the different classificatory systems that are used to enlist schizophrenia subjects at different research sites. More recently, the increased reliance of the DSM system has lead to the use of reliability-determined diagnostic scales which use global scores to ascribe diagnostic status. However, these scales have their own inbuilt inconsistencies. In particular, they tend to lump together significant amounts of clinical case data into atheoretical symptom composites, thus losing potentially important information about symptomological breadth, variance, and history. The outcome can be a reduction in sample validity, and concomitantly a reduction in the power of these investigations to predict, or meaningfully explain, differential clinical phenomena. At a general level of research methodology, the biological assumption on which the disease 'theory' of schizophrenia is garnered is further supported by the hypothetico-deductive model of scientific research which

predominates in psychiatric research. It has been argued that this methodology is flawed in that operationally there is an attempt find support for the hypothesised construct, which even if detected does not validate the theoretical assumptions from which the construct has been effected (Cohen, 1992; Cohen, 1994; Dracup,1995; Ward, Vertue & Haig, 1999). As applied to schizophrenia research, the underlying assumption is that a core neuro-developmental physiological anomaly is the cause of the illness, and all research outcomes are evaluated in terms of the support that they offer this theoretical model *qua* validating the 'schizophrenia' construct.

While sustained interest in the possible biological underpinnings of schizophrenia may have some pragmatic utility, it can be argued that fascination with this aspect of mental illness has lead to artificial constraints on cohesive and integrated theory level development. For example, Flaum (1995) observes that psychiatric nosology currently operates with "tentative agreements to agree" (p.84) regarding the boundaries between mental disorder categories during the current interim, and from which it is 'expected' that syndromes will be validated as diseases. However, a question that appears to be consistently overlooked is whether 'schizophrenia', as a research focal point, maintains construct validity. The fundamental science of determining validity, particularly at an ontological level, is largely ignored in the literature, giving a sense that most researchers and practitioners in the field accept the assumption that the 'schizophrenia' construct is an observable reality. The reason for this may be that schizophrenia is a category of mental disorder, and therefore is treated accordingly the same as all components of the current diagnostic system, which as discussed above is explicitly atheoretical while implicitly supporting the disease model of mental disorder. Bentall, Jackson & Pilgrim (1988) suggest that the lack of progress in discovering the etiologically distinct indicators for schizophrenia may have precipitated attempts to redefine the concept of schizophrenia, a move which would appear to both theoretically and operationally strike at the heart of main stream psychiatric approaches to mental disorder.

Even so, Bentall, Jackson & Pilgrim (1988) note that there has been a resurgence of interest in Kraepelian ideas which has resulted in professionals new to the field being trained to see abnormal behaviour as a series of prototypes, prototypes that may have no scientific value but which serve their profession. In the clinic, this move has served psychologists as well as psychiatrists, as it has enabled psychologists to migrate to outpatient settings to treat the neuroses, while psychiatrists treat inpatient psychoses, thus suggesting a comfortable

professional division (Bentall, Jackson & Pilgrim, 1988). Even so, many of the major critical analyses of psychiatric nomenclature have come from psychologists, certainly reflecting calls such from researchers such as Moskowitz (1993), "it is incumbent upon psychologists to reexamine the implications of continuing to adhere to a medically-driven, categorically based system of nosology, particularly one of questionable validity" (p.11). The issue is not necessarily the political one of which professional body oversees the management of severe mental illness. The issue is to what degree the current dominant paradigm regulates the development of quality research directives, and thus influences how the manifestations of mental disorder are conceptualised and represented. This issue is currently significant as the National Institute of Mental Health "has avowed that a central feature of psychiatric research in the twenty first century will be a focus on the brain, pursued through molecular and cellular biology, along with molecular genetics, complemented by sophisticated cognitive and behavioural science" (Judd, 1998, p.7, in Widiger & Sankis, 2000), and "mental illnesses are real, diagnosable, treatable brain disorders" (Hyman, 1998, p.38, in Widiger & Sankis, 2000). In its favour, the current system has demonstrated that data can be reliably obtained and analysed, and attempts to understand the physiological components of mental illness are highly justified, and have come a long way. However, studying a hypothetical construct such as mental disorders can be no less scientific than studying 'natural' elements, nor do psychological constructs need to be reduced to biological mechanisms to be "scientifically viable", and "optimal scientific progress might be obtained by a more integrative perspective (Widiger & Sankis, 2000, p.380).

A consistent finding which highlights the difficulty in rigidly applying current diagnostic categories, has been the failure of studies employing functional analyses to describe a dividing line between the symptoms of schizophrenia and the affective disorders (Bentall, Jackson & Pilgrim, 1988; Kendall & Brockington, 1980; Moskowitz, 1993). In their six year follow-up study Kendall & Brockington (1980) found that a diagnosis of schizophrenia was unreliable as a predictor of outcome, even though statistical significance was present. They found that this was particularly so for first admission cases who were likely to be diagnosed with an affective disorder at follow-up. In support of the multivariate studies, a subsequent study by Kendall & Brockington (1980) found that no line could be drawn between schizophrenia and affective disorders on the basis of outcome. A further indicator of the lack of selectivity in the schizophrenia diagnosis is the hypothesised arbitrary borderline between neurosis and psychosis, and the continuum that appears to exist between normality and

schizotypal personality disorder, when schizotypal personality features indicates the presence of some symptoms of schizophrenia (Bentall, Jackson & Pilgrim, 1988).

In summary, psychiatry as the major paradigm used for organising and defining the 'schizophrenia' construct, is arguably too narrowly constrained by the disease model of mental disorder to account for the well documented heterogeneous nature of this syndrome. Put more strongly: given the lack of validity as discussed, it is difficult to understand how schizophrenia has survived as a valid diagnosis; "this doctrine would appear as little more than an article of faith" and that "researchers have been pursuing a ghost in the body of psychiatry" (Bentall, Jackson & Pilgrim, 1988, p.314). It may be the weak nature of this biological model which is most limiting for schizophrenia research, as a more strongly stated biological model would be less acceptable to many mental health professionals, and would provide specific propositions that could be countered with other conceptual models. This may ensure a diversity of equally strong models, leading to more robust theories of schizophrenia. It is to alternative conceptualisations and methodologies that we now turn.

Chapter 4 Symptoms and culture: searching for new data in old diagnoses

A potential alternative to the disease model of schizophrenia is the symptom-based approach. This alternative clinical method is presented and critiqued, along with discussing the value of using this approach in light of new understandings of the social–person interaction that is emerging from the field of cultural psychopathology. This chapter discusses some potential 'inner workings' for a methodology that can potentially integrate a wider range of schizophrenia related phenomenology, and sets the scene for the following chapter in which the biopsychosocial model is offered as one potential framework for the method discussed here.

Bentall (1990) has argued that one of the problems with the diagnostic approach to schizophrenia is that the result is hypothetical syndromes are the focus of the clinical investigation, rather than symptoms of disorder. Therefore, much valuable data is lost. This is particularly so, for the psychoses, as a diagnosis is mostly made on the basis of *signs* (data) of disorder, rather than subjectively reported *symptoms* (phenomena) (Alpert, 1985), meaning that some information may never have been gathered. Further, when syndromes are given greater implications, such as explanatory power, than the actual set of statistically associated symptoms that they represent (Bentall, 1990), the search for relationships between the symptoms is deferred, and it is in understanding relationships that coherent theories of psychopathology emerge. In general, a symptom based approach to psychopathology is arguably more able to adapt to the recognition of measures of normality and abnormality lying on a dimensional scale, rather than being categorically separable (Widiger & Sankis, 2000). And this approach seems also to be in keeping with Ward & Hudson's (1998) recognition of behavioural micro-theories as providing critical data from which other higher order theories can be generated and evaluated.

One area of research, cultural psychopathology, is an area that perhaps most highlights the inability of categorical diagnostic criteria to capture the heterogeneity of mental disorder. Cooper (1994) argues that a diagnostic label is a useful tool for communication and

organising information, however, it is not an object that can be transported across cultures easily as it is essentially a social construct. When diagnoses are applied in cross cultural research, the taxonomy used reflects western beliefs and ideals, such as the DSM, and that may not apply to other cultures (Thaker, 1997). For example, searching for schizophrenia in non-western cultures can provide information on the proportion of individuals who match the western criteria, a type of western cultural determinism (Thaker, 1997). It cannot thoroughly describe the manifestation of severe mental illness in the target culture, or, subsequently help reveal meaningful relationships between intrapersonal, interpersonal, and environmental factors on which all psychopathology research is premised. The DSM system has made a major advance in giving attention to cultural issues in its fourth edition, based on recommendations by the NIMH funded Culture and Diagnosis Task Force. However, much of what was to be included was finally axed, leaving only a partial reflection of the dynamic role of culture in psychopathology. Operationally, few specific symptoms were included, symptoms that could be both normative experiences or signs of distress, and culture bound syndromes were put in an appendix and did not include axis I disorders such as Anorexia nervousa which is a western manifestation (Lopez & Guarnaccia, 2000). Lopez & Guarnaccia (2000) argue that the DSM-IV approach gives the implication that cultural issues only apply to cultural minority groups, while cultural researchers "view culture as infusing the presentation of all disorders among all people" (p.575).

An alternative approach would promote the assessment of symptoms rather than syndromes. This may assist in breaking down the cultural barriers that preclude the integration of diverse reasons for, and expressions of, behaviour and cognition. Symptoms are observed phenomena, and therefore, assuming there is no observer bias, they can represent the processes occurring for individuals, rather than representing abstract cultural constructs. Where this alternative system may work best is in tracking symptom change over time, and building models that enrich our understanding of what ameliorates or exacerbates mental illness, and establishing theoretical relationships between illness related events. For example, while no single population or culture is free of schizophrenic symptomology, developing countries have a better course and outcome for schizophrenia and other disorders than do western cultures, an effect not accounted for by the severity of the disorder (Jablensky, 1995). However, what underlying factors may be responsible for the cultural differences remains an unresolved issue (Jablensky, 1995). Potentially the alternative framework could resolve this issue by seeking out relationships between psychosocial factors and observed symptoms. The

same benefits may also apply for schizophrenia within western culture, particularly given the well-documented heterogeneity. At the level of 'micro-culture', family systems may also express themselves through the behaviour and cognition of individual family members. Fabrega (1989) defines culture as "consisting of learned systems of meaning, communicated by means of natural language and other symbol systems having representational, directive, and affective functions, and capable of creating cultural entities and particular senses of reality" (p.415). It could be argued that such a process occurs within many social groups, including families, and where the family system is significantly different from the predominant social system, family-specific entities and senses of reality could occur. For example, one of the case histories of first onset psychosis presented in chapter 17 (case two) reads "Given her exposure to her mother's schizophrenic processes, the members of the father's first family may have developed a culture of magical thinking, particularly as both her and her brother experience the presence of the dead brother."

Persons (1984) has suggested five advantages of the symptom-based approach: 1) the avoidance of problems of diagnosis and classification (this is particularly relevant for first-presentation cases of psychosis); 2) the focus on phenomena that are usually ignored; 3) the facilitation of theoretical development; 4) the recognition that clinical phenomena are related to normal behaviour; 5) the potential improvement in classification which might follow from a better understanding of individual symptoms. To this could be added at least two other advantages, the facilitation of more inclusive case formulations which can be used to guide ongoing evaluation and treatment, and the development of research protocols that are unconstrained by non-valid ad hoc concepts.

Persons (1984) notes that the symptom-based approach has its own flaws, such as the heterogeneous nature of some symptoms impacting on validity in a similar way to categories of symptoms. However, it is also plausible that appropriate studies could show that such problematic heterogeneity may be overcome in one of two ways, decomposing the symptom into its constituent manifestations, or by contextualising the symptom and valuing it in terms of its relationship to other clinical phenomena. While this approach may at first glance appear to come dangerously close to proposing that single-case formulations are the only valid way to describe a psychosis, this expectation is arguably due to the currently poor theoretical development associated with schizophrenia. It could be argued that that the symptom based approach would make professional communication, as well as identifying research

populations difficult. However, within the current diagnostic system possible parallel pathways exhibiting similar phenomena are not explicitly accounted for, making clinical treatment decisions equally difficult. As categories are valid scientific tools for organising information, it would be expected a new model would incorporate different levels of information, but guided by a theory that incorporates parallel or heterogeneous processes. Certainly, focusing on issues of reliability in the diagnosis of schizophrenia has done little to promote theories of the psychological processes that underlie and interact with the symptoms of schizophrenia. Therefore, concurrent with a shift to assessing and tracking symptoms to understand mental processes, theory development is requisite. This is consistent with Hempel's original concern that scientific advancement requires both empirical data and theories to guide data gathering efforts (Hempel, 1965; Thakker & Ward, 1998).

Going back a little to explore the notion of describing psychotic phenomena via single-case formulations, one gets the sense of the balance that needs to be struck between having standardised categories that facilitate professional communication and research, and the elicitation of more fine grained phenomenology inherent in clinical treatment. The clinical vs. research requisites are recognised in the DSM system, as it provides five different assessment axes, and states that a good description of phenomena is more important than making specific diagnoses. However, beyond dictates of professional conduct, the reality of working in time and resource limited treatment settings, adhering to the requirements of third party funders, and the ease of lumping information into higher order categories arguably produces an atmosphere in which diagnoses are applied to cases with little concern given to discriminating individual differences (Macrae & Bodenhausen, 2000). T.McGlashan (personal communication, 14 January 2000) has stated that the two traditions, clinicians and researchers, cannot easily be integrated. That is, the requirements that each has for fulfilling their roles are significantly different (see Appendix I for original). Integration can occur, however, with the encouragement of training psychologists in the use of scientific practice in clinical settings (Stricker & Trierweiler, 1995). Perhaps an initial step may be the use microtheories of client's experiences so that the detail laden explanatory models negotiated between client and clinician could be used for professional communication, and developing patterns of phenomena in research. Arguably, this level of flexibility would require considerable theoretical development, probably resulting in a multi-model, multi-level system, to preempt untenable assertions being made.

Persons (1984) suggests that a symptom-based approach is crucial to the theoretical advancement of schizophrenia as: 1) if symptoms are ignored, investigators risk committing to theories that do not account for observed phenomena; 2) the symptom approach makes it easier to formulate hypotheses about underlying mechanisms; 3) the symptom approach allows for tighter, more elaborated links between proposed underlying mechanisms and the overt phenomena that are hypothesised to be accounted for by those mechanisms; 4) the symptom approach allows investigators to study interrelationships among symptoms, a strategy that facilitates psychological theorising; 5) the emphasis on diagnostic categories can make it surprisingly difficult for the researcher to be clear about the hypothesis his or her study was designed to test. This last point is particularly relevant to hypotheses based around psychological phenomena as even 'core' symptoms of schizophrenia may or may not be present in individual cases, or may readily cross diagnostic category boundaries.

A return to the investigation of specific symptoms may benefit the generation of more valid and possibly complex syndromes, but the selection of symptoms to be investigated requires the presence of a firm theoretical understanding of the roles these symptoms play in the diathesis, etiology, and pathogenisis of schizophrenia (Costello, 1993). Costello (1993) states "what is absent at present is a well-developed theory as to how these experiences and behaviours are related and how they originate. The more thorough investigation of these experiences and behaivours, their relationships with one another, and their relationships to other variables may eventually suggest a more useful construct or constructs than that of schizophrenia" (p.292). One entry point proposed by Knight (1987) is that of cognitive dysfunction, the proposal being to "generate cognitive deficit types as our independent variables, we can study symptoms as our dependent variables, and we can move from the stronger theoretical base of cognitive models in our attempts to make sense of the morass of symptoms of schizophrenia" (p.26). Currently, the cognitive model is already making inroads into broadening the understanding the function and maintainence of delusional belief systems by accessing the subjective meaning in the content of the beliefs, and building personal explanatory models around this information (Bentall, 1996).

In summary Bentall, Jackson & Pilgrim (1988) argue that "schizophrenia is not a valid object of scientific research, and that psychologists should consider new ways of researching into abnormal behaviour". Further to this, Boyle (1990) iterates "no amount of reliability can confer validity and no amount of sophisticated technology can compensate for conceptual

weaknesses. Nor is it likely to enlighten us as to the cultural and social origins of some of the bizarre behaviour presently attributed to 'schizophrenia'. It may be that, at least in the interim, taking a descriptive / symptom based approach to cases presenting with mental disturbances, and giving such descriptions meaningful structure by collecting this data under the umbrella of a model that includes a range of physiological and psychosocial factors, will prove fruitful to the understanding of severe mental disorder, if not challenging. Taking such an approach, a potentially wider range of phenomenology could be integrated into the framework of vulnerability – stress interaction, and recognition of the change in symptomology over time. This, it would seem from the above analysis, would provide greater avenues to understanding the psychopathologic heterogeneity currently subsumed under the schizophrenia diagnostic category than perpetuating research focusing on between category differences. At a clinical level it is likely to help promote the building of individual explanatory models, that may enhance the clients understanding and management of their subjective experience, as well as reminding clinicians of the dynamism of person – illness interactions in the clients they see. It is to the biopsychosocial model of schizophrenia that we turn to now.

Chapter 5 Integrating models of psychosis: the biopsychosocial model

This chapter discusses the integration of biological and psychosocial models of schizophrenia into a comprehensive biopsychosocial model. This model is presented as a framework for structuring data pertaining to the interrelated processes inherent in the psychoses, for highlighting vulnerability–stress interactions in schizophrenia, and for understanding changes in clinical presentation over time. In particular, the specific biopsychosocial model proposed by Ciompi is critiqued.

Biological models of schizophrenia have, by virtue of their focus on physiological mechanisms and events, taken a disease oriented approach. Concomitantly, separate biological oriented models have developed around the familial, latent vulnerability, neurological/structural, biochemical, and functional aspects of schizophrenia, based on what is known to be a general disease process. However, as discussed previously, there has been little equivocal evidence presented to support the notion that the etiology and maintenance of schizophrenic symptoms is entirely accountable to an independent physiological disease process. Arguably this lack of evidence may simply be due to a lack of the technology required to track the neurophysiological aspects of this disorder. However, with the growing interest in the degree of influence that environmental factors have over psychobiological functioning, the disease model may be unnecessarily limiting potentially more conclusive research initiatives (Widiger & Sankis, 2000). In particular, in opting for reliability over validity, both psychiatric research and practice appears to have been focusing on the disease *process* of schizophrenia as communicated between informant and clinician. Arguably, this approach may be somewhat contrived as it actively disengages itself from significant information regarding a range of disease *initiating* and disease *maintaining* factors; i.e. the psychosocial dimension of severe mental illness that psychologists are trained to apprehend and use to make sense of the presenting phenomena. The clinical relevance of this may be most evidenced in the disease model's approach to treatment which appears to disregard the accompanying illness-related psychosocial challenges that impact on treatment prognosis.

Psychological approaches to explaining schizophrenia spectrum disorders have their roots in the early psychoanalytic movement. While Freud gave little direct attention to schizophrenia due to his pessimism over treatment outcome for this population, the notion that somatic disturbance can be associated with psychological factors lead to an increased effort to understand the schizophrenic process (Shapiro, 1981). Jung (1907/1944) however took a more optimistic approach and developed the first psychological model of schizophrenia. This model proposed that schizophrenia was psychogenic in nature, based on the premis that the psychoses were activated by a toxin, which in turn was a product of hysteria and other neuroses. Between 1930 and 1940 this psychoanalytic approach was employed by Sullivan and Fromm-Reichman in their work with schizophrenic individuals at Chestnut Lodge. At this time Sullivan (1953) developed his "interpersonal theory of psychiatry" which enabled him to explore the family's role in the genesis of schizophrenia. Fromm-Reichman also made reference to a broader etiological system when he coined the term "schizophrenogenic", a term which has historically, and rather pejoratively, been applied to mother-schizophrenic child interactions. While work at Chestnut Lodge helped establish psychoanalysis as a major treatment modality for schizophrenia, outcome studies have not supported its use for this severe disorder (Stone, 1986), and where in the literature successful intervention is reported, the treatment duration and content appear exhausting. Arguably, the explosion in pharmacotherapy for schizophrenic spectrum disorders and concomitant biological approaches further strengthened the perception of long-term psychoanalytic psychotherapy as an inappropriate treatment option.

Contemporarily the growth of behavioural interventions, followed by the complimentary cognitive strategies, has led to renewed psychological interest in the schizophrenic process and treatment thereof. Key areas of attention that supported this shift of emphasis include 1) research showing an association between the level of familial expressed emotion and psychotic relapse indicating an interplay between biological / neural patterns and environmental factors (Kolb & Wishaw, 1996); 2) clinical use of social skills training to prepare patients for general social functioning (Bellack, 1997; Penn & Mueser, 1996), 3) the application of systematic cognitive therapeutic strategies, especially for delusional states (Hole, Rush & Beck, 1979), 4) the use psychoeducation to inform the patient of their disorder and its implications (Nightingale & McQueeney, 1996), 5) a greater understanding of the role that anxiety plays in the psychotic process (Widiger & Sankis, 2000), 6) the inclusion of family therapy in treatment packages (Halford & Hayes, 1991), and 7) recognition of the

iatrogenic effects, frequency of refractory symptoms, and medication non-adherence associated with neuroleptic intervention (Den Boer, Westemberg & Verhoeven, 1987; Fleischhacker, Meise Gunther & Kurz, 1994).

Presently Cognitive Behavioral Therapy (CBT) is increasingly being offered to this population as a significant adjunctive therapy (Bellack, 1997; Birchwood & Tarrier, 1992; Brenner, Boker & Genner,1997; Haddock & Slade, 1996; Kingdon & Turkington, 1994; Liberman & Green,1992). CBT has also recently been recommended when developing treatment plans for those cases experiencing their first psychotic episode, and it is hoped that in the future such an approach may be used for treating prodromal cases to prevent the onset of psychosis (McGorry, 1998). Therefore, a comprehensive treatment approach requiring attention to both biological and psychological management is now emerging.

The incorporation of both biological and psychological factors into an understanding of the schizophrenic process has been facilitated by the emerging biopsychosocial model of mental functioning (Engels, 1977). The biopsychosocial model was presented by Engels as a more phenomenologically valid approach to the study of human thought and behaviour within the field of psychiatry, and was generated in a response to the prevalent reductionist approaches of biomedicine and behaviourism. Engels underlying argument for the model is that models derived from scientific methodology that are empirical and mechanistic are unable to capture, and often ignore, the complex and interrelated nature of human experience, and as such breed clinical insensitivity. While there is some criticisms of the biopsychosocial model in terms of its validity as a 'model' in the strict sense (McLaren, 1998), its value and applicability lie in its assistance in linking many existing semi-independent models of mental functioning. In this instance the model may be better thought of as a 'biopsychosocial frame' (Muir, 1998), the specificity of which may be heightened when it is employed in the descriptive analysis and reporting of the major mental processes seen by clinicians. Indeed, it is the organisational quality of this model that denotes its utility in presenting a multifactorial descriptive model of psychotic experience, such as that to be presented in the present thesis.

The integrative approach of the biopsychosocial model allows it to readily convey the idiosyncratic nature of individual cases that present with long-term courses and consequences. In this respect the model's ability to describe processes related to the psychoses are no exception, given the range of distal and proximal factors that eventuate in the individual's

contextually unique experience of psychosis. At its fundamental level this model poses the individual who has a psychotic break as having been at risk for some time due to a measure of biological and life-event vulnerabilities, and whose break with reality has been precipitated by events which have most likely been perceived as aversive. However, such a vulnerability, even in association with stressful events, need not determine such decompensation (Ciompi, 1988), probably due to the mediatory role of individual differences and culture.

A major prediction of the biopsychosocial model is that psychosis develops via the interaction between vulnerability and stress mechanisms. Based on data from three major European long-term outcome studies on schizophrenia Ciompi (1988, 1989) has presented a three phase biological-psychosocial model of the long-term evolution of schizophrenia (see Appendix II). The first phase is one characterised by the premorbid evolution, and requires the presence psychosocial (social, familial, cultural) and biological (genetic, perinatal) factors. These factors provide a premorbid vulnerability to acute psychosis, while continuing to impact at all points of the process. The second phase is one of psychotic decompensation during which the acute facets of the disorder are observable. Thirdly, a phase of long-term evolution emerges and involves residual symptoms, which in turn act on the pre-existing vulnerabilities, and other earlier phases of the process if and when they emerge in the future. While this model represents plausible relationships between factors known to be associated with the long-term course of schizophrenia, support is largely based on theories related to domains of human functioning (e.g. stress, vulnerability-hypothesis), and data generated from the study of specific areas in the development of psychosis that have been deemed as 'separable'. The broad based nature of this model alone may preclude formal testing, however such a model provides a useful indicator of key areas where ongoing research may be aimed.

While Ciompi's model provides an expanded version of the evolution of psychosis, and how a complicated clinical psychosis potentially lays the foundation for longer term schizophrenia, each area of his model requires further development and potential differentiation. In particular, for the purposes of the present thesis two areas require comment: 1) Ciompi's insistence on using the term 'residual', and 2) the degree to which vulnerability is a dynamic process. Firstly, in phase three, Ciompi's conception of an acutely psychotic period being followed by either recovery or residual states clearly indicates his firm belief in the disease model of schizophrenia. To regard the presence of psychopathological symptoms at post-psychotic assessment points as a 'residual state' of a previously diagnosed index disease is to

negate the dynamic nature psychosocial functioning and brain – environment interactions. To redress this limitation, an easy addition to Ciompi's model may be a fourth phase which represents the emergence of secondary symptoms and psychological processes that are present for some sufferers. Likely secondary problems would be those of mood (i.e. post-psychotic depression), anxiety, trauma, and panic symptoms. However, even this addition is simplistic as the changes to psychosocial functioning are unlikely to be linear as portrayed in this model, leaving the necessity for feedback and maintaining loops to extend between the outcome phase(s) and all preceding points – interpersonal, cultural, familial, stress-coping, and neurophysiological status.

Secondly, what the literature considers to be the 'premorbid vulnerability' remains unclear, particularly in relation to the proposed mediatory role of stressful life events in the precipitation of a psychotic break. The most commonly cited vulnerability factor is a genetic predisposition, but as discussed earlier the genetic argument has been disputed, along with references to associated differences in brain structure anomalies which may appear as consequences of illness-related changes rather than existing as precipitating factors. Proponents of the biopsychosocial model, such as Ciompi, appear to have taken a middle-ground approach, acknowledging that biological factors interact with psychosocial factors to activate the latent (biological) vulnerability; that 'vulnerability' is not an all or nothing entity. However, the actual processes that cause and / or moderate the hypothesised vulnerability remains beyond the schizophrenia / psychosis literature. Questions such as, is the vulnerability static, with variability in symptom activation being contingent on changes in psychosocial processes, or does the vulnerability itself change over time? For example, in considering the effects of psychosis treatment, it is unclear to what degree there is a proactive effect on the underlying predisposition, or whether clients and case managers are able to manage the contingencies that activate the vulnerability, or even which components of the treatment effect particular biopsychosocial changes for which cases. Given the findings from the treatment of depression, there is a strong suggestion that cognitive-behavioural therapy does reduce the underlying cognitive-schematic vulnerability (Hollon, DeRubeis & Evans,1996) suggesting that a neuro-organisational component of vulnerability is modifiable. The point here is that brain functioning and structure appear to be interrelated and mutually modifiable. Therefore, taking this spectrum of interdependent processes into account, the conceptualisation and description of pre-acute and relapse vulnerability requires the inclusion

of non-linear multifactorial aspects of functioning, including cognitive, behavioural, and emotional stress-coping responses to environmental changes.

Overall, the biopsychosocial model offers a way to highlight the different factors that combine and interact to produce patterns of psychopathology, as well as allowing for the heterogeneity that is found within these symptom patterns. Certainly, in terms of the hypothesised interdependent nature of underlying vulnerability, stress-responses, emergent psychopathological states, and illness maintaining factors, the case for more comprehensive treatment approaches can easily be made. One area in which the biopsychosocial model has significantly influenced contemporary treatment is the growing number of services providing early interventions for psychosis (Birchwood & Tarrier, 1992). In this area the vulnerability – stress conceptualisation has lead to an increased interest in secondary prevention (McGorry, 1992), and as a first step in managing the presentation of psychosis in young adults, and as a method for minimising symptom severity and psychosocial maladjustment (Birchwood, Todd & Jackson, 1998; McGlashan & Johannessen, 1996; McGorry et al., 1996). In extending the discussion of psychosis, part 3 will further explore the literature pertaining to negative symptoms.

Part Three

The Positive and Negative Symptom Dichotomy

Chapter 6 The history of positive and negative symptom constructs

This chapter introduces the differentiation of schizophrenia into two distinct syndromes: positive / excess symptoms, and negative / deficit symptoms, and discusses how each relates to diagnosis of the primary schizophrenic disorder.

The historical practice and theory of schizophrenia and the psychotic disorders has seen a consistent attempt to systematically define this syndromal disorder by reference to its major symptomatic features. As the core deficit in psychosis is a break-down in reality testing (Trzepacz & Baker, 1993), an unobservable phenomena, the presence of this feature can only be determined through an assessment of the signs and symptoms hypothesised to be a result of this core deficit. Since the writings of both Kraepelin and Bleuler the symptoms of schizophrenia have been clustered generally into positive thought disorder features and deficit features, both having received primary significance at some time, with the neurologist Hughlings-Jackson being the first to formalise this distinction into positive and negative symptoms. Contemporarily, the attempt to define major schizophrenic syndromes has lead to researchers such as Crow (1980) and Andreason (1980; Andreason & Olsen, 1982) to revive and revise the positive and negative symptom dimensions. At a clinical level these two subtypes represent distortions and/or excesses (positive symptoms), or reductions/losses (negative symptoms) in the individual's normal functioning (see table 6.1).

According to the DSM-IV (American Psychiatric Association, 1994) positive symptoms have a primary influence on the diagnosis of both psychosis and schizophrenia. This focus is based largely on the historical emphasis on the presentation of positive thought disorder, delusions, and hallucinations for reliable diagnosis of acute psychosis (Andreason, 1982). Arguably this selective focus has been reinforced by the contemporary finding of the almost selective effect of neuroleptic medications on the positive symptoms (Borison & Diamond, 1997). The recognition that hallucinations and delusions can be found in a range of psychiatric conditions, such as severe anxiety or substance abuse, indicates that such a diagnostic emphasis, especially for differential purposes can be potentially misleading. Further, Lindenmayer and associates (1994a) found that hallucinations, as rated by the Positive and

Negative Syndrome Scale (PANSS) which is a clinician rated measure for psychotic symptoms, accounted for less variability than did negative features or features associated with disorganised cognition.

Table 6.1. Positive and Negative symptoms in Schizophrenia.

Negative Symptoms		**Positive Symptoms**	
Symptom	**Function lost**	**Symptom**	**Function lost**
Alogia Loss or diminution in the fluency of thought and speech.	speech fluency	Hallucinations Abnormal perceptions occurring in any sensory modality.	perception
Affect Blunting Loss of the capacity to express emotions fluently.	emotional expression fluency	Delusions Aberrant interpretations or subjective meanings given to perceptions.	inferential thinking
Avolition – asociality Loss of motivation; inability to initiate or persist in tasks.	volition and drive	Positive formal thought disorder Distortions in the form and content of speech.	language
Anhedonia Loss of the capacity to feel pleasure, particularly expressed socially.	hedonic capacity	Bizarre behaviour Unmodulated behaviour, eg. Social, sexual, stereotyped.	organisation and control
Attentional impairment Cognitive and or motivational dysfunction.	attention	Inappropriate affect eg. unmodulated mood incongruent affect.	affective fluency and expression

Modified from Andreason (1990), p14.

Conversely, diagnostic reference to negative symptoms has been avoided in psychiatric nosology due to a lack of reliability in measuring this construct, largely due to the lack of psychometric instruments for this, a situation that has recently been more favourably addressed (Fenton & McGlashan, 1992b; Andreason, 1982). Instead of diagnostic importance, the role of the negative symptoms in defining schizophrenia has been more thoroughly described in terms of illness vulnerability, such as when a prodrome of deficit symptoms is present, and illness duration (chronicity / residual dysfunction). Negative symptoms are reported to be associated with an identifiable prodrome, especially marked by anxiety symptoms and social withdrawal, and an illness course that is marked by chronicity (Andreason, 1990). Therefore a diagnosis which includes the presence of negative symptoms would prototypically suggest a case characterised by gradual and insidious onset, one which

leaves the individual vulnerable to relapse, and which in the extreme is marked by its refractory nature. However, in terms of diagnosis, a singular emphasis on positive symptom criteria, as is presently the case, or equally for negative symptoms, can not be justified for diagnositic purposes given the polythetic nature of schizophrenic spectrum disorders (Andreason, 1982). Certainly, as will be discussed below, such a narrow diagnostic emphasis lacks the credibility of being able to account for causal pathways that will impact on long-term treatment outcome, for individual's subjective experience of mental illness, and which could lead to unnecessary clinical pessimism regarding treatment outcome.

Chapter 7 Challenging the symptomological dichotomy

Having discussed the two major dimensions of schizophrenia, this chapter considers the relationship *between* the two symptom clusters, and offers an alternative five-factor model that highlights the need to pay attention to a range of functional domains within clinical cases. The chapter finishes with a reminder that the key to understanding the clinical picture is the characteristics of the client themselves, and that the importance of this is in going beyond diagnostic entities in forging understandings for mental illness.

While positive and negative symptom dimensions appear to be readily separable, the majority of cases of schizophrenia seen clinically present as mixtures of these 'pure' subtypes. As such Fenton & McGlashan (1992a) have posed positive and negative symptoms as semi-independent domains, the interactions of which lead to the observed clinical variability between schizophrenic patients. This variability also is accounted for by the phasic nature of this spectrum of disorders, with positive and negative symptoms becoming more profound at different points in the course of the illness (Fenton & McGlashan, 1992b). Positive symptoms are typically observable in the early phase of psychotic illness, whereas negative symptoms tend to appear in the subacute and subchronic phase, with the symptomatic effect becoming more profound and stable in parallel with chronicity.

While the multidimensional nature of schizophrenia has been acknowledged in the positing of positive and negative symptom domains, some researchers have looked for other domains to explain case variability. Lindenmayer and associates (1994a; 1994b) have proposed a five-factor model of schizophrenia based on a factor analysis of 240 cases diagnosed with schizophrenia (typically with a gradual onset) and rated on the Positive and Negative Syndrome Scale (PANSS). They found that 57.5% of the variance in their analysis was accounted for by five components with good internal consistency - negative (α = .86), positive (α = .80), excitement (α = .76), cognitive (α = .79), and anxiety/depression (α = .69). Of these, the negative component accounted for 23.7% of the variance, and had high internal consistency supporting an assumption of structural unity. Replication studies implementing

changes such as drug free (1 week washout), age, and inpatient vs. outpatient, as presented by the authors, found the same core items for the negative factor (emotional withdrawal, passive/apathetic social withdrawal, lack of spontaneity, poor rapport, active social avoidance, and blunted affect), and supported the five-factor model in general. While the addition of the excitement and depression factors is largely an artefact of the PANSS general psychopathology scale, as argued by the authors, it does highlight the need to pay attention to a range of functional domains within individual cases, so as to access their deficits and strengths. Certainly it highlights the need to avoid simplistic subtyping of schizophrenia, and the assumptions contained within such approaches.

A significant challenge to a purely two-fold conceptualisation of schizophrenia is the lack of validity in the schizophrenia construct, as already discussed above in detail. To recap, Boyle (1990) has proposed that schizophrenia is an ill-defined construct that has been promoted by selective attention to, and distorted use of, biological components of the illness, as a means of supporting the prevailing medical model of mental illness. Interestingly, while this model is promoting greater consensus in the diagnosis of mental disorder, via the DSM system and its adjunctive rating scales, Andreason (video interview), who is a strict adherent to the psychiatric model as well as a co-developer of DSM-IV criteria for schizophrenia, considers DSM criteria insufficient for conceptualising all the aspects associated with this disorder. Andreason iterates that clinicians need to go beyond the symptoms and assess areas such as daily functioning, family setting, work experiences, insight, and the inner experience of the patient, thus avoiding "losing the human face of the profession [psychiatry] through oversimplified diagnostic criteria." This point echoes the observation made by Kraeplin, that "we will have to look for the key to the understanding of the clinical picture primarily in characteristics of the individual patient...his expectations play a vital role" (Kraeplin, 1920, in Bentall, 1996).

More recently Strauss (1992), in a review of his own clinical development as a psychiatrist, highlights the importance of taking what he describes as a 'person - key' approach to understanding schizophrenic psychopathology. That is, trying to understand human functioning by placing the person (as a representation of the dynamic interactions between psychology, biology, and experience) at the core of clinical activity, and treating notions of pathology and normalcy as empirical issues. The approach outlined above, by Strauss's admission, calls for a shift in the existing psychiatric paradigm, one which grounds the

patient's unique symptomology as a derivative of their expectations and experience of life, potentially transcending current diagnostic categories via non-specificity of a number of symptoms, and which suggests that new psychosocial avenues to treatment should be considered. As already reviewed, the biopsychosocial has made some inroads towards such a comprehensive client-centred, and symptom-centred approach. In taking the next step toward a biopsychosocial model of negative symptoms, particularly as related to stress – related factors, part 4 will explore in more detail the negative symptom construct.

Part Four

The Negative Symptom Syndrome

Chapter 8 Validity of the negative symptom construct

This chapter discusses the validity of the negative symptom construct, beginning with the inroads made with the advent of structured psychometric instruments for measuring negative symptoms, and the subsequent definition of the construct as a 'valid' entity. This proposition is critiqued, and the lack of well grounded theoretical development, and therefore validity, is discussed. Methodological issues, hierarchical inclusion, and the commonality of negative symptoms with other disorders is discussed in support of the need to conceptually revisit notions of causality for the negative symptoms, an area followed up in chapter 11.

Negative symptoms have been observed to cluster together in a consistent fashion, and have thus been referred to as a syndrome. For instance, Crow (1980; 1985) proposed a type II syndrome based on the consistent clustering of a group of symptoms characterised by their deficit qualities. These symptoms were measured by 2 items on the Manchester Scale, an early measure of schizophrenic symptoms: affective flattening and poverty of speech; later this was expanded to include behavioural deterioration and abnormal involuntary movements.

The validity of the negative symptom dimension as a clinically definable construct has recently received considerable attention, a period Andreason (1982) refers to as a renaissance of interest in negative / Bleulerian symptoms. Construct validity is an essential component in the identification and proper utilisation of the parameters around a clinical entity as it lays the groundwork (i.e. data) upon which assumptions of generalisability can be made. Without determining such parameters the outcome for studies of negative symptoms will largely be of applicability only to the researched client / subject group. Further, using a 'goodness of fit' model in which client's presentations are 'fitted' according to the nearest recognised category of disorder, both specificity and sensitivity are likely to be low for some clients, and treatment may be misdirected producing possible iatrogenic effects for clients already under considerable duress.

Foremost in this resurgence of interest has been Andreason's (1984) construction of the Scale for the Assesment of Negative symptoms (SANS), which measures specific negative symptoms in patients with schizophrenia, followed by Kay et al.'s (1987) Positive and Negative Syndrome Scale (PANSS), which measures schizophrenic symptomology and includes a general psychopathology scale. The SANS has been shown to have good inter-rater reliability and internal consistency and measures five global subtests of what are considered to be reliable symptoms of negative schizophrenia: affective flattening, alogia, avolition, anhedonia, and attentional impairment. The scale has been criticised on several grounds (Den Boer et al., 1987), specifically that 1) cognitive impairments are found in both positive and negative schizophrenia, 2) attentional impairments may be a motivational effect, and 3) that anhedonia may represent secondary rather than primary processes. However the SANS has been successful in allowing for the 'independent' measurement of negative symptoms, and is sensitive to changes in symptomology over time, therefore providing a standardised instrument for clinical research (Andreason, 1982; Fenton & McGlashan, 1992). Similarly, Lindenmayer and associates (1994a; 1994b) have described a core set of negative symptoms, using the PANSS, which is comprised of emotional withdrawal, passive / apathetic social withdrawal, lack of spontaneity, poor rapport, active social avoidance, and blunted affect, items which indicate an interaction between deficit states and the social context.

While the negative symptom has gained a certain 'face validity' (Stampfer, 1990, p.518) by virtue of its inclusion within psychiatric nosological since Bleuler's writings, surprisingly little in the way of formal validation has been attempted. It may be argued that the validation of a construct such as the negative symptom syndrome is made fundamentally difficult by the multi-presentational and broadband nature of what is currently described as the negative symptom construct. Further, optimal operational reliability (for construct parameters to be generalisable and have predictive power) construct validity needs to be based on an independent criterion or criteria found in all cases, typically biological in nature. However, such a criterion has not been demonstrated for any schizophrenic syndrome. More generally, these types of validation problems are common to psychopathology generally, making validity studies 'lengthy and unpopular' (Sommers, 1985, p.375). This difficulty in extracting definitive parameters is likely to be especially prominent when the construct being measured is syndromal (i.e. negative symptoms), as by their very nature they are typified by inter-individual variability. Therefore, it can be argued that validation of the negative symptom

syndrome requires a closer scrutiny of patterns of sub-categories, and for the syndrome to be maintained as "a hypothesis to be tested rather than a fact to be explained" (Sommers, 1985).

The current hypothetical status of the negative syndrome, and the associated problems of validity, is highlighted by the lack of an empirically based explanatory model of negative symptoms in the current literature. For example, Kay & Lindenmayer (1987) note that baseline negative symptoms were the strongest predictor of functional outcome in their two-year prospective follow-up of the early course of 19 subjects who had schizophrenia. However, consistent with other studies focusing on symptom presence and duration, little in the way of a formal explanation of precipitating and maintaining factors for negative symptomology was made available, other than an acknowledgment of the confounding effects of neuroleptic induced states, and depressive and reactive features. Clearly then, there is room for the initiation of more descriptive studies to help extract both subtle and discrete sub-categories of observable behavior (eg illness vs. treatment-related vs. psychosocial vs. intrapsychic) within the negative syndrome as it is presently understood, as well as to integrate existing data that is embedded in a range of diverse areas of psychopathology research. Already there is a strong argument made for the differentiation of secondary (reactive) and primary deficit subtypes, and these will be discussed in more detail in chapter 11. Along with Zubin (1985) it is suggested that this type of research will provide the groundwork for highlighting independent or semi-independent constructs characterised by negative affective states, as has been demonstrated for the positive – negative symptom dimensions.

A methodological issue concerning construct validity is the use of cross-sectional studies of phenomenology (Andreason & Olsen, 1982), as identifying sub-categories can only enlighten similarities or differences in underlying causal mechanisms if a longitudinal process can be tracked (eg Fenton & McGlashan (1992) who used a 9 month assessment window). For example, it may eventuate that a single underlying process is present for negative symptoms, which leaves these individuals at risk for reacting to adverse experiences with a negative behavioural and affective pattern. Conversely what may be being witnessed is a diverse pattern of illness related events that, especially when chronic, result in neurobiological changes. To date such a differentiation can not reliably be made (Zubin, 1985). Social science research has identified that simple correlations are not robust predictors of causation (Howell, 1992), and correlations generated in cross-sectional designs are arguably more fallible when

generating theories of complex pathological causes and events. Therefore, the hypothesis that negative symptoms are an effect of the primary psychotic disorder is not a more valid assertion than stating a theoretical link between negative symptoms and affective disorder, or any other theoretically plausible interactive process.

Taking a step back from viewing the phenomenology of negative symptoms through a lens that has been largely focused by schizophrenia research, it may be observed that negative affective states characterised by flattened affective responsiveness and social withdrawal are not entirely restricted to schizophrenic pathology. Certainly, given the relationship between psychosocial stressors and a number of negative symptoms of schizophrenia (Strauss, 1992), coupled with what is now known about human psychological coping processes (see chapter 14), it has been suggested that negative symptoms may not be indigenous to psychotic illness (Zubin, 1985), or pathognomonic of schizophrenia (Andreason, 1982), or even more generally to severe psychiatric illness. For example, several studies have reported negative symptoms in depression inpatients at rates as high or higher than for inpatients with schizophrenia (Hafner & Maurer, 1995). Clearly, the transportable nature of negative symptoms across diagnostic categories is reflective of, and may underlie, the problems of validity already discussed for this syndrome. In summary, Sommers (1985) states "The omission of validity data in negative symptoms research, however, is a serious flaw....Moreover, it implies a major change of attitude, with the negative symptoms construct treated as a hypothesis to be tested, rather than a fact to be explained" (p.376).

The following two chapters will explore this 'transportable nature' of negative symptoms. Firstly the general effects of illness severity on symptom expression will be reviewed, and secondly a review will be made of the potentially manifold causal pathways, appearing at different points of the illness course in relation to the acute psychosis, that may be involved in the presence of negative symptoms in first-onset psychosis.

Chapter 9 The hierarchical effect of severe psychopathology: exploring severity

As stated above, negative symptoms are found in other diagnoses, making the description of causal processes difficult, particularly when cross-sectional designs are used (Sommers, 1985). One diagnostic issue relevant to this commonality in symptoms is the hierarchical nature of diagnostic categories (Foulds & Bedford, 1975; Wing, 1995). The model proposed by Foulds & Bedford describes categories of disorders as being rated hierarchically, with those categories higher up overriding those below, and persons with a particular disorder will have also present with symptoms from lower class levels. The model recognises dimensional as well as categorical concepts, which may potentially enhance the understanding of relationships between psychiatric disorders (Wing, 1995), and the presence of one major disorder should alert clinicians to the possibility of secondary syndromes (Lundy, 1992). In support of the hierarchical model, Wing (1995) reports that in the International Pilot Study of Schizophrenia (IPSS) a large proportion of people with schizophrenia would have been classified as affective if symptoms discriminating schizophrenia had been left out. Wing also states that at least some of the affective symptoms in the (IPSS) study were so common in acute schizophrenia that they "must be reactive to the stress of the primary experiences" (p.10), thus recognising that the 'rule' of symptom inclusiveness for the highest order class of disorder could be due to two different processes: a) lower order symptoms can act as vulnerability factors for higher level classes of disorder, and b) potentially lower order symptoms can constitute a secondary response to severe pathology.

One potential benefit of applying the notion of disorders as classes on a hierarchical scale is the descriptive power this has for issues of severity in the psychoses. Severity here is used to depict changes in the intensity, frequency, and duration of symptoms. In syndromal disorders such as the psychoses, this definition of severity is added to with changes in the *range* of psychotic phenomena. While an individual sufferer has many events that concur to produce psychopathology, the addition of notions of hierarchy have the benefit of describing and

structuring the compounding effect of physiological and psychosocial changes, and how severe changes to functioning can lead to a 'snowballing' of symptomology. Arguably, an increasing range of pathological symptoms could be exponentially related to increased dysfunction across a number of domains, and by definition pushing the person up a hierarchy of functional severity. This proposition is supported by Chapman & Chapman (1973) who have suggested a "generalised deficit" in patients with schizophrenia, this deficit being due to their severe impairment in so many areas, and that this makes them difficult to compare with other groups due to the high overlap of dysfunction.

Methodologically, a generalised state of dysfunction means that conceptually causal mechanisms will be closely entwined with other symptomatic factors. In this instance the presence of deficit symptoms, such as withdrawal or flat affect, could be taken as partial evidence for a primary psychosis, while equally being acceptable as correlates of poor psychosocial functioning. For example, consider the case of a person with an inpatient admission with acute psychosis, who's presentation includes mixed periods of hyperarousal or amotivation and flattened affect, and has a past history of sexual abuse, combined with current stressors. Without, a) longitudinal assessment, ideally predating the psychotic admission, or b) a coherent multi-faceted theory of the interrelationship between psychosis and other life events, and without c) prematurely foreclosing on assumptions of causality for specific symptoms when they appear to 'fit' with an overarching syndromal disorder, it would be difficult to determine whether the hyperarousal and amotivation were components of the index psychosis, or a co-occurring disorder such as PTSD. The implications for treatment strategy being different for each. Further, the differential effects of co-occurring disorders may be confounded by the effects that severity of illness has on subjective ratings of the intensity of events. For example, psychiatric patients tend to rate events as more stressful than non-patients, thus making the relationship between stress and psychopathology difficult to disentangle (Dohrenwend, 1979).

This type of problem has plagued multivariate analyses of studies using samples taken from hospitalised cohorts, as correlations between symptoms can become over inflated (Bentall, Jackson & Pilgrim, 1988), again due to the generalised deficit in the more severe inpatient. One example is Jablensky's (1995) description of robust predictors for good or poor outcome in schizophrenia (see Appendix III). While Jablensky has justifiably categorised the factors into several separate domains of functioning, a review of the most predictive factors could

suggest an alternative grouping for both good and poor prognosis based on different levels of illness severity and social support (see table 9.1), with the exception of the *extrovert or cyclothymic personality* factor, although this could potentially be related to social support. This proposition is supported by the IPSS study which found that work, social relationships, severity of symptoms, and need for hospital admission (severity) were found to have longitudinal contiguity between the pre-admission assessment and two and five year follow-up. Arguably, the presence of work as a stable factor, as reported by both Jablensky and Strauss (1992), may not only load on social support, but also potentially on psychodynamic issues such as social identity and motivation – purposefulness. While the argument here is that many of these predictive factors are potentially reducible to more basic process, or at least difficult to disentangle from the hierarchical effects of severe illness, the extreme version of 'diagnosis is prognosis' is not argued for (Strauss, 1992).

Table 9.1. Reconfiguration of Jablensky's (1995) prognositc factors.

	Poor outcome
High level of illness severity	Longer duration of preindex illness. Insidious. Negative symptoms on first admission.
Low social support (and maladjusted social – identity)	Single divorced separated. Social isolation.

	Good outcome
Low level of illness severity	Shorter duration of preindex illness. Acute, associated with excitement, elation, perplexity, anxiety or depression (possibly a shorter reactive onset).
High social support (and resilient social – identity)	Married. Good work, social, and sexual adjustment.

The point of making such a regrouping as demonstrated here is to highlight the difficulty in disentangling the generalised effect of severe pathology from distinct and significant illness related processes, as predicted by the hierarchical model. There is a need to not only identify the range of biopsychosocial factors, but to try and disentangle them from their combined and potentially additive effects (severity). Without such a model, indicators such as those

highlighted by Jablensky remain as single factors with poorly developed conceptual properties, not least of which is the inability to competently describe the interrelationships between factors. Jablensky noted that in the IPSS study (WHO, 1979, in Jablensky, 1995), the best five predictors were only able to explain 30% of the two year outcome variance, suggesting the importance of change in the predictor variables. Arguably, such changes are not random events, but may well reflect the inclusion of greater ranges and types of symptoms, as well as the interactions between them.

Chapter 10 A broader exploration of the negative symptom construct

Having highlighted that at least some negative symptoms are associated with mental states other than the psychoses, negative symptoms remain as significant elements in the psychoses. For instance, Kay & Lindenmayer (1987) reported that in their two-year follow-up of acute schizophrenia, the strongest overall predictor of favourable acute outcome in acute schizophrenia was baseline negative syndrome at the time of index admission. However, hypothetically there may be conceptually different factors at work in the production of negative symptoms, other than as a manifestation of primary schizophrenic processes, arguably so in more complicated clinical cases. This chapter explores the theoretical multi-causality of negative symptoms, and lays the ground work for part 5 which explores the hypothetical relationships between life events and specific negative symptoms, as mediated by stress - related processes.

As already discussed in part 3, the positive and negative symptoms found in schizophrenia are at least semi-independent (Carpenter et al., 1988), not withstanding that the majority of cases present as mixtures of these two clinical subtypes. This indicates that causal processes may also be associated with independent factors for each subtype of schizophrenia, although such an assertion needs to be approached cautiously (Hafner & Maurer, 1995). For example, efforts to uncover the neurobiology of schizophrenia has indicated different expressions in the brain for the two subtypes, 1) positive symptoms are associated with increased dopamine activity in the prefrontal cortex - 'hyperfrontality', as well as the lack of structural anomalies, whereas 2) negative symptoms are associated with decreased dopamine activity in the prefrontal cortex - 'hypofrontality', and the presence of structural anomalies such as increased ventricle size and cortical atrophy (Kolb & Wishaw, 1996). Although, these physiological processes may in themselves be found to be signs of a common dysfunction (Hafner & Maurer, 1995). Even so, signifying potential differences in causal mechanisms and symptomatic processes is clinically relevant in determining the treatment strategies to be used (Ward, Vertue & Haig, 1999). This point is important to the syndrome of negative symptoms because, a) prominent negative symptoms only occur in a proportion of schizophrenic patients (Fenton & McGlashan, 1992), b) different processes may result in similarly expressed negative symptom states (Carpenter et

al., 1988), and c) negative symptoms have been shown to not necessarily end in an unremitting disease course, indicating that they may be best used descriptively in clinical settings, avoiding assumptions of permanence or pathogenisis for the observed syndrome (Carpenter, 1985).

While negative symptoms have been associated with schizophrenic spectrum disorders since the early writings of Beuler, Kraeplin and Hughlings Jackson, there is no definitive notion regarding their etiological mechanism. Different researchers have attributed the negative symptoms they have observed to a range of factors related to the experience of schizophrenia, such as a manifestation of the core illness, drug-induced akinesia, institutionalisation, depression, positive symptoms (e.g. paranoid avoidance) (Andreason, 1990), emotion focused-coping (McGlashan et al, 1975; Strauss, 1989a), neuroleptic-induced depression (Carpenter, Heinrichs & Alphs, 1985), demoralisation (Carpenter, Heinrichs & Wagman 1988), a return to pre-morbid personality features (Zubin et al., 1985), post-psychotic trauma (Jefferies, 1977; McGorry et al., 1991), and institutionalisation (Wing & Brown, 1970). The association of these factors to negative schizophrenia suggests that either, 1) each may provide a pathway to a similar presentation characterised by negative symptomology, or that 2) each is a discrete syndrome marked by functional deficits, and for which differences become apparent only after stepping back from a cross-sectional analysis of the presentation. Andreason et al (1994) note that it is because negative symptoms can be the result of a wide variety of factors that they are difficult to study.

This variety of causal mechanisms that has been associated with a presentation of negative symptoms indicates that there is a need to identify possible differences in symptomological patterns between clients. One such re-categorisation has been proposed by Carpenter et al. (1985) who have differentiated between secondary and primary negative symptoms. Secondary negative symptoms are thought of as reactive in nature and are thought to be due to a lack of motivation, withdrawal, and resignation. Conversely, the primary negative symptoms are considered to reflect a true deficit state that is attributable directly to the psychotic disorder. This distinction can be considered to reflect illness-intrinsic and illness-extrinsic negative symptomology (Mundt, Barnett & Witt, 1995), an important distinction for treatment, given that the secondary symptoms are likely to be more amenable to treatment (Carpenter et al., 1985).

Carpenter et al. (1988) propose that an operational definition can be made between secondary and primary negative symptoms based on a systematic assessment of the clinical history and presentation, and based on this have developed a structured assessment instrument for which adequate reliability has been shown. Their model (Carpenter et al., 1985) demonstrates that the main criteria to be applied is that the client must display prominent negative symptoms for at least 12 months, and in the absence of secondary causes to be categorised with a primary negative syndrome. Otherwise the client's negative symptom's can be considered as due to secondary processes. However, as the criteria appears to represent 'duration of negative symptoms', it is potentially confounded by issues of severity as discussed in the previous chapter. Further, another operational problem is that in the majority of cases where clinicians must make a distinction based on clinical judgement, reliability is poor. This effect was demonstrated by data from the Psychotic Disorders Field Trial of DSM-IV which provides an approximation of routine clinical settings, and resulted in kappas of <0.5 for all symptoms when differentiating between primary and secondary negative symptoms (Andreason et al,1994). Andreason et al (1994) caution that there is obvious concern about the ability of clinicians to make this sort of distinction, and suggest strategies relying on informative populations (i.e. drug-naive patients), longitudinal observation, and measures of potential causes of secondary negative symptoms. Further, because of the "considerable amount of sheer clinical talent required in making clinical judgements...in non-paradigmatic "grey area" cases....Raters who lack extensive theoretical and direct clinical knowledge...may unknowingly generate as much noise as signal" (Sommers, 1985, p.374). Taken together, Andreason's and Sommer's comments indicate the need for both good methodology, and the discriminatory power provided by an inclusive and contextualised theory of negative symptomology.

The above issues demonstrate the potential difficulty with trying to determine differential subtypes with largely the same symptom expression, particularly when longitudinal assessment is unavailable. However, rather than negating the potential explanatory power of distinguishing between primary and secondary negative symptoms, arguably it highlights the need for clinicians to make a thorough assessment of the background to the illness, rather than focusing largely on the presenting symptomology. Certainly, Carpenter et al. (1985) suggest avoiding premature assumptions of a primary disease syndrome, due to the clinical pessimism associated with chronicity in this group.

Being able to determine more treatment responsive cases of negative symptoms would also be an important component of an early intervention strategy, particularly given the common occurrence of negative symptoms prior to acute onset of psychosis. For example, Hafner & Maurer (1995) present retrospective epidemiological data on symptomological presentation during the longitudinal course of first-onset psychosis. Using their Interview for the Retrospective Assessment of the Onset of Schizophrenia (IRAOS), a measure of schizophrenia symptoms, they found that negative symptoms were the first pathological signs to appear in 70% of their sample cases, both negative and positive symptoms appeared within 1 month of each other in 20% of cases, and positive symptoms only appeared in 10% of cases. Two phases were identified in the early course of the cases interviewed, 1) a pre - psychotic phase characterised by negative and non-specific symptoms and a mean duration of 3.1 years, and 2) a psychotic phase with a mean duration of only 1.6 years (median 0.1 years). Following the acute episode, at both six month (Biehl, 1986, in Hafner & Maurer, 1995) and 12 month follow-up (Pogue-Geile & Harrow, 1985, in Hafner & Maurer, 1995) it was found that negative symptoms had reduced by around 50%, and appear to correlate significantly and positively with positive symptoms (Hafner & Maurer, 1995). At their own two year follow-up, Hafner & Maurer (1995) found that the negative symptoms were comprised of affective blunting in 40-50% of cases, avolition – apathy in 40-60% of cases, anhedonia in 50-60% of cases, and alogia and attentional impairment being comparatively low at around 20%. These findings reported by Hafner & Maurer are consistent with the prodromal, acute, and post-acute phases of psychosis proposed by Ciompi's biopsychosocial model.

Given the implications of the secondary and primary negative symptom differentiation, and individual variability in symptoms and illness course, an argument can be made for exploring different potential etiological mechanisms for negative symptoms. In support, Fenton & McGlashan (1992b) note that "The different character and course of negative symptoms in early vs middle to later phases of schizophrenia strongly suggest that they may be generated by different underlying mechanisms" (p.68). Based on a recognition of the multi-factorial nature of the negative symptom construct, four general pathways are proposed. Firstly, negative symptoms could be due to the index psychotic disorder, reflecting an inherent variability of an underlying dysfunction, such as a biological vulnerability. This would be represented by Carpenter et al.'s proposed primary negative symptoms, and be related to poorer outcome. Secondly, the negative symptoms may be expressions of other physiological (biological, sociobiological, or psychobiological) and / or psychosocial processes that predate

the psychosis, or that emerge in parallel with the preacute psychotic phenomena. For instance, given the proposed continuity between normal functioning and psychosis (Strauss, 1989a), dysregulation of mechanisms that mediate stress may produce increasing levels of psychological distress and subsequent symptom reactions (Baumeister, Heatherton & Tice, 1994), and eventuate in the triggering of a psychotic vulnerability. Thirdly, the negative symptoms may be a reaction to the primary psychosis, either passively, such as functional retardation, or actively as a form of coping, such as a temporary withdrawal coined as 'woodshedding' by Strauss (1992). Mayer-Gross (1920, in McGlashan et al., 1975) defined four modes of reacting to psychotic experience, 1) denial of the future (despair), 2) creation of a 'new life' in the post acute phase, 3) denial of the psychotic experience, and 4) 'melting' of the illness into a continuous set of life values. Similarly, McGlashan et al. (1975) propose integration (unity of personality organisation) and sealing over (denial) as psychodynamic coping responses to psychosis. In terms of co-occuring disorders, post-psyhcotic depression and PTSD have been recent focuses of research (McGorry, et al., 1991). Fourthly, the negative symptoms may be due to emerging neurological changes in the brain due to psychotic brain processes or the effects of medication (Den Boer, et al., 1987), as well as changes in the brain as result of adapting to new psychosocial and environmental demands and challenges (Widiger & Sankis, 2000).

These pathways are not necessarily independent, in severe or complicated cases arguably there is the potential for more than one to be activated. Even in cases where an observed negative syndrome was long standing, indicating its primary nature, given an understanding of the client's subjective experience of psychosis as potentially distressing, secondary responses may be clinically significant. For example, Strauss (1992) describes one patient following a near full remission of psychotic symptoms as, "sitting in a darkened room in her house, and had not worked or had contact with her friends for most of the time since I had seen her...two years before" (p.20).

While much of what has been discussed regarding *initiating, maintaining,* and disease *processes* is largely hypothetical, particularly regarding any proposed associations between illness related factors, what can be stated clearly is the variability in symptom presentation, outcome, and as argued above, causality (Sommers, 1985; Strauss, 1992). However, considerable work is still needed in developing an integrated account of these factors and other potential candidate factors, and how they may interact and affect each other, either

singly or via the additive effect of symptom clustering found in higher order classes of disorder. Due to the confounding effects of the high symptom overlap in syndromal disorders, taking the symptom based / descriptive approach, as discussed in chapter 5, may provide a clearer account of how the processes involved in negative symptoms may be represented theoretically. Following a review of the stress literature in part 5, this approach will be used in part 6 to explore potential relationships between life – events and negative symptoms, as highlighted by various areas of psychopathology research.

Part Five

Overview of Stress and Posttraumatic Stress Disorder

Definitions II Clinical terms related to stress

This chapter will provide definitions for the main terms clinical terms referenced in this part of present thesis.

Stress

A particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well – being (Lazarus & Folkman, 1984).

A relatively disruptive level of negative emotional experience which results from negative thoughts about our environment (King, Stanley & Burrows, 1987).

Trauma

From the Greek word for *wound*, within the field of psychopathology trauma refers to psychological injury caused by extreme emotional assault (Reber, 1985).

The application of the term "trauma" in psychiatry has its earliest beginnings in the writings of Freud, and is associated with various individual responses to a range of experiences. Psychodynamically, it is described as the experience of "helplessness on the part of the ego" (Gallagher, Leavitt & Kimmel, 1995).

Posttraumatic Stress Disorder (PTSD)

A psychological disorder associated with serious traumatic events. It is classified as one of the anxiety disorders (Reper, 1985).

PTSD is essentially the development of characteristic symptoms following exposure to an extreme traumatic stressor involving direct experience of actual or threatened death or serious injury, or threat to one's personal integrity, or witnessing any of these events or another, and the response must involve intense fear, helplessness, or horror. The characteristic symptoms

include re-experiencing of the traumatic event, avoidance, and increased arousal (American Psychiatric Association, 1995). Refer to appendix IV for DSM-IV diagnostic criteria for PTSD.

Chapter 11 Introduction to stress

This chapter explores the multi-faceted nature of stress, and its effects on the human organism. Included is a review of the subjective nature of stress – person interactions, and a differentiation between short term stress and long-term stress, with both positive and dysfunctional outcomes noted.

The term stress refers to an experience with several component processes, each of which has been termed 'stress' depending on the context of the situation, or the point being made in regard to stress processes (King, Stanley & Burrows, 1987; Sarafino, 1998). The first component is the presence of a stressor (event), either internal or external. Historically the concept of stressors has been researched in association with life change events. However, in the past two decades stress has been differentiated to include events such as chronic stressors, daily hassles, macrosystem stressors such as economic downturn, 'nonevents' – the nonoccurence of expected events, as well as various forms of childhood and adulthood traumas (Wheaton, Roszell & Hall, 1997). Secondly, there is the person's perception of the intensity of the demand to overcome the situation, and associated with this is the persons evaluation and beliefs surrounding their ability to cope. Thirdly, stress can refer to a consequent negative emotional response, a state of tension or strain. King, Stanley & Burrows (1987) note that it is this aspect of the stress experience that is properly called stress or anxiety. A fourth component is the transaction between the person and the environment, a dynamic process by which people actively influence the impact of a stressor via a variety of cognitive, behavioural, and emotional strategies. Given that the process of stress can include any or all of the above factors indicates that stress is a *conditional* experience, the same stressor being experienced differently due to subjective components of the process. For example, cognitive and transactional models of stress particularly emphasis the perceived meaning of events (primary appraisal) and the perceived availability of coping resources (secondary appraisal) in predicting the outcome of the individual's well-being (Lazarus & Folkman, 1984). The subjective components of stress can also be separated into psychological meaning, physiological reactivity, and interpersonal mediating processes, thus distinguishing stress as a biopsychosocial process (Sarafino, 1998).

In highlighting the conditional nature of stress is the finding that stress can differentially enhance or degrade individual performance in the short-term. Stress can have the positive effect of optimising performance depending on the individual's own optimal level of arousal. Too little, or too much stress-related arousal either side of this optimal level tends to result in degradation of performance outputs due to either reductions in drive, or the overwhelming of cognitive and physiological functions (Hebb, 1955, cited in Sarafino, 1998). Interestingly, while the low end of this spectrum appears to be marked by a lack of stress, an alternative description may consider the effects of low levels of stimulation as too little arousal or stimulation can have the ironic effect of increasing anxiety (King, Stanley & Burrows, 1987). In particular, boredom has been found to result in the experience of anxiety in the same way that over-stimulating adversity does. However, it appears that it is not the level of under-stimulation that is the contingent factor, but the degree to which the level of environmental stress is perceived to be under the person's control (King, Stanley & Burrows, 1987).

Stress also interacts with individuals in differential ways in the longer-term. For example, the experience of adversity in the formative years for some people has been associated with a more robust personality structure. Cognitive models have accounted for this process via the development of schemas about the world that integrate good and bad features of the environment and are therefore more durable, as well as schemas of the self characterised by positive mastery beliefs from past coping successes (Garmezy & Rutter, 1983). In this respect Wheaton, Roszill & Hall (1997) have argued that prior stress can produce a state of 'immunity' to other subsequent stress experiences. However, adversity can also result in negative changes in functioning due to factors including the type and severity of the stressor, its timing in relation to psychosocial development and culmination with other adversities, personality structure, absence of protective factors, and the types of coping mechanisms employed (Freedy & Hobfoll, 1995). Further, adversity may potentially result in chronic vulnerability to further negative experiences via a number of biopsychosocial stress-responses (Horowitz, 1986; Perry & Plate, 1994). The non-linear nature of long-term stress reactions cannot be overstated as the above mediating factors are interactive, the differential outcomes of coping style – social context interactions alone may be significant. For example, Wheaton, Roszill & Hall (1997) have noted that stressors may occur as part of a "matrix of disadvantage" (p.51), in which clusters of semi-independent stressors co-occur, producing

stress laden environment. For instance, these authors speak to the occurrence of abuse, "coming from a background of abuse probably does not describe sufficiently the array of stressors at issue in these cases" (p.70).

Such environments have been associated with negative belief systems that are characterised by thoughts of uncertainty, loss, helplessness, and hopelessness – cognitions considered to be critical in the development of negative emotional states such as anxiety and depression (Alloy et al., 1990) and defensive self-protecting coping styles such as avoidance, withdrawal, and dissociation (Horowitz, 1986). These factors have been hypothesised as acting on the individual's belief / self-system via the perception of one's self-efficacy as being insufficient to ward off the perceived threats, or to cope with the associated fear. For instance, Alloy et al. (1990) propose an 'aroused anxiety syndrome' as a result of uncertainty regarding one's ability to cope. This syndrome occurs when "the future is perceived as dangerous and threatening and one is likely to become hypervigilant, searching the environment for cues relevant to one's control over future outcomes" (p.525). In the context of severe stress-responses, this process may include the compounding effect of alternations in the cognitive processing of traumatic memories and avoidance of internal and external stress triggers (Horowitz, 1986).

One of the conceptual implications of viewing long-term stress outcomes as being the result of a network of interrelated factors is that research initiatives may need to utilise longitudinal methods. For example, gathering data on individual variables across development, as well as for the type, spacing, and ordering of the events may be critical components in ascertaining why individuals differ in their reactions to traumatic events. Again, both the conditionality as well as cumulative effects of stress indicate the multi-factorial nature of this construct. As will be reviewed in the following chapter, the extreme pathology represented by the syndromal diagnosis of PTSD demonstrates this multi-factorial challenge, particularly as this diagnosis appears to suffer from the same quality of conditionality as do conceptualisations of lesser subjective states of stress.

Chapter 12 Trauma and posttraumatic stress disorder: a biopsychosocial perspective

This chapter explores the nature of extreme stress reactions, such as trauma, and reviews Horowitz's two-factor model of traumatic stress responses. This is followed by a discussion of the diagnosis of PTSD, critiquing problems associated with different types of stressors and the high overlap of co-occurring symptomology and syndromes. Finally, a review of relevant neurophysiological data is made, with the occurrence of traumatic events during the formative years implicated as a biopsychosocial vulnerability to psychopathology in general.

As discussed above, stress can occur at different levels, particularly as distress can be conceptualised as lying along a continuum with well-being (King, Stanley & Burrows, 1987). At the extreme end of this spectrum distress can become severe and debilitating, resulting in mental illness and possibly a psychiatric diagnosis. The various outcomes of distress have been dubbed as the psychiatric equivalent of the common cold, typical being expressed through states of depression and anxiety (Mirowsky & Ross, 1989). This chapter explores this extreme end of the stress spectrum, introducing the concept of traumatic stress, and presenting the PTSD diagnosis.

The term traumatic stress has been applied to severe responses to stress, and is largely an outcrop from the psychodynamic tradition, the term "trauma" having its earliest beginnings in the writings of Freud and Janet. Eth & Pynoos (1985) state that trauma occurs "when an individual is exposed to an overwhelming event resulting in helplessness in the face of intolerable danger, anxiety, and instinctual arousal" (p.38). Horowitz (1986) has described this process as a 'stress response syndrome' within his comprehensive cognitive / information processing model of trauma. This model is a two-factor model that proposes that traumatic stress results in a cyclic alternation between the intrusion of traumatic material into conscious perception or memory, and avoidance or denial of this material when the person begins to feel overwhelmed. This process has been described by Briere (1999) as a 'healing algorythim' in

which the mind attempts to heal itself through cognitive and emotional habituation. Briere notes that this process is successful in around 20% of cases, however for others the intrusion phenomena can result in chronic re-traumatisation. Horowitz proposed a number of signs and symptoms that are characteristic of each of these factors (table 12.1). It could be argued that these two groups of symptoms roughly fit into the same dichotomous categories of positive and negative dysfunction as does schizophrenia, probably reflecting the chronic syndromal nature of each disorder (McGorry, 1991).

Table 12.1. Operational definitions of the signs and symptoms of intrusion and avoidance.

Signs and Symptoms of Intrusion.	**Signs and Symptoms of Avoidance.**
Pangs of emotion	Avoidance of associational connections
Rumination or preoccupation	Numbness
Fear of losing bodily control, or hyperactivity in the bodily system	Reduced level of feeling responses to other stimuli
Intrusive ideas in word form	Rigid role adherence
Difficulty dispelling ideas	Loss of reality appropriacy of thought switching attitudes
Hypervigilance	Unrealistic narrowing of attention
Re-enactments	Vagueness
Bad dreams	Inattention
Intrusive thoughts or images while tying to sleep	Inflexibility or constriction of thought
Intrusive images	Loss of train of thought
Startle reactions	Loss of reality appropriacy of thought by sliding meanings
Illusions	Memory failure
hallucinations	Loss of reality of appropriacy of thought by disavowel
	Warding of reality-orientated thought by use of fantasy.

Adapted from Horowitz (1986).

The longitudinal process described by Horowitz is one of immediate coping following a stressor, then outcry which results in possible panic, dissociative, or psychotic states, followed by the intrusion – denial cycle, the long-term outcome being either working through of the traumatic material or degradation in psychosocial functioning and personality structure. As discussed above, one of the effects of stressful experiences may be the development of vulnerability to further stress, and in the extreme, psychiatric illness. One of the expressions of this vulnerability may be severe anxiety such as PTSD. For example, individuals who are prone to experiencing anxiety, depression, family history of psychiatric difficulties with behaviour

problems of childhood, or negative personality factors have been found to have an increased risk for PTSD (Davis & Breslau, 1994). Further, for many cases, the trauma / PTSD symptomology increase in magnitude, causing long lasting changes to behavioural and physiological reactivity (Southwick, Yehuda & Charney, 1997).

In preparing for the release of the DSM-III in 1980, the American Psychiatric Association used Horowitz's two-factor model as the basis for the inclusion of the new axis I category of posttraumatic stress disorder (PTSD). PTSD has since been retained through to the current DSM-IV (see appendix IV for DSM-IV criteria), and is a unique category as it is one of the very few DSM-IV diagnoses that has etiology as one of the criteria (Green et al., 1985). The main criteria include a) a stressor event and the subsequent subjective emotional response, b) re-experiencing the event, c) persistent avoidance, and d) hyperarousal (American Psychiatric Association, 1995). However, one of the notable features of the PTSD category is that while the criteria appear straightforward, particularly given the stressor as a diagnostic 'anchor', thus indicating an uncomplicated diagnostic picture, complex presentations appear common (Briere, 1999). Two factors that appear to represent the complicated nature of this disorder are consistently reported in the literature – difficulty in determining what the stressor event is, and extensive psychiatric comorbidity.

Scott & Stradling (1994) note that "PTSD is unusual in including etiology as part of definition....the presumption, supported by the syntax of the definition –'the event', 'the trauma' – is thus of a single, acute, dramatic episode" (p.71). However, not all people who experience a PTSD type stressor will receive this diagnosis, suggesting it is not sufficient, and conversely, some people who would otherwise be eligible for the diagnosis are excluded on the basis of not having one significant reportable event in their psychosocial history, suggesting it is not necessary (Scott & Stradling, 1994). For many individuals the experience of a trauma laden event does not develop into a regressive manifestation such as PTSD or panic disorder. While dependent on the type, duration, and intensity of event, typically only a small minority of individuals present with a psychopathological reaction such as PTSD (Alarcon, Glover & Deering, 1999). The reasons for this may be due to mediating factors similar to those discussed above for stress-reactions. In particular, the diagnostic requirement of the presence of both a quantifiable stressor event as well as a severe reaction indicates that the presence of PTSD is a conditional event, and therefore open to individual difference. For example, it is now emerging

that traumatic stress does produce changes to certain areas of the brain, and that these changes are long lasting and can provide a vulnerability to hypersensitive reactions to future stress (Perry & Plate, 1994).

For those individuals who are adversely affected by life events, trauma can be associated with a long-term trajectory of problematic life factors, which not only represent the presence of traumatic experience, but also potential vulnerability to further victimisation. Therefore, the histories of people with PTSD can show a great range of stressful life events, particularly when associated with interpersonal trauma in childhood (Herman, 1992). Further to this, given the conditional nature of the person – environment interaction, individual differences in stress tolerance, and accumulation of stressful events, it is possible for otherwise innocuous events to be subjectively traumatic (Scott & Spalding, 1994). These types of problems have led Terr (1991) to categorise adverse events into type I and type II traumas. The type I traumas are the result of discrete and sudden events, which is more representative of the classification of PTSD. Terr distinguishes type II traumas as the result of ongoing or repeated events that have a cumulative impact on the individual. Garmezy & Rutter (1983) report that in children stress effects are multiplicative rather than additive as each time a risk factor is introduced into a persons life it potentially doubles the level of subjective psychosocial distress. While these multilple events include objectively severe stressors, they may also be more subtle, yet their personal significance can be dramatic (Widiger & Sankis, 2000). For example, Scott & Spalding (1994) report cases from the literature in which PTSD symptoms occurred as a result of employment related stressors, such as reprimands, often with the precipitating stressor being anything but unique, rather the last of a series of events. To Terr's type I and II traumas, Gallagher et.al. (1995) propose a type III classification to address the often found caretaker-child relationship failures that often predate and/or co-occur with the repetitive trauma. Overall, these problems of assigning etiology to an episode that is both discrete and categorically severe is particularly limiting in terms of applying the diagnosis to the full gamut of stress responses (Briere, 1999). Further, the range of different potential etiological stressors has meant that research on trauma has been largely conducted independently on particular types of trauma, resulting in parallel trauma literatures (Wheaton, Roszill & Hall, 1997) reminiscent of Kalmar & Sternberg's (1988) discussion of segregated theory.

In response to these problems of diagnosis, Herman (1992) has proposed a new diagnosis of 'complex PTSD' which refers to a more complex and serious form of PTSD as the result of prolonged and repeated trauma events. Typically the clinical implications of complex PTSD include: a) a more diffuse and tenacious clinical picture, including dissociation, somatisation, and chronic depression, b) characterological changes associated with interpersonal functioning and changes to self-identity, and c) a tendency for vulnerability to self harm or harm from others. McGorry (1995) notes that while this conceptualisation deliberately blurs the distinction between changes in state and trait related functioning, it offers good face validity for clinicians working with complicated cases of trauma, particularly for trauma histories stemming from childhood.

Related to difficulties in describing PTSD according to the stressor criterion is the difficulty in presenting the phenomenology of trauma as being restricted to a specific disorder. That is, there is a high overlap between PTSD symptomology and the symptoms subsumed by a number of other mental disorders. Brady (1997) reports that 16% of PTSD cases have one other disorder, and nearly 50% of PTSD cases have three or more diagnoses. When dissociation was taken as a comorbid disorder, it appeared in 82% of current cases of PTSD, and was particularly associated with cases involving childhood abuse. Further, in a psychiatric setting, those with PTSD are more likely to have substantial comorbidity than those without. The comorbid diagnoses for PTSD included affective disorders, anxiety disorders, somatisation, substance use, dissociative disorders (Brady, 1997), and borderline personality disorder (Briere,1999). Conversely, Meuser et al. (1998) in their study of 275 patients (inpatient and outpatient) with severe mental illness rated PTSD in 58% of depression cases, 54% in borderline personality disorder cases, 40% in each of bipolar and personality disorder cases, and 37% and 28% respectively for schizoaffective disorder and schizophrenia cases. In response to these figures, Brady has suggested that the term comorbidity when applied to PTSD is a misnomer, and van der Kolk et al. (1996) have suggested that PTSD and its comorbid conditions should be considered as "complex somatic, cognitive, affective and behavioural effects of psychological trauma" (p.85) rather than separable conditions.

The outcome of chronic PTSD has been described by Alarcon et al. (1999) as being able to be characterised by a cascading of neurobiological and psychological events. Their contention is that PTSD is a dynamic process that does not follow a static or unilinear course, but which is

modified over time by the individual variables and external events, even when these factors appear unrelated. The central point that Alarcon et al. (1990) make is that much of the psychiatric comorbidity that is seen with PTSD is better represented as co-occurring dysfunctions that are manifested secondary to the primary trauma reaction. That is, to treat them as comorbid entities is a misrepresentation, as according to Feinstein's (1970) definition, the term comorbidity is used to describe an additional clinical entity that occurs at the same time as the index illness, not to describe the multiformity or secondary diagnoses of a disease (Winokur, 1990). To use the example of Diabetes Mellitus, while high blood sugar levels due to pancreatic anomaly is the central dysfunction, the disease results in many secondary manifestations, such as neuropathy, that cannot be considered to be anything other than incipient components of the index disease.

As interest in the psychosocial nature of trauma has increased in the past two decades, so too has interest in the physiological components. In general it has been found that the experience of trauma is a good example of how psychological states influence physiology at a functional level, resulting in symptoms such as panic and dissociation, as well altering physiology at a structural level (Perry & Plate, 1994; Southwick, Yehuda & Charney, 1997; Van der Kolk, 1999). Researchers have long understood the role of catecholamine (norepinephrine) response via sympthetic nervous system activation – fight, flight, or freeze, as well as hypercortisolism via the hypothalamic-pituitary-adrenal axis (HPA axis) when stress is long standing (Southwick, Yehuda & Charney, 1997). However, more recent research findings indicate that the long term effects of PTSD have a more profound biological effect than that already found for stress. In particular, PTSD patients are found to have lower cortisol levels, as well as other HPA axis anomalies, suggesting a heightened sensitivity of this axis to stress. Also, there is an increase in receptor binding sites for glucocorticoid, an effect associated with decreases in the presence of cortisol (Southwick, Yehuda & Charney, 1997).

Perry & Plate (1994) have reported that trauma, particularly occurring during the formative years, can produce these long-term trauma related brain changes are particularly significant when they. These authors report that both the autonomic nervous system and the HPA axis can potentiate long-term sensitivity via cognitive processes as "concentration and arousal tune out nonsignificant information and focus completely on the external threat" (p.137), and if during development these systems are required to be persistently active, they become over active and

hypersensitive, 'mirroring' the environment. The result is a persistent state of hypervigilance or low level alarm, and resulting in modulation of the chronic arousal via dissociative states. Thus, "what was once a core neurobiologically mediated defense against acute stress may become maladaptive in its persistence and generalisation to non-threatening stressful experiences" (p.136-137), and which may act as a vulnerability to further traumatic states or experiences. Or the descent into severe psychopathology, such as schizophrenia (Harrop, Trowe & Mitchell, 1996). Such propositions are supported by the finding of the brain's plasticity of psychological events, which paves the way for an ongoing interplay between psychological and physiological events in the development of brain functioning (Harrop, Trower & Mitchell, 1996).

Briere (1999) states that whether trauma is acute or chronic is predicted by the *level* of avoidance, particularly when substances are used, or dissociative states or numbing is present, as habituation to the fear is blocked. The phenomenology of avoidance, particularly numbing is an inability to concentrate on or feel anything (Harrop, Trower & Mitchell, 1996). While numbing is rated as an avoidance strategy by Horowitz, and in the DSM-IV category of PTSD, it has recently been reconceptualised as belonging to the same autonomic process as anxiety related hyperarousal. Clinically numbing has been found to be the predominant stress response following a significant stressor event, and this has resulted in the DSM-IV including the diagnosis of acute stress disorder, which recognises differential immediate and long-term emotional reactivity (Briere, 1999). Further, numbing appears to have a threshold effect that is nonlinear (intrusion and avoidance symptomology are linear), thus indicating that it may be a physiological defense mechanism (Briere, 1999). Van der Kolk (1999) reports that freeze / numbing responses serve the function of blocking the conscious experience of overwhelmingly stressful situations, and that this process is governed by stress induced endogenous opiodes and norepinephrine systems. Briere (1999) reports that the presence of numbing in the early stages of trauma is predictive of chronicity and severity of PTSD as its physiological substrate makes numbing difficult to consciously control, because the presence of numbing typically indicates a perpetual state of being overwhelmed, and because little habituation can occur. Further, conceptualising numbing or dissociation as a functionally deficit state suggests that it may act as a psychosocial vulnerability factor. This may be characterised by deficits in conscious learning, particularly in the context of associated hypervigilence as reported by Perry & Plate (1994), as well as subsequent psychosomatic withdrawal and potentially interpersonal distrust reducing opportunities to gain protective social support.

Overall, this chapter highlights the endemic nature of stress in human functioning, the extreme examples of which are now recognised as being sufficiently distressing to warrant classification in the DSM system. However, the variability in the factors resulting in onset of the PTSD as a chronic disorder, as well as the high overlap with other disorders, indicates that the full impact and range of trauma related experience needs to be further explored. Some progress is being made with the recognition of the cumulative and longitudinal effects of stressful life events, represented by such propositions as a 'complex PTSD', and in developing an understanding of the physiological workings of trauma in the context of human development. In particular, combining the phenomenological data and the neurophysiological data, arguably the occurrence of trauma, particularly in the formative years, offers a vivid description of how psychological factors can elicit changes in brain functioning, which in turn can produce a long term biopsychosocial vulnerability to severe reactive changes in psychosocial functioning. Given the possible explanatory power of trauma related processes to psychopathology generally, the potential for further theoretical and empirical research is arguably enormous (McGorry, 1995). In this context, the part 6 explores the relationship between trauma and psychosis, highlighting some of the elements of severe stress that may be involved psychiatric illness, specifically the negative symptoms associated with psychosis and PTSD.

Part Six

Exploring the Relationship Between Negative Symptoms and Stress

Chapter 13 The association between stress, illness and psychosis

In this chapter the relationship between stress and psychosocial functioning will be further explored, first by observing the effects of stress and coping in general medical illness, an area in which health psychology researchers have been particularly interested. This interest follows the general finding that even life threatening events, such as coronary heart disease or diabetes mellitus, do not just 'happen' to people. People adapt to the illness according to their experience, perception, and appraisal of the illness, as well as a host of distal and characterological factors. This process of illness – person interaction is then applied to mental illness, in particular psychosis, highlighting the potential for traumatic stress to be a precipitant and an outcome of severe mental illness – each process colouring the subjective and quantifiable nature of the presenting disorder. To finish, it is argued that the process of stress responses is characterised as a complex system, in which a cascade of events may lead to 'jumps' into states of psychotic decompensation, or PTSD, or potentially a combination of both.

Research has found that significant levels of stress can be both a precipitant to general medical illnesses, as well as a response to illness (Sarafino, 1998). Stress can increase the chances of illness via changes in life style, such as increased use of substances, anxiety, and social isolation, as well as physiological changes such as degrading effects of stress on immunological functioning (Sarafino, 1998). Further, the person's response to illness can be coloured by their stage in the developmental trajectory. For instance, adolescents may find it difficult to re-organise their attempts for autonomy and self-identity consolidation around illness factors. In young adults this can cross over with problems in developing intimate relationships as this ability is influenced by people's perceptions about themselves as worthy, competent, and meaningful (Sidell, 1997). Individual's who do not acquire intimacy bonds are also at further risk for poor illness related adjustment due to the possible lack of social support (Sidell, 1997).

In terms of reacting to general illness, the initial reaction has been described by Shontz (1975, cited in Sarafino, 1998) as being characterised by three response states: 1) shock – being bewildered and feeling unreal; 2) encounter – disorganised period including feelings of loss, grief, helplessness, and despair; and 3) retreat – denial or avoidance strategies used until the person accepts adaptations need to be made. Retreat is used as a 'base of operation' from which the person can adapt to reality in their own time. However, illness also affects the person, particularly acting on self-concepts and life expectations for both the immediate future and the long-term, and the greater the threat presented by the illness, the more impact the illness can potentially have (Sarafino, 1998).

Two major psychodynamic factors that stand out are issues of loss – of health, control, and independence, and of uncertainty for the course of the illness, including mortality (Sarafino, 1998). Moos (1982, cited in Sarafino, 1998) has proposed a crisis theory for illness. This theory is a cognitive model in which a range of distal illness and psychosocial factors, and proximal appraisals and coping skills interact with the health crises. Appraisal involves the person's grasping of the meaning or significance of the illness to their life and predicts the formulation of adaptive tasks, the outcome being contingent on the types of coping skills employed. Moos suggests two categories of adaptive tasks. Firstly, illness related tasks include dealing with the pain and other symptoms, dealing with the hospital environment and treatment procedures, and developing and maintaining relationships with health professionals. Secondly, there are adaptive tasks related to psychosocial functioning, including preserving emotional balance, preserving self-image, competency, and mastery, sustaining relationships with family and friends, and preparing for an uncertain future.

The above discussion illustrates that stress and illness are inseparable elements, with the relative contribution of each dependent on individual conditional factors. In terms of responding to illness, arguably some level of anxiety regarding the outcome of the illness would be required to enhance adaptation and coping performance. However, depending on a number of subjective factors, such as idiosyncratic beliefs, distress tolerance, and related defence mechanisms, as well as objective factors such as positive social support and security, stress may intensify the negative effects of the illness, and potentially result in secondary morbidity.

While differences between mental illness and general medical illness would obviously make strong generalisations unwise, it could be argued that the principles of stress (illness) response may be similar due to the psychological factors involved. In both situations the person needs to adjust to an immediate and significant change to personal functioning and expectations for the future, changes in interpersonal relatedness, disruption of self-orientation schemas, as well as intrapsychic negative emotional states. Based on this assumption, some of the general findings from the stress literature, and the stress-illness literature may be able to be applied to potential stress – psychosis interactions. The potential may be for mental illness to be more of an engulfing experience, and subsequently more stressful, than general illness. In support, Estroff (1989) notes that severe mental illness is not just something the person has, but something the person is, or may become.

Studies have found that a number of life events have also been found to correlate with psychopathology, with symptom scores being found to fluctuate with the level and types of stressful life events experienced (Dohrenwend, 1979). Particular disorders that have been evidenced as following life events include acute schizophrenia, depression, and neurosis. However, Dohrenwend qualifies these associations by stating that the retrospective measurement of life events in these populations is problematic as for long-term syndromes the events may be due to the symptoms of the disorder rather than antecedents. While this differentiation is essential for identifying specific etiological factors, arguably, in terms of the longitudinal development of the disorder, either scenario would predict the possible importance of life adversity given the interaction between stress and psychiatric phenomena generally. For example, severity of life events correlates with psychiatric severity, with a greater range, frequency and intensity of stressors related to severe mental illness. Mueser et al. (1998) found in their sample of 275 cases of severe mental illness that PTSD was predicted most strongly by the range of different traumas (on average 3.5), followed by childhood sexual abuse. As noted by Perry & Plate (1994) the occurrence of 'core' traumas in childhood appear to be related to long-term trajectories of mental disorder, at least as significant components embedded in a wider matrix of disadvantage. These traumas include sexual abuse, physical and violence abuse both direct and witnessed, and parental death or divorce (Wheaton, Roszell & Hall, 1997).

Childhood abuse in particular has been found to have a substantial association with psychiatric disorders, potentially increasing the risk of psychiatric illness by two to three fold

(Burnam et al., 1988). Read (1997) in a review of child abuse and psychosis found evidence that "those who have been abused as children enter psychiatric hospitals at a younger age, have longer and more frequent hospitalisations, spend more time in seclusion, are more ikely to receive psychotropic medication, relapse more frequently, are morel likely to attempt suicide and deliberately harm themselves, and have higher scores on measures of global severity of psychiatric symptoms" (p.450). Read notes that given prevalence rates of female child sexual abuse being 20 to 30%, and physical abuse being 10 to 20% of the US population, this constitutes a major potential for the etiology of psychiatric illness. Within the psychiatric population, Read has calculated from studies between 1984 to 1996 that 64% of female inpatients report either physical or sexual childhood abuse (50% sexual abuse, 44% physical abuse, and 29% both physical and sexual abuse). Similar rates are reported for childhood physical abuse in male patients, and a lower rate for childhood sexual abuse, although male patients still suffer this type of insult in childhood at around twice the level of the general male population. Highlighting the potential for relationships to exist between stress and mental illness, Mueser et al. (1998) found that 98% were able to report at least one traumatic event in their histories, suggesting Read's calculations are more likely to be underestimates than overestimating the true rate of abuse in psychiatric populations.

A problem with studies showing trauma – mental disorder associations is that often the research samples consist of the most severe cases, therefore it is unclear if the abuse related symptoms are attributable to the sample, or if the abuse symptoms may have contributed to chronicity (Greenfield et al., 1994). This problem is further confounded by the high overlap of symptom ratings between psychometric instruments that are used to 'independently' rate different disorders such as PTSD, dissociation, depression, and schizophrenia. However, in an attempt to address some of these problems Greenfield et al. (1994) prospectively studied a group of first-onset psychosis for relationships between psychosis, childhood abuse and dissociative symptoms. They found that 53% of their sample of 38 patients had histories of sexual abuse, a proportion consistent with findings for more chronic patients. In summary they note that "our results indicate that childhood abuse may contribute to an atypical constellation of symptoms, which might not affect clinical outcome during the initial episode, but which may contribute to poorer treatment response and illness chronicity" (p.833).

Potentially contributing to the effects of the abuse Mathias & Bulia (1999) found that when violent abuse happened to a person who lived in a group home, had a substance abuse history,

or was diagnosed with schizophrenia, they experienced more negative responses when reporting the victimisation and seeking help. Similar poor responses to patients with schizophrenia, affective disorders, and substance abuse disorder who disclosed a history of childhood abuse were found in a New Zealand inpatient setting by Read & Fraser (1998). Zimmerman & Mattia (1999) found that underreporting of abuse history may be related to clinicians failing to ask about it, resulting in PTSD not being detected when it was not the presenting complaint. In illustration, Mueser et al. (1998) found that 43% of their sample of 275 inpatients and outpatients were able to be categorised as having PTSD, however, only 2% of these cases had previously had this diagnosis recorded.

Stress may also be an outcome of mental illness, the illness itself, or elements of being mentally unwell, being the stressor event, Horowitz (1986) notes that "mental disorders may create a cascading series of stress events, while at the same time impair coping capacity...the individual who has a psychotic reaction, is hospitalised and treated, and then is discharged may also be regarded as having a stress response syndrome....dreadful anticipation of social scorn....intrusive thoughts about the illness episodes....witnessed struggles with staff or demented episodes of other patients in the hospital may also have been stressful events, during a period of relative 'copelessness' " (p.67).

As discussed above, the emergence of acute psychotic phenomena has been associated with both the activation of an underlying vulnerability, as well as the emergence of secondary psychiatric features. In relation to the influence of stress in psychosis, Ciompi (1989) notes the possible influence of the dynamics of complex systems as articulated by Prigogine & Stengers's (1984). This theory postulates that due to positive feedback effects and non-linear escalating processes, complex systems make sudden 'jumps' from states of equilibrium into new 'regimes'. These jumps in the evolution of the system tend to occur following a period of destabilisation, and often depend on several environmental influences, and when the change occurs several 'jumps' may succeed each other in a cascade effect. This concept has been applied to epileptic brain dysfunction, family processes, and dopaminergic feedback activity. Ciompi (1989) suggests that these dynamics may also be applied to schizophrenia, in particular involving the 'switching' from normal states to psychotic states under the influence of stress. Ciompi argues that on the basis of biopsychosocial vulnerability, and immediate stressors such as family conflicts, the at risk individual may react in such a way as to escalate the tension in the social environment, and arguably themselves, until the escalation reaches a

critical point, dubbed by Bleuler (1984, cited in Ciompi, 1989) as the 'point of no return', where "a switching occurs, i.e. an abrupt change in the whole system of thinking, feeling, and behaving toward psychotic functioning" (p.19).

Strauss, Rakfeldt, Harding & Lieberman (1989) also promote the idea of viewing persons with severe mental illness as complex evolving systems operating within biological and social contexts, framing this approach at a more descriptive level. For example, Strauss (1989a) notes the difficulty that individuals with schizophrenia have in managing wide ranges in the level of stimulation, and that above a certain level the individual develops positive symptoms, but below a lower stimulus level the individual develops negative symptoms. To this they apply the notion of a 'stimulus window' in which functional relationships between the 'stimulus level' and the presenting symptoms are likely to be complex. Strauss considers that a range of factors, not least of which is the individual's self-regulatory coping attempts, produce "longitudinal patterns in terms of certain basic needs repeatedly described by patients: the needs for self – esteem, social relationships, psychological organisation, and involvement in productive efforts such as work" (p.24). However, acting parallel to this is the recognition that "the accumulation of undermining experiences may be particularly powerful in schizophrenia. Stigma, discouraging 'therapeutic' messages, social dysfunction, and the problems schizophrenia often generates in functioning cognitively may all interact over time to make remaining engaged, involved, and hopeful particularly difficult" (Strauss, et al., 1989).

If these compatible notions of complex systems in human functioning are put together, arguably it is possible to see how stressful events, stress reactions, and mental disorder may interact to produce a continuum (non-linear) between normal functioning and dysfunction. For example, Strauss (1989a) has proposed that from a systems perspective the severity and type of schizophrenic symptoms may be governed by dynamic regulatory mechanisms that allow for shifts in symptomology, such as the presence of a delusional system may precipitate hallucinatory phenomena under the influence of further stress. Similarly, Bleuler (1911, in Bentall et al 1988) considered one of the characteristic features of schizophrenia to be a disintegration of personality due to a loosening of associations. In terms of the impact of stress, this loosening of associations can be postulated as a parallel conceptualisation of the component process in PTSD whereby the individual fails to make continuous and coherent perceptions due to the effects autonomic system disruption. For example, a narrative taken

from a policeman at the 1989 Hillsborough disaster in Britain where police mismanagement resulted in the deaths of 90 spectators clearly demonstrates the response of a system of complex perceptual, representational, and intellectual modalities to extreme stress "At one point I thought I was going in and out of reality. I felt as if I were seeing things and then drifting off into not noticing things even though they were happening – I just couldn't register them. I can only remember patches. The daft thoughts that enter your head. They're not thoughts from you. I don't know how they get there" (Taylor, Ward & Newburn, 1995).

In the past two decades there has been a number of studies which have theoretically and empirically explored the association of trauma and psychosis as complex processes of overlapping symptomology. In particular three studies have been conducted that demonstrate a presence of PTSD diagnostic criteria being present for a substantial proportion of psychotic inpatients. Firstly, McGorry, et al. (1991) found that in their sample of 36 first-onset psychosis patients 46% and 35% were rated as having PTSD at four and 11 months post admission respectively. Further, they found that patients rated the process being hospitalised as most stressful. Similarly, Shaw, McFarlane & Bookless (1997) found in a sample of 45 inpatients recovering from acute psychotic episode that 52% of their sample met their criteria for post-psychotic PTSD, again with hospitalisation issues being reported as distressing, along with psychotic phenomena, although the authors noted that patients appeared reluctant to discuss the subjective experience of their illness, possibly indicating avoidance and embarrassment. Meyer, Taiminen, Vuori, Aijala & Helenius (1999) found that 11% of their cohort of 46 inpatients met the criteria for PTSD, and unlike the previous two studies, psychotic phenomena was rated as more disturbing than the hospitalisation process, with PANSS positive symptom and depressive / anxious symptom scores being the most predictive of the development of PTSD. These differences may be accounted for by first-onset psychosis vs. chronic samples, as well as different hospital regimes, or case severity. However, together they indicate the potential for different aspects of the psychotic experience, both content and process, to be highly disturbing. This also supports the contentions that 1) the experience of a stressor event may have many anchor points, each potentially being stored in memory separately (Pynoos & Nader, 1993); and 2) that both the novelty of the illness context as well as the illness itself constitute points of adaptation for the individual sufferer (Sarafino, 1998).

Further, a high level of stress may be associated with psychosis via life events that occur prior to an acute episode. Dohrenwend, Shrout, Link & Skodol (1987) note that the number of life

events reported as present prior to acute onset is significantly greater for people with schizophrenia than for control cases, and that 46% of the schizophrenia cases had at least one independent stressor event in the three week period prior to admission. Similarly, Rogler & Hollingshead (1965) found that people with schizophrenia and their spouses experienced more stressful events, including economic problems, physical illness, and interpersonal problems, in the 12 months preceding symptom onset than did controls, and that these difficulties were intimately entwined in their attempts to cope with the deteriorating psychosocial situation. However, there has been equivocal findings regarding the relative importance of a stress related 'trigger' for schizophrenia in the period prior to acute deterioration. Bebbington, Bowen, Hirsch & Kuipers (1995) offer one explanation for this disparity. They note that life events may have their effect over longer periods of time than the few weeks prior to admission used in other studies, a proposition supported by their finding of elevated events over controls over a prior three month period. Conceptually, these pre-episode life events may be components of the prodromal syndrome noted by Ciompi (1989), a phase characterised by the non-specific features of mood changes such as tension, irritability, depression, anxiety, withdrawal, as well as 'vegetative' signs (Malla & Norman, 1994). However, Dohrenwend et al. (1987) caution that while life events and poor social networks (support) may play a role in schizophrenic episodes, these events themselves may have been contingent on more important factors, such as personality characteristics, remote events and circumstances, and family history of mental disorder.

These findings exemplify the complex nature of person – environment interactions, and the need to understand the full range of events and states of functioning across time. In particular, the accumualation of events may be powerful determinants of severe mental illness. For example in psychosis, stigma, discouraging 'therapeutic' messages, social dysfunction, and cognitive deficits may interact in such a way to make being engaged, involved, and hopeful difficult (Strauss, et al., 1989). Arguably, taking a phenomenological approach to these factors, possibly treating them as micro-theories and single factor theories will

Overall, this chapter has reviewed the significant impact that traumatic stress can have, and how this may result in a number of psychiatric conditions, including psychosis. However, despite the significant associative and longitudinal data presented regarding the interaction of psychosocial factors in psychosis, the associations remain to be integrated into models that can competently handle the overlap in primary and secondary illness features. However, the

basis for such a model is highlighted with the recognition of the operation of complex biopsychosocial systems that may include both passive reactivity and active self-regulation as responses to personally meaningful experiences. The implications of this system are noted in the high level of traumatic events in psychiatric samples, the prevalence of co-occurring disorders, and the longitudinal changes in the way these disturbances are expressed. While many valid reasons may exist as to why a detailed phenomenological account of these process is yet to emerge, on reason may be that further theoretical work is required to direct the research needed to signify where significant relationships may be embedded.

One area in which theoretical and empirical interest is now emerging is for the potential overlap between the negative symptoms of schizophrenia and the avoidance and numbing features of PTSD, and area that until the now has received minimal research interest. Arguably, given that models of mental disorder facilitate better understanding and prediction of relationships between psychopathological factors, the phenomenology of negative symptoms would seem a rich area in which to seek some of these relationships, given the degree so psychosocial dysfunction associated with them. This endeavour also has clinical relevance due to the negative symptoms of schizophrenia typically having been viewed pessimistically due to their poor treatment response via traditional schizophrenic intervention methods. Therefore, new conceptualisations, and descriptions of novel theoretical relationships may be warranted, with the trauma literature potentially offering a broad framework in which to incorporate otherwise segregated phenomena. Following from this, the next chapter conceptually goes down a level to "search for the lost data" that may be used to highlight a theoretical interaction between trauma and psychosis-related negative symptoms.

Chapter 14 Stress and negative symptoms: exploring hypothetical relationships

This chapter explores the theoretical associations between the negative symptoms found in psychosis and PTSD. First the theoretical overlap between these two constructs is reviewed, and then this is followed by an exploration of a range of different symptoms and psychosocial factors that may be implicated as fundamentally similar deficit inducing processes. The chapter combines a phenomenological approach that explores not only the behavioural manifestations of avoidance and denial, but also the subjective social and emotional concomitants of deficit like reactions to disturbing events. Further, guided by an integrative approach, such factors as shame, stigma, developmental processes, psychological and social factors, and post-psychotic trauma are discussed in relation to negative symptomology. Overall, it is indicated that there is much material available in the psychological domain that points to a relationship being theoretically and clinically relevant for some cases of psychosis, however, much work remains in developing these links. In an attempt to provide some clarity to the cross-sectional and longitudinal nature of these relationships in clients with first-onset psychosis, case studies that illustrate the focal points will be presented in the following chapter.

Consistent with the research linking the psychosocial impact of adverse life events with people's level of functioning, quality of life, and ongoing mental and physical well-being is the 'rediscovery' of the strong influence exerted by biopsychosocial factors on the phenomenology of the negative symptoms associated with psychosis. As discussed in part 2, different researchers have attributed negative symptomology to a range of factors related to the experience of negative schizophrenia, such as a manifestation of the core illness, drug-induced akinesia, institutionalisation, depression, positive symptoms (eg paranoid avoidance) (Andreason, 1990), emotion focused-coping (McGlashan et al., 1975; Strauss, 1989a), neuroleptic-induced depression (Carpenter, Heinrichs & Alphs, 1985), demoralisation (Carpenter, Heinrichs & Wagman, 1988), a return to premorbid personality features

(Stampfer, 1990), and post-psychotic trauma (Jefferies, 1977; McGorry et al, 1991). Again, the negative symptoms have been differentiated by Carpenter et al (1985) into the two categories of secondary (illness-extrinsic) and primary (illness-intrinsic) negative symptoms, the secondary negative symptoms being construed as reactive, and due to problems of motivation, withdrawal, and resignation, and may be present in a patients with other diagnoses.

It has been proposed that Post Traumatic Stress Disorder (PTSD) is a potential causal mechanism for negative symptoms, via a discrete event such as hospitalisation (McGorry, 1991), disturbing psychotic symptoms (Meyer et al., 1999), appraisals of the social and self-oriented meaning of experiencing psychosis (Strauss, et al., 1989a), or from ongoing cumulative stress, which may also account for the build-up (prodromal phase) of deficit behaviour (Stampfer, 1990). In particular, the areas of emotional numbing, affective constriction, and estrangement from others appears to have the greatest conceptual overlap between the two syndromes (McGorry, et al., 1991). Further theoretical support for an association is the proposed dichotomising of these syndromes into positive and negative symptom constructs, with conceptual and empirical support being present for an overlap between PTSD and psychotic positive symptoms (McGorry, et al., 1991). Also, the association between trauma and negative symptoms is supported by the reported high prevalence of factors that are considered to cause trauma also being present in the histories of individuals with schizophrenia (Meuser et al., 1998; Read, 1997). To date only three articles have appeared in the literature that has shown specific interest in a hypothetical relationship between negative symptoms and PTSD symptomology. Two of those have been theoretical (Strauss, et al., 1989; Stampfer, 1990) and the third has attempted to show an empirical association (McGorry, et al., 1991). In addition to these reports, there are a number of articles exploring PTSD and psychosis interactions across a range of reactive, affective, and perceptual symptomology, as well as more general stress – severe psychopathology relationships. A number of these studies have been cited and discussed above.

However, while the association is documented, it has yet to be fully developed theoretically, and replicated via empirical analysis. Apart from the short history of interest in psychosis and trauma overlap, difficulties exist in the range of methodological problems involved in the measurement of PTSD and psychosis, such as comorbidity, item overlap on psychometric devices measuring PTSD, schizophrenia, general psychopathology, and depression, sample

biases in that those who decline to participate may be the most avoidant or apathetic, small sample sizes, and cross sectional evaluations (Shaw, McFarlane & Bookless, 1997). Further, the equivocal findings for negative symptoms cannot be taken as a negation of the overlap between PTSD and psychosis generally, which has received support. It may however, indicate the complex nature of both measuring and disentangling the deficit symptoms of severe mental illness, given the problems associated with rating an absence of some aspect of functioning (Zubin, 1985), as well as the negative symptoms being common to a range of disorders (Andreason, 1990), thereby diluting their effect when appearing in multiple syndromal presentations, such as PTSD, psychosis, and affective disturbance.

Given the above problems with theorising about negative symptoms and psychosis, two factors indicate the relevance of pursuing the identification of possible correlative and causal associations. Firstly, the number of confounding conceptual and methodological problems arguably speaks to the potential for more theoretical and empirical research to be directed toward this area. Secondly, there is good clinical reason to further explore this area – the enormous impact of negative symptoms in the psychoses in terms of case management and psychosocial disruption that may precipitate future relapse. Further, as noted above, the identification of negative symptoms and their successful treatment is arguably a crucial component in the early intervention for psychosis innovations. Based on these terms of enquiry, this chapter will take a descriptive biopsychosocial approach to symptomology and phenomenology, and will present a number of behavioural and single factor associations that may be implicated in deficit symptomology. It is hoped that not only the largely social and biological effects of psychosis will be highlighted but also the implications that stressful life events and psychosis have for the 'self' will be explored.

Coherence of PTSD and schizophrenia 'deficit' symptoms

It appears that there may be some validity to exploring the overlap between the avoidance or 'deficit' symptoms for PTSD (DSM-IV criterion C) and the negative symptoms of schizophrenia. As argued above, both the PTSD syndrome and the schizophrenic syndrome appear to have similar positive and negative dimensions, and a range of non-specific symptoms that may be related to overlapping anxiety and affective symptomology. As already argued, a symptom based approach may be one method of describing and refining possible

relationships between psychiatric events. This approach will be utilised here to discuss specific symptoms of each syndrome.

An empirical relationship between the avoidance component of PTSD and negative symptoms of schizophrenia was first tentatively indicated by the research of McGorry et al. (1991) in a sample of first-onset psychosis patients at EPPIC. This research group has since found a strong correlation in a replication. McGorry P.D. & Cooper J. (unpublished manuscript, cited in McGorry, 1995) found a similar correlation between PTSD and negative symptoms in a sample of Vietnam veterans suffering from PTSD, and who had never been diagnosed with schizophrenia. This may suggest that avoidance and negative symptoms are phenomenologically the same or parallel processes, or that the instruments used to measure the two constructs have a significant number of conceptually similar items.

In the DSM-IV (American Psychiatric Association, 1995), PTSD avoidance symptomology includes "deliberate efforts to avoid thoughts, feelings, or conversations about the traumatic event and to avoid activities, situations, or people who arouse recollections of it....Diminished responsiveness to the external world....markedly diminished interest or participation in previously enjoyed activities...markedly reduced ability to feel emotions" (p.425). For schizophrenia the symptoms of interest are "affective flattening is especially common and is characterised by the persons face appearing immobile and unresponsive, with poor eye contact and reduced body language....range of emotional expressiveness is clearly diminished most of the time....inability to initiate and persist in goal-directed activities" (p.276). Taken at face value, while the syntax is can be differentiated, particularly with the PTSD criteria contextualised by the trauma event, the representative observable behaviour may be indistinguishable. For example, by definition a client with post-psychotic PTSD could present as not engaging with his or her treatment (interpersonal and medical) or the treatment setting (buildings or staff) as a means of avoiding traumatic recollections, as well as having a range of non-specific negative symptoms that could be related to the psychosis or the PTSD. Arguably, at a behavioural level this client could be considered pessimistically as a case treatment resistant schizophrenia characterised by negative symptomology unless the time was taken to 'discover' the phenomenology of the presentation.

Neale, Blanchard, Kerr, Kring & Smith (1998) in their review of flat affect in schizophrenia note that flat affect as a clinical term can be taken to imply more than is justified for two

reasons. Firstly, they note that clinical ratings may be more of a reflection of interpersonal style or social behaviour than emotion, arguably reflecting personality dimensions in the schizophrenia's. Secondly, they suggest that components of emotion may be dissociated in some individuals with schizophrenia, thus giving an outward appearance of expressionlessness, while emotional arousal may be the person's subjective experience. For example, the authors report that for a subgroup of schizophrenia patients, mainly chronic, skin conductance responses indicate a high level of sympathetic activation, a finding that can be replicated by asking normal subjects to inhibit their emotional expression. Similarly, Harrop, Trower & Mitchell (1996) report that behavioural difficulties exhibited by thought disordered clients become more pronounced when they are asked to focus on emotionally meaningful material. Strauss et al. (1989) report one case in which a man diagnosed with schizophrenia was rated as having flat affect, while at the same time he was writing very emotionally charged poetry. Further, Neale et al. note the high occurrence of anxiety related events in this population, and the finding that precipitants of relapse indicate that these patients may experience intense emotions, and that in 50% of cases a stressful life event was present in the month preceding a relapse. While the authors acknowledge that this data needs to be further developed, it appears that emotional arousal / anxiety and flat affect may be a schizophrenic analogue of the hyperarousal and affective numbing described in cases of PTSD. If this was true for some cases it indicates a novel reconceptualisation of an otherwise 'primary' schizophrenic criteria.

One behavioural manifestation of avoidance could hypothetically be social withdrawal, or reversed, social withdrawal, as well as other forms of behavioural avoidance are likely to have underlying cognitive and affective components. Carpenter, Heinrichs & Alphs (1985) have argued that in cases of impending psychotic decompensation, negative symptoms may be exacerbated as a process of 'defensive withdrawal'. While it is may be difficult to generalise to humans from animals for stress research given the high degree of cognitive mediation, some studies have reported possible monkey analogues to human social withdrawal. Walker, Davis & Baum (1993) have reported that when monkeys are injected with amphetamine, producing hyperarousal similar to that seen in psychosis or PTSD, the predominant effect over a period of days is social withdrawal, as focusing on self rather than events and social relationships appears to be associated with relief from the hypervigilant state. However, while this behavioural response appeared to be adaptive in that it was directed toward maintaining homeostasis, the social effect was isolation, with the social structure of the group failing.

Walker et al. also reported that monkeys that have had portions of the prefrontal cortex ablated, an area of the brain strongly linked to psychosis and PTSD psychopathology, actively avoided social contact with other members of the group, with lesions having their greatest effect in adolescence.

For people with schizophrenia, the behavioural component of social isolation appears to be offset by the subjective desire in the clients to improve their social relationships and become productive members of the social group, indicating that anhedonia and social withdrawal may be at least semi-independent (Walker et al., 1993). It could be argued that a confounding factor may be social impairment conferred by neuroleptic medications may be misconstrued as intrinsic social withdrawal, with the real subjective concerns of the client being overlooked regarding their loss of functioning subsequent to medication treatment. In support of this association Walker et al. report that females typically need less neuroleptic medication than males, and correlated with this is their better social adjustment. Therefore, in terms of conceptualising the causal factors and phenomenology of social withdrawal, arguably many factors may be implicated in the initiation and maintenance of this sign of maladjustment, and in the case of neuroleptic related deficits, the presence of this extrinsic factor could potentially explain some to the refractory nature of deficit symptoms.

Nonspecific symptoms of severe mental illness associated with schizophrenia may also be intertwined with PTSD symtomology. For example, depersonalisation and derealisation (dissociative symptoms), depression, anxiety, anger, poor concentration, and memory impairment are functional states of each disorder (American Psychiatric Association, 1995). One of these symptoms that has received attention is anhedonia. Anhedonia appears to be present for both PTSD and psychosis. For PTSD, complaints of "markedly diminished interest or participation in previously enjoyed activities" is part of the avoidance criteria, and is also included as one of the non-specific negative symptoms associated with schizophrenia. However, given that anhedonia is found to be at least as severe in a cohort of depressed inpatients, it may be that this symptom is truly non-specific, and may be labelling a raft of underlying psychological or biological mechanisms. For instance, in their study of the outcome of psychosis, Hafner & Maurer (1995) found that at one year follow-up, many of the negative symptoms had abated, with the exception that "only anhedonia showed a markedly higher degree of persistence, but it was not possible to determine whether this symptom was

attributable to secondary phenomena – for example, such as social impairment – or whether it represented a primarily personality trait" (p.140).

Shaner & Eth (1989) note that a number of the residual signs of schizophrenia closely resemble symptoms of chronic PTSD. For instance persistent re-experiencing may appear as residual delusions or hallucinations that are no longer 'prominent', and may hypothetically also explain marked peculiar behaviour, ideas of reference, and illusions. Continued psychological responses to these residual positive symptom features may potentially be a contributing factor to other residual negative symptoms.

To summarise, the above symptom similarities may indicate that there may be considerable value in determining if trauma related processes are involved in the presence of negative symptoms in clients with psychosis, either from events before or around the psychosis, or as a result of the psychosis itself. While the above discussion focuses on a limited range of the full symptomology associated with negative symptomology, a review of DSM-IV specific and associated features of PTSD and schizophrenia indicate that the potential symptom cross-over may be more extensive (table 14.1). The associated features are included as they represent the more chronic or residual features in both schizophrenia and PTSD, and therefore are longitudinally relevant. The symptom descriptions provided are, however, simplistic and may miss subtle phenomenological differences, or be representing different underlying mechanisms or dysfunctions. The list may also not be fully inclusive of the full range of potentially overlapping factors given its restriction to DSM-IV signs and symptoms. Further, this set of symptom pairings may be over biased toward identifying similarities between potentially disparate constructs, and therefore missing critical differences. However, given the descriptive nature of the DSM system it could be expected that these similarities may have some clinical relevance. Albeit, at least as indicators for further research and theory building efforts, and to highlight the potential subjective distress that is the person's response to life events and psychiatric illness, rather than behaviour being 'decontextualised' and rated plainly as part of a disease manifestation.

While this cross over of symptoms is hypothetical, the symptoms themselves can be placed in a variety of descriptive single factor theories or specific associative processes (Ward & Hudson, 1998) that may increase the explanatory power of the proposed relationships by providing them with context. That is, contextualising the symptoms or related processes

within known psychopathological processes, such as post-psychotic PTSD, a range of reactive negative emotional states, stigma, and shame.

Table 14.1. Hypothetical overlap between features of schizophrenia and PTSD in DSM-IV.

Schizophrenia.	**PTSD.**
Affective flattening.	Psychic numbing.
Alogia (poverty of speech related to diminution of thoughts).	Psychic numbing.
Avolition (goal-directed behaviour dysfunction).	Psychic numbing / avoidance / dissociation. Difficulty completing tasks.
Inappropriate affect.	Impaired affect modulation.
Anhedonia	Anhedonia.
Depersonalisation and derealisation.	Dissociative symptoms.
Depression / dysphoric mood.	Depression / low mood.
Anxiety.	Severe anxiety disorders.
Suicidality (10% of schizophrenic cases).	Self destructive and impulsive behaviours.
Somatic concerns.	Somatic complaints.
Sleep pattern disturbance.	Sleep pattern disturbance.
Interpersonal relationships dysfunction.	Interpersonal relationships dysfunction.
Social Isolation.	Avoidance / social withdrawal.
Subjective experience of danger to internal stimuli.	Subjective experience of danger to internal stimuli.
Attentional problems / concentration.	Difficulty concentrating or finishing tasks.
Confused and disorientated during active phase.	Acute stress reaction (see Acute Stress Disorder criteria).
Dysphoric states.	Reactive emotions such as guilt, shame, despair.
Substance related disorders.	Substance related disorders.
Neuroleptic refractory.	Neuroleptic refractory.
Early personality dysfunction.	Change in personality characteristics.

Post-psychotic PTSD: a personal "disaster"

Post-psychotic PTSD as a hypothetical clinical phenomena was first discussed by Jefferies (1977) in response to his observation of an ongoing 'amotivational syndrome' that appeared to persist beyond the remission of the acute psychosis. Jefferies proposed "that going crazy is a traumatic experience...that the consequent effects on one's self-esteem and ego-identity may be such as to precipitate a traumatic neurosis....similar to that which follows back injury, or ...coronary infarct" (p.199). McGorry et al. (1991) consider PTSD as a possible "response syndrome" to stress induced by a psychotic episode from two main sources. Firstly, psychosis is highly anxiety provoking potentially as a result of persecutory phenomena, terrifying perceptual disturbances, and threats to the self. Secondly, going through the treatment process can include involuntary hospitilisation that may involve the police, coercion, restraint,

seclusion, or sedation. The context for this experience is a closed environment with other disturbed individuals, at a time when one's own capacities are under challenge from acute symptomology. The impact of hospitalisation appears to have its greatest traumatic effect for those experiencing their first admission (McGorry et al., 1991). Hammill, McEvoy, Koral & Schneider (1989) in their study of seclusion found that patients considered their seclusion to have bothered them more than any other part of the hospitalisation process, with the patients subjective experience being one of negative appraisals and anxiety - feeling humiliated, punished, helplessness, fear, sadness, and anger. These authors noted that seclusion is used most frequently for schizophrenia patients, and typically early in the course of hospitalisation, arguably when the patient is most in need of interpersonal support.

In terms of the association of PTSD with negative symptoms in the post-psychotic phase, a strong correlation was found in a sample of first-onset psychosis clients at EPPIC (McGorry, 1995; McGorry et al., 1991). While this research suggests that the hypothetical relationship between negative symptoms and PTSD in the post-psychotic phase is supported, further replication is required, particularly given the methodological disadvantages of using self-report, and the small sample size, the authors themselves cautioning against premature conclusions based on their findings. A post-psychotic depression has also been considered to be a reactive process in psychosis, either as a response to the illness, a side-effect of treatment, or possibly a hidden feature of schizophrenia which is unmasked during the post-acute phase (Bentall et al., 1988). Similar to the link between PTSD and negative symptoms based on functional deficit states, the depression and negative symptoms appear to co-occur. Similarly, Zlotnick, Warshaw, Shea & Keller (1997) have reported that individuals who have a history of trauma are at risk for chronic depression, and McGorry et al. (1991) found that depression in their sample appeared to have been secondary to the PTSD syndrome. This suggests that at least one association between deficit symptoms may be via depressive symptomology as affective components overlap in PTSD, depression, and schizophrenia. Potentially, depression may be part of a reactive psychological process for some individuals across a variety of disorders (Stampfer, 1990). This contention is also in keeping with Andreason's (1990) observation that negative symptoms are not specific to the schizophrenic disorders, and that negative symptoms can be found at rates as high, or higher in cases of severe depression (Hafner & Maurer, 1995).

One potential problem with the conceptualisation of post-psychotic PTSD may reside in the implicit assumption in the terminology itself, the term *post* indicating that the trauma is a consequence of the nearest severe stressor – the psychotic episode. Certainly, mental health professionals having regular contact with the devastating short-term and long-term consequences of psychosis, may justifiably rate observable PTSD symptomology as subsequent to the psychosis. However, to state that the trauma must be from the psychotic experience may undermine the process of exploration that must go on for each client, to uncover the phenomenological meaning of the client's presentation. For example, multiple stressors, including extraneous variables in the environment such as work, study, and relationships, may themselves have been subjective stressors, implicated in the prodromal phase, and possibly causally implicated in the emerging psychosis. Thus suggesting the importance of attending to the client's own account of their illness and points of subjective distress.

In the long-term, the most serious effects of post-psychotic PTSD may be an increased risk of relapse. According to the vulnerability stress model, biopsychosocial characteristics of vulnerability, such as autonomic hyperactivity, may interact with environmental stressors, resulting in intermediate states of hyperarousal (Shaner & Eth, 1989). These states may produce a vicious cycle of vulnerability, stress, and possibly unwanted coping attempts, such as substance abuse, that increase the risk of further psychotic symptoms, particularly as the inter-episode post-psychotic trauma includes hyperarousal and interference with social supports (Shaner & Eth, 1989). Further, the presence of deficit symptoms may decrease the client's ability to reintegrate their sense of self, and their ability to function socially and gain interpersonal support, this effect being particularly disruptive during adolescent psychosocial transitions.

Psychological and social dynamics

Arguably, one advantage of using a person – centred phenomenological approach is in observing the individual in their interactions with social and environmental stimuli, as well as being able to 'discover' the subjective meanings inherent in significant personal experiences, and how these appraisals relate to coping attempts. Stressful events may have an impact on the person's domains of social functioning, such as work, school, and family. Further, the same event may have multiple meanings for the person experiencing it, depending on the role

domains that are affected by it (Estroff, 1989). These role domains include the various social roles that the person may have depending on the range of interpersonal relationships that are operating – the 'public self', as well as a the individual's 'private self' that makes personal attributions based on the subjective experience of the self (Estroff, 1989). It may be that the more of these domains that are negatively associated with the event, the greater the impact of the event. Further, if the event has considerable impact on the self, then functioning across all the other domains may be derailed via self concept deficits and physiological changes. For instance, Freud (1967) considered overwhelming threats to the self, internal and external, to result in helplessness of the ego, the clinical presentation being one of post traumatic neurosis.

Psychosis has been conceptualised as one stressor that impacts across all of the above domains, not least on the self, given the internal nature of the disorder and the ensuing fragmentation of the mind. Jefferies (1977) pre-empted the current interest in this association when he applied the notion of post traumatic neurosis to the effects of psychotic disorder. More recently, Strauss (1992) reports that for psychosis, perceptions of losing one's mind or going mad have immediate effects on the self-system. A dramatic description of the experience of schizophrenia comes from Honig (1988) "perhaps the true illness is terror, the dread of annihilation and impending doom, and the accompanying psychic pain" (p.431). These descriptions of responses to psychosis for some cases appear to mirror the psychodynamic perspective of trauma as the result of threats to the integrity of the self (Horowitz, 1986).

That people with psychosis can experience the full gamut of biopsychosocial reactions to stress appears to be born out by the high level of co-occurring anxiety, reactive depression, PTSD, substance abuse, suicidality, and interpersonal and social problems as discussed in detail above. Further, the subjective nature of stress may arguably be most notable in trauma related to psychosis as the client is reacting to intrinsic stressor events that may be difficult to differentiate from the self, and that overall symptom severity affects subjective experience (Bradshaw & Brekke, 1999). Therefore, description of the subjective elements of trauma for these people may be critical for a full understanding of the processes involved in the initiation and maintenance of this illness manifestation. Subjective accounts from persons with psychosis reinforces this assumption. Strauss (1992) reports that "some of our subjects with psychotic disorders often lose their sense of being a person..." (p.22). If this loss, or threat to personal integrity, was physical rather than intrapsychic, combined with the fearful subjective

distress reported above the stressor criterion for DSM-IV PTSD would be met. Arguably, while the physical component of the criterion may increase reliability of caseness, it may be detracting from clinical validity, at least in some cases of post-psychotic PTSD.

The effects on the individual's social and private world may change over time as the illness moves from the vegetative and anxiety prone prodromal period, to acutely florid phenomena, and potentially to chronic symptomology. Over time a transformation may occur in which a prior, enduring, known, and valued self may be replaced by a less known and knowable, devalued, and dysfunctional self (Estroff, 1989). Estroff argues that one of the ways that this occurs is through a process of role constriction, in which social contacts and productive activities are lost, along with the positive identities derived from those roles. The new predominant role that emerges may be that of the "schizophrenic", as the person's self-concept becomes engulfed by the new deviant role. High levels of engulfment have been found to correlate with an increasingly demoralised and restricted life, hopelessness, depression, low self-esteem, lack of self-efficacy, and social maladjustment (McCay, Ryan & Amey, 1996). These authors also report that in first-onset psychosis clients, subjective emotional responses to engulfment include confusion, denial, anger, shame, and a sense of alienation from others. Further, they suggests that the critical element in responding to psychosis is the meaning of the events, an explicit and idiosyncratic description of which may be therapeutic and lead back to positive identity formation. For longer term patients, Strauss (1989b) reports that brief relapses are perceived as less threatening as it reassured them that they could get well again, whereas a long relapse produced feelings of helplessness, hopelessness, and result in the loss of skills and stamina.

In terms of the high overlap of depression with negative symptomology, the processes involved appear to indicate the presence of dynamic cognitive processes, as suggested by a complex systems approach. For example, in terms of reactive depression Chadwick, Birchwood & Trower (1996) argue that an important factor implicated in the emergence of post-psychotic depression is the client's attitudes, inferences, and evaluations about themselves and their illness; it is the appraisal of the psychosis, not the severity of the psychosis itself, that predicts favourable coping and outcome. In particular, they argue that it is the global and total condemnations made of the self and others, that have a significant impact on recovery style and emergent depression, and that this vulnerability is rooted in the attachment process from early childhood. Extrapolating on this theme, and their finding of a

significant relationship between recovery style and depression, Drayton, Birchwood & Trower (1998) suggest that the diagnosis of psychosis presents yet another threat to the self, activating an immature defence style that protects against overwhelming anxiety. They acknowledge the simplicity of this model, and the need to disentangle the complex relationships between psychosis, fragility of self, and helplessness depression. However, this is supported by McGlashan et al. (1975) and Strauss (1989a) who have described 'sealing over' and 'woodshedding' respectively as psychodynamic processes that are conceptually linked to the behavioural symptoms of avoidance, denial, and social withdrawal.

Addressing the cognitive-memory processes associated with trauma, McGorry (1995) argues that in the first-onset psychosis population, psychosis is a novel traumatic event that lacks existing schematic structures to anchor the experience to, and therefore the material is prone to being dissociated and handled differently in memory. This type of dissociative processing is similar to what occurs for people who are adversely affected by other types of novel traumas, such as the experience of abuse (Sauzier, 1997). McGorry posits that this may account for the sealing over, denial, and unawareness of illness that is characteristic of some first-onset psychosis clients, and may be a function of numbing and avoidance associated with dissociative symptomology. And that these avoidance symptoms may predate and mask the intrusive symptoms, possibly explaining why the link between PTSD type symptoms and psychotic symptoms is difficult to elicit in these individuals.

Strauss et al. (1989) propose a range of psychological and social sources of negative symptoms as including the pain of relapse into florrid states, loss of hope and self-esteem, the potential for impulsive or bizarre behaviour, problems forming a new identity that does not include a sick role, feeling guilty for past dysfunction, the threat of entering stressful situations, situations where the person is rendered helpless from the disorder or the environment (such as in cases of repeat relapse or low socio-economic background), institutional demoralisation, a need to stay 'sick' to be eligible for the social benefit, and stigma. Further, the dysfunction that can occur in these areas can accumulate into what Alarcon et al. (1999) consider to be a cascading effect which leads to more guilt, lower self-esteem, interpersonal sensitivity, depression and further social isolation. And from a complex systems approach would constitute a significant risk for relapse.

Self-regulatory mechanisms

It has been proposed that the secondary negative symptoms associated with schizophrenia and trauma are primitive self-regulation mechanisms, that is they are not just passive reactions to events, but may have a subjective functional component. Both McGlashan (1990) and Strauss (1989a) both argue that negative symptoms may have an adaptive function, as the person utilises regulatory defences to manage and adapt to increases in stimulation. McGlashan (1990) states that "patients may 'use' negative symptomology to seal off other affect states like anxiety, rage, and shame, or to defend against a stimulus barrier breakdown" (p.179). Similarly, Strauss (1989a) has posited 'woodshedding' as a defence mechanism which is characterised by a long plateau which includes dominant features of apathy and withdrawal. Therefore negative symptoms can be conceptualised as not just passive reactions to intense distress, but for some individuals a dynamic form of emotion-focused coping (MacDonald, Pica, McDonald, Hayes & Baglioni, 1998). This process is conceptually coherent with the types of illness reactions that are predicted by crisis theory as discussed above, including such coping strategies of avoidance and denial. One example of the operation of avoidance as a regulatory mechanism is the observed decrease in interpersonal relatedness and productive activity following discharge from hospital. Strauss (1989b) suggests that this behaviour is employed protectively in an attempt to reduce the demands of the new situation.

This process is conceptually similar to models of cognitive and emotional self-regulation. Baumeister (1991) discusses a clinical presentation that is strikingly similar to negative symptoms, positing such as an 'escape from the self', when the self experiences a lack of internal resources to cope with a given situation and has the sense of failure. Baumeister argues that events that carry threatening implications about the self produce escape needs and behaviours in an effort to reduce the level of internal stress caused by the subjective appraisals. In particular Baumeister posits a concomitant state of cognitive deconstruction as a state in which "the person responds to this unhappy state by trying to escape from meaningful thought into a relatively numb state...." (p.91). Arguably, this process of changes in consciousness may become systematised over time. While the dynamics of escaping dreaded appraisals may vary from a stress response to traumatic internal stimuli and subsequent hypervigilance, theoretically these types of stress responses may be representative of complimentary cognitive and emotional processing of distressing subjective material, with both potentially occurring in psychosis and PTSD. Accepting that numbing or avoidance

could be present via these two different pathways, this may promote the conceptual validity of the non-specific nature of reactive avoidance. It is of note that Baumeister developed his theory in response to the self-destructive behaviours of suicidal and alcohol abusing clients, both pathologies that are common in chronic forms of psychosis and PTSD.

Further, the process outlined by Baumeister is conceptually strikingly similar to dissociation found in trauma cases, and which has been found to be strongly related to psychotic symptomology (Allen & Coye, 1995), as well as physiologically modulated flight, flight, freeze responses (Briere, 1999). Kluft (1992, cited in Allen & Coyne, 1995) has described dissociation "pragmatically as a defence in which an overwhelming individual cannot escape what assails him or her by taking meaningful action or successful flight, and escapes instead by altering his or her internal organisation; i.e. by inward flight" (p.143), the result being states of 'tuning out', 'zoning out', 'foggyness', 'unreality', 'dreamlike' (Allen & Coyne, 1995). One potential process in this relationship may be between HPA axis related hyperarousal and numbing, and behavioural avoidance. For example, Harrop et al. (1996) suggest that thought disorder may be a kind of natural 'antidepressant' that serves to block out, or numb, threatening internal stimuli. In the new DSM-IV diagnosis of acute stress disorder, dissociative symptoms include feeling of detachment, numbing or lack of emotional responsiveness, decreased awareness of surroundings, derealisation, depersonalisation, and inability to remember a significant aspect of the trauma (American Psychiatric Association, 1995). Therefore, it may be argued that dissociation could be considered a syndrome of cognitive and emotional deficits, and perceptual disturbances, the components of which are not unlike the negative symptom cluster, or sealing over / woodshedding, and appear to be employed at some level of consciousness in response to traumatic stress. Further, based on both the dissociation and schizophrenic deficit literature, while dissociation has an adaptive function, both syndromes can become maladaptive, and have been associated with ongoing vulnerability to life events and further distress (Gershuny & Thayer, 1999; McGlashan et al., 1975). For example, positive social and familial support has been shown to provide protection against psychopathology, with both the positive value of this mechanism and the converse risk associated with negative or hostile social environment having been highlighted for schizophrenia and PTSD samples (Erickson, Beiser & Iacono, 1998; Tarrier, 1996). This protective mechanism may be undermined by behavioural avoidance, and cognitive and emotional denial via social isolation and or increased pressure from the family to reintegrate socially and functionally (Hooley, 1985). Arguably, both the negative symptoms and the lack

of interpersonal support may interfere with medication adherence, another critical protective factor for this population (Bebbington et al., 1995).

Stigma

One prominent psychosocial problem that confronts some people with severe mental illness in western culture is social and self-stigmatisation. Katz (1981) considers stigmatisation to be a process "whereby the perception of a negative attribute in a person becomes associated with global disvaluation of the person" (p.2), and the discrediting attribute could be any personal or social feature "that arouses in observers strong feelings of repugnance, disdain, fear, and so on", and that the "stranger who displays a strong the negative attribute will tend to be seen as having other negative attributes as well" (p.119). Byrne (1999) states that for mental illness sufferers stigma can come from the illness, its treatment (i.e. neuroleptics), or attending a psychiatric service unit, and a predominant emotion appears to be one of shame. To this point, Byrne argues that being diagnosed with a mental illness begins a process in which the person begins to perceive themselves as different, and self-stigmatisation may occur. In relation to schizophrenia, Strauss et al. (1989) have listed the stigma of being viewed as deviant (mental patient) as one psychological pathway to negative symptomology, with the severity of the stigma, the severity of the disorder, personality traits, and other stressors or protective factors modulating symptom expression. Further, in a study of 75 mental service user's perceptions of public attitudes "most users perceived the public as overwhelmingly unsympathetic and afraid" (Shepherd, 1998, p.56).

While levels of stigma could be conceptualised as varying with levels of paranoid symptomology, the perception of one's stigmatisation does appear to reflect real tendencies in the general population to view the psychiatrically unwell pejoratively. For example, Patten (1992, cited in Read & Law, 1999) surveyed 1001 New Zealand adults and found the dominant impression to be that psychiatric disorders lead to unpredictable behaviour, a loss of control, nonresponsibility, increased incompetency, unreliability, violence, and dangerousness. Read & Law (1999) report that the predominant stereotype is of the unpredictable and violent 'madman', particularly for those people diagnosed psychotic, even though only four percent of murders are committed in New Zealand are perpetrated by individuals with mental illness.

Shame

As already noted shame, guilt, and anger occur in response to traumatic events, as well as psychosis. Further, shame is associated with chronic illness and loss, and may be an analogue response to guilt when the self is perceived as having been at fault. For example, in survivors of natural disasters, guilt is a significant contributor to maladjustment (Raphael, 1986). In cases of mental disorder, this guilt may possibly be modified into shame as the disaster is personal and internal. Arguably, shame may have particular relevance in adolescence given the perceived social deviance associated with mental illness by sufferers and the wider society. One Totara House case history reports a client who "is sensitised to others laughing around him, and he becomes agitated by thoughts that they are laughing at him, and wants to act out, and when others compliment him he experiences a voice that tells him to say "fuck off". As such shame and stigma may be intimately entwined. Understanding the implications of shame in the phenomenology of negative symptoms may be of particular benefit to clients experiencing this negative affect state.

Taking the approach of Gestalt theory, Stein & Lee (1996) note that people fix their needs and goals in relation to their experience of themselves and their experience of their environment in relation to themselves. However, in chronic illness, their example being multiple sclerosis, the environment is no longer external, as it exists within the body and limits the body, and because of this a particular problem associated with chronic illness is determining what is illness and what is the self. And as people's abilities and sensations change with the illness, the self – environment 'gestalt' is always changing, the result being that the person is constantly faced with disorientation and the unknown. Chronic illness, they suggest, is like "living in a very unfriendly environment – one of pain, disorientation, the need to constantly change one's image of self, and so on" (p.111).

Lewis (1992) states that shame and sadness (depression) share a common cause and exhibit behavioural similarities (gaze averted, hunched shoulders, bodies pushed inward, inhibition, and cognitive deficits), behaviours that overlap with negative symptoms. If the shaming experiences are repeated, the result is typically depression, not just sadness. Lewis argues that depression, rather than socially unacceptable anger, is an emotional substitute for shame in conditions where the self-system is threatened with breakdown. Lewis argues further that "we must be prepared to treat individuals with depressive symptomology, which may reflect

underlying shame, with care, since the removal of the depressive symptomology will uncover the shame, and without any defence the shame may promote more serious disorders, such as schizophrenia" (p.145).

Substance Abuse

A noteworthy feature of both psychosis and PTSD is the high level of co-occurring substance abuse found in each. Substance abuse in either of these disorders is associated with poorer outcome and a more severe illness course (Davison & Neale, 1996). The same features are found in clients who have the combined diagnoses of psychosis and PTSD, and the substance use may be one pathway to increased psychopathology in this group (Cantwell et al., 1999). Arguably, some individuals diagnosed as psychotic, and who use substances, may well be doing so to modulate the emotional processing and stimulation associated with traumatic memories.

In summary, it is indicated that the association between the negative symptom syndromes found in psychosis and PTSD may be relevant in some cases, and that the implications that some of these associations may have for clinicians and researchers need to be further developed. In particular, it is possible that much of the non-psychotic or secondary phenomena can be conceptualised as being reactive to psychosis or the accumulation of other events, and may be associated with the psychosocial sequela of distal and proximal traumatic events. While other readings of the literature may have found further associations, or discrepancies between the proposed associations, taken together the above discussion indicates that theoretical attention to stress – psychosis interactions is warranted. This is arguably so given the range of overlap in psychodynamic and non-specific factors that appear to have some conceptual relevance to the variance in psychotic presentations. Further, in taking a wide sweep of the associated literature, several lines of enquiry into psychopathology appear to cross over. These have included the reconceptualisation of negative symptoms as a self-regulatory state, the links between shame, stigma, and depression, and the interactions between both positive and negative symptoms in PTSD and psychosis. In part seven a discussion of first-onset psychosis is made, in particular highlighting the additional effect of the critical developmental stage of adolescence on responses to potentially threatening or incapacitating mental illness. Further, a comprehensive treatment programme is reviewed, along with case histories illustrating stress related processes in this population.

Part Seven

Life Events in First-Onset Psychosis

Chapter 15 Developmental issues in first-onset psychosis

This chapter explores the effects of developmental stage on symptom expression and psychosocial impact of first-onset psychosis. In particular issues of autonomy and social relatedness are central to the types of disruption that can be the result of psychosis for some individuals.

Developmentally, psychosis could be seen as producing a significant degree of distress as it is a life event that is incongruent with the individual's expectations and appraisals, as indicated by feelings of loss and uncertainty, not only for their lives in general, but also as this event is 'out of time' in the life-span trajectory – chronic illness typically being more associated with older age. Further, adolescent developmental issues of autonomy and identity formation, as well as state dependent cognitive appraisals, are likely to interface with coping attempts. For example, if avoidance is used chronically as a protective self-regulatory mechanism, then the impact may potentially be one of a vicious downward cycle, characterised by the emergence of poor social skill utilisation that has been found in people with schizophrenia (Bellack, 1997), and possibly poor stress tolerance, and subsequent vulnerability to further overwhelming stress experiences additive to the effects of psychosis. Similar effects may be found when substances are used, possibly reflecting one the most dysfunctional forms of avoidance, but which is common in the young first-onset psychosis population. Also, given that the developmental issues of adolescence reflect a significant overhaul of the self-system with the emergence of abstract cognitive abilities, the impact on the self – both personal and related to others, the experience of psychosis and related psychosocial adversity may be particularly distressing.

As first-onset psychosis typically occurs in adolescence and early adulthood, the developmental – psychodynamic issues occurring at this juncture need to be incorporated into the intervention paradigm. The well cited developmental psychoanalyst Erik Erikson (1959, 1968) has proposed that adolescence is a period in which identities are formed, such as for sexuality, work, and culture. The central task of this stage, according to Erikson, is the

consolidation of a self or identity that incorporates past identifications and aligns the adolescent's attributes with the opportunities that society offers. The successful resolution of this "conflict" is a coherent sense of self that allows for further personal growth, and for productive contribution to society. A significant component of this developmental process is an emancipation from dependence on the adolescent's family and other childhood institutions, a process which is negotiated individually by each family unit in western culture (Peterson, 1989), and can result in stress from interpersonal conflicts and attempts to achieve the social milestones of academic success, career, and intimate relationships.

Erikson (1959) describes a strong interaction between the society – culture and the individual in identity formation, and argued that problems in development arise when socialisation does not prepare children for the demands they will face as adults. Erikson's conviction appears to be that each culture promotes different paths of development, for each society functions best with adults of a particular personality structure. It could be argued that western culture has poorly integrated notions of mental imbalance with contemporary social demands and acceptability, possibly premised on the perceived direct and indirect socio-economic costs of mental illness (Miller, Mahurin, Velligan et al., 1995). Therefore, the experience of psychosis or any debilitating mental illness is a psychosocial demand that may be poorly prepared for in western culture, especially in young people facing the challenge of new social paradigms, and forming a cohesive sense of self. In particular, at a time when autonomy, new social allegiances, and career issues are to the fore, these individuals may find themselves in a new form of dependence that is 'out of time' for their expected developmental stage. For instance, Jefferies (1977) states that "sometimes explicitly, but more often implicitly, schizophrenics [*sic*] are seen as being "like children" and the expectations consequently put Sandra them are those that one would put on a child rather than on an adult" (p.202). This process may occur both in the clinic and in the family, particularly if resistance to autonomy is the family's reaction to the illness as it swings into a protection mode. Arguably adding a unique psychosocial dimension to their illness manifestation.

Another developmental adjustment during the adolescent years that is associated with forming a new social identity is the increasing attachment to a peer group. However, identifying with a peer group has a two-fold effect for adolescents, 1) an opportunity to develop mastery skills outside of previous socially dependent relationships, and 2) the peer group in turn exerts its own ideals, expectations, and need for conformity on the developing individual (Peterson,

1989). Sarafino (1998) has observed that adolescents need to be liked, and to feel accepted by their peers, potentially leading to tensions both within the individual and between the individual and the group when the new attachment is poor, such as when the individual becomes unwell. While in many instances when a member of a peer group is perceived as aberrant this may be met with proactive support, for others this normative process can be disrupted, leading to problems of self-ness and relatedness for the individual, particularly when vulnerabilities, such as shyness or anxiety, are already present. The peer group can also be the place where the individual begins to develop intimate relationships, intimacy being the next 'conflict' in Erikson's stage model (Peterson, 1989). Therefore, a breakdown in adolescent social bonding can co-occur with a disability in the formation of adult relationships and behaviours that are part of the normative developmental process. Again, this may be particularly so for people attempting to cope with challenges to the integrity of their mental health. In support, Sarafino (1998) argues that young adults typically resent not having had the opportunity to develop their lives in the direction they planned - to get married, have children, or enter a particular career. This loss then can have a circular effect, producing problems in coping with their illness (Sarafino, 1998).

Related to these developmental issues is the influence of developmental stage on the types of symptoms present for specific disorders. For example, it is generally agreed that adolescents become depressed as adults do. However, irritability, agitation, and anxiety are common symptoms found in adolescents with low mood (Weiner, 1992). This developmentally specific presentation may be due to adolescent's life stage. Alloy, Kelly, Mineka & Clements (1990) present evidence for a pathway of psychopathological severity that progresses from anxiety, to a mixed anxiety – depression syndrome, and then to a more pure depression; in psychodynamic terms the progression is from uncertain helplessness, to certain helplessness, to hopelessness; in psychoanalytic terms the progression is from protest to despair. Alloy et al. argue that the presence of anxious or activated symptoms in adolescent depression is because their reactions to life experiences have typically not progressed to the chronically "hopeless" form of depression. Instead the mix of activation and deficit symptoms is reflective of depressed adolescent's vacillating certainty in their "helpless" cognitions. The same general rule applies for first-onset psychosis; the presentation is tempered by the idiosyncrasies of the individual's developmental stage.

An issue that is related to both adolescent psychopathology and first-onset psychosis is the lack of symptomological stability in this population. In general, around 40% of clients with first-onset psychosis have their diagnosis changed in the first three to four months, typically as a result of the high overlap of bipolar affective disorder and schizophrenia, the 'schizomanic patient', and the heterogeneous forms of psychosis (Joyce, 1984). This point can be made even without introducing the understated effect of personality on the presentation of severe mental disorders. Further, the labelling to mental illness can be a two edged sword; a diagnostic label can externalise the illness, giving the client and family an antagonist to confront, however, if the diagnosis elicits feelings of helplessness, shame, and subsequent stigmatisation, this can be harmful and degrading in the early stages of the illness (McCay, Ryan & Amey, 1996). Therefore, it appears that it may be beneficial to avoid applying diagnoses of schizophrenia, with its stereotypes of chronicity and unpredictability, and to use preliminary diagnoses of one of the psychotic disorders. For example, from one of the case histories presented in chapter 17 that is taken from Totara House, a first-onset psychosis intervention programme, the consultant psychiatrist clearly states "...it is very difficult to make a definitive diagnosis in this age group, and we do not like to make a diagnosis of schizophrenia because of the lack of stability of this diagnosis in this age group". However, at the two-year discharge, it may be that a diagnosis of schizophrenia is introduced to ensure that the client has time to adjust to the consequences of the emerging long-term patterns in their illness, and to their clinical management shifting to a community sector team.

Overall, it is highlighted that the challenges associated with adolescence and early adulthood are likely to interact with other illness-related processes, with the potential for significant distress and symptom variability as these clients attempt to adjust to the significant changes in functioning a life expectations. The range of issues that exist for these clients is further highlighted by shifts to comprehensive treatment programmes in an effort to treat existing psychopathology and prevent long-term risk. One such programme, Totara House, will be reviewed in the following chapter.

Chapter 16 Totara House: a case study in the intervention of first-onset psychosis

This chapter offers a case study exploration of an early intervention for psychosis program. The guiding paradigm is reviewed, particularly indicating its potential usefulness, over and above medication-only regimes, as a structure that can competently work psychological processes into case management plans. In particular, Totara House is used to illustrate how such services can apply the biopsychosocial model in a multi-disciplinary outpatient setting. Issues related to the adolescent – early adulthood age range serviced by this programme are also discussed in the context of providing a comprehensive, yet clinically sensitive intervention.

Totara House is a specialised unit in Christchurch, New Zealand, that is dedicated to the treatment and community management of first-onset psychosis. This chapter describes the types of interventions offered at Totara House, and the reasoning behind using the approaches outlined. Integral to this discussion is an understanding of the age range of the individuals seen at this service; how the individual's stage of psychosocial developmental impacts on illness presentation and management. However, the actual operationalisation of the biopsychosocial treatment of psychosis is not covered in this the present thesis. For psychological treatment strategies the reader is referred to Birchwood & Tarrier (1994), Haddock & Slade (1996), and Kingdon & Turkington (1994).

As stated the preceding chapters, the predominating disease model of schizophrenia has largely shown a disregard for the premorbid and post-acute psychosocial challenges faced by individual sufferers. One contemporary exception to this is the application of the biopsychosocial framework to the clinical management of first-onset psychosis. As discussed above, the biopsychosocial model proposes that while individuals who experience psychosis have an underlying physiological vulnerability to developing such a disease, the process is actualised and maintained via a range of psychosocial stressors. Therefore, the options for

treatment focus broaden depending on what the activating and maintaining factors are formulated as being for these cases. Recently, this model has been used to provide a guiding model for re-conceptualising and treating first-onset psychosis by treatment providers in handful of countries, including Finland (Integrated Treatment for Acute Psychosis – API), Australia (Early Psychosis Prevention and Intervention Centre - EPPIC) and New Zealand (Totara House). Initial data that is now coming out of research at EPPIC in Melbourne suggests that use of a more comprehensive treatment based in a specialised early intervention for psychosis unit successfully intervenes in the positive symptoms of psychosis, as well as the negative symptoms that are associated with chronicity (McGorry, Edwards, Mihapoulos, Harrigan & Jackson, 1996).

Totara House is a specialised outpatient unit that operates under the umbrella of Healthlink South services in Christchurch. It is comprised of a multi-disciplinary team, including psychiatrists, clinical psychologists, specialist nurses, an occupational therapist, social workers, and a researcher, and provides comprehensive clinical management to predominantly young people (16 years to 30 years) who are diagnosed as having psychosis. The intervention has a duration of two years from the time of acceptance into the programme. The goal of the Totara House program is to respond to the symptoms, events, loss of psychosocial functioning, and co-occurring morbidity associated with psychosis, thereby avoiding the poor outcome and treatment resistance that is associated with untreated psychotic episodes. Further, this goal is applied to individuals, rather than a diagnostic group, so that the treatment made available to individuals is tailored to their recovery style. Referrals are received from other Healthlink South units (emergency, acute inpatient, and community sector teams) as well as General Practitioners, tertiary institutions, high schools, and self-referrals. The education of professional outside the Totara House team is an important part of the Totara House service, particularly in making the service a known point of referral for appropriate candidates. Once a client has been accepted they receive intensive case management, psychiatric monitoring on minimal dose new generation medications to promote adherence, as well as access to a range of individual and group interventions, according to the client's presenting problems and situation. Groups that are typically made available to clients include:

- Focus Group - a cognitive behavioural group intervention that explores the nature of psychosis, vulnerability – stress interactions, post-psychotic trauma and anxiety, stigmatisation, identifying illness indicators, and relapse prevention.
- Drug and Alcohol Group – is an educational and motivational intervention that explores the impact of substance abuse on psychosis. 70% of Totara House clients present with drug and alcohol problems, making this a significant management issue.
- Anxiety Group – a cognitive behavioural intervention aimed at the management of anxiety, especially the frequent presentations of social anxiety and trauma.
- Family Group – is a psychoeducational group for the families of Totara House clients.
- Recreational and Life Skills Groups – Women's Group, Men's Group, Art Group, and Walking Group, provide personal outlet, and social skills and networking to clients.

Clients, and family members where needed, also have access to individual therapy sessions with a clinical psychologist and social workers, regular contact with the psychiatrist where medication regimes can be discussed and negotiated, and an open door policy with the service / case managers is promoted as an appropriate self-management strategy. Totara House also refers clients on to community agencies, and inpatient service, when appropriate, and case managers advocate for clients in a number of areas of social service.

The foundation on which the provision of early intervention services ride is the advent of community based care of non-acute / extreme mental illness. As previously discussed, the schizophrenia spectrum disorders carry a large cost to society and the individual sufferer. Contemporarily deinstitutionalisation has been found to reduce the monetary cost of the management of these individuals (Goldberg, 1991), albeit partially because the flow on of funding to community-based care has not been fully realised (Andrews, 1991). Goldberg notes that as well as the economic advantage of community based care, randomised controlled trials have indicated that acute psychosis can be treated cost effectively in the community. One of the keys to the success of this outpatient approach to first-onset psychosis has been the use of intensive case management. For example, at Totara House the case manager is assigned at the time a referral is accepted into the service, and that manager ideally works with the client throughout the course of treatment, providing a consistent point of contact. For the relationship between case manager and client to work most effectively the case manager needs to be open and aware of the full range of factors present in the clients life

that may be impacting on their functioning. This allows case managers to preempt exacerbations of the index illness, and suggest to and guide the client toward appropriate life-changes that will promote stability of thought and behaviour.

In summary, the Totara House program employs interventions aimed at both the need for the psychiatric control of psychotic and mood symptoms, and psychological input into a range of illness related factors (presenting and historical). Medication is used to treat active symptoms, and to provide a therapeutic window in which any co-occurring disorders and psychosocial issues can be addressed. Medication is utilised with an understanding of that clients are not impersonal recipients, and that adherence is enhanced when drugs work, education is available, and side effects are minimised. The intensive and comprehensive psychosocial management and interventions are flexible, and encompass a number of issues and secondary phenomena that may impact on the index illness. Further, the value of tailoring the treatment to the individual's recovery style enhances the face validity of the intervention, making it more acceptable and accessible to clients. For example, McGlashan (1987) has identified two styles of recovery, each with its own treatment needs, a) *integrating* clients can be encouraged to use insight into the meaning of the illness in their self-management, b) clients who *seal over* (maladaptive emotional – focused coping) can be encouraged to utilise behavioural skills to minimise stress.

Overall, the system of illness management that is emerging for the early intervention of psychosis needs to be dynamic and sensitive to the individual client's symptom 'picture', as well as their individual needs. Arguably, a biopsychosocial frame provides a good reference for this undertaking. However, in part 2 the argument has been made for more theoretical development to proceed for concepts of schizophrenia and factors related to this diagnosis. While the biopsychosocial frame represents a move in this direction, there is still considerable room in the vulnerability – stress model for further delineation of illness related processes and relationships, and the unpacking of potentially over-simplistic or unidirectional causal and maintaining factors. In relation to the present thesis, the association between negative symptoms and stress has been noted as being particularly significant in people who are experiencing their first psychotic episode (McGorry, 1995). This is a potentially crucial area of interest given the high level of anxiety found in the early symptoms of psychosis (Lader, 1994), and the developmentally harmful nature of socially debilitating events, such as social withdrawal, anhedonia, low mood, and cognitive disturbance, to name but a few (Strauss,

1989). In an attempt to descriptively illustrate some of these factors at a phenomenological level, the following chapter presents six case studies of first-onset psychosis that are hypothesised as including stress-related features.

Chapter 17 Case studies in first-onset psychosis: observing the hypothetical stress-illness relationships

In this chapter case histories are presented for six clients of the Totara House programme who had been referred for management of their first-onset psychosis. The cases presented here were not selected randomly from the Totara House client list, rather they were suggested for inclusion by case managers based on criteria provided by the author. In general, this criteria included either current or historical presence of anxiety features, such as trauma related to historical events or to the psychotic presentation, as well as a presence of negative symptoms, such as social or emotional withdrawal. One case also includes the presence of borderline intellectual functioning to highlight potential clinical implications for these complex cases. As such, it is not expected that the cases presented here are representative of the first-onset psychosis client base at Totara House. Rather, that they demonstrate a relationship between life events and symptom features associated with psychosis for clients who may have these symptom constellations, or reactive features in general, in their presentation. All clients who agreed to participate did so on a voluntary basis, and approval was gained from the Canterbury Ethics Committee. The names given used in the cases are pseudonyms, and a balance has been sought in presenting the data between detailing the client's history while ensuring their anonymity. Data presented in the case histories were gleaned solely from the client's Healthlink South file, and incorporates clinical notes from case managers, psychiatrists, psychologists, and specialist nurses, as well as assessment and treatment reports, and management reviews. This information has been presented in approximately chronological order, thereby offering at least an approximation of the longitudinal evolution of the client's mental health status. In particular, issues surrounding anxiety, trauma, stigma, shame, dissociation, and avoidance have been focused on. Each case is followed by a short summary of the potentially important aspects illustrated by it.

An argument may be made for more robust forms of gathering and utilising client information than relying solely on case notes, such as grounded analysis. However, the goal of including case histories was to illustrate some of the theoretical associations presented in the preceding chapter, rather than attempting to 'prove' a theory. That is not to say that case studies are unimportant tools of inquiry. Mental health workers interact with single case data on an hourly basis, and doing in clinical research promote the description and understanding of both the subjective and objective phenomenology of mental disorder, as proposed by Strauss's (1992) person – centred approach. Rating signs and symptoms of mental disorder, and searching for significant correlations may provide reliable data in the latter medium to latter stages of theory building – particularly for testing theoretical constructs. However, in the initial stages of developing a set of relationships, such as with validating the relationship between trauma and psychosis, this approach may miss too much valuable data. Therefore, using a data rich approach such as case studies, particularly for a fledgling relationship such as between negative symptoms and trauma, may prove valuable for uncovering tentative patterns in the events observed in and described by clients. From this perspective, the cases presented here are organised as descriptive explanatory models that are unique to each client's psychosocial and psychiatric history, in an attempt to powerfully illustrate the features and associations central to this chapter.

Case 1. Chris

Chris is a young Caucasian male who has generally presented as tidily dressed, verbally concrete, and avoidant of emotional issues. He has lived all his life both his parents, and older siblings. He continues to live with his parents. Chris's mother has described him as having had a 'happy' childhood, with no obvious stresses or trauma. While he was born into an apparently high achieving family, Chris experienced poor co-ordination during his childhood years, and academic problems emerged in his 5th form year, when he struggled to keep up with the work. Around this time he sustained a head injury after an assault, and while he was cleared following a scan, there may have been some changes to his personality and concentration as a result, including reduced motivation, passive – aggressive, and moodiness. He also stole numerous cans of Coke and received Police diversion, and these stressors contributed to a deterioration in his sleep and high anxiety. He left school at the end of the sixth form having gained School Certificate Art and Music, and has had part time jobs since this time, one lasting two years. He currently spends his time on the internet, playing guitar, or with friends, and he has become increasingly isolative in the home environment.

During a family trip away Chris experienced a change of functioning with symptoms equating to severe anxiety and panic, and psychotic phenomena, resulting in an acute admission to hospital on returning home. While away Chris became agitated and anxious, crying and inconsolable, voicing obsessional thoughts, persistently talking about organising his own funeral, and joined his parents in bed one evening saying "I love you", hugging them and

looking for safety. During the return car trip he appeared to suffer a prolonged panic attack, finding the confines of the car unbearable. Retrospectively, family members were able to describe that over the preceding months there had been a deterioration in mood and social behaviour (increasing isolation and reticence), unusual ideas and preoccupations, a degree of psychomotor retardation; but no overt oddities of behaviour. At admission Chris was described as extremely unwell, with extreme chaos, catatonia and facial grimacing, visual and auditory hallucinations, believed himself to be the devil, and ideas of reference. On the ward he was agitated, tearful, distressed, and repeatedly attempted to hug patients and staff inappropriately. He was hypervigilant to the point that "despite obvious tiredness, fighting the need to sleep for reasons known only to himself", as well as spending periods staring into space, possibly due to the presence of a stress related dissociative state. On his second night of admission the clinical notes indicate the high level of stress he was experiencing at that time:

> "When informed he would be staying the night, looked upset and asked "what have I done wrong?"....Chris remained scared, frightened of hospital seeking hugs and given reassurance that he was safe and everything would be alright.... Chris was able to settle and sleep intermittently if nurse held his hand....Chris remained driven, boundaryless, insecure, constantly seeking out nurse...."

After around 15 days as an inpatient Chris's symptoms began to settle, and he became embarrassed by the mention of his past hallucinations, stating that he can't really remember these, suggesting the emergence of avoidance strategies. However, after a period of around 25 days of inpatient care had elapsed, again Chris's mental state took a marked deterioration into "florid psychosis and anxiety, with thoughts being reported as more morbid and sick in nature....scared anxious and troubled by voices, which he does not want to discuss as this increases his anxiety". He was totally disorganised to the point he couldn't shower himself, and his attempts to leave the ward increased. At this time Chris was treated under a Compulsory Treatment Order. While he was indicated for seclusion at this time, he was so distressed by the thought of this he was nursed one to one on the ward.

At discharge Chris was diagnosed as First Psychotic Episode – most likely schizophrenia , and referred to Totara House for outpatient treatment. Review at Totara House indicates that Chris has experienced a rapid on-setting psychosis with a strong presence of co-occurring anxiety, and trauma reaction to his psychotic condition, in the context of no previous trauma or obvious psychosocial difficulties. The embarrassment and trauma he has for his acute inpatient admission have interfered with his attendance of Totara House psychosocial intervention group, as a way of avoiding memories of what happened. However, his stress response has been balanced by a very supportive family who visited him constantly while he was on he ward, as well as the lack of disruption to Chris's network of peers; his friends apparently accepting and understanding his hospitalisation. Alternatively, questions have been raised about Chris's lack of motivation being a component of his premorbid personality, particularly in the context of his previous head injury. Further, Chris was assessed as being in the Borderline range of intellectual ability following concerns about his concrete approach questions and identifying emotional issues. The Totara House psychologist notes:

> "It appears that Chris may struggle to effectively process complex abstract information and problem-solve in respect to daily issues, therefore it is likely that his psychotic experiences may have been particularly distressing. It is likely that the frightening and confusing nature of Chris's emerging psychotic experiences may have been exacerbated by his inability to effectively understand or organise this influx of

> information from several modalities....Chris may have accordingly felt overwhelmed and confused....Currently, it appears that Chris is managing the fears and confusion associated with his psychosis and hospitalisation through cognitive and emotional avoidance of these issues. While this approach may allow Chris to reassert some sense of personal control over these experiences in the short-term, it is unlikely to ameliorate the associated distress, confusion and his own sense of self-efficacy in the long-term."

Currently, the positive psychotic symptoms have abated, and there has been no other panic attacks, obsessive-compulsive behaviour, or prominent social anxiety. His mood has increased and he is sleeping well, he has more energy and has begun attending the gym, and is actively seeking employment.

Summary

This case illustrates a post-psychotic trauma reaction, complicated by personality and cognitive factors. Chris has a premorbid history that is uneventful apart from a head injury sustained during an assault. Around the same period in which the assault occurred there was a decline in functioning, including personality, motivation, and affect. Following this Chris became increasingly anxious and socially isolated after being caught stealing by police. From this a rapid emergence of florid and severe psychosis with catatonic features and morbid thoughts occurred, to which he responded with agitation, tearfulness, behavioural and cognitive disorganisation, and distress. He was scared and frightened of the hospital and became distressed and unable to rest when he could not leave. Hypervigilance was also present, as well as periods of staring into space, and subsequently he has used cognitive and emotional avoidance to manage his distress over his psychotic experience. This case appears to clearly point to a traumatic reaction to psychotic decompensation, accompanied by hypervigilant, dissociation, and behavioural avoidance and denial. However, the overlap between premorbid personality, brain injury, and concrete emotional strategies related to borderline intellect, may also be implicated in the amotivational features of Chris's presentation, particularly given the lack of chronic social avoidance that is commonly present. The lack of social withdrawal may offer some strength to the notion of positive and accepting social supports as offering protection against secondary morbidity, possibly via the deactivation of stigmatising and shaming self-appraisals. This case highlights the complex issues at work for clients with these features, particularly in trying to disentangle the cognitive dysfunction from the psychodynamic dysfunction, each with different management implications. Of note, the psychosis has not lead to any disruption in his familial and peer relationships, and he is currently goal-driven to gain employment.

Case 2. Belinda

Belinda is a young Caucasian female who generally presented as casual and tidily groomed, with minimal eye contact, often wearing sunglasses, and tearful. She lives at home with her father and stepmother, as well as siblings and stepsiblings. Her parents separated during her primary school years due to the disruption caused by her mother's schizophrenic illness, and from that time she been under the care of her father, which she found hard. She initially got on well with her stepmother, but not recently, and she only identifies with one of her siblings. The father and stepmother have been supportive of her mental health needs, although at times when she is unwell Belinda has been unreceptive to their input, and this has caused conflict within the family that Belinda has not coped with. Belinda's mental health problems began when she was between 14 and 15 years of age following the death of her brother by suicide. She returned to school the following year without any overt problems. However, at 17 years of age she began to feel increasingly low in mood, and missed half of her sixth form school year. Since this time things have become substantially worse for her, to the point where she only watches television or listens to the radio. She did hold a part time job for a period, but was unable to manage this and resigned. Her self-identity also took a dramatic shift with her rebelling against being a "goody-good" and now preferring to be "sad and angry" as opposed to being her previous "happy and pretty" self. From this time Belinda has not developed a social network of friends; she does not feel inclined to engage in intimate relationships as her past relationships have invariably been characterised by themes such as rejection and inconsistency, and which have impacted on her ability to trust others.

Following a period of not communicating with the family, isolating herself, and expressing suicidal thoughts, Belinda was assessed at home and referred on to Totara House when it became apparent that she was severely depressed and experiencing symptoms of psychosis. At the assessment she presented with increased tearfulness, irritability, hopelessness, a great loss of self-esteem and the ability to get enjoyment from life, and poor energy, memory, and concentration. Psychotic phenomena included auditory hallucinations, such as derogatory voices telling her "not to look good", perceptual experiences centred around the ongoing presence of her dead brother, and receiving messages from Alanis Morisette. Belinda was experiencing hearing tapping in the walls, noises, shadows that follow her, and "feeling" her dead brother's presence as evidence that he was still around. This was reinforced by her other brother having similar experiences of the dead brother's continued presence. Belinda feels that the dead brother has been around since his death, and that it is really cruel of him to appear so long after he died as she feels "spooked and confused" by the contact with him. As well as her depressive symptoms, she has ongoing anxiety, with episodes of panic symptoms, and describes herself as having always been an anxious person. She also experiences strong anger and agitation, possibly as an expression of activated depression, with urges to hit people if they appear to be staring at her intrusively, or in frustrating interpersonal situations. Belinda has described having a 100% belief in a "dark force", that she has the devil in her, and that this force strongly influences her life.

Belinda is fearful of ending up with a diagnosis of schizophrenia after having experienced some of the effects of her mother's illness. Belinda has only negative memories of her mother and she worries that she will end up being lonely and strange like her mother. She also has reported that her brother teases her about being "fucked in the head like their mother", and she "knows her father feels the same way". Belinda has felt that the only way to avoid becoming like her mother is by being in love, but she feels isolated because she is the only person she

knows who is not in love. She also feels singled out and abnormal due to a belief she has that she is physically unable to smile. She suffers a considerable degree of self-stigmatisation, and is personally worried about being crazy "all of the time", and that her thinking will never return to normal in the face of thought disorder. This self-stigmatisation may have precipitated her belief in a dark force, as such an omnipotent and external antagonist may be a more acceptable explanatory model of her illness, rather than a dispositional explanation. Belinda has reported a number of traumatic thoughts and experiences, as well as intrusive thoughts, and understands the benefit of therapeutically processing these. She uses a significant amount of avoidance when confronted with distressing personal material, such as closing down and becoming constricted in her affect, and leaving interviews prematurely. It may be that Belinda's own illness sets off traumatic memories of her mother's schizophrenia.

During much of her 18 months with Totara House, Belinda has remained reluctant to engage in Totara House groups, is anxious in social situations, and often wears dark glasses and is constricted in her affect. She has had continued problems with energy, motivation, concentration, and memory. Boredom and agitation have been persistent factors, at times precipitating interpersonal conflicts within the family. On returning to school she found she was unable to cope with the stresses and pressure, with an exacerbation of low and anxious affect, and sleeping a lot to get relief from the voices of demons. Belinda has been described as having an atypical presentation, both in terms of her psychosis and anxiety, and her presentations have observed to be very changeable and unpredictable over time. For example, apart from the decompensation observed at the time of her referral, the feeling that her dead brother is in the house is the only consistently observed evidence for psychotic experience, and appears in the absence of any thought form disturbances. Given her exposure to her mother's schizophrenic processes, the members of the father's first family may have developed a culture of magical thinking, particularly as both her and her brother experience the presence of the dead brother.

Currently, with intensive input from her case manager, mediating between Belinda and her father, getting her up and encouraging her to attend groups etc., Belinda has appeared much warmer and reactive, although thoughts of being 'undeserving and a bother" are surfacing. Her case manager has stated that Belinda responds very positively to "TLC" (tender loving care), and that family relationships are improving over time. She is increasingly socially active, and while fears of negative evaluation persist she is increasing her social circle and going out to new places. Her mood remains problematic along with her anxiety. Her belief in the dark force is being challenged as she develops a new explanatory model of her experiences with the Totara House psychologist, and she is increasing in her ability to control it in some situations. Her major concerns revolve around the similarity of her own experience to her mother's schizophrenia, and thoughts about her brothers death which she acknowledges as a major trigger to feeling unwell for herself. She does not receive messages from Alanis Morisette at present, and states that thinking about Alanis Morisette helps her when she is feeling suicidal as she admires her attitude of doing what she wants no matter what others think.

Summary

This case illustrates a number of non-specific psychosocial factors that can be related to cases of psychosis and trauma. Following her brother's death by suicidal means, Belinda's mood became increasingly low, her academic performance and motivation declined markedly, and

she engaged in little productive or social activity, to the point where she became totally isolated even from her family while still at home. Her self-identity also shifted to negative themes. This state predated her reporting of psychotic phenomena which included derogatory voices and experiences related to her dead brother. She felt spooked and confused by these, as well as being anxious, tearful, agitated. She also experienced anger that appears to be related to shame and self-stigmatisation. Intrusive and trauma laden thoughts have also been reported, and behavioural avoidance continues to be used when she is confronted with distressing personal material. This is in a background of loss from parental separation, and the psychodynamics of having a parent with schizophrenia. The presence of trauma in this case may be from a number of events: the child's witnessing of potentially bizarre behaviour from the mother, the brother's suicide and subsequent beliefs and perceptions related to the brother, Belinda's own psychosis, as well as the interaction of these events, particularly if appraisals of her own illness are triggering emotions or memories related to schizophrenia in her mother. Addressing the meaning of her experiences appears to be associated with improvement, with underlying themes of worthlessness emerging, while avoidance behaviour remains variable.

Case 3. Louise

Louise is a young female of mixed Pacific Island decent, who generally presented with a well-groomed casual appearance, and who engaged well. Louise has received minimal support from her family since becoming unwell, and is currently living in sheltered accommodation away from home. She has experienced severe chaos in her childhood and adolescent years, characterised by physical and emotional abuse by both parents, lack of nurturing, and situations where she wanted to protect her siblings but ended up feeling guilty when unable to do so for fear of her own safety. During her early years Louise witnessed her parent's violent relationship, and took on a parental role toward her younger siblings. At eight years of age Louise's mother remarried, again with the relationship being characterised by violence and abuse; one incident involved witnessing her mother and boyfriend rolling around on the floor half naked with her mother being beaten up. She picked up a shoe to protect her mother and was told that if she didn't get out she would be killed. Louise's mother was highly critical and physically abusive toward Louise, including highly confusing situations for Louise, such as her mother pulling her into a room and was about to beat her, then joking about it. Louise felt that it was at this time that problems with low mood emerged for her. At ten years of age Louise returned to live with her father, again experiencing chaos in the form of parental alcoholism and sexualised behaviour, as well as a loss of contact with her cultural identity. Louise described her father as talking about sex a lot, informing her when he was about to masturbate, and on occasion making her remain in the room while he had sex with women. From the age of 12 years she has felt that people were looking at her in a sexual way, has often had sex on her mind, and realised that her mother was weak and could not protect her. At 13 years of age Louise's mother returned for a number of months and the abuse and violence from her toward Louise resumed. At one stage Louise moved to a girl's home as she felt disgusted by her fathers continued sexualised behaviour toward her and her sister.

Louise's first contact with mental health services occurred when she was 15 years of age, at which time she had two inpatient admissions. At this time she was under a Temporary Care Agreement by CYPFS because of an abuse claim made against her father. Over a period of months, Louise's mood and social and academic functioning had been in decline; once happy and engaged in school she had become increasingly socially withdrawn and depressed. Immediately prior to admission she was described as being in a world of her own, and exhibiting totally uncharacteristic behaviours such as actively initiating sexual behaviour with boys. At admission she was diagnosed as Bipolar Affective Disorder with psychotic features; elevated mood, grandiosity, and oppositional behaviour. She also exhibited generalised anxiety with panic attacks, and was ashamed of her own sexual behaviour while unwell, this shame being considered as a precipitant factor of her low mood. Louise's mental health improved quickly, and at discharge she moved to her uncle and auntie's having refused to return home. Later in the same year, after a period in which she excelled at her new school, Louise's condition again began to deteriorate. At this time she began to report to her case manger that her uncle was attracted to her, having feelings of not being liked and being evil, and asking for her appearance to be repeatedly checked. Louise was documented as having 'paranoid, and erotomanic delusions, plus delusions of reference, with marked decompensation, in the presence of withdrawal'. Shortly after this, Louise was molested by her uncle, and shifted to supportive accommodation.

At age 17 years Louise's case management was taken over by Totara House, during which time she has adhered to her medication regime, joined Totara House treatment groups, and made contact with the Totara House psychologist. During this time Louise has detailed that when she feels significantly 'trapped' in situations where she perceives her sexual safety is at risk it has contributed to a deterioration in her mental health. This is further compounded by her hypervigilance to sexual cues (non-delusional) from males, and has resulted in two shortly spaced instances where she has voiced concerns over the inappropriate 'sexual' nature of the behaviour of male caregivers. These issues around safety and sexuality, and other psychodynamic issues around relationships, defensiveness against rejection, stigmatisation / shame, and worthlessness, have been explored. Louise has had occasional exacerbations of her symptoms, which have been linked to potential psychosocial stressors and indicate an ongoing psycho-biological vulnerability. However, in the past 12 months her mood and self-esteem have increased, she is brighter, more cheerful, and more active, has resumed school, and has explored intimate relationships with males safely. Reflecting on previous case notes, Louise's psychologist has written:

> "Unfortunately, these reports by Louise have, on some occasions, been interpreted and labelled by mental health professionals as signs of unwellness, and that such thoughts represent erotomanic delusions. It is therefore paramount that the interpretations of Louise's reports of sexual-related cues/issues take into account her reported history of sexual abuse, and her possible tendency to pick up on such cues either correctly, or incorrectly due to hypervigilance; this may not necessarily be indicative of delusions of a sexual nature, but possibly more represent the sequeale of sexual abuse"

Summary

This case illustrates the potential relevance of early traumatic interpersonal experiences in the presentation of psychosis. Louise has experienced severe chaos in her formative years, including the a lack of parental nurturing, parental separation, 'double whammy' (Hughes, Parkinson & Vargo, 1989) of direct and witnessed emotional and physical abuse, as well as familial sexual abuse, and on at least one occasion she was told she would be killed. Her response included low mood, disgust, a decline in social and academic functioning, becoming increasingly socially withdrawn, and at times being 'in a world of her own', symptoms that may be related to dissociative symptoms of numbing and avoidance. Louise also exhibited uncharacteristic behaviours including sexualised themes at times, possibly as expressions of a self-system that is deconstructing, and shame appears to be a subjective factor impacting on her mood states. This case could arguably be conceived of as a reactive psychosis marked by severe affective features as a result of a biopsychological vulnerability resulting from trauma experiences in childhood and adolescence, and lack of protective familial social support.

Case 4. Luke

Luke is a young Caucasian male who looks younger than his years, and who generally presented as tidily dressed, pleasant, rather intense, and at times with mood incongruent affect (giggling and smiling). Luke is currently living in sheltered accommodation, but has lived most of his life at home with both his parents. His family has a strong religious background, and Luke himself is a respected member of the church. He is an only child who has parents who are supportive of his mental health needs. From three years of age Luke has experienced significant disruption within the family due to both parents' frequent hospitalisations. From his seventh year the family lived on a "knife edge" due to the father's medical condition, which was accompanied by the father's "totally unpredictable" emotional reactions and anger. At these times, when Luke would try and rectify the problem he would be pushed away; the message he gave himself was "I'm not good enough". The role he and his mother took was one of "keeping the peace" and avoiding emotional expression for fear of upsetting the father and putting his health at risk. To this end, Luke's mother has been referred to as a "somewhat cold, problem-solving lady who certainly is not into emotionality" with Luke having learnt that one is meant to deal with problems in a logical and non-emotive way. A social worker's report from Luke's first admission reads:

> "Luke has had a family role of 'making other people feel OK', so there is little space for him to have needs of his own. There is a tendency for him to take responsibility for other's feelings (ie mum's anger). He needs to learn to tolerate pain / uncomfortable feelings, and to give self permission, including to go to tertiary institution and 'succeed' where parents 'failed'. On one occasion Luke left home following a family conflict in which his own anger dominated, at this time he attempted to harm himself with glass from his broken watch. Following this both Luke and his parents requested that he be admitted, and were unable to accept that the issue was 'psychodynamic' with

> Luke's own rigid attitudes acting to wall off his pain, and his parents difficulty in accepting anger as an emotional expression."

More recently, a report from Luke's Totara House psychologist reads:

> "Luke's need to avoid or insulate himself from emotional fluctuations sees him conceptualise and mislabel normal mood fluctuations in a psychiatric manner – with an expectation that this fully accounts for change in his mood. This enables him to avoid examining psychological precipitants possibly associated with those normal mood fluctuations. This mislabelling of normal mood fluctuations is also associated with fear and stress of him becoming manic or clinically depressed and being unable to cope; this fear and stress increases the risk of the emergence of a psychiatric mood state.... This tendency thwarts his early utilisation of adaptive coping strategies."

Luke's early years were characterised also by many moves to different cities, which he coped with by isolating himself and tending not to make friends. At the age of 10 years the family settled in Christchurch, however, following this Luke only formed one close friendship, and that person moved away. He was also bullied and teased at high school, which impacted on him greatly as it came from within his peer group. Luke's first contact with mental health services followed a period of depression when he was 15 years of age, during which he attempted to kill himself by hanging and falling off his bike in traffic. At this time his constant negative self-appraisals had become so severe "he was almost hallucinating". He was also not coping with family arguments, and his mother's threats to leave, and ran away. Luke was diagnosed as having Bipolar Affective Disorder – II, and was treated successfully as an outpatient. He went on to finish school close to top of his class, reflecting his self-imposed high academic expectations, as well as his use of hard work as a coping mechanism to block out what was happening around him. Luke then went on to tertiary education, but did not achieve to the level he expected. At this time he again became depressed, and took and overdose of anti-depressants in an attempt to kill himself. Again he was treated as an outpatient, followed by several years symptom and medication free, during which time he was employed, and trained to be a missionary. During his first missionary trip as a health worker in Bangladesh he became physically and mentally unwell within two weeks of arriving. He was admitted to hospital there with manic symptoms including irritability, agitated behaviour and "incongruent and irrelevant thoughts and behaviour, sleeplessness, paranoid ideas and also violent acts on other persons prompting necessity to tie him up on one occasion". At the time he admitted he was on a "high", and believed that he had special powers, including that he was Jesus Christ. In his mission work he had begun to act in culturally insensitive ways putting himself in physical danger. Immediately following his return to New Zealand Luke was admitted to hospital at which time he was extremely psychotic, exhibiting religious and paranoid delusions. He was aggressive and agitated with staff initially, particularly when he was blocked from doing something, and he needed to be sedated on a number of occasions. He experienced auditory hallucinations, such as being told to run in front of cars, as well as other perceptual disturbances.

At discharge Luke was accepted into the Totara House. At this time there were no overt symptoms of psychosis. However, Luke was at times experiences "racy thoughts", an overwhelming desire to remove his clothes in public, or to stop cars with his superman powers, both of which he found funny at the time, but scary in hindsight. He also had intermittent thoughts of walking into traffic, attacking others with a knife, and sexual thoughts about children (in the absence of arousal), all of which he found distressing. Luke was encouraged to become more independent, and he moved from home to supported

accommodation. He began attending Totara House group interventions, and engaged well in these. While preparing a narrative of his illness experience he experienced "flashback" experiences of events around his psychosis, and felt like he had "got it off his chest" after finishing. He also has had flashback experiences at other times, of the events in Bangladesh that he is embarrassed about.

Within eight weeks of discharge Luke was again admitted to hospital with elevated mood, agitation, and thoughts of running naked in front of cars. One instance has seen him go to a local school and remove his clothing, then attempting to use his "super powers" to stop cars, before becoming scared by his own actions and returning home. At presentation he denied having any psychotic phenomena, but his mood was at times incongruent with the conversation. He was described as "very different presentation from the usual manic patient, in that he was very insightful and fearful of what he may do due to his irritability and elevation in mood." During this admission Luke had to be restrained and secluded or sedated on a number of occasions for his own safety due to his repeated absconding from the ward. This behaviour was driven by impulses to kill himself by running in front of cars. Luke's subjective experience of this is as follows:

> "When I feel impulsive I just want to run, breakout and kill myself by running in front of cars on the motorway. It doesn't matter if the doors are locked the impulses are so strong and I don't know where they are coming from. I don't want these thoughts or impulses and I don't know how to control them."

Within six weeks of discharge Luke was again required an admission with similar problems to the previous admission. It appears that his mental health began to deteriorate while on home leave due to the family not coping with his intermittent mood fluctuations. At this admission a pattern was noticed whereby an elevation in Luke's mood occurred during the weekend home leaves, with questions regarding the influence of family dynamics on his mood states. Again he was discharged to Totara House.

Currently Luke has ongoing sleep disturbance, lack of motivation, and realistic concerns for his future. He lacks skills for labelling, experiencing, and expressing feelings/emotions. He continues to have a long standing tendency to feel that he is not an active participant in his life, that events simply happen to him, that he does not feel connected with people or events, and has difficulty finding meaning in the changes to his mood, assuming they are solely a response to chemical changes. This is compounded by episodes of feeling intensely "irritated, angry, drained, and uptight", often without an awareness for a precipitant, suggesting the presence of high stress, anxiety, and activated low mood. He has subjective feelings of closing down emotionally into an 'emotional limbo', especially around fears of becoming high again, and has a sense of disappointment and sadness at avoiding things that he feels may make him go high again. His emerging personality profile suggests the presence of schizotypal and possible borderline traits, along with clinical elements of anxiety, dysthymia, and post traumatic stress. He has very little social contact with his peers, and limited self-confidence, however he has made the transition from his parents home to supported accommodation, and has been able to set some long term goals in the face of realistic fears of further failure to meet his expectations.

Summary

This case illustrates an example of post-psychotic trauma response within a context of potentially stressful distal personality and family dynamics, and poor emotional regulation skills. In his formative years Luke has experienced unpredictable violent behaviour and emotional denial in his parents, bullying at school, and personal negative cognitions centred on themes of failure, and perfectionistic achievement. There was also a background of affective disturbance and self-harm attempts, including one uncompleted suicide attempt. Luke's initial psychotic episode included physical and mental dysfunction in a foreign country, and his subjective distress resulted in his being tied up. On returning to New Zealand he was extremely psychotic, and his inpatient treatment included several aggressive periods that resulted in sedation. He also experienced command hallucinations centred on killing himself, and impulses to commit bizarre and uncharacteristic behaviours, both of which he was unable to control at times, again resulting in sedation, or seclusion. Luke found these experiences distressing and has experienced intrusive phenomena related to his psychosis. He also has ongoing sleep disturbance, lack of motivation, low mood, and the subjective feeling of being in a state of 'emotional limbo' – particularly in response to fears about becoming acutely unwell again. He is socially isolated, has limited self-confidence, and problems processing personally emotional material. The above phenomena may be related to trauma responses characterised by a stress-related vulnerability, intrusion, and hyperarousal, psychic numbing, and behavioural avoidance. It may be that Luke has learnt to use 'sealing over' as a mechanism for regulating overwhelming affective and meaning components related to experiences and memories from his childhood and the psychosis. However, this strategy appears to be maladaptive for Luke, in that his negative symptomology is chronic, and environmental stress is managed poorly, for example, resulting in mood elevations during home visits.

Case 5. Sandra

Sandra is a young female of European and Asian decent who looks younger than her years, and who generally presented as casually dressed, well groomed, co-operative, but anxious and with poor concentration. Sandra is currently living at home with her mother, mother's partner, and her older brother. Her parents separated when she was four years of age, and she still has regular contact with her father. Her family has been supportive of her mental health needs, and her mother has been actively involved in her management. Sandra's early years saw her witness her father's physical abuse toward her mother, and she is unsure whether he was violent toward here. However, on one occasion the father threatened to leave her and her brother on the side of the road if they misbehaved. On visits to her father, he was often angry

and verbally abusive toward her, telling her she was stupid, and using other derogatory terms. In her later childhood she was physically abused by her brother, with this abuse only stopping when her bruises were noticed when she started High school. Sandra's mother has been described as overprotective and over-involved, and excessively worried, irritable, and quite strict. At school, Sandra had few friends, missed many days with complaints of stomach pains due to her dislike of school, anxiety around other children, and fear of harm coming to her mother, and achieved at a low level.

At the end of her sixth form year she became unwell after a period of six months of low mood. Prior to the school ball in June of that year Sandra became convinced her friends were talking about her, and spreading rumours that she was a "slut and a lesbian", apparently precipitated by her distress over not having a partner for the ball. One month prior to her admission at the end of the year severe auditory hallucinations started, and resulted in her becoming uncharacteristically agitated, aggressive, and yelling and swearing at the voices. The voices were derogatory, telling her she was a "whore, molesterer, and lesbian", as well as commanding, and at times telling her to harm herself by burning herself with hot water or over-dosing on medication, and threatening to do this to her themselves. She believed that compliance with these demands would have killed her. Sandra also became more irritable with family members, and more withdrawn and socially isolated. Retrospectively, the Totara House psychologist commented on the development of Sandra's psychosis:

> "...the formation of an insecure attachment style and a working model of interpersonal relationships as volatile and constituting potential threats to physical and psychological safety, as well as a perception of self as inadequate....At a time in development when individuation and formation of identity through association with peers and establishing romantic relationships are major tasks, Sandra's difficulties in forming close relationships may have become a more prominent concern for her, particularly after Sandra realised she would not have a boyfriend to attend the prom with as she wished....Perhaps as a means of preserving her self-esteem she appears to have projected her self-reproach onto her friends, becoming convinced that they were criticising her and making people reject her."

Sandra was admitted with low mood, psychotic symptoms, and paranoia, as well as insight and considerable distress by her condition. She was very scared and bewildered by her situation, and described occasions when she became so frightened of the voices that she could not get out of bed. Sandra described feeling powerless to change or stop the voices, she felt worried that the voices might never end, and that she felt sad at the loss of her quality of life. On her first home leave she felt paranoid and the voices became louder, and she was confused, and returned to hospital where she felt much safer. At this time Sandra's stepfather asked the pertinent question of "how long can she cope with these voices without a deterioration in her mood and thoughts of self-harm?" However, her symptoms subsided over a period of one month, and she was discharged with a referral to Totara House. She has since had one other admission, due to a combination of neuroleptic induced side effects (extra-pyramidal and rashes), persistent derogatory voices and chirping, and anxiety with a possible panic attack (feeling her throat closing over).

Sandra's assessment at Totara House indicated that she has a psychotic disorder which is only partially responsive to antipsychotic medications, and that she also has a physical fragility to most psychotropic medications which has impacted on the management of her symptoms. She was also noted to have an underlying generalised anxiety which is exacerbated by a deterioration in her mental health, leaving her vulnerable to depression. The stress of high

anxiety and low mood, coupled with a poor self-efficacy, then appears to further exacerbate her psychotic symptoms. Her vulnerability to stress was considered to be related to rejection fears related to her early abuse history, dependence on her mother, minimal coping skills and stress tolerance, and withdrawal – avoidance, and or aggressive, when a crisis develops. In conjunction with the Totara House psychologist, Sandra's treatment included debriefing memories related to her experience of psychosis, and she was able to gain insight into the nature of her internal dialogue, which revolved around feeling angry with herself and a strong desire for more autonomy and self-directedness. Near the end of the two year intervention period at Totara House, Sandra's psychotic symptoms were the best they had been – ongoing, almost consistent low-grade 'murmurs' in the background. She has learnt to cope with these symptoms by using self-supporting skills such as using positive thoughts plus distraction. She has gradually felt more powerful over the voices, particularly when she can laugh at the content of the voices, and resist acting on their commands. Sandra was able to identify that when she resists what the voices are saying she feels powerful, and when she gives in to them, or feels unable to stop them, she feels very sad and powerless. She no longer avoids social situations, but anxiety symptoms remain mildly uncomfortable and she endures them with discomfort. However, she appears to engage in more subtle avoidance, such as decreased eye contact and not talking. Currently, she has returned to school for part time study, and her social network is increasing. She continues to be managed by a community outpatient team, and is maintained on medication regime for her residual symptoms. However, given her psychological response to her experience of psychosis, the Totara House psychologist has stated:

> "Clinical psychology contact should be made available for Sandra on an occasional basis while she remains well, but if her mental health deteriorates, then this contact should increase to weekly. This contact can assist in safety monitoring, plus help empower Sandra to continue to utilise the adaptive cognitive behavioural coping strategies she has adopted."

Summary

This case illustrates the co-occurrence of trauma factors as a consequence of historical life-events and psychosis. Sandra's formative years include her parents separating subsequent to a period of physical abuse, which was witnessed by Sandra. Since then she has received verbal and emotional abuse from her father and brother. She has subsequently experienced significant and generalised anxiety, and interpersonal insecurity reflected in expectations for relationships to be volatile and constituting a personal threat to safety. These insecurities appear to have triggered a decompensated state following an interpersonal stressor, resulting in the emergence of derogatory and persecutory voices that she found difficult to resist, agitation, and aggressive behaviour – typically directed at the voices and family members. Her subjective response included significant distress, with her becoming so frightened it interfered with her ability to function, and she felt powerless and a sense of loss. She became socially withdrawn and isolated, including subtle avoidance when in interpersonal situations. Sandra's experience of powerlessness appears to be directly related to the amount of control

or resistance she has over the voices. Debriefing memories related to psyhosis have helped her gain insight into her own response to being ill and losing autonomy and self-directedness, and behavioural avoidance is declining. However, Sandra's illness is chronic requiring ongoing medical and psychological management, possibly due to elements of residual psychotic dysfunction, a return to premorbid personality features, or ongoing anxiety.

Case 6. Michael

Michael is a young Caucasian male who generally presented as casually dressed, typically with dysphoric mood, yet pleasant and co-operative. He is currently living at home with both his parents, and siblings. Michael has supportive parents who have accommodated his change of functioning into the family system, while actively challenging disruptive behaviours. Michael is described as having never been an affectionate child, was not very communicative, and showed few signs of emotional reactivity as he grew. He did well at school and had a wide circle of friends until changing school at 11 years of age. At the age of 12 or 13 Michael became involved in an antisocial peer group, getting in trouble with police, taking drugs, and satanic worship. Following a fight at school when he was 14 years of age, Michael became extremely distressed and began to believe that other pupils were also plotting to get him. Approximately six months later he was at a party and after drinking heavily and smoking cannabis, he became suddenly convinced that he had died and gone to hell, and that other people were demons and were coming to get him. At this time Michael began to sit in his bedroom armed with a knife to protect himself. Michael also began having recurring dreams of confrontations with other students, which he found distressing and impacted on his motivation to get out of bed. A voice appeared and started saying that people were after him and telling him that he would be beaten or tortured. The voice, which identifies itself as the Devil, frequently makes derogatory comments such as "you're ugly" or issues commands telling him to kill himself. Songs also began to hold messages for him, usually with a morbid theme such as "kill yourself and those you love". He developed the belief that he was in hell, that the people around him were demons, and that he was being punished for trying to give up Satan worship. Subsequent to this he felt there was no one he could trust and he began to avoid all social contact, including leaving school. He has had no social contact outside the home since this time, although he has a developing social network via the Totara House client base.

Michael was referred on to Totara House when he was 17 years of age, once it became evident that his psychotic symptoms were not remitting following the successful outpatient treatment of his substance abuse. At intake he was given a preliminary diagnosis of Major Depressive Disorder with psychotic features; Michael has a long standing history of low mood and poor self esteem that covers most of his formative years, and which has intensified in the last two years following his experience of first-onset psychosis and subsequent anxiety and hopelessness. This has left him feeling "slowed up" all the time, and that he has trouble "feeling" things, that he has to work hard to feel anything at all. Michael's symptomology includes extreme negativity, low mood, auditory hallucinations, low energy, withdrawal, agitation, anger, and pervasive feelings of wanting to die. Michael also has severe anxiety that is described as bordering on paranoia, and which drives active avoidance behaviour, particularly in social situations. This anxiety does not appear to be related to overt fears of negative evaluation, instead related directly to his persecutory fears, and in the past has

resulted in marked panic attacks. In relation to this, the Totara House psychiatrist noted in a report that:

> "We are finding very much that the boundary between anxiety and paranoia is very much blurred, and a number of our clients' paranoia has responded very well to cognitive behavioural techniques."

During Michael's contact with Totara House it has been observed that his state of mental health is contingent on the amount of stress he is experiencing subjectively. For example, when stressed Michael can acknowledge a sequence of events including becoming sad, irritated, angry, and suicidal, compounded by the voices returning and telling him to harm himself. This sequence occurs most easily when his energy and motivation are low. A component of this stress is the shame he has reported for having a mental illness. Michael is sensitised to others laughing around him, and he becomes agitated by thoughts that they are laughing at him, and wants to act out, and when others compliment him he experiences a voice that tells him to say "fuck off". While Michael knows that psychosis is an illness, he believes that the illness is due to his involvement with drugs and with the devil, and that it is fate and he deserves it, and that this has made him fearful of the unpredictability and uncontrollability of the illness. In one instance, he reported a frightening dream following which he awoke believing he was having a 'relapse' or psychosis. This combination of his ongoing fear, and the belief that its causes are the product of supposedly omnipotent supernatural forces appear to have led to a degree of hopelessness with regard to his future. Further, Michael has experience post trauma symptoms such as flashbacks, avoidance, startle response, and hypervigilance, and these have been noted as decreasing over the time he has spent in treatment.

Currently Michael is still experiencing low mood, energy and motivational problems, difficulties with concentration, and anxiety symptoms. However, his self-confidence has increased, and he has been able to engage in social events and intervention groups at Totara House. He has started developing a social network outside of his family, and is planning on returning to part time study. He is now able to manage the level of stress in his life, and subsequently the voice has been less prominent. Further, Michael has been able to work with the Totara House psychologist to explore and challenge the meaning of his beliefs about the devil, and being persecuted by others, to the point that they have a less limiting impact on his life.

Summary

This case can be conceived of as illustrating a presentation of post-psychotic trauma response. Michael's psychotic symptoms appeared at a time of distress related to being harmed by other people, and being in hell, and was subsequently contributed to by the appearance of a derogatory and persecutory voice. This is in the context of a background of mood problems and poor self-esteem which may have impacted on the developing personality structure, and style of emotional coping. Michael responded to the psychosis with mistrust, shame, stigma, social avoidance, poor concentration, hopelessness, and a cluster of 'amotivational' symptoms. Coupled with this is severe anxiety directly related to his persecutory fears, his

level of subjective distress cyclically impacting on his mental health status. He has also experienced an intrusive dream and flashbacks, the content reflecting psychotic material. In particular, the anxiety and mood problems have remained, with social avoidance attenuating over time, and successful management of stress appears to be associated with increased well-being, and less psychotic phenomena.

Overall, these cases descriptively indicate that at least some negative symptoms, and probably other positive and non-specific symptoms as indicated by the literature, found to occur in cases of first-onset psychosis may be conceptually reframed as trauma related dysfunctions. Many of the process and associations already discussed in the part 6 were noted to exist both within each case, and were well represented across all six cases. However, given the limitations in number and selection criteria of this sample, generalisations to other cases of first-onset psychosis may be difficult to make. Arguably, the cases do demonstrate that reactive processes related to extreme stress, across the life course, specifically in adolescence, and at the time of the acute psychotic episode, may have considerable explanatory power for some cases, the implications of which will be discussed in part 8.

Part Eight

Discussion

Chapter 18 General discussion

The discussion in this part of the thesis follows three main areas. Firstly a general discussion is made of the disadvantages and potential advantages of the arguments made in the thesis. In chapter 19 clinical implications are discussed, with an emphasis on the treatment of subjectively distressing components of psychosis. Finally in chapter 20 the implications for theoretical development in the psychoses are explored.

While the causal and phenomenological associations presented in this thesis appear to have some degree of face validity, a number of potential difficulties exist. Firstly, the overlapping processes presented are based on descriptions of correlations. Therefore, it is difficult to determine if the conceptual similarities actually reflect 'real' similarities, or whether the co-occurrence of factors would remain consistent, or theoretically plausible, over the course of the illness. Further, when extracted from the literature these correlations themselves may be conceptually flawed due to the non-specificity of many deficit symptoms and the use of cross-sectional research methods. This suggests the use of longitudinal designs to validate the descriptive data. Secondly, as the propositions described here have been proposed for negative symptoms that are secondary and reactive to life events, the theory may not hold for other cases of psychosis or negative symptoms, and therefore individual symptom associations are likely to be low in generalisability. This is highlighted by the cases presented in chapter 16, which represent only a small proportion of the clients managed at Totara House. However, if this theoretical exploration was extended to include a range of protective factors, rather than solely risk factors, then even more variance may be accounted for. Thirdly, the difficulty in ranking symptoms dimensionally to allow for multi-causality and non-specificity, while retaining standardised diagnoses that facilitate research and treatment, is highlighted by the type of associational re-conceptualisation of severe psychopathology presented here. Fourthly, the present thesis has been biased toward seeking out and describing theoretical associations that indicate commonality between distress and negative symptoms, as it is expected that this approach may indicate where the explanatory gaps are while promoting interest in the arguments presented. However, for a meta-theory to remain valid for the greatest number and range of clients, it would be important to also include differences and points of divergence that represent the rich variety of functioning in severe disorder.

In their article on theory construction Ward & Hudson (1998) make the observation that theory development does not just occur, but that it is a response to an existing knowledge system which is found to lack adequate explanatory power. That is, the multiplicity of the observed phenomena are not fully understood or represented, suggesting the need for new or re-conceptualisations of relationships between events to be generated. Ward & Hudson propose that in the initial stages of theory development researchers ask themselves two questions (a) "Where are the explanatory gaps?", and (b) "What level of theory construction would be most helpful in advancing understanding at this point?" (p.61). The present thesis has attempted to respond to both of these questions in relation to theoretically describing psychosis as a complex psychopathological process that includes both trait (biopsychosocial vulnerability) and state (interactive secondary / reactive processes) factors. In particular, two main research areas were explored. Firstly, a description was made of a number of potential problems with existing theories, implicit assumptions regarding the psychosis as a disease 'entity', and the DSM system's reliance on clusters of symptoms for diagnosis – many of which are low in specificity. Secondly, the associations between negative symptoms and stress were explored, with an emphasis on similarities at the level of phenomenology and illness-person interactions.

Overall, a number of arguments taken from the above focus of enquiry have been potentially realised at the theoretical level and at the level of descriptive association. In terms of the arguments related to the underdevelopment of current models of psychosis and schizophrenia, the potential disadvantages of following the DSM model for research guidance were highlighted. In particular, it was suggested that the DSM may be overly committed to a weak biological model of schizophrenia, and therefore limited in its ability to provide an unbiased and multi-factorial framework for complex disorders.

While it is acknowledged that categorising mental disorder using the DSM system has many advantages, such as communication and standardisation, the use of syndromal clusters as representations of disease 'entities' has largely failed to account for the huge variability and overlap of psychiatric symptomology. It has been argued that proponents of the DSM system have retained, and proliferated, the current categorical disorders as their 'reliability' provide an air of validity to psychiatric 'diseases entities'. Which in turn justifies the domination of psychiatry in the management of mental illness, while excluding alternative

conceptualisations of disorders. Therefore, calls to represent the dimensional qualities that exist between disorders via shared symptoms remain unheeded. For example, Moskowitz (1999) has argued that prior to the publication of the DSM-IV (American Psychiatric Association, 1995), research studies described a significant overlap between schizophrenic disorder and severe affective disorder, suggesting that these disorders may represent a dimensional spectrum of severe illness. Similarly, the present thesis, in looking at PTSD, psychosis, and affective disorder, highlighted the extensive co-occurrence of a number of features from these 'separate' disease categories, and it was suggested that the commonality between them was due to process related to stress responses to environmental and internal events.

From the above issues a number of theoretical arguments were extrapolated. In particular, it was argued that the popular assumption of biological supremacy in the etiology of the psychoses is a theoretically untenable position. Not because biological factors fail to be implicated, but because a number of psychological processes are equally valid and deserving of research attention. The alternative approach promoted is the re-conceptualisation of the psychoses as dysfunctional states generated within complex systems, and being mediated by multiple interactive factors, distal and proximal. Support for this assertion comes from a range of theoretical sources. Firstly, Kalmar & Sternberg (1988) and Ward & Hudson (1998) have both reported on the proliferation of theories that may have important conceptual overlaps, but which fail to utilise each other in the building of meta-theories with greater explanatory power. For example, many theories exist for each of the DSM disorders, yet few attempts have been made to coherently describe the associations between them, and associations which may exist can be hidden by descriptions of symptoms which are unique to specific diagnoses. The alternative approach suggested by these authors is emphasising the value of theory development as a research endeavour, and integrating existing theories into more encompassing frameworks of psychopathology.

Secondly, it was found that even in disorders of severe psychopathology, the person's subjective states are important factors to be considered. In particular, Strauss (1989) has argued that people have a relationship with their disorder that influences course and outcome. This suggests that the self is an important determinant in expression of mental illness that has been lost in some psychiatric accounts of disorder. While existing models describe various biological and psychosocial interactions in disorder processes, such as loss of functioning and

illness-environment interactions, few models include the person as a dynamic factor in the milieu of mental disturbance, potentially negating a rich source of data. This point is highlighted by the finding that people's illness attributions impact on adaptive responses, and long-term outcome (Sarafino, 1998). Also, developmental stage has been presented as having particular impact at critical points in the person's life course, with adolescence being a time in which both self-identities and social roles are influenced by current events. Thus, a wide range of phenomenological factors need to be assessed as integral components of a complex system, and taking a symptom-based approach may be one method of facilitating this conceptual shift in theory development.

Overall these theoretical arguments are taken as tentative support for developing comprehensive meta-theories that act as explanatory models for the full gamut of illness initiating and maintaining factors, as well as descriptions of the illness process, the components of which themselves will feed back into the 'system' via changes to brain functioning, social skills, and the person's own self-system. As put by Estroff (1989) "we proceed ethnographically and inductively to learn the concepts, meanings, and experiences of our expert informants. At that point our tasks as researchers are to seek associations, causal links, patterns, and implications" (p.195). In the present thesis the association between stress-response processes and psychosis generally, and negative symptoms specifically, was presented as evidence for re-conceptualising some psychotic symptoms, and thus providing new information from which to propose more general or meta-processes in severe psychopathology. The importance of including biological and psychosocial factors was supported research that indicates that psychological processes influences long-term brain functioning, and can potentiate an illness vulnerability (Widiger & Sankis, 2000).

The negative symptoms found in psychosis appears to be one area within the field of psychopathology which offers a clinical picture characterised by multi-causal and multi-factorial presentations. Further, the negative symptom syndrome has clinical importance in that they are associated with the early stages of psychotic illness, and severity of this syndrome tends to predict long-term outcome. The discussion of associative processes between stress and negative symptoms highlighted a number of relevant general processes which may be 'lost' when the client's presentation is fitted to the available diagnostic categories. In particular, given the lack of heterogeneity and lack of validity and specificity for the negative symptom syndrome, as well as the phasic nature of psychotic symptomology,

four semi-independent causal pathways were described; 1) an expression of the index psychotic disorder; 2) expressions of other biological or psychosocial processes that predate the psychosis or emerge in parallel to the psychosis; 3) a response syndrome (passive or active) secondary to the primary psychosis; 4) a result of changes within the brain as a result of psychotic brain processes, medication, and / or changes to environmental contingencies.

The implications for representing these pathways in a comprehensive model are that in some cases negative symptoms may be better conceptualised as secondary morbidity, either to the psychosis or other life events that are capable of eliciting changes in complex systems, and that these reactions can have psychological, social and physiological analogues resulting from distal and proximal events. Further, while existing models of psychosis include biopsychosocial factors, including the presence of stress as a precipitant to psychosis, it was argued that they are overly simplistic and constrained by the 'disease' hypothesis of psychosis. In the present thesis, the exploration of the overlap between secondary negative features and stress-related processes, such as PTSD, resulted in the description of a number of symptoms and processes associated with deficit phenomena that are not entirely specific to the psychoses. Firstly, DSM-IV diagnostic criteria for negative or avoidance symptoms, as well as associated features, for PTSD and schizophrenia were found to have a high degree of descriptive overlap. While this descriptive correlation does not provide evidence of causal similarity, it is taken as suggestive of similar processes in both PTSD and schizophrenia, and therefore indicating the potential for further theoretical exploration.

Two single-factor theories were highlighted as potential candidate processes. Firstly, the proposed post-psychotic trauma syndrome appears to offer have significant explanatory potential for subjective distress responses to psychotic phenomena and treatment contingencies, and has significant potential in guiding components of prevention and treatment programmes for severe mental disorder. Further, it highlights the need to put the person before the diagnosis so that symptomology related to distress is not lumped *ad hoc* into an overarching diagnostic category that 'hides' therapeutically critical information. Secondly, negative symptoms were conceptualised as primitive self-regulatory mechanisms which serve an adaptive function for the client. Similarities between the sealing off of affect found in schizophrenia, and deficit components of the dissociative 'syndrome' were highlighted. Both appear to represent a process in which the person escapes distress by

altering an internal emotional or cognitive organisation, the outcome of which includes behavioural expressions of avoidance, such as flat affect.

Other common processes that are less specific include a constriction of roles – self and social, the taking on of a sick role, subjective terror, shame, stigma, depression, lack of insight as a result of anomalies in encoding memories of the distressing events, helplessness and loss of self-esteem, and the potential for impulsive or bizarre behaviour. These factors have been considered to interact with the person's developmental stage, and potentially with their micro-culture and wider culture, resulting in symptom expressions that are relevant to the distressing events, the person's expectations, and the socio-cultural context. Further, perhaps the full subjective and psychiatric impact of the above factors is only realised when they are posited as accumulative events, rather than singular, resulting in a cascading into dysfunction and risk for relapse.

Overall, these findings have been taken to suggest that a correlational relationship between traumatic events, subjective distress, and negative symptoms may exist for a number of clients. Extending this theoretical exploration to potential causal associations it can be argued that stress related processes could be implicated in the phasic nature of negative symptomology in the psychoses. The timing of negative symptoms with the prodromal and post-acute phases may be conceptually relevant to stress-person interactions. Given the high co-occurrence of abuse histories and life events preceding acute decompensation, the psychotic prodrome may reflect an increasing exacerbation of cumulative adversity in the client's life, with negative symptoms being contingent on the subjective distress being experienced. Similarly, negative symptomology in the acute and / or post-acute period may reflect a trauma reaction or cognitive deconstruction in response to 'threats to the self' experienced during the florid phase, both from the illness itself as well as treatment contingencies.

Following from the evidence highlighting brain changes that result in increased sensitivity to stress via HPA axis anomalies, the long-term consequences of these distressing experiences and losses of functioning has been proposed as an enduring vulnerability to psychotic decompensation, both prior to and following the initial episode. These long-term changes would provide longitudinal pathways to mental disturbance, and would explain how events from the past could interact with present environmental contingencies to produce alterations

in mental states. Also, it would appear to demonstrate one potential pathway to negative symptoms (avoidance) that are chronically maladaptive, and difficult to manage. Further still, based on the assumption that personality, like many mental disorders, reflects the internal organisation of person-environment interactions (Paris, 1999), an underlying brain mechanism that is responsive to stress-related process may account for characterological changes associated with chronicity, and may even be the chronicity (as in a return to pre-morbid personality styles following acute psychosis). This potential for long-term maladaptive changes in the brain and functioning may be at a critical stage in adolescence when identity formation is occurring, thus forging one pathway to long-term negative symptoms and relapse vulnerability.

Taking a cue from the stress-illness literature, it could be argued that a common element in these processes is the mediatory role of cognition. In particular, the present thesis has indicated a significant amount of cognitive processing in response to distressing and overwhelming events that can be placed in the two general categories of appraisal and adaptation. In a dynamic model these processes would not represent fixed time points relative to the stressor event, but ongoing interactive evaluation-behaviour routines. Appraisal cognitions would include the evaluation of the personal and social meaning of the event, particularly appraisal of threat, evaluation of coping resources, thoughts of loss, uncertainty, and uncontrollability, and formulation of adaptive tasks. Adaptation responses would include changes to self-concept and experience of self, such as role constriction, engulfment, and shame. Adaptation would also include a range of cognitively mediated self-regulatory changes in functioning, including emotional and behavioural / instrumental adjustments in functioning and social relatedness, such as sealing over of affect, dissociation, cognitive deconstruction, avoidance, social withdrawal, and substance abuse. It could be argued that for clients with psychosis, resolution of these cognitive tasks may be difficult due to illness related reductions in available processing capacity (Nuechterlein, 1987).

Overall, it appears that some negative symptoms, and other related symptoms that occur in first-onset psychosis may be conceptually reframed as trauma related dysfunctions. In particular, this conceptualisation appears to account for the longitudinal phases and high overlap of co-occurring symptomological factors in first-onset psychosis, and may offer considerable explanatory power for some cases of psychosis that include reactive features. More generally, it appears that processes intrinsic to stress or trauma may provide a

framework for re-conceptualising psychopathology generally. In this instance, the inclusion of biopsychosocial factors that are informed by phenomenological descriptions and relevant psychodynamic processes may present a wide array of research opportunities, and enhance the face validity of interventions – reducing the incidence of iatrogenic and secondary effects, and increasing client adherence to the programme.

Chapter 19 Clinical implications

This chapter explores the clinical implications for treatment provision and specific trauma therapy requirements as highlighted by the theoretical association between negative symptoms and trauma related factors.

The combination of early intervention in the psychoses as a secondary prevention and community based care has resulted in the development of a number of comprehensive first-onset psychosis programmes. At present, as an outcropping of the success of EPPIC in Australia, and paradigmatic shifts to community based secondary prevention strategies, each major New Zealand city now has one of these programmes operating. However, it is worth noting that only in 1996 the *Schizophrenia Bulletin* ran an issue on early intervention in schizophrenia that described EPPIC as an "exciting effort...the only early detection and intervention program in the world today" (McGlashan, 1996, p.199), and as discussed, the initial outcome data for EPPIC is promising (McGorry et al., 1996). This highlights the infancy of both the treatment programme and the research that is driving these ventures, therefore it is worth reviewing the implications for treatment efficacy that may be related to the theoretical assumptions in the present thesis.

Firstly, as suggested by the subjective impact of life adversity and psychosis, as well as the importance of subjective nature of stress-related coping, the client's presentation and therapeutic needs are central to the management of psychosis, rather than treating the diagnosis. In this instance McGorry's (1992) suggestion of developing individual explanatory models may provide an informed structure to service delivery, as well as intrinsic meaning for the client. At a service level, the explanatory models could be used to develop micro-theories regarding the factors involved in a client's presentation, and could be evaluated over time to determine if they were efficacious in guiding the implementation of specific treatment components. For example, a similar approach has been taken to the content of delusions, with delusions being regarded as carrying personal meaning and symbolising beliefs or schematic structures (Bruno, 1993). This approach has allowed delusional and hallucinatory material to be used as components of unique explanatory models for clients, rather than disregarded as unpredictable features of a psychotic decompensation (Haddock et al., 1998). In a broader sense, using a person-centred phenomenological approach in observing the person in their

interactions with social and environmental stimuli, as well as being able to 'discover' the subjective meanings inherent in significant personal experiences, and how these appraisals relate to coping attempts, may indicate the potential for cognitive therapies to be included in treatment programs for psychosis.

Secondly, the use of intensive case management is likely to promote close monitoring by clinicians of changes in the client's mental and behavioural functioning, and provide proactive opportunities for the client to role model self-management skills. Further, coping skills, both emotional and instrumental could be introduced and potentially sustained through close contact between staff and the client. These opportunities may circumvent the overuse of subjective regulatory strategies that may become maladaptive and interfere with psychosocial integration. Perhaps most importantly, as much that is psychotherapeutic is contingent on the working alliance that develops between the clinician and the client, regular contact with the case manager may allow the client to begin to work through some of the traumatic memories they have accumulated. This may happen either directly with the case manager or through a case management referral to more experienced clinician. Therefore, training case managers in detecting distress and behavioural avoidance, and in helping clients discuss and reframe this phenomena as part of a normal reaction to overt fear and uncertainty may be a significant step in attenuating the presence and progression of negative symptomology. In relation to an explanatory model, exploring the subjective phenomenology of mental illness may also help the client to see that not all of what they experience or feel is 'the illness', that at least some of their experience reflects processes that are anchored in the continuum with normality. This approach may have the buffering effect of offering hope in a time that for some is marked by feelings of helplessness, and potentially some degree of insight into the link between stress and psychosis that can be utilised in relapse prevention. Although this approach needs to be balanced against protecting the client from potentially unattainable expectations regarding the return to their past roles and capabilities.

Thirdly, secondary prevention is a theme that runs through both the above treatment implications. However, in terms of reducing the impact of trauma events, distress, and the theoretically related regulatory processes, there may be a number of components of first-onset psychosis intervention that may protect against unnecessary symptomology. As clients may have reactive symptoms associated with a range of life events, including premorbid adversity, psychotic phenomena, and subsequent treatment contingencies, these areas need to be

regarded as critical to preventive strategies. One overt source of trauma in psychosis has been found to be the processes associated with hospitalisation. This indicates the potential for reworking the way in which already highly distressed clients are managed in an inpatient setting. For instance, at EPPIC the programme has a large enough catchment area in which to sustain a specialist first-onset psychosis inpatient service that is informed by the psychosocial issues for this population. In response to the initial study which highlighted the distress caused by hospitalisation, they were able to restrict the use of subjectively punitive methods such as seclusion, and subsequently in their replication lower levels of PTSD were found (McGorry, 1995). Another change in these services has been to resist applying diagnostic labels to clients when this may have an unnecessary shaming and stigmatising effect, particularly as the diagnosis is changeable in first-onset psychosis. Similarly, outpatient treatment, particularly if it is based away from the local 'mental hospital', is arguably less likely to evoke subjective identification with being seriously mentally ill and socially deviant, or shame in being seen entering the hospital. However, this needs to be balanced by the availability of the inpatient service as a place of safety for the client when their mental health deteriorates. Also, the judicious use of neuroleptics, along with regular medication reviews, again are approaches which can reduce the negative impact of toxicity, side effects, and subsequent treatment non-adherence.

Overall, the implications of taking the time to 'unpack' the clients illness to determine where the points of distress are for the client may enhance treatment adherence, particularly for people experiencing a schematically novel and potentially terrifying first episode of psychosis. This point was made by McGorry (1992) who suggests that client develop explanatory models of their illness, and particularly in the early stages this can be at odds to the implicit model used by staff, a situation which may account for the often reported lack of 'insight' in this population. Arguably, within a comprehensive treatment service for first-onset psychosis, in which cases can often be complicated by a range of illness related factors, taking a dynamic symptom based approach may assist in guiding individual cases to the appropriate treatments, and would facilitate the role of individual case management. For example, Hogarty et al. (1995) have described a treatment approach for patients with schizophrenia in which 'models of the person' are developed to facilitate the management programme.

Another component of treatment that would be indicated, particularly for cases with severe anxiety / trauma features, negative personal appraisals, and associated negative symptoms, is some form of trauma therapy, both as a primary intervention and for secondary prevention. McGorry (1991) has suggested that trauma therapy be offered to some clients with first-onset psychosis. The implications for the treatment of psychosis that stand out from the above discussions, is the need to identify and treat possible PTSD symptoms to avoid exacerbation of the illness, and the possible ameliorative effect this may have on otherwise protracted negative symptoms. Within the domain of early intervention this approach may be critical (Birchwood, Todd & Jackson, 1998), given the exacerbation of trauma in response to psychotic phenomena found in younger cases (Meyer et al., 1999), and in avoiding disruption to psycholoigical and social development in adolescence – problems that are associated with the experience of illness in this age group. Further, the recognition and treatment of trauma features may have a beneficial effect on treatment adherence if it means less avoidance behaviour is utilised by the client, particularly when encouraged to discuss psychotic experiences, or to utilise staff and medications.

However, little work in the way of trauma therapy for individuals who have, or are, experiencing psychosis has been presented in the literature. Currently, generic cognitive behavioural methods have been transported to this population, such as at Totara House, or the EPPIC cognitively-oriented psychotherapy for early psychosis (COPE) (Jackson et al., 1997), with trauma issues being covered more in terms of psychoeducation than actual trauma work. What is not present is a validated therapy in which traumatic processing can occur, particularly one that is safe in these acutely vulnerable clients.

Arguably, two problems may impede the transporting of existing methods of trauma resolution in this population. Firstly, the need for insight based coping methods, reasonable affect regulation, goal directed motivation, and the presence of a therapeutic alliance in traditional cognitive behavioural methods, and which may not be present for some clients, particularly so in the early in the psychosis. However, it may be in the early stage that trauma therapy may enhance relapse prevention as it appears that receiving appropriate treatment in the few months during and after a first admission for psychosis may have a critical impact on prognosis (Birchwood, Todd & Jackson, 1998). This is supported by the stress literature which indicates that the outcome of distress depends on how the situation is dealt with at the time, particularly whether coping attempts result in adaptation or humiliation (Garmezy &

Rutter, 1983). Secondly, many clinicians may be concerned about exacerbating the psychotic phenomena via an increased focus on internal stimuli that trauma therapy often utilises. Perhaps with the move toward the use of modified cognitive behavioural techniques for more complex psychiatric illnesses, such as Briere's (1999) Self-Trauma model, trauma therapy may be more readily adapted to the psychoses.

In respect to negative symptoms, Den Boer et al. (1987) argue that "until now no adequate therapy has been developed and the association of negative symptomology with structural brain abnormalities...bears the risk of leading to an unjustified therapeutic nihilism" (p.110). Arguably, whether or not the negative symptoms are primary or secondary to the index illness, reframing them as part of the psychosocial phenomenology of psychosis may encourage clinicians to promote adaptive changes to emotional and behavioural coping that will confront these deficits over time. However, in terms of negative symptoms being possible self-regulatory mechanisms, such as sealing over of affect, a caveat for treatment may be that clinicians work *with* the client, rather than *over* the client, to ensure that psychosocial reintegration is achieved without the client becoming overwhelmed and symptomatic. The point here is one of pacing the intervention so that the many cognitive, social, and psychodynamic challenges associated with post-psychotic recovery can be managed by the client. Thus avoiding the emergence or continuation of maladaptive regulatory strategies that are associated with chronicity and risk of relapse.

The implications for neuroleptic interventions may also need to be considered. A client presenting with a mixture of psychotic and trauma features may respond poorly to anti-psychotic medications. While neuroleptics may be used to treat the prominent psychotic symptoms, persistent use of these for residual symptoms more reflective of trauma may lead to unnecessary toxicity and side effects, as well as possibly neuroleptically intensified negative symptoms (Shaner & Eth, 1989). As the danger of not treating the symptoms of psychosis with medication is unacceptable, this concern needs to be addressed empirically.

In summary, this chapter has highlighted the implications an association between negative symptoms and trauma may hold for clinical management of first-onset psychosis. In particular, a descriptive method of presenting current and longitudinal factors in the client's presentation may promote therapeutic continuity and comprehensiveness, particularly in mulit-disciplinary clinics. Further, this approach may facilitate the goals of secondary

prevention in the management of the first-onset psychosis population, and potentially lead to a reduction in the long-term effects of avoidance behaviour, stigma, denial, and changes to the self-system. Therefore, an advantage of describing negative symptoms as processes contingent on the person's history, current cognitive and behavioural skills, and the context in which the person-illness is embedded, and thus potentially modifiable, may be in the rethinking of pessimistic assumptions regarding treatment prognosis. Also, as the presence of trauma in the client's history would indicate a treatment component that addresses trauma resolution and integration of significant life experiences, more attention is needed to developing trauma therapy for the psychoses.

Chapter 20 Research implications

This chapter discusses the implications for developing multi-factorial models of complex psychopathology. Such a reconceptualisation was supported in the discussion, which highlighted the theoretical underdevelopment in psychology, and of paradigms of severe mental disorder. The theoretical association between negative symptoms and life adversity is taken as tentative evidence of the multi-causality of psychosis, as well as indicating the unique matrix of events associated with first-onset psychosis, thus suggesting complex models may be better than linear models. The underdeveloped status of the Ciompi's (1988) biopsychosocial model is recapped, and alternative methods and models are reviewed, emphasising the need to include person-centred, symptom-based approaches. The chapter ends with a brief discussion of problems with the assessment of symptoms, particularly for differentiating negative symptoms and trauma processes.

As stated, the building of a theoretical multi-factorial model of severe psychopathology, one that implicates stress-related process in the initiation and maintenance of psychosis, is beyond the scope of the present thesis. However, a number of points have been raised which may indicate some features that could be included in such a model or models. Generally, it has been noted that for a model to have sufficient explanatory power (i.e. to represent the workings of a complex system) would require the inclusion of multiple pathways to mental disorder, as well as allowing for multiple expressions of illness, particularly as the severity of the illness increased. Further, the inclusion of feedback loops that represent maintaining factors and pathways to future decompensation are arguably critical if issues of causal primacy are to be disentangled from secondary phenomena in the presentation.

Already it has been discussed that the relationships between these types of factors are typically discussed in clinical case formulations, however, extending the same comprehensiveness to general models, or research assessments, may prove formidable. Particularly given the problems with comorbidity, and implicit causal assumptions embedded in many current clinician rating scales, such as the Brief Psychiatric Rating Scale (Overall & Gorham, 1962), as well as the heterogeneity in the research sample that can be hidden by syndromal diagnosis.

In part two, Ciompi's (1988) biopsychosocial vulnerability-stress model of psychosis was presented. This model posits stress as a precipitant to psychosis, via unspecified biological mechanisms. However, it was argued that this model is overly simplistic and constrained by the 'disease' hypothesis of psychosis, and appears to be characterised by weak or general associations, rather than providing clarity via the inclusion of detailed feed back loops and illness perpetuating factors. This model also represents psychotic decompensation as a linear 'event', rather than embedding the client in a matrix of observable and subjective adversity. It was argued that this theoretical failing has the effect of 'decontextualising' the illness, and is likely to perpetuate the assumption that the illness overrides all other factors in the client's life. And thus the concerns of the illness (observable symptoms), and by proxy diagnosis, are potentially treated as more important than concerns for the client's experience. This point was made clearly by Bannister (1968), who criticised researchers for their tendency to "erect psychologies of schizophrenics as if they were a logically distinct species" (p.183). This theoretical disadvantage may not be realised in an informed clinic, but may be perpetuated at the nosological and ontological levels, potentially limiting research directives and the development of meta-theories. This type of issue has lead Boyle (1990) to call for 'schizophrenia' free research, so that new models and relationships can be developed without the potential for constraint from some current conceptualisations.

From this discussion, three critical issues appear to stand out in terms of extending the biopsychosocial account of psychosis: 1) determining which symptoms or associated factors indicate the etiological specificity in the presentation, 2) the potentially problematic shift from a model of detailed and individually relevant phenomena to a model that can be applied more generally, and 3) being able to evaluate the model. The discussion below addresses some of the arguments associated with these issues.

Methodologically, a generalised state of dysfunction means that causal mechanisms will be closely entwined with other symptomatic factors. This problem is arguably greater for the psychoses as the notion of severity can present researchers with a potential circularity in determining what is the illness and what are the effects of the illness. Particularly as studies in this population typically compare groups using measures rating chronicity, such as inpatient vs. outpatient, and which rely on cross-sectional, decontextualised methods. In particular, attempting to disentangle symptomology in disorders characterised by functional severity, and thus ranked high in a diagnostic hierarchy, may be difficult as the instruments used will

invariably share many items due to the hierarchical inclusion rule. It is probable that these items are the ones often referred to as non-specific symptoms. Arguably, depending on the disorder, or the researchers paradigmatic bias, symptoms may be labelled differently, but at a behavioural level they may be indistinguishable. In this instance, the presence of deficit symptoms, such as withdrawal or flat affect, could be taken as partial evidence for a primary psychosis, while equally being acceptable as correlates of poor psychosocial functioning, or a component in a stress-response syndrome.

Conceptually, longitudinal research would potentially provide a plausible method with which to capture the interweaving of relationships, and indicate which events may have causal primacy, or at least temporal primacy. For example, as already discussed, Strauss et al. (1989) have proposed a longitudinal assessment for determining whether negative symptoms are primary or secondary. Further, a longitudinal approach would allow for the observation of patterns in the data that are not available in cross-sectional designs, processes which may extend over several years, the average prodromal period for first-onset psychosis being around three years (Hafner & Maurer, 1995). This method may be particularly useful in differentiating processes that characterise first-onset psychosis, other psychoses, and chronic forms of schizophrenia, and in attempting to determine what factors are specific to each of these, but not to severe mental illness generally (i.e. specific vs non-specific factors). Longitudinal designs can be either prospective or retrospective, with advantages and disadvantages applying to both (Venables, 1990). The disadvantages appear to be significant, and may indicate that this methodology should be used with caution in schizophrenia research. Prospectively the target population is small even when indicators are used to narrow the sample selection process, and dropout can decrease the power of the data. Retrospectively, the data that is coded is typically collected to meet agendas different to the research focus, and the historical fact of changing research criteria can lead to distortions in the data.

Another potential avenue for developing an understanding of causal pathways is to research the relationships between normal functioning and psychiatric dysfunction, potentially highlighting many of the contingencies which result in psychopathology. A number of advances in this respect have already been put forward in the stress and coping literature, with concepts of resiliency, hardiness, personality styles, and ability to elicit social support being functions that potentially differentiate those that achieve adaptation vs. those that fail (Sarafino, 1998; Weibe, & Williams, 1992). In the psychoses it has been noted that

hallucinations can appear on a continuum with normality (Strauss, 1989a), and affective disturbances, severe enough to produce functional retardation and suicidal behaviour, occur in the general population in response to distress (Davison & Neale, 1996). Therefore, this broad based research could identify where mental divergences can occur, and which of these appears to predispose the individual to further problems. However, a caveat with both of these research methods, longitudinal and searching out commonalities and divergences between psychopathological and normal states, would require considerable resourcing, and would likely be untenable to clinics wanting to get involved in researching their client base.

An alternative method put forward in the present thesis which may make smaller research programmes more manageable is the symptom based approach, in which diagnostic categories are set aside, and patterns of symptoms are studied free of overarching diagnoses (Bentall et al., 1988; Persons, 1986). A number of advantages of this approach have been discussed in detail in part 2, however if combined with the person-centred approach, this methodology could provide a rich array of data regarding the client's history and present state of functioning, with assessment methods using both behavioural observation as well as subjective reporting, without losing data to a diagnostic label. For example, Costello (1993) states that "it is only by a close investigation of the relationships between the symptoms that we will discover the extent to which negative symptoms are the result of independent causes or the result of causal processes that produce positive symptoms...." (p.297). Further, a long term advantage to this method would be the detailed data that would be made available for future retrospective longitudinal studies. If symptoms were detailed client-wise, using consistent descriptive terms, rather than terms artificially tied to diagnostic assumptions, then patterns between the symptoms, and co-occurring events, could potentially be observed over time. Also, if this approach were to be used over time, then emerging patterns could be potentially used in the development of new diagnostic categories and terms.

An argument that has been put forward in the present thesis is that in assessing and managing mental disorder, care needs to be taken to keep the illness contextualised by the client's experience of his or her self, their past and immediate environment, and their socio-cultural paradigm. Taking a symptom approach, phenomena significant to the client, both subjectively and in terms of risk for relapse, could be highlighted and assessed, and in terms of prevention be built into relapse management plans. Further, in terms of treatment this approach may

enable the illness to be broken down into manageable 'mini-disorders' rather than treating many symptoms as meaning-less components of the primary disorder.

However, taking this approach has its own risks, one caveat being that symptoms would need to be assessed longitudinally, and in the context of a multi-faceted framework to be able to disentangle the many overlapping illness features. Further, a potential confounding influence is the effect of the build up of symptom severity, with increasingly wider ranges of symptoms and psychopathological processes having a 'snowballing' effect. For example, the client's decent into madness may be rooted in events earlier in the history, as stressors from the past can affect mental states and personality in the present (Stampfer, 1990), with other more recent adversities acting to maintain the illness and perpetuate the risk for further episodes of decompensation. However, this approach appears to have the potential benefit of retaining the full gamut of data that may be relevant to the clinician and researcher, and in the initial stages of client contact may provide a comprehensive assessment on which to base future predictions, and on which to reflect future evaluations. Further, associations can be forged, and causality indicated between events on the basis of the explanatory and predictive power of the relationships, what Kalmar & Sternberg (1988) refer to as persuasiveness, thus allowing for any one of a number of biopsychosocial factors to have causal primacy. In terms of the biopsychosocial model, this would provide a degree of flexibility in conceptualisation of psychopathological processes.

One application of the symptom-based approach may be in the use of symptoms and posited multi-factorial relationships to build explanatory models of the client's illness, as discussed in the previous chapter. McGorry (1992) has proposed that this approach could have the two-fold benefit of breaking the vicious cycles of distress experienced by the client as a result of appraisals regarding the meaning of the illness. Explanatory models may have their benefit in providing heuristic approaches to making a number of symptomatic behaviours and phenomena, illness related assumptions, cognitions, and responses explicit to both the client and the clinician. In particular, this model may help in providing meaningful organisation to the cognitions reported by clients, which can then be referenced against their affective and behavioural symptomology and coping attempts to potentially identify patterns of functioning and dysfunction. As these models may also help the client order their own thoughts, it may enhance the reliability of their self-reports over time.

Extrapolating from McGorry's (1992) initial suggestion regarding the clinical application of explanatory models, these models may provide an interface mechanism between the different levels of theory proposed by Ward & Hudson (1998) and the different levels of abstraction in handling clinical data. In general, it could be argued that one of the problems with disentangling the confounding effects of diagnosis, severity, and co-occurrence of symptomology in psychopathology, is that the different levels at which aspects of mental disorder can be viewed are not made explicit. This argument is similar to that offered by Ward & Hudson in terms of theory construction generally. The argument here is that explanatory models of psychopathology can be constructed at any one of the meta, single factor, or micro levels, and if based on valid abstractions / theories will remain consistent between the levels for any one client, or at least relevant components of the models will remain valid. For example, at the meta-theory level would reside multi-factor theories of psychopathology, such as the vulnerability-stress model, or McGorry's (1995) proposition for using stress as an overarching framework for conceptualising psychotic phenomena. At the single factor theory level, models may include processes such as post-psychotic trauma, stigmatisation, and self-regulation of affect. At the micro-level, models of immediate distress responses to feeling overwhelmed, or paranoid, or immediate family conflict, or being faced with taking undesirable medication, to name a very few, may be present. To these levels can be added the level of grand theory, which may be untestable. For argument's sake, an example would be posing negative symptoms as an evolutionary significant expression of the sick role, in that deficits behaviours may communicate to others a need to receive care, thus potentially ensuring survival of the unwell member of the social group.

As can be seen from this conceptualisation, with increasing levels of abstraction also comes increasing complexity, and therefore a range of methodological difficulties in evaluating the model's validity also occur. Thus potentially providing an analogue to present difficulties in conceptualising and testing multi-factorial models in psychopathology generally. However, in making the levels explicit, potential starting points for validation can be highlighted. For example, it would be expected that particular elements from all three levels would be used to construct the multi-factorial explanatory model that best reflects the client's illness manifestation. It may be that when a client enters a particular programme the explanatory model would be simple, based on tentative cross-sectional information at the micro and single factor level, and only becoming a personal meta-theory over time as more information emerges. This useage is similar to existing rating scales and symptom inventories, with the

addition of being able to represent the interaction between factors, currently or over time. Thus it may provide an alternative to cross-sectional descriptions of symptomology for which relationships are left implicit, and typically tied to the weak psychiatric paradigm.

Returning to the issues of nosology, a problem that appears to pervade much of the above discussion regarding the representation of multiple factors, is the influence of the incumbent diagnostic system on the research outcomes, particularly when diagnoses are represented without reference to the significant component features. The main problem is that much data is lost in diagnosis which does not allow for patterns of events and symptoms, and the total variance accounted for by the research objectives are diluted by the heterogeneity in the research sample. While this problem could be overcome by the use of rating instruments to select more homogenous groups, as a means of 'standardising the diagnostic standard', this may lead to even more lost data, particularly in terms of causal associations. Therefore, as patterns of relationships are the cornerstone of valid theories, the problem of standardisation is potentially a circular one, similar to that of illness severity, which results in reliability at the expense of validity and generalisability. It is this sort of problem that has lead Boyle (1990) to argue that a rethink of how phenomena are grouped or conceptualised is required, as classification itself represents the search for order or patterns.

The explanatory model system outlined above may have some advantages for nosological development in that diagnostic entities could be represented as not just a cluster of symptoms, or symptoms plus duration, but could include the missing dimension of symptom interaction via summaries of the three theoretical levels. The potential here could be for both dimensional and categorical aspects of mental disorder to be represented. However, in retaining a degree of standardisation this system would need considerable theoretical and empirical validation. Two specific disadvantages may be the reliance on subjective reporting to gain all of the data that is relevant (i.e. subjective components of the illness), as well as the need for considerable work to be done on defining symptoms at an operational level. For example, as discussed in part six dissociation, while at face value may be considered a symptom of trauma, is itself a syndrome of other differential symptoms and signs. Explanatory models may however, at least initially, have success in describing a greater range of interactive client variables in clincial settings, or areas of fledgling research, with generalisability outcropping from development over time. In particular, the model may provide a heuristic tool for 1) providing ongoing indicators for assessment and treatment areas, 2) indicating areas that may be of

interest to researchers operating within the biopsychosocial paradigm, and 3) integrating research findings regarding causal processes that are outside the bounds of standard diagnosis formats.

While the present discussion has not included issues regarding the actual identification and assessment of symptoms and illness-related factors, that does not mean these issues are not problematic. For example, in terms of empirically measuring the trauma component in first-onset psychosis, the psychometric instruments used would need to avoid sharing the significant number of common symptoms in these groups of clients. Particularly because rating a presence is only a half-measure, specific factors high in explanatory power need to be identified also to start building up a causal model that indicates what types of therapeutic initiatives will best help the client. In terms of rating presence of trauma free of other overlapping constraints, the Trauma Symptom Inventory (TSI) appears to be a conceptually valid instrument with good psychometrics (Briere, 1995), and has been used with patients with schizophrenia without conceptual or reading difficulties (Briere, 1999). However, in rating the presence of trauma, for research and therapeutic purposes the stressor, or trauma anchor, is also of crucial interest. At present there is no validated instrument available that rates traumatic events specific to the types of experiences of people presenting with psychosis. Further, given the multiple events that can result in traumatic distress, the scale would need to be able to rate a number of situations deemed by the client to be subjectively disturbing. The author has developed one such self-report scale, the Stressful Life-Events Questionnaire (SLEQ) (see appendix V). This scale has not been validated, and was developed from the literature reviewed in part 6. However it does provide a range of events that can be rated separately, and each event can be rated on a 0 – 3 lickert scale. This scale may potentially be used qualitatively to help the clinician build up trauma components in a client's explanatory model, as well as indicating anchor points for trauma severity ratings. Further, in their reliability study of self-reported victimisation and PTSD by individuals with severe mental illness, Goodman et al. (1999) found that their sample of 50 people showed relatively little shifts in reporting over a two week period, suggesting that self-report measures in this population is reliable, at least over short periods of time.

Regarding assessment of factors more generally, the overarching argument that has been presented here, albeit implicitly so far, is that many if not most of the problems with disentangling specific illness factors from more general processes related to severity and

diagnosis, is due to the underdeveloped state of theory in psychosis. The potential outcome being the promotion of invalid associations, while other more fruitful relationships are left aside because they do not fit the research limitations. Arguably, this leaves researchers who are exploring new relationships in psychopathological processes with either losing critical data to inflexible diagnoses, or left calling for further theoretical development in their area of interest so that new methods can be generated.

In summary, this chapter has highlighted the significant potential for theory building in psychopathology generally, in terms of making a paradigmatic shift to contexutalised and data rich methods. Also, the same potential exists for psychosis, particularly in the differentiating of causal and secondary factors. Using explanatory models both in the clinic and for research was highlighted as an area of development which may provide a new dimension of complexity to descriptions of mental illness. Further, the explication of different levels of abstraction in these models may increase their descriptive and predictive power. While such an approach may be criticised for its lack of organisational categories, it may balance the discussed lack of validity in existing syndromal categories used for diagnostic purposes. Overall, the descriptive approach may not be an end in itself, however, at least in the interim it may provide a first step in the integration of theoretical, research, and clinical demands, via its utility for observing and explaining specific symptoms without losing any relevant data, and without being constrained by overarching categories of mental disorder. Within the first-onset psychosis population this approach may be particularly useful in delineating the many factors impacting on illness presentation and outcome, and may result in new ways of subtyping psychotic disorder, reducing heterogeneity in schizophrenia, and potentially highlighting a trauma related group with specific treatment needs.

Conclusion

Severe psychopathology presents a rich field of data from which to derive increasingly integrated theories that represent the complexity of human function and dysfunction. In the present thesis theory building as an enterprise crucial to psychology has been presented and critiqued. This discussion then shifted to an exploration of the negative symptoms found in psychosis, highlighting problems with syndromal diagnosis and the proposed multi-causality of 'secondary' negative symptoms. A theoretical relationship between these symptoms and stress-related events was proposed and illustrated, and is taken as support of the multi-causality hypothesis of negative symptoms. Support is also taken for the argument indicating the need to re-conceptualise psychosis as a disorder characterised by *interacting* vulnerabilities and stressors, rather than trying to assign paradigmatic dominance to either diathesis or the stress components of psychosis.

Overall, a number of theoretical similarities and associations between negative symptomology and PTSD were indicated. These included specific signs and symptoms taken from the DSM-IV, as well as a range of associated features and biopsychosocial processes. Many of these associations and related features, such as a history of significant stressful life events, were found to exist in case studies of first-onset psychosis clients. One single factor theory to be supported by the present thesis was the phenomena of post-psychotic trauma which represents the extreme distress, horror, and threat to the self that the subjective experience of psychosis is for some persons in the throes of their first episode of acute madness. In particular, cognitive mediatory factors were highlighted as fitting well with the psychological, psychodynamic, and social aspects of responding to adversity, as the person evaluates the stressor, and their ability to have some control over it. Similarities between the negative symptom 'amotivational syndrome' and self-regulation processes were highlighted, and it appears that the functionally debilitating symptoms of social withdrawal and behavioural avoidance, seen in both psychosis and PTSD, may be the observable analogues of a complex response to overwhelming affect.

In finding these types of theoretical associations, wholesale pessimism associated with poor outcome in cases with prominent negative symptomology may be unwarranted, particularly when the deficits are part of a wider matrix of secondary responses to adversity. Therefore

indicating the symptoms may be contingent on processes that are modifiable. Further, this can be taken as supportive of secondary prevention efforts that attempt to circumvent long-term biopsychosocial dysfunction, via the identification and attenuation of contingencies which perpetuate or add risk to the disorder. The theoretical association between PTSD and psychosis also indicates that developing trauma therapies, and single-case explanatory models of illness events, may assist clients in cognitively debriefing from trauma events, as well as processing meaningful components from the illness via psychodynamic integration.

In terms of theoretical development, these associations support the use of stress / trauma models as a framework for including a raft of functional processes in psychosis. It has been highlighted that these processes can be identified as operating at three different levels of theoretical abstraction. Developing symptom-based explanatory models which can operate at all of these levels depending on the descriptive requirements of the client, treatment service, or researcher. Potentially this approach could manage multiple layers of information, as well as provide categories of behaviour via the meta-level theories. Further, it may facilitate the prediction of complex person-illness-behaviour routines, indicate and track treatment, and be useful in the early stages of research when guiding data and theories are being collated. Also, setting aside current methods may result in less confusion regarding symptom overlap, comorbidity, and heterogeneity, as the three tier explanatory model explicitly seeks out these data rich points of inquiry, in the progression toward multi-factorial meta-theory representations. As the goal of theory building and integration is the increasingly accurate approximations of the functional complexity that is being human.

Appendix I

Email letter from T. McGlashan to the Totara House researcher

14 January 2000.

Dear Mr Turner

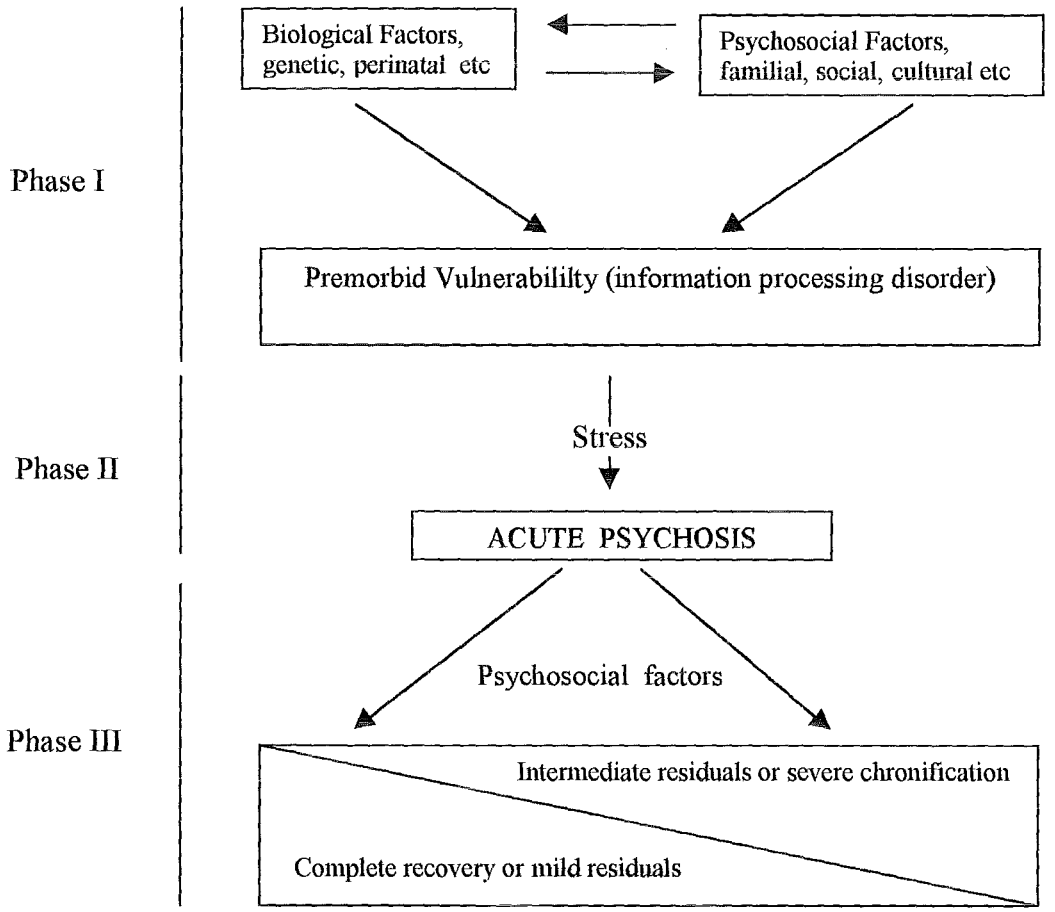
This is in reply to your letter of December 20, 1999. I am afraid you have been asked to do an impossible task, ie, do research in clinical settings without research instruments and research resources. After 30 years in this business as both a clinician and a clinical researcher I know through repeated experience that the two traditions cannot be integrated with the strategies you outlined. You need to go back to whomever gave you this charge with the news that decent research cannot be done without extended assessment forms and training and that you cannot ever expect clinicians to do research. Sorry to be the bearer of bad news but you don't need to come to the same conclusion yourself after a long period of frustrating experience.

Sincerely
T.H. McGlashan

Thomas H. McGlashan, M.D.
Professor of Psychiatry
Yale University School of Medicine
Director, Yale Psychiatric Institute

Appendix II

Biopsychosocial Model of Psychosis, modified from Ciompi (1989)



Appendix III

Synopsis of predictors of course and outcome in schizophrenia, modified from Jablensky (1995).

Poor Outcome	Good Outcome
Sociodemographic and family	
*Single, divorced, separated	*Married
Male sex	Female sex
High EE (short term)	Low EE
	Affective disorder in relatives
Premorbid personality and adjustment	
Schizoid personality	*Extrovert or cyclothymic personality
Poor psychosexual adjustment	*Good work, social and sexual adjustment
*Social isolation	Social contacts outside the family
Adjustment problems in adolescence	Precipitating stress or life events pre-onset
Past episodes and treatment	
*Longer duration of preindex illness	*Shorter duration of preindex illness
Mode of onset	
*Insidious	*Acute, associated with excitement, elation, perplexity, anxiety or depression
Initial clinical state	
*Negative symptoms on first admission	Affective features confusion, clouding
Affective blunting	
Primary delusions	Secondary delusions
Bizarre delusions	
Somatic delusions	
Voices from body	
Conversing with voices	
Hearing own thoughts	
Haptic and tactile hallucinations	Soft neurological signs
Social withdrawal	
Other variables	
Abnormal MRI	Good initial response to neuroleptics
Cortical atrophy on CT	
Street drug use (cannabis)	Response to placebo

* 'Robust' predictors (replicated across multiple studies); EE, expressed emotion; MRI, magnetic resonance imaging; CT, computerized tomography.

Appendix IV

DSM-IV Symptom criteria for posttraumatic stress disorder

A) The person has been exposed to a traumatic event in which both of the following were present:
   (1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
   (2) The person's response involved intense fear, helplessness, or horror. Note: In children, this may be expressed instead by disorganized or agitated behaviour.

B) The traumatic event is persistently re-experienced in one (or more) of the following ways:
   (1) Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: In young children, repititive play may occur in which themes or aspects of the trauma are expressed.
   (2) Recurrent distressing dreams of the event. Note: In children, there may be frightening dreams without recognisable content.
   (3) Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening when intoxicated). Note: In young children, trauma-specific re-enactment may occur.
   (4) Intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
   (5) Physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

C) Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
   (1) Efforts to avoid thoughts, feelings, or conversations associated with the trauma.
   (2) Efforts to avoid activities, places, or people that arouse recollections of the trauma.
   (3) Inability to recall an important aspect of the trauma.
   (4) Markedly diminished interest or participation in significant activities.
   (5) Feeling of detachment or estrangement from others.
   (6) Restricted range of affect (eg, unable to have loving feelings).
   (7) Sense of a foreshortened future (eg, does not expect to have a career, marriage, children, or a normal life span).

D) Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
   (1) Difficulty falling or staying asleep.
   (2) Irritability or outbursts of anger.
   (3) Difficulty concentrating.
   (4) Hypervigilance.
   (5) Exaggerated startle response.

Appendix V

Stressful Life-Events Questionnaire

Below is listed a number of stressful events that people may experience over the course of their lives. Please check each item, showing how **distressing** each event was for you **at the time it occurred**.
Note that if there are events you have not experienced, mark the "9" box for that item.

0. Not stressful **1.** Quite stressful **2.** Extremely stressful **9.** Not had this experience

1. Having had a hospital inpatient admission	0	1	2	9
2. Being placed in seclusion or forcefully restrained while in hospital	0	1	2	9
3. Because of the psychosis I felt that I was 'losing my mind'	0	1	2	9
4. Being able to see, hear, or feel unpleasant things that were part of the psychosis	0	1	2	9
5. Feeling like everyone must know I have a mental health problem	0	1	2	9
6. Because of the psychosis my family is no longer close to me	0	1	2	9
7. Having heard about or seen someone close to me be victimised	0	1	2	9
8. Being beaten when I was younger	0	1	2	9
9. In the past being made to feel small or fearful by other's comments	0	1	2	9
10. Being forced to behave sexually when I did not want to	0	1	2	9
11. Feeling my life has been threatened by my own or other's actions	0	1	2	9

In a few words, briefly describe the most stressful experience you have ever had.

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