A Common Factor for Childhood Syndromes

and Disorders

Empirical Evaluation and Associated Risk Factors

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Declaration

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Statement of Ethical Conduct

The research associated with this thesis abides by the international and Australian codes on human and animal experimentation, the guidelines by the Australian Government's Office of the Gene Technology Regulator and the rulings of the Safety, Ethics and Institutional Biosafety Committees of the University.

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"By the worldly standards of public life, all scholars in their work are of course oddly virtuous. They do not make wild claims, they do not cheat, they do not try to persuade at any cost, they appeal neither to prejudice nor to authority, they are often frank about their ignorance, their disputes are fairly decorous, they do not confuse what is being argued with race, politics, sex or age, they listen patiently to the young and to the old who both know everything. These are the general virtues of scholarship, and they are peculiarly the virtues of science." Jacob Bronowski

It is with those words above in mind that I have engaged in a truly engaging, albeit challenging PhD candidacy. Those who have shared my journey understand fully both the joy, and the difficulty that is inherent to this experience. Such a task could not be achieved without the guidance and support of many others, and I ask for the indulgence to take the opportunity to acknowledge them here.

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Abstract

There is strong evidence of excessive levels multivariate comorbidity for the major childhood internalizing and externalizing syndromes and/or disorders. The overall aim of this thesis is to propose a general factor to capture the common variance for the major internalising and externalising childhood syndromes and disorders. In this context, the thesis reports three empirical studies to evaluate the plausibility of a common factor, and potential risk factors that may be associated with it.

Study 1, examined if childhood psychopathology could be characterised by a bifactor model, which would provide support for a common factor. The bifactor structure was tested on data from a clinical sample of 974 parents and their children referred to the Royal Children's Hospital, Melbourne. Data was gained from the Syndrome and DSM-Oriented scales of the Child Behavior Checklist (CBCL), and the Anxiety Disorders Interview Schedule for Children-Parent Version. The model was also tested on the data from the Achenbach System of Empirically Based Assessment (ASEBA) Reference Group; an epidemiological sample used to validate the CBCL. The bifactor model showed excellent fit and substantive support for the hypothesised common factor.

Study 2 extended Study 1 and investigated whether the major childhood syndromes/disorders conform to a circumplex structure, which would provide support for the presence of a common factor underlying these syndromes/disorders. The circumplex was tested using the same data and same measures as Study 1, and results demonstrated that child psychopathology conforms to a circumplex structure,

with more than fifty percent of all variance accounted for by the common factor. This provided strong support for the presence of the hypothesised common factor.

Study 3 aimed to investigate the nature of this hypothesised common factor, and investigate which constructs are associated with it. The association of three constructs – negative affective temperament, parental psychopathology and familial functioning – on the general factor specified by the bifactor model was investigated. The results demonstrated that negative affect and parental psychopathology, both individually and in interaction, appear to be key risk factors associated with the common factor.

Overall, results suggest that, in contrast to current conceptualisations of childhood psychopathology as discrete and distinguishable entities, there is a common liability to all psychopathology in childhood. Such a common liability helps explain the high level comorbidity of childhood disorders and syndromes, because the liability suggests that all manifestations of psychological illness may, at least in part, have some common geneses. The results here suggest that negative affect and parental psychopathology are key risk factors in understanding this common liability factor.

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Introduction

In psychological terms, comorbidity is the coexistence of two or more distinct psychological disorders in the same individual at the same time (Achenbach, 1990/1991). Since first being defined by Feinstein (1970) more than forty years ago with regard to medical diseases, it has captured the interest of researchers in psychopathology, sparking research, considerable disagreement and at times pointed controversy (Krueger & Markon, 2006).

Within the clinical child psychiatry/psychology literature, it has been argued that understanding the substantive reasons for comorbidity is one of the key challenges facing mental health professionals and researchers (Kendall & Clarkin, 1992; Lilienfeld, 2003). This is largely because comorbid presentations are the norm rather than the exception in clinical practice, and rates of comorbidity are universally found to be significantly and substantially greater than would be expected by chance alone (Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003; Krueger & Markon, 2006). While arguments have been made that comorbidity is an artefact of nosological imprecision or of problematic research methodology, these arguments cannot singularly or collectively be demonstrated to fully explain the concept (Baldwin & Dadds, 2008; Milberger, Biederman, Faraone, Murphy, & Tsuang, 1995; Wolff & Ollendick, 2006).

Given the rebuttal of arguments regarding comorbidity as an artefactual phenomenon, there has been a specific need to consider more substantive explanations of comorbidity. While many theories have been proposed, the hypothesis that comorbidity between psychological disorders/syndromes may be a

result of the fact that the risk factors for the two disorders are correlated appears to have the greatest research support (Lee & Bukowski, 2012; Lilienfeld, 2003). Despite the fact that comorbidity is multivariate in nature – that is it occurs between and across all disorders in the spectrum of psychopathology - most research into comorbidity to date, has been bivariate in nature, looking at individual disorder pairs and attempting to provide explanations about the causes of comorbidity in that pair of disorders (Batstra, Bos, & Neeleman, 2002; Krueger & Markon, 2006). This has been a serious limitation of comorbidity research to date, and though recently comorbidity research has progressed towards multivariate conceptualisations, such research is still in its infancy.

Issues have arisen in progressing bivariate comorbidity research to the multivariate domains. Firstly, when moving research to the multivariate domain, more complex and sophisticated analysis techniques than have often been used are required, though techniques such as structural equation modelling (SEM) are available to fill this gap. Other issues such as model parsimony are less easy to deal with however, because when extending currently supported bivariate models to the multivariate, the complexity grows exponentially rather than additively (Krueger & Markon, 2006), and as a result models become hard to test, and lack clinical utility. One interesting model that is related to the supported bivariate hypothesis that comorbidity is caused by correlated risk factors between the disorders; that is that comorbidity/co-occurrence between psychological disorders/syndromes may be influenced by a single higher-order factor (Lilienfeld, 2003). This model, proposed by Klein and Riso (1993) and operationalised by Neale and Kendler (1995), hypothesises that comorbidity occurs because the comorbid disorders are alternate manifestations of a

'single' common liability factor, sometimes referred to as a *psychopathological liability*. This 'single' common liability is in fact conceptualised as derived from a multifactorial combination of heritable and environmental risk factors. This model differs from classic bivariate models of correlated risk factors because it proposes a single liability factor underlying all comorbidity, whereas the classic correlated risk model assumes that each comorbid pair of disorders has its own individual shared liability. This common factor model has significant parsimony over classic correlated risk models, but has generally shown to be a poor fitting in bivariate research (Neale & Kendler, 1995; Rhee, Willcutt, Hartman, Pennington, & DeFries, 2008). This thesis will outline an argument that any attempt to test a model proposing a single common factor within a bivariate domain would by definition, ignore a wide range of potential covariates, such as other disorders which may significantly impact an overall model. Thus the lack of support for a common factor model may be an artefact of a failure to assess the phenomenon multivariately, rather than the particular model being a poor model per se (Krueger & Markon, 2006). Surprisingly, there have been few attempts to investigate a common factor model within the multivariate domain of comorbidity, and it is the aim of the thesis to investigate a common factor model of multivariate comorbidity as implied by the common liability models.

The Aims of Thesis and Empirical Studies to Address These Aims

The overall aim of this thesis was to propose a general factor to capture the common variance for the major internalizing and externalizing childhood syndromes and disorders. Three empirical studies were conducted to evaluate the plausibility of such a common factor, and the potential risk factors that may be associated with such a

common factor. The first study examined if childhood psychopathology could be characterised by a bifactor model. The bifactor model allowed for the modelling of a common psychopathological liability factor, but also allowed for consideration of domain specific factors that are necessary to consider in the context of multivariate psychopathology; in this case the well validated internalising and externalising domains of child psychopathology. The bifactor model was tested for two different types of diagnostic structure; a categorical diagnostic structure, based on the DSM-IV (American Psychiatric Association, 1994), and derived from Anxiety Disorders Interview Schedule-Parent Version (ADIS-IV-P: Silverman & Albano, 1996); and a dimensional empirically validated syndrome structure, in the form of the Child Behavior Checklist (CBCL) from the Achenbach System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001). Both the Syndrome Scales and DSM-Oriented scales were used from the CBCL. The model was tested on clinical data from parents and their children referred to the Royal Children's Hospital, Melbourne, as well as using the ASEBA Reference Group data, which is the epidemiological sample used to validate the CBCL. Study 2, using the same data as study 1, further tested for the common factor, to provide cross-validation. Specifically it investigated whether child psychopathology conformed to a circumplex structure, which if validated would provide compelling support for a common factor underlying all childhood psychopathology.

Study 3 aimed to investigate the nature of this hypothesised common factor, and investigate which constructs may have associations with this common factor. The association of risk factor constructs was specifically investigated, because if significant associations between risk factors and the common psychopathology factor

were found, then it would imply that the common factor is most likely a liability factor as proposed by Klein and Riso (1993) and Neale and Kendler (1995) in their common liability factor models. Therefore, the study tested the association of three risk factor constructs – negative affective temperament, parental psychopathology and family functioning – with the common factor in the bifactor model.

Organisation of the Thesis Chapters

The three empirical studies form Chapters 3, 4 and 5 of this thesis. These are accompanied by two literature review chapters and a general discussion chapter. Chapter 1 provides a review of the concept of comorbidity, and its importance as an area of clinical research. The chapter outlines the existence of comorbidity at significantly greater than chance levels, and rebuts arguments that suggest comorbidity is merely an artefact of nosology and methodological problems with research. It will outline the need for substantive explanations for the causes of comorbidity, and the limitations of the predominantly bivariate research and models to date, and highlight the need for multivariate models. It will also discuss the potential utility of a common factor model to explain comorbidity, based on a common liability model.

Chapter 2 investigates this idea of a common liability which is conceptualised as a multifactorial combination of heritable and environmental causes, or risk factors. It explains why any common factor model based on a common liability should show meaningful associations with a range of risk factors. It provides a discussion of three candidate risk factors – negative affect, parental psychopathology and poor family functioning – and presents empirical evidence demonstrating that each of these

factors has been implicated with the development of psychopathology, and comorbidity. It will also provide an outline of the limitations of the current empirical literature on risk factors for comorbidity, and explain why a multivariate approach to risk factor research should be undertaken, and the need for modelling complex interactions between risk factors for comorbidity.

Chapters 3 through 5 present the background, method, and results of the three empirical studies, as well as discussion of the results. Chapter 6 is a General Discussion chapter, and provides a summary of findings across all three studies along, with a discussion of the implications of the results of these studies as a whole. This chapter also discusses the limitations of the thesis and suggestions for future research, with a brief overview of the conclusions of the thesis.

Chapter 1 - Comorbidity

The term "comorbidity" was first coined by Feinstein (1970) more than forty years ago, and originated in literature on the epidemiology of medical diseases. In the time since, it has captured the interest of researchers in psychopathology, sparking a myriad of research, considerable disagreement and at times pointed controversy (Krueger & Markon, 2006). The interest in comorbidity developed quickly within the clinical psychiatry/psychology literature, leading Kendall and Clarkin (1992), to argue that comorbidity was "…the premier challenge facing mental health professionals in the 1990s" (p. 833), a view that has continued through the next twenty years (see Angold, Costello, & Erkanli, 1999; Krueger & Markon, 2006; Lilienfeld, 2003).

Lilienfeld, Waldman, and Israel (1994) indicate that the concept of comorbidity took root in the psychiatric/psychological literature in the late 1980s, coinciding with the development of the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III: American Psychiatric Association, 1980) and the subsequent revision, DSM-III-R (American Psychiatric Association, 1987), in which a greater emphasis was placed on the existence of discrete mental disorders (Mineka, Watson, & Clark, 1998). Since then there has been an exponential growth of research in the area as evidence by a cursory search of research databases. Using the term "comorbidity" as a keyword, a search of the American Psychological Association's PsycINFO® database for the period 1872 to June 2012 produced approximately 19500 results from peer reviewed journals alone, of which over a third

(approximately 7400) were within the last five years. Clearly, the phenomenon has become a major topic of research.

This review chapter will outline the concept of comorbidity, and its importance as an area of clinical research. The first section of the chapter will define the concept of comorbidity and how it relates to psychological disorders. There will be a discussion of what makes comorbidity a worthy area of substantive investigation, specifically related to the significantly above chance level of occurrence, and the negative effects of comorbidity, in terms of severity, prognosis, and treatment outcome for individuals. The second section will provide a discussion of arguments that suggest comorbidity is entirely an artefact of nosological imprecision. It will outline the specious nature of these arguments, and how comorbidity exists regardless of whether the psychopathology is assessed using dimensional or categorical systems of assessment. The third section will review explanations of comorbidity as an artefact of research methodology, and explain that while these arguments possess merit, they cannot singularly or collectively be demonstrated to be the entire cause of the broad spectrum of the comorbidity phenomenon. As a result the need for substantive explanations for comorbidity will be demonstrated. The fourth section will outline the potential substantive explanations for comorbidity, along with the need to extend current substantive explanations to embrace multivariate models. It will cover the three broadly popular explanations for comorbidity; the causative model where one disorder is thought to cause the other, a model which views comorbid disorders as a third independent disorder, and a model that suggests comorbidity is caused by shared etiological or risk factors between disorders. It will demonstrate that only the last of these theories has solid empirical support. The final section will critique the

current state of the literature, and discuss how most research into comorbidity to date, has investigated individual disorder pairs. It will be argued that for comorbidity research to move forward, multivariate approaches considering the broad spectrum of psychopathology must be undertaken. This is justified because the phenomenon occurs across the broad spectrum of disorders and syndromes, and not just between certain specific disorder pairs. It will briefly outline the problems of statistical analysis and poor parsimony encountered by transferring bivariate models of comorbidity to the multivariate domain, and argue that Structural Equation Modelling can help overcome statistical limitations and identify problems with parsimony. Finally there will be an argument for a need to consider a hybrid model of comorbidity, based on shared etiological or risk factors between disorders, and which hypothesise a single common factor underlying psychopathology and comorbidity.

Defining Comorbidity

In his classic paper Feinstein (1970) offered a number of slightly different definitions of the concept "comorbidity", and most papers discussing comorbidity today use one of the variations Feinstein provided. Krueger and Markon (2006) argue that the most distinct and succinct definition was that for "…a patient with a particular index disease, the term co-morbidity refers to any additional co-existing ailment" (Feinstein, 1970, p. 467). However, similar definitions are more often cited and a quick review of seminal papers in the area shows that the most common definition used defines comorbidity as "any distinct additional clinical entity that has existed or that may occur during the clinical course of a patient who has the index disease under study" (Feinstein, 1970, pp. 456–457). In effect this definition argues that

comorbidity is the coexistence of two (or more) distinct disorders in the same individual at the same time. Feinstein's definition was initially only referring to medical (biological) disorders. However, given the phenomenon was also noticeable within the context of psychological disorders, the definition was considered to be equally applicable to co-existing clinical psychological disorders (e.g. ADHD, Generalised Anxiety Disorder) derived from a classification system such as the DSM-IV-TR (American Psychiatric Association, 2000) and or ICD-10 (World Health Organization, 1993). In transferring Feinstein's definition from the more general medical domain to the specifically psychopathological, comorbidity was thus defined as two or more distinct psychopathological entities (derived from a single diagnostic conceptualisation/system), existing in a single individual, at a single time point (Achenbach, 1990/1991, 1995; Angold et al., 1999; Caron & Rutter, 1991; Lilienfeld, 2003).

Debate over the Nomenclature in Psychopathology

The definition of comorbidity is not without debate within the clinical psychology literature. Feinstein (1970) developed his definition(s) of comorbidity in the context of chronic disease, consistently making reference to 'distinct clinical entities' or similar language (Mineka et al., 1998). Controversies emerge from the difficulties in attempting to extend this definition to psychiatric disorders, because they do not easily correspond to the idea of a 'distinct clinical entity' (Lilienfeld, 2003; Lilienfeld et al., 1994). Psychological and psychiatric clinicians deal with the concept of the 'disorder'; a behavioural/psychological syndrome that is in some way deviant from a specified standard of normality (American Psychiatric Association, 2000). This is fundamentally different from the medical concept of clinical entities which refer directly to clearly validated disease entities. Angold et al. (1999) argue

that this is a critical distinction, because psychological comorbidity between
disorders may imply that there is some problem with the classification system, rather
than any meaningful association between underlying disorders indexed by that
classification. However this does not invalidate the idea of comorbidity in a
psychiatric or psychological context; rather the nature of any classification system
must merely be taken into account when considering psychological comorbidity.

Further debate about the use of the term in a psychological context, arises because
Feinstein's definition implies that true comorbidity occurs only when the two (or
more) diseases that are present have distinctive aetiopathogenesis (i.e. distinct causes
and developments), or at a minimum effect distinct medical systems (Vella, Aragona,
& Alliani, 2000). This has led Blashfield and colleagues (Blashfield, Keeley, &
Burgers, 2009; Blashfield, McElroy, Pfohl, & Blum, 1994) and Caron and Rutter
(1991) among others to emphasise that for the definition of comorbidity to be valid
in a psychological context, that definition requires a provision relating to evidence
that there exists some underlying causal distinctiveness between the conditions.
However, as both Lilienfeld et al. (1994) and Meehl (2001) indicate, this is almost
impossible in psychological medicine, because, as argued above, mental disorders
are not usually defined in terms of causes, but symptoms (Krueger & Markon, 2006).

A semantic debate around the use of the term comorbidity has thus developed, as
outlined by Lilienfeld (2003). He argues that use of the term comorbidity reifies
current mental disorder constructs, implying a conceptual clarity that is not present.
Lilienfeld (2003) argues that clinical disorders are not bona fide categories with well-
understood and discrete aetiologies, and proposes the use of the more precise terms

'co-occurrence' when considering individuals, and 'co-variation' when considering close (statistical) relationship between disorders or syndromes at a global level (see also Keiley et al., 2003; Krueger & Markon, 2006; McConaughy & Achenbach, 1994). Such views are supported by van Praag (1996) who argues that use of the term "comorbidity" creates a lack of clarity because the term can be used to encompass too many conceptually distinct phenomena. However, while many researchers have been sympathetic with the need for greater precision in use of the term, there is general view that abandoning the term "comorbidity" would be both premature and counterproductive. Indeed, Rutter (1994) argues that this dilemma has actually served as the impetus for research on both comorbidity and the validity of diagnostic constructs. Spitzer (1994) further argues that there are many disease entities in medicine which do not possess clear-cut aetiologies or pathophysiologies, and thus using the term 'comorbidity' is perfectly reasonable for describing co-occurring entities even if they are not authentically distinct entities. Thus while the debate about the use of the term within psychological contexts is worthwhile, the debate itself does not invalidate use of the concept. Thus at the end we are left with the original definition from Feinstein (1970) as a baseline definition for comorbidity in a psychological context, succinctly summarised by Achenbach (1990/1991): "comorbidity is the coexistence of two or more distinct disorders in the same individual" (p. 271).

Why Study Comorbidity?

Comorbidity as the Rule in Clinical Psychology/Psychiatry, Not the Exception

As outlined in the opening section, a cursory investigation of the research literature shows that the "comorbidity" phenomenon has become of great interest. There are

many reasons why comorbidity has become such a great focus of research within the psychological and psychiatric research communities. By far the most obvious reason is that within clinical practice, comorbid presentations are very common; indeed in clinical practice, adult, childhood and adolescent clinical cases *without* psychiatric comorbidity are the exception, rather than the rule (Andrews, Slade, & Issakidis, 2002; Angold et al., 1999; Hall, Lynskey, & Teesson, 2001; Lilienfeld, 2003; Merikangas et al., 1998).

Many studies have also demonstrated the prevalence of comorbidity, within both clinical and epidemiological samples. A recent paper demonstrably highlights the large comorbidity prevalence of DSM-IV-TR diagnoses in adolescent epidemiological samples. Merikangas et al. (2010) reports on *The National Comorbidity Survey–Adolescent Supplement*; a representative face-to-face survey of over ten thousand adolescents aged 13 to 18 years in the continental United States of America. Using a modified version of the World Health Organization (WHO) Composite International Diagnostic Interview Version 3.0 (CIDI: Kessler & Ustun, 2004), they found that 42 percent of their sample with one class of disorder also met criteria for another class of disorder, and nearly 20 percent were eligible for three or more diagnoses (Merikangas et al., 2010).

Despite some acknowledged limitations, including restricted age range, lack of full parental reports and the nature of cross-sectional assessment (Merikangas, Avenevoli, Costello, Koretz, & Kessler, 2009), the prevalence rates reported in Merikangas et al. (2010) closely approximate many other research findings over the past 25 years. For instance, Anderson, Williams, McGee, and Silva (1987), using a

community sample of children, found that 55 percent of children meeting criteria for a DSM-III-R disorder met criteria for at least one other diagnosis as well. Similarly, data from the British Child and Adolescent Mental Health Survey in 1999 (Ford, Goodman, & Meltzer, 2003), which surveyed over 10000 children aged 5-15, showed that approximately 30 percent of children meeting criteria for one disorder were diagnosed with at least one more condition (see Angold & Costello, 1993; Angold et al., 1999; Caron & Rutter, 1991; Klein & Riso, 1993 for a full review). Studies investigating the rates of comorbidity among children and adolescents are not as readily available outside predominately English-speaking populations and countries, though those available show similar rates of comorbidity, even if overall prevalence of disorders are higher or lower (Belfer & Rohde, 2005). General prevalence studies in Brazil (e.g. Fleitlich-Bilyk & Goodman, 2004), Yemen (Alyahri & Goodman, 2008) and in Native American populations (Whitbeck, Johnson, Hoyt, & Walls, 2006), as well as studies of specific externalising disorders in Brazil (e.g. Souza, Pinheiro, Denardin, Mattos, & Rohde, 2004), and China (Leung et al., 1996) have consistently shown that the 20 to 40 percent of children meeting criteria for one disorder had comorbid conditions; similar rates as English-speaking samples.

Comorbidity as an "Above-Chance" Phenomenon

It must be acknowledged that by itself, the phenomenon that any given person may legitimately qualify for more than one clinical diagnosis is in itself not particularly interesting. The key to the concept of comorbidity that has captured the interest of psychopathology researchers is that the rates of comorbidity are much more likely than one would expect simply by chance alone; consequently, the number of cases

with only a single disorder are almost universally found to be significantly and substantially less than would be expected by chance (Keiley et al., 2003; Krueger & Markon, 2006). The degree to which comorbidity exists at above chance levels is perhaps best illustrated using the data of Newman, Moffitt, Caspi, Magdol, and Silva (1996) who investigated the prevalence of psychological disorders in adolescents aged 11-21, in a longitudinal study. In their sample, 31.2 percent received a diagnosis of a DSM anxiety disorder, and 21.7 percent received a diagnosis of a DSM mood disorder. If comorbidity were simply a matter of chance alone, the expected rate of co-occurrence of this syndrome pair in the sample, would be the product of 31.2 percent and 21.7 percent, or around 6.8 percent. However, overall rates of co-occurrence for this syndrome pair observed in this study were around 9 percent. Such findings are consistently replicated across a wide range of studies, across a wide range of disorders. This has led to a conclusion that observed comorbidity of these apparently distinct psychiatric disorders far exceeds that expected by chance alone (Wolff & Ollendick, 2006), and it is these non-coincidental levels of comorbidity that has created such substantial interest in the phenomenon.

Homotypic and Heterotypic Comorbidity

The substantially above chance levels of comorbidity is not the only aspect that has created such research interest in the area. Another notable feature of comorbidity is that it affects all diagnostic and syndrome groupings, not just some (Krueger & Markon, 2006). In childhood psychopathology, comorbidity is commonly seen between disorders with similar diagnostic criteria. For example, there is a high level of comorbidity between major depression and dysthymia (Donaldson, Klein, Riso, & Schwarz, 1997). Such comorbidity within diagnostic classes, in this case mood

disorders, is referred to as *homotypic* comorbidity (Angold et al., 1999). In itself, this may not be particularly surprising that major depression and dysthymia are highly comorbid, as both are mood disorders with similar symptoms, and in fact differ more in symptom severity and duration rather than symptomatology. In such a situation comorbidity much be regarded as a failure of categorical diagnoses to adequately describe separable syndromes (Angold et al., 1999; Lilienfeld, 2003), or as Achenbach (1990/1991) eloquently explains it, that existing models of classification are "not carving nature at her [sic] joints" (1990/1991, p. 272; see also Wittchen, Höfler, & Merikangas, 1999; see also later discussion of nosological artefacts).

High levels of comorbidity are also noted between disorders that are within different diagnostic classes. For example rates of comorbidity are quite high between depressive disorders and anxiety disorders, with prevalence rates for children varying between 20 and 75 percent in clinical samples (Angold & Costello, 1993; Angold et al., 1999; Milberger et al., 1995). Anxiety and mood disorders may be seen to be related with research evidence leading to a conceptualisation of these two classes of disorders being part of a higher order factor often called "internalising" disorders (Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Krueger & Finger, 2001). Internalization here is the propensity to express distress inwards, and include mood disorders (e.g., major depressive disorder, dysthymia) and anxiety disorders (e.g., generalised anxiety disorder, separation anxiety disorder, phobias, obsessive-compulsive disorder) among others. Thus such comorbidity between anxiety and mood disorders may be seen as homotypic.

Similarly, research has found that for children with Attention Deficit Hyperactivity Disorder (ADHD), 20-67 percent also meet criteria for Oppositional Defiant Disorder (ODD), and 20 to 56 percent also meet criteria for Conduct Disorder (CD) (Burt, Krueger, McGue, & Iacono, 2003; Mash & Barkley, 2003). Again however, research evidence has led to a conceptualisation of these three classes of disorders being part of a higher order factor often called "externalising" disorders, where externalization describes the propensity to express distress outwards, or behaviourally (Cosgrove et al., 2011; Krueger, 1999; Krueger et al., 1998; Krueger & Finger, 2001).

Research has also demonstrated high levels of *heterotypic* comorbidity, or comorbidity between disorders within different diagnostic classes (Angold et al., 1999). Such an example would be comorbidity between ADHD (externalising) and anxiety or depression (internalising). Research has found that for children with ADHD, 10-40 percent also meet criteria for an anxiety disorder, and a majority of studies finding covariation with mood disorders at between 20 and 30 percent (Mash & Barkley, 2003; Milberger et al., 1995). Similarly Greene et al. (2002) found that more than 30 percent of clinically referred children diagnosed with major depression also met criteria for conduct problems.

Negative Effects of Comorbidity for Individuals

Another reason for continued research interest in comorbidity is that presence of comorbid conditions is associated with a range of negative outcomes for individuals in terms of symptom severity, service utilisation, treatment effectiveness, and prognosis. Research has consistently demonstrated that, compared to individuals with

only a single disorder, individuals with comorbid disorders have increased symptom

severity (Krueger, 1999; Manassis & Menna, 1999), and higher rates of distress from

their symptoms (Andrews et al., 2002; Angold et al., 1998). As an example, Newman

et al. (1996) gathered longitudinal mental health data from ages 11 to 21 and created

a scale of impairment covering seven indices including suicide attempts, need for

medication, and self-reported interference in life activities caused by the illness. This

impairment scale was strongly and positively correlated ($r=.61$) with the absolute

number of diagnoses made.

Comorbidity has also been clearly demonstrated to be associated with increased rates

of service utilisation (Angst, 1996; Kessler, 1995), and significantly poorer treatment

response (Emmanuel, Simmonds, & Tyrer, 1998; Kessler, 1995). The reasons for this

increased service utilisation and poor treatment response are two-fold. In part, this

may be because additional disorders may not be diagnosed and thus remain

untreated. Persons with more than one mental disorder are also likely to have a

greater number of psychological issues to treat, than persons with a single disorder.

Similarly, persons with comorbidity tend to have poorer treatment compliance, with

individuals with comorbid anxiety and depression more likely to terminate

antidepressant treatment than patients with depression alone (Brown, Schulberg,

Madonia, Shear, & Houck, 1996; Davis, Barlow, & Smith, 2010; Hall et al., 2001;

Kendall, Brady, & Verduin, 2001). Prognosis following treatment is also poorer with

comorbid presentations, with relapse more common in comorbid presentations

(Rohde, Clarke, Lewinsohn, Seeley, & Kaufman, 2001). It must be noted however,

that some research has shown that the effects of comorbidity outlined in the past two

paragraphs are not definitive, with some research indicating that persons with

comorbidity do not always show greater symptom severity (Tsao, Lewin, & Craske, 1998; Tsao, Mystkowski, Zucker, & Craske, 2005) and worse treatment outcome (e.g. Olatunji, Cisler, & Tolin, 2010). However, the weight of research does suggest that comorbidity is still more likely to have negative effects for many individuals in terms of severity, prognosis and outcome.

The Need for Explanations of Comorbidity

Given the prevalence of comorbidity, and its impacts, research to find the potential causes of comorbidity has become a major focus of research in psychopathology. Researching this phenomenon is extremely important because it has implications for the validity of past and future classification systems, etiological theories, treatment outcome research, and treatment recommendations (Keiley et al., 2003). However, before substantive explanations can be considered, questions about the reality of the concept arise.

Is Comorbidity Real, Or An Artefact of Nosology?

In examining the comorbidity rates and comorbidity patterns, almost all early studies, and a large number of recent ones have used a categorical approach to measuring the prevalence of psychopathology, and therefore comorbidity. Such categorical approaches have classically been derived from classifications system such as the American Psychiatric Association's Diagnostic and Statistical Manual (DSM) and or the World Health Organisation's International Classification of Diseases (ICD). However, the very use of these classification systems has been argued to be the root cause of comorbidity. That is comorbidity has been argued to be merely an artefact of the classification system used, rather than a 'real' phenomenon.

Comorbidity and Categorical Measurement of Psychopathology

Historically, psychopathology has been conceptualised in terms of putatively distinct categories (Krueger & Markon, 2006)*.* Such systems, often referred to as categorical systems, or medical systems, are categorical-polythetic systems, in that psychopathology is regarded as consisting of a number of categories, though there are multiple ways of meeting criteria for category membership. That is, various combinations of symptoms are sufficient to qualify a person as a member of a category, so long as the correct numbers of symptoms are present. These systems are (almost) always dichotomous in nature, in that an individual either has a disorder if the predetermined criteria for that disorder are met, and does not have the disorder if the predetermined criteria for that disorder are not met. Categorical models are also designed to be inferential in nature, involving the identification of qualitative differences in behaviour that are based on clinical observations and careful history taking to categorise the patient by type of pathology. Even when a specific aetiology cannot be identified, an underlying defect or deficit is often postulated to account for observed signs and symptoms (Ferdinand et al., 2004; Kamphaus, Rowe, Dowdy, & Hendry, 2006).

Categorical systems within clinical psychology are most commonly exemplified by the American Psychiatric Association's Diagnostic and Statistical Manual, now in its revised fourth edition (DSM-IV-TR: American Psychiatric Association, 2000), and at the time of writing soon to be in its fifth edition (DSM-5: American Psychiatric Association, 2013)*,* and the World Health Organisation's International Classification of Diseases, now in its tenth edition (ICD-10: World Health Organization, 1993) with an eleventh edition due in 2015 (ICD-11: see World Health Organization,

2013). Traditionally, categories within these classification systems were initially derived from clinical impressions (Gould, Bird, & Jaramillo, 1993), and are mainly the result of consensus among experts. As a result, disorder categories are defined by a rather arbitrary set of criteria, though over time these categories have been refined through application of experimental paradigms. Furthermore, these systems, as a result of their dichotomous nature, do not allow the provision of more information than the presence or absence of the disorder, and cannot provide information regarding the severity of a disorder, or the number of symptoms met (Ferdinand et al., 2004).

It is the dichotomous nature of DSM-like categorical systems that is most notably critiqued in the literature. Achenbach and McConaughy (1992) noted that the lack of ability to account for severity means that the shift between 'normal' and 'abnormal' cannot be well understood within a categorical framework. As a result, sub-threshold psychopathology, or psychopathology just below the level required for diagnosis, cannot be classified or indeed investigated using such systems (Cantwell, 1996). This is especially true of high prevalence problem behaviours in children (e.g. inattention, hyperactivity, depression), as research indicates that there is no distinct shift from the normal to the abnormal for these behaviours. Rather it appears that these child behaviour problems fall along continua in the population, and thus maybe more appropriately measured using a continuous measurement system, rather than a dichotomous one (Hudziak et al., 1998)

The current categorical systems have also been critiqued based on its inability to discriminate between disorders accurately. According to the DSM-IV-TR (American

Psychiatric Association, 2000), categorical classification is appropriate when all members of a diagnostic category are homogeneous, and when different categories are mutually exclusive with clearly defined boundaries. However, these criteria are not met when using the DSM as a classification tool, with diagnostic categories often showing sub-optimal construct and discriminant validity (Jensen et al., 1996). Indeed the DSM-IV-TR and previous editions, explicitly acknowledge this point by indicating that individuals with the same diagnosis are likely to be heterogeneous (American Psychiatric Association, 2000). As a result, reliability and validity of DSM diagnoses tends to be lower than when using other diagnostic systems (Sroufe, 1997; Widiger, 1992). This, combined with the high rate of atypical presentations, such as those frequently diagnosed with the 'not otherwise specified' label, points to the shortcomings of current categorical psychiatric classification systems, and a lack of fit between the system and clinical reality (Jablensky, 1999a, 1999b). These limitations are directly related to the attempt to argue that comorbidity is purely an artefact of the nosology of the DSM (or ICD) categorical system, though a couple of distinctly different explanations of the cause of this alleged artefact are proposed.

Comorbidity as an Artefact of Symptom Overlap in Categorical Diagnostic Systems?
Several authors have noted that comorbidity could be generated by the fact that individual "nonspecific" symptoms are shared by disparate diagnoses (Caron & Rutter, 1991), with the result that a certain amount of overlap is built into the diagnostic system. In other words, the argument is that comorbidity does not occur at greater than chance rates in reality; rather it is argued that it merely appears to occur at these levels because the diagnostic nomenclature lacks specificity (Wolff & Ollendick, 2006).

Certainly, some pairs of diagnoses do share some overlap in nomenclature. For example, irritability is a common symptom in children with depressive disorders, but significantly is also a common primary or secondary feature of Oppositional Defiant Disorder (ODD: American Psychiatric Association, 2000). If such an overlap was the sole cause of the apparent comorbidity between these two disorders, then the coexistence of the two disorders would be present only in those manifesting the shared symptoms and not in those whose disorders did not involve such symptoms (Angold & Costello, 1993). This can, and has been, easily tested by controlling for overlapping symptoms. Biederman, Faraone, Mick, and Lelon (1995), controlled for overlapping symptoms in ODD and depressive disorders by removing the overlapping symptoms, and then re-evaluating the diagnosis, using the same threshold criteria for diagnosis (e.g. 5 symptoms for a diagnosis of depression). Using this method, called the symptom subtraction method, the rates for comorbidity did decline, though the majority of individuals still maintained their comorbid diagnoses, and the rates were *still* substantially above chance levels. Similar results were obtained by Milberger et al. (1995) looking at comorbidity between Attention Deficit/Hyperactivity Disorder (ADHD) and other disorders, leading to a conclusion that comorbidity rates, while perhaps inflated by the non-specific nomenclature, were not just an artefact of overlapping diagnostic criteria.

Despite this evidence, some discussion in the literature has focussed on the fact that there were notable drops in the rates of comorbidity when using the symptom subtraction method. It has been suggested that this could still indicate that the vast majority of comorbidity may still be an artefact of nosological overlap (see

Biederman et al., 1995; Milberger et al., 1995). However, this substantial drop is actually more an artefact of the subtraction method used in the above cited studies, rather than evidence for overlapping nosology. Milberger et al. (1995) argue that the subtraction method is overly stringent, because while it reduces the number of possible symptoms that can be endorsed, it does not equivalently reduce the number of symptoms that need to be endorsed for a diagnosis. For example, the criteria for Generalised Anxiety Disorder (GAD) require three of a possible six symptoms for diagnosis in adults (50%), or one of a possible six symptoms for diagnosis in children (16%). However, if using the subtraction method to correct for the two overlapping symptoms of with ADHD, the stringency of the criteria for diagnosis increases to three of a possible four remaining symptoms for adults (75%), or one of a possible four in children (25%). This increase in stringency can be alleviated using an alternate method, called the proportion method (Milberger et al., 1995; Spencer et al., 2001). In the proportion method, overlapping symptoms are still not counted, but the diagnostic threshold is lowered to require that the same proportion of symptoms from the reduced set as is required for the original diagnosis (Spencer, et al., 2001). In the example cited above, this would mean that only two of the remaining four symptoms would be required for a diagnosis of GAD in adults.

Milberger et al. (1995) used this proportion method to assess the influence of overlapping ADHD symptoms on the diagnosis of comorbid disorders for a range of DSM-III-R disorders, and found 98 percent of children maintained a diagnosis of ADHD when overlapping depression symptoms were removed, with 83% maintaining their diagnosis of major depression when overlapping ADHD symptoms were removed. Similar results were found in adult samples as well. These results,

taken together indicated that while nosological overlap might create a slight inflation to the comorbidity statistics in certain cases, the comorbid conditions themselves were not an artefact of symptom overlap (Milberger et al., 1995).

Indeed, to propose such a hypothesis to explain all comorbidity is misleading. When considering the broader range of psychopathology, there is a lack of possible nosological overlap for many diagnostic pairs. For example, when considering conduct disorder (CD) and depressive/anxiety disorders there is no overlap in diagnostic criteria (though some associated features can be similar), and given that CD has substantive comorbidity with depressive disorders and anxiety disorders, overlapping symptom lists cannot be used as a plausible explanation (Angold et al., 1999). Indeed the diagnosis of a disorder almost exclusively requires multiple symptoms to be present, making this explanation an unlikely candidate for explaining the comorbidity (Wolff & Ollendick, 2006; Zoccolillo, 1992). Thus while some overlap may be perceived, the vast majority of disorders have an acceptable, if imperfect level of specificity.

Comorbidity as an Artefact of the Artificial Nature of "Splitting" Categories?
There have also been arguments put forth in the literature that comorbidity reveals fundamental problems in the way that contemporary nosologies attempt to categorise psychopathology (Caron & Rutter, 1991). DSM or ICD diagnoses are argued to superimpose artificial distinctions on an existing category, because of a convention to define diagnostic entities into many specific narrowly-defined disorders, or engage in 'splitting', rather than defining diagnoses into a few broadly-defined categories or 'lumping' (First, 2005; Meehl, 1995, 2001). Figure 1.1 outlines the conceptual

difference between the 'splitting' and 'lumping' approaches to dividing clinical

presentations. The left hand side of Figure 1.1 illustrates a diagnostic system that

'splits' the 'diagnostic pie' into many narrowly-defined disorders, along the lines of

the strategy used by DSM/ICD. The right hand side outlines a diagnostic system that

'lumps' presentations into four broadly-defined diagnostic categories.
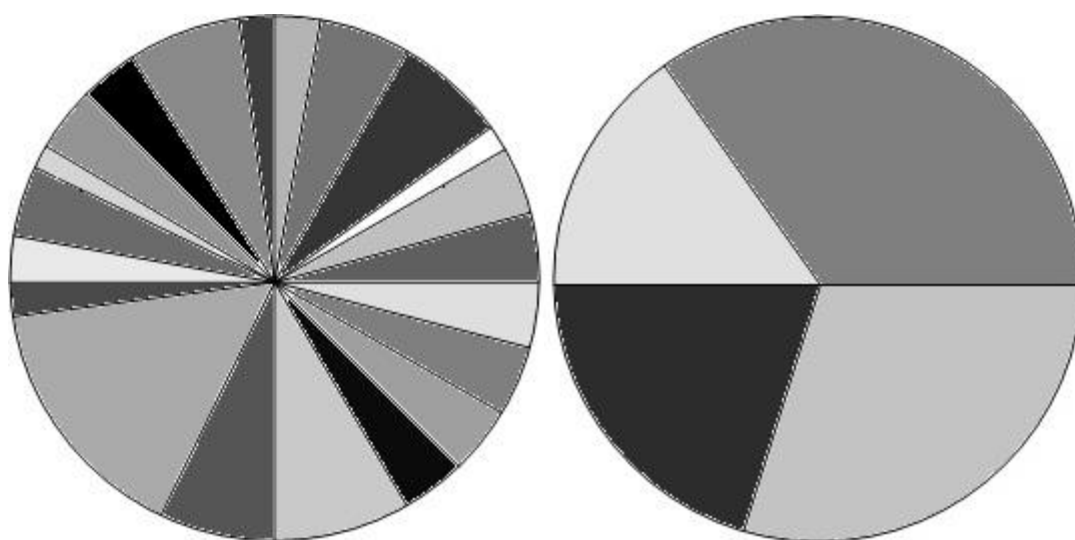
Figure 1.1. Diagrammatic conceptualisation of the 'splitting' (left panel) and

'lumping' (right panel) approaches to the division of clinical presentations.

Probably the most obvious example of 'splitting' in the DSM relates to the substance

dependence disorders. DSM divides substance dependence into ten categories based

on the class of substance so that a patient who is dependent on three substances gets

three comorbid diagnoses (e.g., opioid dependence, cannabis dependence, cocaine

dependence) rather than a single diagnosis of 'multi-substance dependence' (First,

2005; Hall et al., 2001). Such issues also occur in disorders more likely to be found

in childhood, such as the mood disorders where Major Depressive Disorder and

Dysthymia are considered separate disorders (Donaldson et al., 1997), despite the fact that they share the same basic features of depressed mood (American Psychiatric Association, 2000). It is indeed very notable that Major Depressive Disorder and Dysthymia are highly comorbid (Donaldson et al., 1997).

There are many arguments, and a considerable evidential base to support the idea that comorbidity is a pure artefact of the way the diagnostic categories are 'split'. At its simplest, Maj (2005a, 2005b) argues that the mere fact that some disorders rarely occur in isolation is indicative of a system imposing categorical distinctions that do not exist in nature (Meehl, 2001). Further to this argument is that certain comorbidities, like that between major depression and dysthymia, are markers of a single disease process. Some studies in adults suggest that major depression, dysthymia, and the apparent comorbidity of the two, represent separate manifestations of the same disorder (e.g. Keller et al., 1992; Klein, 1990), while others have suggested that comorbidity between major depression and dysthymia is merely a marker of increased severity (Angst & Dobler-Mikola, 1984). While arguments against comorbidity being an artefact of the artificial nature of diagnostic "splitting" can be made, the overall hypothesis is not easily rebutted when considering comorbidity purely within classic categorical systems. However, arguments against comorbidity being an artefact of nosology are more easily made when considering other diagnostic systems.

Dimensional Measurement of Psychopathology

Categorical systems such as those found in the DSM are not the only approach to the measurement of psychopathology. Alternative classification schemes, often referred

to as dimensional systems, utilise a multivariate statistical approach, hence they are often referred to as "empirical," "quantitative," or "statistical" systems. Dimensional classification methods have their roots in psychometric assessment, and are developed on the assumption that there are a number of behaviour traits that all individuals possess. It is argued that these traits exist along a continuum (Kamphaus & Frick, 2002), with individuals varying to degrees and existing anywhere along these dimensional continua (Scotti & Morris, 2000). Dimensional systems thus attempt to measure latent traits/constructs, based on input from multiple indicators (or items) of behaviour.

As a result of the underpinnings of dimensional systems, psychopathology within this system is viewed as a quantitative deviation (e.g. extreme score) from normal, rather than as a discrete disorder (Edelbrock & Costello, 1988). Unlike categorical systems, these traits or dimensions of behaviour are typically derived from quantitative measures, such as behavioural rating scales or symptom inventories completed by parents, teachers, or adolescents. Through the use of multivariate statistical procedures such as cluster analysis and factor analysis (Gould et al., 1993), individual items on these scales are clustered to form specific narrow dimensions. These dimensions summarise information about a group of descriptive variables, into an abstract, higher order variable (Blashfield, 1998). Sometimes the narrow dimensions are further grouped into superordinate broad factors (e.g. internalising, externalising), with the purpose of translating underlying latent traits into a relatively small number of categories to offer an effective communicative tool, and an alternative to existing classification schemes such as the DSM-IV-TR (Kamphaus et al., 2006). In effect, dimensional systems view behaviours in children such as

inattention, hyperactivity and depression not as disorders, but as latent constructs that are more or less characteristic of individuals.

Advantages of Dimensional Approaches

There are certainly some distinct advantages of such dimensional approaches. These approaches, because of their empirical derivation, have demonstrated greater predictive validity (Fergusson & Horwood, 1995) and statistical reliability (Cantwell, 1996) than categorical models, as well as producing more reliable, homogeneous groupings. This is in part because they minimise the need for clinical judgment and inference (Achenbach, 1990/1991; Jensen et al., 1996), and that cut-off points are not referenced against an arbitrary number of criteria, but rather against the general population (Gould et al., 1993). As a result, decisions on the number and severity of problems can be based on actual distributions of scores in populations rather than on arbitrary criteria (Ferdinand et al., 2004). Such empirically based scales also avoid any sense of nosological confusion by using statistical methods to identify distinct entities, and separate symptoms, such that they are only identifiable within one syndrome. That is symptoms that cross-load onto multiple syndromes are eliminated from consideration as they do not allow determination of distinct entities (Kendall et al., 2001).

However, dimensional methods are not consistently used, nor widely sanctioned for use to diagnose mental disorders, mostly because they have not been fully incorporated into the predominant DSM/ICD systems (Kamphaus et al., 2006). This is a result of the limitations of the dimensional models of classification, most pertinently the lack of consensus regarding the optimal dimensions that should be

used for classification purposes (Clark, Watson, & Reynolds, 1995). Dimensional

models have also been critiqued because of a lack of a theoretical basis for forming

the dimensions, and reliance on statistical distributions, rather than impairment in

determining disorder membership (Burger & Neeleman, 2007; Gould et al., 1993),

and for their inability to assess rare psychopathologies (Achenbach, 1980; Edelbrock

& Costello, 1988). However, they provide another system within which the concept

of comorbidity can be considered, and this warrants further consideration.

Comparing Categorical and Dimensional Measurement of Psychopathology

Before considering comorbidity within dimensional models however, it is necessary

to consider another issue. Given their clear divergence in development and

methodology, the question arises: is there any similarity between categorical and

dimensional measurement of psychopathology? Ideally if comorbidity is to be

considered in the context of two separate nomenclatures, then some similarity

between them would be advantageous, otherwise any explanations related to the

cause of comorbidity could not be universal, but rather be specific to a particular

classification system.

Certainly research suggests there is far more similarity than divergence. Edelbrock

and Costello (1988) investigated the correlation between DSM-III Axis I diagnosis

derived from the diagnostic interviews and scores from an early version of one of the

more popular, widely used, and well validated behaviour problem scales; the Child

Behavior Checklist (Achenbach & Edelbrock, 1983). Point-biserial correlations

between each DSM diagnoses and scores for CBCL syndromes suggest substantial

convergence between two different approaches. Similar findings were ascertained by

Gould et al. (1993), and Jensen et al. (1996) who further noted that there was no

evidence of the superiority of either system, and that "controversies about 'best' assessment strategies may be artificial" (p. 151). Indeed Kamphaus et al. (2006) argues that both categorical and dimensional methods both reasonably, if imperfectly, attempt to categorise individuals into homogeneous groups, which is the ultimate goal of classification or diagnosis. As a result, arguments that these approaches are entirely discrete are overstated.

Comorbidity within Dimensional Classification Systems

Given that dimensional systems allow a different approach to investigating comorbidity, but show some relationship to categorical systems, the existence of comorbidity within dimensional systems must be considered. Findings similar to those found in research using dimensional systems are found in empirically derived syndromes, such as those from the Child Behaviour Checklist (CBCL: Achenbach & Rescorla, 2001). McConaughy and Achenbach (1994) investigated the comorbidity between the eight empirically based CBCL syndromes in matched general population and clinical samples of over 2500 children aged 4-18. They found significant correlations between all CBCL syndromes, indicating consistent comorbidity across the broad spectrum of empirically-based dimensional psychopathology. Similarly, Döpfner et al. (2009) found rates of CBCL syndrome co-occurrence of between 7 and 34 percent. Indeed the Döpfner et al. (2009) study, which used a German general population sample of 4- to 18-year-old children, clearly shows the cross-cultural generalizability of comorbidity within dimensional models, just as noted within categorical models. The rates of such co-occurrence in statistically derived syndromes are also fairly consistent with those using DSM categories, in both English and non-English speaking populations (e.g. Crijnen, Achenbach, & Verhulst;

X. Liu et al., 2001; McConaughy & Achenbach, 1994; Verhulst & van der Ende, 1993; Weiss, Süsser, & Catron, 1998; Yang, Chen, & Soong, 2001). Rates of comorbidity also do not seem to vary greatly as a function of the empirical system used, with Ruchkin, Sukhodolsky, Vermeiren, Koposov, and Schwab-Stone (2006) finding co-occurrence rates using the Behavior Assessment System for Children syndromes (BASC: Reynolds & Kamphaus, 1992) to be similar as for the CBCL based syndromes. Heterotypic comorbidity is also found for empirically derived syndromes with many studies showing weak to moderate correlations between internalizing and externalizing latent factors in the confirmatory factor analytic studies with adults and adolescents (Cosgrove et al., 2011; Krueger, 1999; Krueger et al., 1998).

Comorbidity also exists at above chance levels when considered within dimensional classification systems as illustrated using the data of Döpfner et al. (2009). Döpfner et al. (2009) found 4 percent of adolescents were within the clinical range of the delinquent (rule-breaking) behaviour, and 5.1 percent were within the clinical range for aggressive behaviour. Again if comorbidity were simply a matter of chance alone, the expected rate of co-occurrence of this syndrome pair in the sample, would be the product of 4 percent and 5.1 percent, or around 0.2 percent. However, overall rates of co-occurrence for this syndrome pair observed in this study were over 20 percent. Most research findings show comorbidity at similar rates, and at significantly above chance levels, whether considered in terms of dimensional systems or categorical systems.

Resolving Nosology as a Cause of Comorbidity: Empirically Validated Systems

The fact that comorbidity is found at significantly above chance levels within dimensional, empirically based systems also allow the rejection of the arguments made that comorbidity is *purely* an artefact of nosology. While again it must be acknowledged that the arguments for comorbidity being an artefact of a lack of nosological clarity are worth noting, and indeed may create a degree of statistical inflation in the rates of comorbidity within categorical systems, they cannot be considered as a primary explanation of comorbidity. It is certainly accurate to suggest that a small degree of artefactual comorbidity may result from the type of classification system used in a given study, whether this is from the inadequate nature of the "splitting" within nosological systems, or from symptom overlap between diagnostic entities (Caron & Rutter, 1991). However, as indicated earlier, empirically based scales avoid any sense of nosological confusion by using statistical methods to identify distinct entities, and separate symptoms such that they are only identifiable with one syndrome. Given comorbidity still exists within dimensional, empirically based systems, we can be certain that comorbidity still exists despite the nosological issues outlined above.

Comorbidity or Co-Occurrence? Further Debate over Nomenclature

Further to the definitional confusion outlined earlier in the chapter is that "comorbidity" and "co-occurrence" are often used to separate the idea of diagnostic disorder entities such as those in the DSM-IV-TR (American Psychiatric Association, 2000) and factor-analytically derived syndromes, such as the Achenbach System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001). In this context, comorbidity refers to coexistence of DSM-IV type

disorders, and co-occurrence to the coexistence of factor-analytically derived
syndromes. For the purposes of this thesis, such a distinction between comorbidity
and co-occurrence will be employed where appropriate, but such definitional
differences do not substantively alter the original definition of Feinstein (1970), as it
is equally applicable to co-existing clinical disorders (e.g. ADHD, Generalised
Anxiety Disorder), derived from a classification system such as the DSM-IV-TR
(American Psychiatric Association, 2000) and or ICD-10 (World Health
Organization, 1993), or to empirically derived clinical syndromes (e.g. attention
problems and anxiety problems) such as those from the Achenbach Scales of
Empirically Based Assessment (Achenbach & Rescorla, 2001).

Summary

The previous sections have outlined a definition of comorbidity and outlined that its
above chance level of occurrence makes it a worthy area of substantive investigation.
Almost all modern epidemiological or clinical studies have shown that there is a
relatively high degree of overlap, regardless of whether the psychopathology is
assessed using dimensional or categorical systems (Andrews et al., 2002; Döpfner et
al., 2009; Merikangas et al., 2010; Weiss et al., 1998). Within the literature, evidence
suggests that comorbidity rates are even higher in childhood and adolescents than in
adults, and the patterns of comorbidity are different from those of adults (Yang et al.,
2001). Indeed the negative effects of comorbidity, in terms of severity, prognosis,
and treatment outcome for individuals, means that understanding the root causes of
comorbidity is vital for the development of clinical psychology. The previous section
has also demonstrated that arguments that comorbidity is entirely an artefact of

nosological imprecision are spurious. As a result substantive explanations for the
aetiology of comorbidity must be considered.

Is Comorbidity An Artefact of Research Methodology?

Before considering substantive explanations however, Rutter and Sroufe (2000)
argue that one main question that must be addressed is whether comorbidity may be
an artefact of methodological problems in research; that is, it is artificially created by
problematic research techniques? This differs from the previous arguments
surrounding nosology, because the arguments centre not on the systems used to
assess psychopathology, but on the research methodology employed in investigating
comorbidity. Such discussions are considered here because they are equally relevant
to both categorical and dimensional nomenclatures, and thus affect *all* research on
comorbidity. Certainly many researchers have argued that comorbidity is merely an
artefact of the research methodologies used to measure the prevalence of mental
illness, while others have argued that comorbidity is at least in part due to the
methodologies employed (see Lilienfeld, 2003, for a review). A number of potential
methodological causes for comorbidity have been hypothesised, include sampling
biases, method covariance, and issues surrounding the use of bivariate statistics when
comorbidity is a multivariate construct. Each major explanation shall be discussed
presently.

Selection and Berksonian Biases?

Early research in comorbidity was often based on clinical samples (Angold et al.,
1999). Since this research did not use population-based epidemiological samples,
there was considerable argument that the findings of above-chance levels of

comorbidity were likely to be an artefact of selection bias and, or Berkson's bias, which produce higher comorbidity rates in a clinic population than those that existed in the general population. Berksonian bias (Berkson, 1946) is mathematical artefact derived from the fact that an individual with multiple disorders can obtain treatment for any disorder(s). To illustrate Berkson's bias, suppose that a person with particular disorder, X, has a probability of referral p_a, and a person with a different disorder, Y has a probability of referral p_b. This would mean that a person having both disorders X and Y, would have a probability of referral of $p_a + p_b$. This higher probability of referral for people having both disorders would increase their likelihood of being selected in a clinical sample, unless the sample contained all subjects with a particular disorder; a practical impossibility (Caron & Rutter, 1991). By nature therefore, such a sample would inflate the estimate of comorbidity above what would be found in non-referred or community-based populations (Achenbach, 1990/1991). A related concept is referral bias, also known as clinical selection bias (e.g. Du Fort, Newman, & Bland, 1993), and results from the fact that individuals with two (or more) disorders may be especially impaired and therefore more likely to seek treatment than are individuals with only one disorder. Although some authors (e.g. McConaughy & Achenbach, 1994) do not differentiate Berksonian and clinical selection bias, they are subtly different and are worth differentiating on methodological and theoretical grounds. (Lilienfeld, 2003).

Certainly, there is some evidence implicating these biases in the inflation of comorbidity statistics. Children with comorbid conditions are significantly more likely to present to treatment facilities than children with only one condition (Costello et al., 1996). Indeed, as discussed previously, in clinical settings,

individuals with a single diagnosis are very rare (Brown, Campbell, Lehman,

Grisham, & Mancill, 2001). Similarly, clinic-referred individuals usually have more

severe symptomatology, and are more impaired (e.g. Angold et al., 1998). Finally,

the rates of comorbidity found in research using clinical samples always tend to be

significantly greater than in research using general population samples (Angold et al.,

1999). Certainly, there is clear evidence that Berksonian and clinical selection biases

are at play in comorbidity research.

However, of themselves, Berksonian and clinical selection biases cannot adequately

account for comorbidity. This is because data from general population studies, such

as outlined earlier in the chapter (e.g. Döpfner et al., 2009; Merikangas et al., 2009),

also find significant and substantial heterotypic and homotypic comorbidity/co-

occurrence, and this indicates comorbidity is present at all levels of

symptomatological severity. Angold et al. (1999) noted in their meta-analytic review

of comorbidity that although referral biases certainly increase reported rates of

comorbidity, the simple fact that comorbidity is found in nonclinical samples

strongly suggests that there is more than simply Berksonian and clinical referral bias

artefacts (Baldwin & Dadds, 2008; Lilienfeld, 2003; Milberger et al., 1995).

Method Covariance

Method covariance, in terms of comorbidity, occurs if the correlation between two

disorders may be partly attributed to correlated error (Brown, 2006; Lilienfeld,

2003). These correlated errors could result when the measurement of each disorder is

derived from a shared mode of assessment. In childhood psychopathology research,

parent report is the most common form of assessment used in diagnosis, and this may

produce inflated estimations of comorbidity. This is because parent reporters (or any others) may be more likely to report a second illness in the presence of another, or may biasedly report on symptomatology because of personal views about the nature of association between two disorders, thus inflating estimates of comorbidity (Baldwin & Dadds, 2008). A wide range of research however, mostly using Structural Equation Modelling (SEM) techniques, has shown that the effects of method covariance, while present, are minimal. SEM allows the investigation of the effects of method covariance, by allowing error components to correlate and thus the effect of this correlation to be estimated. Research, such as that by Keiley et al. (2003), has consistently shown the comorbidity is still present when using procedures to control for method covariance. As a result, comorbidity cannot be viewed as a pure function of method covariance.

Indeed, it should be noted that such biases can be bidirectional in nature, as method covariance can just as equally lead to a deflation of correlation estimates, as it can lead to inflation of correlation estimates. For example, there has been a noted under-identification of inattention in girls who exhibit no other problematic behaviour (Gershon, 2002). Similarly, teachers have been shown to only notice inattention in the context of disruptive behaviour. Thus method covariance, while potentially affecting the precise statistical values of comorbidity, cannot be seen as a causal effect of comorbidity, and thus further explanations must be found.

Artefacts from Using Multiple Informants

Lilienfeld (2003) noted that over the past few decades, researchers have recommended multi-informant assessments to obtain a complete diagnostic picture of

children (see also Mash & Barkley, 2003; Ollendick & Hersen, 1993). This has been in part a way of overcoming issues related to method covariance, as outlined above, as well as providing a triangulation in diagnosis. However, Jarrett and Ollendick (2008) argue that the use of multiple informants in itself may create serious problems when it comes to assessing comorbidity. In particular, reliance on varying opinions and interpretations of problem behaviour means that information must be combined, and some decision making process is required (Youngstrom, Findling, & Calabrese, 2003). Wolff and Ollendick (2006) indicated that an 'or rule' has often been used in research, in that a disorder is considered present if one source endorses the disorder, even if the other does not. However, this means that a single behaviour could be interpreted differently by different informants, and thus two disorders could be endorsed based on interpretations of the one behaviour (Wolff & Ollendick, 2006). Similarly, informant sources could be rating behaviours that are only exhibited in one setting (e.g. home, school, etc.), and are thus may be a situationally specific response to an environment and not necessarily indicative of psychopathology (Jarrett & Ollendick, 2008).

Angold et al. (1999) outline several cogent reasons why multiple informant use is not the reason for above chance rates of comorbidity. Firstly, they note that adult studies also find high rates of comorbidity despite the propensity for adult studies to rely on a single informant. Similarly, studies using only child self-report (e.g. Lewinsohn, Klein, & Seeley, 1995; Rohde, Lewinsohn, & Seeley, 1996) or comparing differences in the number of comorbid diagnoses reported by parents and children find no substantive difference in comorbidity rates (Jensen et al., 1999; Jensen et al., 1995) though such findings are not always completely universal (see Youngstrom et

al., 2003). Thus on the whole, it cannot be argued that comorbidity is the result of the use of multiple informants and indeed Wolff and Ollendick (2006) argue that estimates of co-occurrence may be *more* accurate when based on multiple sources of information, because of the potential biases evident from use of a single reporter (as outlined in previous section).

'Epiphenominal Correlations'

Several researchers, such as Angold et al. (1999), have suggested that the high comorbidity rates and correlations found in epidemiological and clinical samples may be in fact epiphenomenal in nature. By epiphenomenal, it is argued that the real rates of the correlation/comorbidity/co-occurrence are in fact at chance levels, because the correlation of two disorders/syndromes may be explained by the correlation of each of them with one or more other syndromes (Döpfner et al., 2009).

Usually, quantification of comorbidity/co-occurrence has been made using pairwise concordances among diagnoses. However, if using pairwise concordances/correlations when three conditions are statistically associated, it is possible that one pairwise association is purely a mathematical function of the pairwise associations between the other two. This possibility is referred to as epiphenomenal comorbidity (Angold et al., 1999). This possibility has been directly investigated by several child/adolescent psychological researchers. Döpfner et al. (2009), in a study of child and adolescents, investigated this possibility using partial correlations among CBCL syndrome pairs, and showed that many partial correlations between syndromes, which by nature exclude influences of other syndromes, are much lower than base correlations. This was especially true for heterotypic co-

occurrences. Similarly, Ford et al. (2003), in a cross-sectional study of designed to describe the prevalence of DSM-IV disorders in British children and adolescents, noted that after adjusting for the presence of a third disorder, there was no longer significant comorbidity between some disorder pairs such as anxiety and conduct disorder.

Two points regarding these and similar studies must be noted however. Firstly, though this was more noticeable for homotypic than heterotypic co-occurrences, the partial correlations found by Döpfner et al. (2009), and Ford et al. (2003), were still non-zero and still above chance in almost all cases, and thus stating that the correlations between pairs is entirely epiphenomenal does not hold entirely true. Secondly the use of a rather 'blunt' statistical instrument such as partial correlations must be critiqued. While the statistical approach can be deemed appropriate, the use of correlations, even partial correlations, can still be considered to be a use of bivariate-style statistic in attempting to understand multivariate comorbidity. The use of specifically multivariate modelling techniques, such as SEM, is a far more sophisticated way of assessing the potential for epiphenomenal correlations. Partial correlations are still conducted one symptom pair at a time, whereas when using SEM, all disorders can be considered *simultaneously*. This provides a more comprehensive explanation of comorbidity than use of other statistical techniques. Batstra et al. (2002) note that when multivariate analysis techniques that take epiphenomenal associations into account are used, truer comorbidity/co-occurrence rates can be estimated, and in fact are still found above that which would be seen to be *purely* epiphenomenal. Thus while comorbidity/co-occurrence rates may be

inflated by the use of pairwise concordances, there is still some degree of comorbidity/co-occurrence that needs substantive explanation.

Summary

This prior section has reviewed explanations of comorbidity as being an artefact of methodology. While there is some merit to these explanations, the possible methodological explanations for comorbidity, singularly or collectively, cannot be demonstrated to be the entire cause of the broad spectrum of the comorbidity phenomenon (Angold et al., 1999). As such we can be sure that substantive explanations for comorbidity need to be sought. In the next section, some potential substantive explanations are outlined, along with the need to extend current substantive explanations to embrace multivariate models.

Substantive Explanations for Comorbidity

Given that arguments regarding comorbidity as an artefact of nosological imprecision or methodology are specious, there is a specific need to consider more substantive explanations of comorbidity. The purpose of this section is to carefully examine some of the more common substantive explanations for observed comorbidities. There have been many substantive explanations proposed, though they generally fall into three main categories. The main categories of explanation proposed include: a) one disorder causes another; b) that comorbid disorders should be treated as a third independent disorder; and c) that comorbidity is caused by shared etiological factors.

Each of these explanations will be discussed in turn. The key point for each of these categories of proposed explanation, is that they are equally valid for both categorical comorbidity between DSM or ICD based disorders within categorical diagnostic systems, and for covariation between empirically validated syndromes within dimensional diagnostic systems. Indeed the evidence for and against each of these theories is drawn from studies using both DSM disorders and empirically validated syndromes. It is also important to note, that despite over two decades of research, the study of comorbidity in terms of its aetiology and development is still what Loeber, Burke, Lahey, Winters, and Zera (2000) describe as an "embryonic state" (p. 1475), and thus the research evidence for these theories is not comprehensive.

Disorder A Causes Disorder B: Is Comorbidity a Result of One Disorder Causing (or Putting An Individual at Risk For) The Other Disorder?

One of the initial and most common substantive explanations for comorbidity is that comorbidity occurs because one disorder causes or puts an individual at risk for the other (Lilienfeld, 2003). In effect this is a purely causative theory. Within this explanation however, there exists three potential associations between any pair of disorders (Wolff & Ollendick, 2006). Firstly, Disorder A may be a direct cause of Disorder B. Secondly, Disorder B may be a direct cause of Disorder A. Thirdly, Disorder A and Disorder B may reciprocally cause each other, influencing each other in a transactive fashion. Despite the three potential associations however, the underlying assumptions for each explanation are the same; one disorder is seen to temporally precede the other, and resultantly puts an individual at elevated risk for the development of the second disorder (Seligman & Ollendick, 1998).

Empirical Support for Causative Models

Certainly this group of explanations do have some empirical support. For example, looking at the notable heterotypic comorbidity between ADHD and anxiety, it is difficult to conceptualise how anxiety in a child could cause the development of ADHD. However, it certainly has been shown that ADHD in children can significantly increase the risk of anxiety symptoms. Biederman et al. (1996), following a group of children and adolescents over four years, showed the prevalence for developing anxiety disorders over the four year period was substantially higher for children with an initial ADHD diagnosis, than those without. Many potential pathways for this increased risk have been outlined (see Baldwin & Dadds, 2008), but there is some support for the idea that mediators such as peer rejection may play a part, with heightened levels of peer rejection for boys with ADHD being related to higher rates of anxiety disorders (Greene, Biederman, Faraone, Sienna, & Garcia-Jetton, 1997). However, the relationship between ADHD and anxiety may not be unidirectional. Anxiety has been shown to significantly alter the expression of ADHD with Pliszka (1989) showing that children with comorbid anxiety and ADHD can be less impulsive than children with ADHD and no anxiety disorder. Baldwin and Dadds (2008) also argue that it is possible that anxiety early in life might lead to decreased concentration and consequently lead to ADHD-like symptoms of inattention.

Similarly, Patterson and Capaldi (1990) proposed a similar causative model for the relationship between Conduct Disorder and anxiety. They posited that conduct problems lead to failures in social situations that gradually lead to depression and anxiety. Capaldi (1992) found support for this hypothesis in that sixth grade boys

with conduct problems showed significantly more depressed mood symptoms in

eighth grade, with no reciprocal between sixth grade depressed mood and eighth

grade conduct problems existing. A related theory, Glaser's (1967) theory of masked

depression, has been tested by Ritakallio et al. (2008). This theory suggests

depression precedes CD/ODD because depressive symptoms lead to acting out

behaviours, in part because of a paradoxical decreased concern about the adverse

consequences of antisocial actions (Capaldi, 1991). Ritakallio et al. (2008) found that

depression predicted subsequent antisocial behaviour among girls, but conversely,

antisocial behaviour did not predict subsequent depression in a 2-year prospective

follow-up study.

Empirical support for the causative models has also come from studies using SEM

techniques. Neale and Kendler (1995) investigated the comorbidity between Major

Depressive Disorder (MDD) and Generalised Anxiety Disorder (GAD) using models

for this explanation drawn from the work of Klein and Riso (1993). Their study,

using 2,163 female twins from the population-based Virginia Twin Register

(Kendler, Neale, Kessler, Heath, & Eaves, 1992), showed mixed findings. Certainly

a causal model of MDD causing GAD showed adequate fit, and was a parsimonious

model. However, causal models of GAD causing MDD and of reciprocal causation

fitted very poorly.

Evidence Against Causative Models

While there is some evidence that the causative model may hold true for some

disorder pairs, the model has been investigated for many disorder pairs, and does not

hold well across all possible comorbidities. The above cited research by Neale and

Kendler (1995; see also Rhee et al., 2008) showed there were other better fitting

models which explained the data more effectively, and parsimoniously. Indeed, this

theory, while simple to model when considering disorder pairs in isolation, is not at

all parsimonious when considering all comorbidity or co-occurrence as a whole, as

will be discussed later.

Another problematic aspect of the theory relates to the issues raised by Seligman and

Ollendick (1998). For causative models to be considered accurate there must be clear

evidence that one disorder temporally precedes the other. One often investigated

example of comorbidity that highlights this, is the comorbidity between conduct

problems and depression. The National Comorbidity Survey Replication study

(Nock, Kazdin, Hiripi, & Kessler, 2006) showed that for around three-quarters

children showing comorbid conduct problems and depression, conduct problems

manifested first (Nock et al., 2006), a commonly found result (Biederman et al.,

1995). However, this finding is not universal with Kovacs, Paulauskas, Gatsonis, and

Richards (1988) finding that it was more common for depression to manifest prior to

conduct disorder. Contrary to both findings, more recent studies (e.g.Lahey, Loeber,

Burke, Rathouz, & McBurnett, 2002) suggest that the relationship between the

disorders is reciprocal with the severity in one disorder positively related to the other.

Thus in summation, while there is some evidence that could be used to suggest that

the simple causative theory may be plausible for the relationship between conduct

disorder and depression, the fact that no clear temporal link can be identified is

problematic. Such lack of temporal specificity is also noted across a range of

disorders (Seligman & Ollendick, 1998).

Furthermore, Fergusson, Lynskey, and Horwood (1996) demonstrated that much of

the relationship between conduct problems and depression can be better explained by

the presence of common risk factors than causality. Indeed this is evidently plausible,

because any causal links between disorders, such as that between ADHD and anxiety

or CD and depression anxiety outlined earlier, are almost exclusively explained in

terms of (an)other mediating variable/s (e.g. peer rejection), and as will be discussed

later, the idea that it is these risk factors that are causal, rather than just mediative,

shows greater support. A similar issue has been encountered in the prior mentioned

example of ADHD and anxiety (Baldwin & Dadds, 2008). Thus, there is increasing

evidence to suggest that simple causal hypotheses may not easily explain the

comorbidity/co-occurrence in totality (Teesson, Degenhardt, Proudfoot, Hall, &

Lynskey, 2003), and other models are sought.

Three Independent Disorders Model: Are Comorbid Disorders in fact a Single Separate Disorder Rather Than two Co-Occurring Disorders?

In the previous sections of this chapter, a methodological argument for comorbidity

was discussed that suggested comorbidity was resultant from the artificial

distinctions made through the current nosological tendency to 'split' disorders by

narrowly defined them, rather than defining diagnoses into a few broadly-defined

categories or 'lumping' (First, 2005; Meehl, 1995, 2001). While this nosological

argument has been refuted as an entire cause of comorbidity, some childhood

research suggests an alternative to the above argument that is in fact substantive in

nature rather than purely nosological. This argument proposes that it may be that the

nature of the splitting, not the fact that there is splitting in nosology that is the

problem. Under this proposal, comorbidity does not represent the combined presence

of two disorders, but rather a third distinct disorder. This model, often referred to as the three independent disorders model, proposes that comorbidity is in fact a misleading concept because the apparent comorbid disorders are in fact a single disorder separate from either disorder occurring alone. For instance, researchers have suggested that ADHD and bipolar disorder occurring together may represent a nosologically distinct entity from other ADHD subtypes (Faraone, Biederman, Mennin, Wozniak, & Spencer, 1997; Wozniak, Biederman, Mundy, Mennin, & Faraone, 1995). Similarly, in the previously cited case of the common comorbidity between major depression and dysthymia, it has been suggested that a diagnostic category of "double depression" be considered (Kovacs, 1996; Kovacs et al., 1984).

Empirical Support for Three Independent Disorders Model

Some support for this theory comes from the work of Biederman and Faraone and colleagues (Biederman et al., 1992; Faraone, Biederman, Jetton, & Tsuang, 1997; Faraone, Biederman, Keenan, & Tsuang, 1991; Faraone, Biederman, & Monuteaux, 2000) across nearly a decade of family based genetic research investigating the aetiology of comorbid ADHD and CD in families. They repeatedly found that relatives of persons with comorbid ADHD and CD had significant cosegregation of ADHD and CD. Cosegregation is a genetic concept, which refers to situations where genotypes have a heightened tendency to be inherited together. In this case, relatives of persons with comorbid ADHD and CD were more likely to develop both disorders than just one, providing support for the aetiological distinctness of ADHD, CD and comorbid ADHD and CD as three distinct conditions.

Evidence Against Three Independent Disorders Model

The evidence for the three independent disorders model is sparse however. If this theory were to be true then the correlates of the combined disorder should be different to that of the independent disorders. Put another way, if this theory is not accurate, and two distinct disorders exist, correlates of both disorders should be evident irrespective of whether there is comorbidity. Biederman and colleagues (Biederman et al., 1999; Biederman et al., 1997), investigating the comorbidity between Bipolar Disorder (BPD) and CD in youth made many findings that cast doubt on the utility of the three independent disorders model. For example, the age at onset of mania did not differ if there was comorbid CD, and the characteristics of mania were similar (irritable mood, chronic course, mixed with symptoms of major depression) irrespective of the presence of CD. Furthermore, the age of onset of CD was similar in children with and without BPD. In summary, the clinical characteristics and correlates of CD and BPD were similar, irrespective of whether there was comorbidity or not. These findings strongly suggest that children manifesting symptoms of BPD and CD have both disorders and not a third independent disorder that is the combination of the two. These findings were similarly demonstrated in the investigation of comorbidity between BPD and ADHD in prepubescent children (Wozniak, Biederman, Kiely, et al., 1995) suggesting that the three independent disorders model is not particularly valid.

Further evidence against the three independent order model comes from the previously cited research of Neale and Kendler (1995) in comorbidity between MDD and GAD, and from Rhee et al. (2008) in comorbidity between ADHD and CD. Both researchers found that structural models investigating three independent disorder

models were the poorest fit of any models tested. Indeed the findings of Rhee et al.

(2008) directly contradict the previously cited work of Faraone and colleagues

(Biederman et al., 1992; Faraone, Biederman, Jetton, et al., 1997; Faraone et al.,

1991; Faraone et al., 2000) as they were both directly investigating ADHD and CD.

Thus overall the three independent disorders model cannot be supported.

Is Comorbidity Caused by Shared Underlying Causal Risk Factors Between Disorders?

The third major substantive explanation for comorbidity provides the hypothesis that

comorbid disorders occur because the disorders share underlying causal aetiological

factors. That is, comorbidity between psychological disorders/syndromes may be a

result of the fact that the risk factors for the two disorders are correlated (Fergusson

et al., 1996; Lee & Bukowski, 2012; Lilienfeld, 2003). There are many variations on

the common risk factors hypothesis in providing an explanation for comorbidity,

though they generally share the same underlying premise. This premise is that if

disorders are predominantly the result of a set of risk factors and these risk factors

are similar for two disorders, then comorbidity reflects the fact that the pathways by

which one disorder develops are the same as those for the other disorder (Teesson et

al., 2003). Alternatively, even if the two disorders may have apparently different risk

factors, these risk factors may correlate with each other leading to the comorbidity

(Wolff & Ollendick, 2006). What these causal factors are is the source of much

debate, and will be outlined in more detail in the subsequent chapter, but they are

thought to include genetic, personality/temperament, social and environmental

factors in combination (Angold et al., 1999; Caron & Rutter, 1991; Lilienfeld, 2003).

This explanation has much empirical support as many risk factors have been shown to be common in comorbid disorder/syndrome pairs. Genetic or quasi-genetic risk factors have shown to link many disorders. O'Connor, McGuire, Reiss, Hetherington, and Plomin (1998) showed that depressive symptoms and antisocial behaviours share a common genetic liability accounting for nearly half of their observed co-occurrence. Similarly, certain temperament factors such as negative affect, which are thought to at least in part be genetic in nature, are hypothesised to be a factor related to a range of comorbid psychopathology. Keiley et al. (2003) investigated common risk factors underlying co-occurring internalising and externalising syndromes as assessed by the CBCL using longitudinal data from 585 children from the American Child Development Project (see Dodge, Pettit, & Bates, 1994). They found that children with a high trait level of a childhood temperament factor they refer to as 'difficultness', but which is closely related and includes negative affect, were rated by parents as having higher rates of comorbid internalising and externalising syndromes. Similarly, Baldwin and Dadds (2008), found negative affect was associated with the co-occurrence of ADHD and anxiety symptoms. Negative affect is also thought to be at least one factor that links both anxiety and depression, according to the Tripartite model of affective and mood disorders (Clark & Watson, 1991). Harsh parenting and a stressful family environment were also shown to be related to higher rates of comorbid internalising and externalising syndromes in the Keiley et al. (2003) study, and have been associated with comorbidity between specific disorder groups such as ADHD, CD, and ODD as found by Burt et al. (2003).

Operationalised Models of the Shared Risk Factors Hypothesis

In addition to individual research on the shared risk factors, there also exist a range of operationalised models of this explanation. Probably the most comprehensive and operationalised models come from the work of Neale and Kendler (1995), who operationalised models initially developed by Klein and Riso (1993). One specific model that fits the concept of shared risk factors is the 'Correlated Risk' model. The 'Correlated Risk' model, also referred to as the correlated liabilities model, proposes that there is a continuous relation between the liability to one disorder and the liability to the second disorder (Rhee et al., 2008). Thus, the model suggests that an increase in liability for one disorder is directly related to increase in liability for the second disorder.

Research by Neale and Kendler (1995), tested both the 'Correlated Risk' model in relation to the comorbidity between Major Depressive Disorder (MDD) and Generalised Anxiety Disorder (GAD), on a sample of 2,163 female twins from the population-based Virginia Twin Register. The model of correlated liabilities was compared to other specific models including a model of the three independent disorders hypothesis and the direct causal hypothesis discussed previously in this section. The 'Correlated Risk' model was demonstrated to fit well by absolute measures (χ^2), though was not necessarily a parsimonious model according to Aikake Information Criterion (AIC). Similarly, Rhee et al. (2008) tested the Correlated Risk model in regards to the comorbidity between attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (CD), using the same models as Neale and Kendler (1995). Using 110 MZ twin pairs and 181 DZ twin pairs recruited from the Colorado Learning Disabilities Research Center Twin Study. They showed the correlated risk

model was a good fitting model by all measures, and was better than either the three independent disorder or the causative models. All in all there is evidence to suggest that the model of shared risk factors has particular potential for explaining comorbidity.

Derivatives of Basic Shared Risk Factors Model

Derivatives and variants of the basic causal risk factor models have also been developed. One interesting possibility is that comorbidity/co-occurrence between psychological disorders/syndromes may be influenced by a single higher-order factor (Lilienfeld, 2003), which for the purposes of this paper be referred to as *psychopathological liability*. This model, operationalised by Neale and Kendler (1995), the 'Alternate Forms' model, hypothesises that comorbidity occurs because the two comorbid disorders are alternate manifestations of a 'single' common liability. This 'single' common liability, is in fact conceptualised as derived from a multifactorial combination of heritable and environmental risk factors, and implies a continuous spectrum of liability to disorders. An individual develops a disorder when the degree of liability present reaches a threshold level (Lilienfeld, 2003; Neale & Kendler, 1995; Rhee et al., 2008), and that person A manifests disorder X while person B manifests disorder Y because of chance or differences in their combination of genetic and environmental risk factors. Thus if two individuals have the same overall genetic liability but are exposed to different environmental risk factors, the two individuals may develop differing disorders. In this model comorbidity occurs because the comorbid disorders are alternate manifestations of a single psychopathological liability spectrum, each with a differing threshold on this spectrum. Thus if a person reaches the threshold for multiple disorders, then they

may manifest them separately and comorbidly. This model differs from the 'Correlated Risk' model, in that this model assumes a single liability factor underlying all comorbidity, whereas the 'Correlated Risk' model assumes that each comorbid pair of disorders has its own individual shared liability. However, the aforementioned studies by Neale and Kendler (1995) and Rhee et al. (2008) that tested the Correlated Risk model, also tested the 'Alternate Forms' model, and found it to be generally poor fitting. However, alternatives of the shared risk factors hypothesis continue to be proposed in attempt to better explain comorbidity.

Summary

This prior section examined some of the more common substantive explanations for comorbidity. Two of the widely tested models of comorbidity, a causative model where one is thought to cause the other, and a model that hypothesises that comorbid disorders are in fact a third independent disorder, while having some merit, generally lack research support. However, a model that suggests comorbidity is caused by shared etiological or risk factors, has considerable research support. Indeed the support is strong enough for Lilienfeld (2003) to suggest that such explanations are potentially the most interesting for comorbidity.

Limitations of Current Bivariate Research and The Need for Multivariate Models of Comorbidity

The previous section demonstrated that a model that suggests comorbidity is caused by shared etiological risk factors shows great promise as a substantive explanation of comorbidity. However, most research cited in the previous section, and indeed most

research into comorbidity to date, has been bivariate in nature. That is, most research looks at individual disorder pairs and attempts to provide explanations about the cause of such comorbidity at the level of individual disorder pairs. Bivariate modelling research is undoubtedly important, and has its uses both clinically and theoretically. Theoretically, bivariate modelling can also help with the development of theoretical approaches to understanding comorbidity that can then be extended. In clinical context, knowledge about causes or risk factors can be useful clinically and theoretically, with information about specific disorders such as that outlined above is undoubtedly important. Indeed Krueger and Markon (2006) argue that bivariate modelling research provides a "…focus on the fundamental elements of comorbidity: two disorders and their combination" (p. 121).

However, as Batstra et al. (2002) note, the phenomenon of comorbidity is global and multivariate in nature; that is comorbidity occurs across the broad spectrum of disorders and syndromes, not just between certain specific disorder pairs. This focus on bivariate research has been argued by some, including Krueger and Markon (2006), to be a great limitation of the current research on comorbidity. Thus for comorbidity research to move forward, multivariate research on comorbidity must be undertaken, and multivariate models of comorbidity must be developed. That is, models and research must consider comorbidity beyond individual disorder pairs, and focus on explaining comorbidity across the broad spectrum of psychopathology. This would provide a more comprehensive explanation of comorbidity than straightforward bivariate modelling, and potentially provide enlightenment about the nature of comorbidity/co-occurrence in a way that bivariate models cannot. Moving to multivariate research and modelling is not entirely straightforward however. When

considering multivariate models of comorbidity, several issues arise, including statistical methods, parsimony, and the need to consider other factors, that are not always of concern in bivariate modelling.

Limitations of Bivariate Analysis: The Need for More Sophisticated Techniques for Multivariate Research and Modelling

Batstra et al. (2002) note that this bivariate research tends to use common (bivariate) statistical methods in studying comorbidity, such as odds ratios, correlations, and regression. However, if the phenomenon of comorbidity is multivariate in nature, any statistical modelling approach used should be capable of mapping the concept of what Krueger and Markon (2006) refer to as "multimorbidity", or multivariate comorbidity. Statistical techniques such as odds ratios, correlations, and regression are simply not capable of such analysis. There has been some use of extensions of bivariate statistics, such as the use of partial correlations in some multivariate analysis of prevalence statistics, to overcome this problem. Partial correlations assess the relationship between two variables as per a Pearson's correlation, but controlling for other intervening variables. The purpose is to find the unique variance between two variables while eliminating overlapping variance from others (Field, 2013). However, partial correlations are not particularly powerful, and are still a bivariate-style statistic, being used to understand a multivariate phenomenon. Thus there is a clear need to move to other sophisticated psychometric techniques, such as structural equation modelling and item response theory.

Structural Equation Modelling

Structural equation modelling (SEM) is a confirmatory and occasionally exploratory statistical technique for testing and estimating causal relations, using a combination of statistical data and qualitative causal assumptions (Hoyle, 1995). SEM is in essence, a multivariate form of regression models, designed to test models that attempt to explain the covariation among variables. Unlike the more traditional regression models, variables can often be simultaneously a predictor and outcome variable within a single model (Byrne, 1998). Indeed, variables in an SEM may influence one another reciprocally, either directly or through other intermediate variables, and thus complex multivariate models can be tested (Schumacker & Lomax, 2010).

One key component to SEM models is that they usually focuses on modelling the interplay between latent constructs - abstract variables like 'intelligence' or 'attitude' that are not (and often cannot be) measured directly, but are rather inferred or estimated from several measured or manifest variables (Bentler, 1980). By not relying on single manifest variables to measure a construct, but rather using several related manifest variables to measure a single construct, SEM allows the modeller to explicitly capture the unreliability that is inherent in all measurement, especially psychological measurement. Thus a more accurate measurement of the desired construct can be made, and as a result, a more accurate assessment of the structural relations between that construct and other latent variables can be made (Brown, 2006).

There are two main components of an SEM: the structural model showing potential causal dependencies between latent variables, and the measurement model showing the relations between a latent variable and the manifest variables (also referred to as indicators) assumed to represent the latent variables (Schumacker & Lomax, 2010). The model is then tested against obtained measurement data to determine how well the model fits the data – or how well the model is able to reproduce the obtained data, based on the constraints imposed (Byrne, 1998). While there are basic SEM models, there are also a range of sophisticated models such as Latent Class Analysis (LCA), Latent Growth Modelling (LGM), Factor Mixture Models (FMM).

The key aspects for SEM when considering multivariate comorbidity, is that it allows for explicit tests of competing models, and even more importantly, allows the exploration of multivariate relationships in an integrated manner. In effect, these techniques allow modellers to *simultaneously* assess impacts at both the level of an individual disorder pair, and across the broad spectrum of disorders. This allows for comprehensive and integrated explanations of comorbidity that simple bivariate modelling, even using partial correlations cannot provide. There has been relatively limited use of more sophisticated techniques such as structural equation modelling (SEM) in comorbidity research, and even then it has mostly been used within bivariate research. However SEM and associated psychometric techniques (e.g. Item Response Theory), have begun to be considered more frequently in recent times (Krueger & Markon, 2006).

Use of Advanced Psychometric Techniques in Exploring Comorbidity

Those studies that have used such techniques have provided interesting insights into comorbidity. Fergusson, Horwood, and Lynskey (1994), used latent class analysis to investigate the nature of comorbidity among 'problem behaviours'; behaviours related to oppositional and conduct problems. They found evidence for four classes of individuals; one class showing very low probabilities of any problem behaviour; one class showing high oppositional behaviour with some conduct problems with a further class showing the inverse; and one class showing high or elevated risks for all problem behaviours. This finding is an interesting idea for psychopathology in general, and may suggest that people with high levels of comorbidity may be a distinct group from those without comorbid disorders. Similar results were noted by Neuman et al. (2001) investigating ADHD and associated comorbidities.

Hale III, Raaijmakers, Muris, van Hoof, and Meeus (2009) investigated adolescent anxiety and depressive symptomatology using Latent Growth Modelling (LGM). They were interested in ascertaining if the comorbid symptomatology was best described by a model that assumes one general factor underpinning development, or by a model that assumes there are two distinct disorders with parallel growth processes. They found that a model assuming two distinct process of development was a superior fit, but noted some interrelationship between the two processes, with the initial symptom severity of either anxiety or depression being predictive of the development of the other. This unique insight into the development of internalising disorders can only be ascertained through techniques such as LGM, and clearly demonstrates the utility of psychometric approaches in gaining unique insights into comorbidity.

One of the more interesting uses of psychometric techniques was the use of item

response theory (IRT) by Krueger and Finger (2001). They hypothesised that anxiety

and unipolar mood disorders are often comorbid because each disorder was an

indicator for a common, higher order factor. Using a clinical subsample from the

National Comorbidity Survey (Nock et al., 2006) they demonstrated that a one-factor

model, with a latent factor they called 'Internalising' fit the correlations among all

seven anxiety and unipolar mood disorders they investigated. They then used IRT to

explore how each diagnosis mapped onto the internalizing factor, and noted that

dichotomous diagnoses measured the higher end of the factor, with high scores on

this factor being associated with increased social costs.

Model Parsimony

Clearly the use of more sophisticated psychometric techniques, such as structural

equation modelling has great potential in helping create multivariate models of

comorbidity. However, when considering multivariate models of comorbidity, issues

such as model parsimony arise, that cannot be apparent when considering

comorbidity bivariately. Indeed it has been the use of sophisticated psychometric

techniques such as SEM that has demonstrated this lack of parsimony from some

models in multivariate domains. It is certainly true that a vast number of multivariate

models can be treated as extensions of bivariate models, while simply specifying a

greater number of disorders. Issues arise when extending the models however, as

extra parameters must be added for each disorder. For some theorised models, this

growth is not additive but exponential, meaning the wider the range of

comorbidity/co-occurrence that one attempts to explain, the more complex the model

becomes. This is problematic as the more parsimonious a model is, the more useful it

is likely to be, and indeed, the more likely it is to be a true population model (Vereshchagin & Vitanyi, 2004).

The problem of parsimony is especially true of models such as the causative and three independent disorders model. In the case of the causative model, when extended to multivariate form, the model becomes almost unspecifiable, because the direction of the causative path for each disorder pair must take into account the direction of causative paths for all potential comorbidities involving one or more of the disorders/syndromes within the pair. In the case of the three independent disorders model the number of new disorders or syndromes grows exponentially, making any classification system almost completely unworkable. Even models of shared risk factors, such as the 'Correlated Risk' model become overly complex. The 'Correlated Risk' model in multivariate form would by definition (assuming a unique liability for each disorder pair), need to include a parameter representing every correlation between each combination of disorders (Krueger & Markon, 2006). This means that the wider the range of comorbidity/co-occurrence that one attempts to explain, the more complex the model becomes. This complexity is problematic, and makes the 'Correlated Risk' model very difficult to examine, and lacking some utility (Krueger & Markon, 2006).

Other variant models of shared risk factors do provide parsimony when considered multivariately. The 'Alternate Forms' model, hypothesising a single common factor (Lilienfeld, 2003), or *psychopathological liability* certainly has such parsimony over the 'Correlated Risk' model at the multivariate level. While the 'Correlated Risk' model includes a parameter representing every correlation between each combination

of disorder (Krueger & Markon, 2006), the 'Alternate Forms' forms model assumes a single common factor underlying *all* disorders, and thus includes only one parameter for each disorder. This parameter represents the influences of the single liability on each disorder. This apparent parsimony advantage of the Alternate Forms model in multivariate form is however undermined by its apparent lack of utility as previous research (e.g. Neale & Kendler, 1995; Rhee et al., 2008) has shown the 'Alternate Forms' model to be a generally poor fitting in bivariate research. In defence of the 'Alternate Forms' model however, is that a model proposing a single common factor to explain multivariate comorbidity may not be truly testable in the bivariate domain (Krueger & Markon, 2006). Any attempt to test a model proposing a single common factor within a bivariate domain would by definition ignore a wide range of potential covariates, such as other disorders which may significantly impact an overall model, were that model to be considered multivariately. Thus it is plausible that a common factor model, or indeed any model of comorbidity, may not show overall fit bivariately as a pure function of not assessing the phenomenon in a multivariate manner, rather than the particular model being a poor model (Krueger & Markon, 2006).

Surprisingly, given its obvious advantage from a parsimony perspective, there have been relatively few attempts to investigate multivariate models of comorbidity based on a common factor model. Two studies have investigated single liability/common factor models of comorbidity, but only in the externalizing spectrum, and both involving adults. Krueger, Markon, Patrick, and Iacono (2005) using adult parents from a twin-family study, and Markon and Krueger (2005) in a US population study found some support for a continuous liability such as that proposed by the Alternate

Forms model, but this support was very mixed, and neither study considered comorbidity in a truly multivariate way (i.e. externalising and internalising disorders). As a result of its limited testing multivariately, and in light of the common factor model having substantial parsimony over other models of shared risk factors, such as the 'Correlated Risks' model, a common factor model is worth testing in the multivariate domain.

Hybrid and Novel Models within the Multivariate Domain

Research in multivariate comorbidity does not need to be limited to just extensions of those models which are shown to work at a bivariate level. As indicated, most models can be modelled in multivariate terms, though with a risk of poor parsimony. In extending such models to include more disorders in a multivariate approach, the opportunity arises for developing new and or hybrid models that cannot be specified when simply investigating bivariate pairs of disorders (Krueger & Markon, 2006). The flexibility of SEM and associated techniques in allowing new/hybrid multivariate models could provide more comprehensive explanations of comorbidity than either bivariate models, or current multivariate models. Such new hybrid modelling allows comorbidity to be investigated using sophisticated and novel methods, which Pesenti-Gritti et al. (2008) argues is vital to develop an understanding of the aetiology of comorbidity, given the current state of the field. Krueger and Markon (2006) outline how hybridised models may work, and how they can be tested using SEM and associated techniques, but at the time of writing of this thesis very few new or hybrid models have been developed, or tested.

The Studies in This Thesis

It is the purpose of the first two studies of this thesis to present and test a hybrid model of multivariate comorbidity, based on the common factor as implied by the common psychopathological liability model outlined previously. This will be done using previously under-considered psychometric SEM techniques. In Chapter 3, a model of multivariate comorbidity that will be proposed based on the SEM concept of the bifactor model, with Chapter 4 presenting a confirmatory test for a common factor, using the SEM circumplex structure. The model presented, can be regarded as a hybridised synthesis of Alternate Forms/shared risk factors models, but extended to explain multivariate comorbidity.

The common factor models hypothesised are based on the idea of shared aetiological risk factors. Specifically the basic common factor model assumes that comorbidity occurs because of a single underlying factor, a psychopathological liability, which is derived from a multifactorial combination of heritable and environmental risk factors. In order to test if any common factor found is indeed this *psychopathological liability* factor, the associations between any common factor and risk factors believed to be part of this liability factor must be established. This is the aim of the third study in this thesis. Given this, it is necessary to discuss the concept of 'risk factors' and this will be undertaken in the next chapter. This chapter will outline the concept of risk factors, identify key factors that may contribute to an overall *psychopathological liability* as suggested by a common factor model, as well as expand on how their contribution to a liability can be tested using SEM.

Chapter 2 - Risk Factors Associated with Multivariate Comorbidity

In the previous chapter, research evidence was presented that indicated the most promising explanation for comorbidity was that it was caused by shared aetiological, or risk factors. However because the phenomenon of comorbidity is definitively multivariate in nature, and the majority of research has been bivariate in nature, the argument was presented that multivariate models of comorbidity should be the focus of future research. One promising conceptualisation of 'shared risk factors' within the multivariate conceptualisation of comorbidity was the idea of a common liability factor, or *psychopathological liability*, that underlies all psychopathology. Psychopathological liability in this context was defined as a multifactorial combination of heritable and environmental causes; also known as risk factors (Neale & Kendler, 1995). The psychopathological liability concept is promising when considering comorbidity in a multivariate context, in part because the concept of liability itself is multivariate in nature. The previous chapter also argued that structural equation modelling (SEM) can provide a powerful approach to understanding patterns of comorbidity in a multivariate manner, because it also allows current bivariate models to be hybridised and extended to the multivariate, and because it allows new, more novel models to be considered. However, before any new, novel or hybrid multivariate models that assume common risk factors can be developed, a discussion of the nature of 'risk factors', both conceptually and practically needs to be considered. This is because any multivariate model of comorbidity that is based around the idea of shared risk factors or a common

liability, should show meaningful associations with a multifactorial combination of appropriate risk factors.

Thus the aim of this chapter is to provide a definition of the concept of risk factors, in the context of a psychopathological liability, and outline various conceptualisations of categories of risk factors. The chapter will outline some of the most likely candidates that could form a multifactorial psychopathological liability in the context of a multivariate model of comorbidity. It will be demonstrated that three constructs – negative affect, parental psychopathology and familial functioning – should be considered as prime candidates to be part of this psychopathological liability, and thus should show associations with any common factor underlying psychopathology. Empirical evidence will be presented to show that each of these factors has been implicated with the development of particular psychopathology, both within the internalising and the externalising domains, and also potentially, the development of comorbidity. Discussion of the limitations of the current empirical literature will also be made, and it will be demonstrated that, just as for the research on comorbidity, a multivariate approach to risk factor research should be undertaken, especially in the context of comorbidity. An explanation of how SEM can be used to test the association between risk factors and a common factor within a multivariate model of comorbidity will also be made.

The Risk Factor Concept

The concept of 'risk factors', is not new in psychology. However, research into risk factors for the development of psychopathology has recently come to be seen as a separate discipline within the field of developmental psychopathology (Nelson,

Stage, Duppong-Hurley, Synhorst, & Epstein, 2007). Risk factor research is seen as a move toward "comprehending the causes and determinants, course, sequelae, and treatment of childhood disorders" (Cicchetti & Toth, 1995, p. 373). In a manner similar to the exponential increase in research on comorbidity, there has been a substantial growth in the risk factor research in the last 15 years (Frick, 2004; Nelson et al., 2007).

What is a 'Risk Factor'

A risk factor can be best defined as a personal characteristic or environmental experience/event, that if present, is associated with an increase in the probability or 'risk' of a particular outcome, over and above the underlying probability of that outcome in the general population (Kraemer et al., 1997; Mrazek & Haggerty, 1994). Thus risk factor research in developmental psychopathology is the study of the antecedent characteristics and conditions to psychopathology, the psychological sequelae (e.g. psychopathology), and the ways in which these antecedent characteristics and conditions may be interrelated with each other and with protective factors (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). Protective factors refer to antecedent characteristics and or conditions associated with a decrease in the likelihood of psychopathology or those factors that may assist in positive adaptation to adverse circumstances (Kazdin et al., 1997). In the context of comorbidity, risk factors are more commonly considered than protective factors (Appleyard, Egeland, van Dulmen, & Sroufe, 2005), and it is in this context, in combination with theories surrounding the nature of common liability factor models, that further discussions within this chapter shall take place

Categories of Risk Factors

Risk factors can be derived from diverse domains within the genetic/biological, and psychosocial/environmental, and have often been framed within the classic 'nature versus nurture' debate in the wider scientific community (Pennington, 2002). Traditionally, research has focused on the role of genetic and environmental variables in the development and maintenance of childhood psychopathology. A range of biological, genetic and behavioural-genetic factors including parental psychopathology and childhood temperament have gained considerable interest within the field of developmental psychopathology. Similarly, environmental factors such as mother–child interactions and family cohesion have also been the subject of much interest as psychopathological risk factors. However, simple designation of many risk factors as environmental or biological/genetic is somewhat overly simplistic and misleading.

The risk factor of parental psychopathology is often considered to be primarily genetic in nature, yet such a variable contributes both genetic and environmental influences (Wolff & Ollendick, 2006). While there is a known familial genetic risk for some anxiety disorders, children who live in a family environment where there is parental hypervigilance may also learn and model the anxiety related behaviours displayed by their parents (Yehuda, Halligan, & Bierer, 2001). Thus Deater-Deckard, Dodge, Bates, and Pettit (1998) argue against the use of putative categories of environmental or biological, instead indicating that psychological constructs of risk for psychopathology can be regarded as falling into four broad domains; child specific, environmental, parenting, and peer-group experiences. However a more parsimonious categorisation of these factors is provided by Crawford, Schrock, and

Woodruff-Borden (2011) who indicate that categories of child traits and behaviours, maternal (parental) traits and behaviours, and environmental risk may be more appropriate, as most constructs can be considered to belong more primarily to only one. Thus when considering the most likely risk factors that may contribute to multivariate comorbidity, it is necessary to consider aspects from all these categories, and this chapter will consider these in light of the Crawford et al. (2011) categories.

What Risk Factors Might Contribute to Comorbidity and a Common Liability Factor?

There is a very large and wide-ranging literature on risk factors, with considerable evidence for significant and substantial genetic influences on the development of most DSM-IV disorders. Similarly there is equivalent evidence that environmental influences have equal, or in some cases greater contributions than genetic influences to the development of psychopathology (Kazdin et al., 1997). There is also substantial evidence for high genetic contributions to all Child Behavior Checklist (CBCL: Achenbach & Rescorla, 2001) syndromes, with environmental influences playing a significant role as well (Derks, Hudziak, van Beijsterveldt, Dolan, & Boomsma, 2004).

However not all of this body is applicable to the comorbidity research. The vast majority of risk factor research is univariate in nature looking at individual risk factors and their association in the development of individual disorders. Where the research does look at risk factors for comorbidity, the tendency, just as in comorbidity research, is for the research to investigate risk factors for bivariate comorbidity, or risk factors associated with development of a pair of comorbid

disorders. Comparatively little research has investigated risk factors in the context of a multivariate model of comorbidity, though this is largely a function of the small amount of research in multivariate comorbidity. Thus when considering risk factors that may contribute to a multivariate model of comorbidity, it is necessary to consider those variables from the broader risk factor research that show universal associations with a wide range of disorders, and a wide range of bivariate comorbidities.

From a wide-ranging list of potential risk factors, three key psychological constructs will be considered; the temperamental construct of negative affect, parental psychopathology and family functioning. These three constructs can be considered as prime candidates to form part of any common liability in a multivariate model of comorbidity and cover each of the three categories of risk factors outlined by Crawford et al. (2011): child traits and behaviours in the form of the temperamental construct of negative affect, maternal (parental) traits and behaviours in the form of parental psychopathology, and environmental risk in the form of familial functioning. Each of these risk factors will be considered in turn, with justification for their consideration in terms of common risk factors in multivariate comorbidity models.

Negative Affect as a Child Trait Risk Factor

While there are several broad-band dimensions that are promising candidates for risk factors as part of any common factor of psychopathological liability, by far the most widely researched and considered risk factor is that of childhood temperament, and in particular negative affect. Temperament has long been known to influence a

child's development in a number of important ways (Kagan, 1998), but recently there

has been a very focussed attention on how certain temperamental styles can be a risk

factor in the development of various forms of child and adolescent psychopathology

(Frick & Morris, 2004). Indeed several journals have devoted entire issues to

examining the contributions of temperament to the development of psychopathology,

including in the Journal of Clinical Child and Adolescent Psychology (Frick, 2004).

Temperament and Models of Temperament

The concept of temperament, and theories of its impact, has a long history in

developmental psychology. Despite the long history and the depth of research focus,

there is still no universal agreement on a single definition of temperament, or

agreement on a universal model of the construct. However, while there are differing

conceptualisations of temperament, most present-day definitions of temperament

share common characteristics. These common characteristics include that

temperament is related individual (behavioural) differences in children that appear

from soon after birth, that temperament has a strong genetic or neurobiological basis,

and that it remains relatively stable across the lifespan (De Pauw, Mervielde, & Van

Leeuwen, 2009; Ţincaş, Benga, & Fox, 2006).

Thomas and Chess Model of Temperament

The genesis of temperament research has long considered to be the work of Thomas

and Chess (Thomas & Chess, 1977), beginning some 40 years ago, most especially

through their New York Longitudinal Study (NYLS). They studied a large sample of

families with infants, and undertook a content analysis on mother's reports of their

children's behaviour during the first six months of life, as well as later observing

infant interaction with their parents. They observed and described nine key temperament dimensions, including children's activity level, threshold for reaction, rhythmicity of eating, sleeping and bowel movement patterns, intensity of response, approach versus withdrawal to new situations, general mood, adaptability to change, distractibility and attention span/persistence. They argued that these dimensions were key to comprehending the 'how' of individual differences in infant behaviour

The Thomas and Chess dimensions showed some utility, although the nature of the dimensions was critiqued, in part because they were developed primarily for clinical purposes, and were seen to lack some applicability to non-clinical spheres. The nine dimensions also showed poor psychometric properties, in part due to the dimensions showing considerable conceptual overlap (Rothbart & Mauro, 1990). This conceptual overlap is reflected in research findings, which show the general mood and adaptability dimensions consistently show overlap with each other, as well as with the approach-withdrawal dimension (Rothbart & Jones, 1998). Indeed a review of factor analytic studies by Martin, Wisenbaker, and Huttunen (1994), suggests that the Thomas and Chess dimensions could best be described by a smaller number of factors including negative emotionality, approach/positive affect, attentional persistence, and activity level (Rothbart & Bates, 1998).

Buss and Plomin Model of Temperament

Buss and Plomin (1975) modified Thomas and Chess's theory, by conceptualising temperament as a developmental predecessor of adult personality. They indicated that temperaments had to be heritable stable characteristics that were in part retained into adulthood. Based on these criteria, the authors proposed four broad temperament

dimensions: emotionality, activity level, sociability and impulsivity though the latter

has been suggested to be omitted from subsequent revisions due to questions about

heritability (De Pauw & Mervielde, 2010). Similarly subsequent revisions have also

suggested that sociability may be two separable dimensions (shyness and sociability,

e.g. Boer & Westenberg, 1994), though no firm conclusion has been reached.

Similarly, questions remain about the structural variability of the dimensions across

cultural and age groups (Gasman et al., 2002).

Rothbart Model of Temperament

The inconsistencies and lack of discriminant validity among the dimensions of the

Thomas and Chess models, as well as the revised models of Buss and Plomin, led

Rothbart and Derryberry (1981) to take a different, more psychobiological approach

to the conceptualisation of temperament. This conceptualisation sees temperament as

individual differences in reactivity and regulation, which have a constitutional base

but which are influenced by inheritance, maturation, and experience (Laredo et al.,

2007). Reactivity, which has two components, positive reactivity and negative

reactivity, involves the underlying response of physiological and behavioural

systems, including motivation and basic attentional processes to sensory stimuli.

Regulation is the activation of neural systems involved in higher order attentional

processes and cognitive control (Zeanah & Fox, 2004). This latter element (which

includes attention, approach/withdrawal, behavioural inhibition, and self-soothing) is

thought to modulate reactivity throughout development.

Common Dimensions in Temperament Models

Despite the differences in their conceptual development, a relatively clear number of common dimensions emerge from these prominent models of temperament. Mervielde and Asendorpf (2000) in their review of temperament models, demonstrate clearly that almost all independent models of temperament have variations on at least three common dimensions; neuroticism/negative affect, extroversion/positive affect, and conscientiousness (Caspi & Shiner, 2006; De Pauw & Mervielde, 2010; De Pauw, Mervielde, & Van Leeuwen, 2009). Therefore what can appear to be a fragmentation of temperament conceptualisations, may actually reflect a tendency for researchers to name similar constructs differently, rather than a total lack of agreement over the primary constructs (Rothbart, Ahadi, & Evans, 2000; Tackett, 2006).

Negative Affect

The three common dimensions (neuroticism/negative affect, extroversion/positive affect, and conscientiousness) common to the major models of temperament have all been investigated as potential risk factors for psychopathology, though one construct is very prominent in the research. Negative affect, also commonly referred to as negative affectivity, negative emotionality or neuroticism in various models of temperament and personality, is widely implicated in psychopathology and comorbidity, and is universally considered to be one of the keys in developmental clinical psychology. This is in part because, unlike other temperament constructs, it is consistently associated with the broad spectrum of psychopathology (i.e. both internalising *and* externalising disorders). Negative affect was defined by Watson and Clark (1984) as a pervasive disposition to experience unpleasant affective states

such as guilt, anxiety, irritability, self-dissatisfaction, and a sense of rejection and sadness (see also Tellegen, 1982). Rothbart, Ahadi, Hershey, and Fisher (2001) further indicate that negative affect in children has been associated with specific biologically based patterns of reactivity, with children with high trait negative affect having consistently greater frequency and intensity of anger, sadness, discomfort and fear experiences (Caspi & Silva, 1995).

Negative Affect and Related Constructs

Providing definitional precision for negative affect is difficult, because many models of temperament used in research subsume negative affect within higher order constructs. A wide body of research relevant to any discussion of negative affect investigates a construct most commonly known as behavioural inhibition (Kagan, 1998). Behavioural inhibition is defined as an initial negative emotional and motor reactivity to novelty (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984), and when assessed in toddlerhood, is manifested in vigilant and withdrawn behaviour in response to novel people and situations (Calkins, Fox, & Marshall, 1996). While research has demonstrated that negative affect only shows partial overlap with behavioural inhibition (Vreeke & Muris, 2012), negative affect and behavioural inhibition are still forms of the same underlying temperamental dispositions.

A large body of research has also examined the 'difficult temperament' construct. The 'difficult temperament' construct combines a number of dimensions into one global index of 'difficultness', but all definitions universally contain characteristics of negative affect (Frick & Morris, 2004). West, Schenkel, and Pavuluri (2008) indicate that a difficult temperament style characterises children with high activity

level, distractibility, high intensity and a low sensory threshold; characteristics that are somewhat inconsistent with negative affect. However, West et al. (2008) also indicate that a difficult temperament style is universally characterised by withdrawal from, or negative reaction, to new or unfamiliar things, as well as overall negative mood; characteristics entirely consistent with negative affect. Indeed these latter constructs are central to the construct of 'difficultness', and thus while the 'difficult temperament' construct is a superordinate construct, the construct clearly has strong theoretical relationships with negative affect. Thus while the focus of the proceeding section is to demonstrate that negative affect is a likely candidate for a common psychopathological liability factor, it will draw on literature investigating overlapping and or superordinate constructs such as behavioural inhibition, and difficult temperament. It must also be noted that much research has investigated multiple temperament constructs and interactions between these constructs. While such interactions are noteworthy for investigating the pathogenesis of individual disorders, such interactions are not commonly associated with comorbidity between disorders and thus will not be discussed here.

Negative Affect as a Risk Factor for Psychopathology

Research has consistently demonstrated that the temperament dimension of negative affect (or equivalent constructs) is involved in the aetiology and development of child psychopathology (Lonigan & Phillips, 2001), and it has been proposed that internalising and externalising behaviour share this factor in common (Lahey & Waldman, 2003; Lilienfeld, 2003). Indeed De Pauw and Mervielde (2010) and Whittle, Allen, Lubman, and Yücel (2006) both provide summary research reviews of the links between temperament and psychopathology and both indicate that high

levels of trait negative affect have consistently been associated with both internalising and externalising psychopathology. As an example, John, Caspi, Robins, Moffitt, and Stouthamer-Loeber (1994), using a sample of 350 twelve and thirteen year-old boys, showed high trait negative affect was associated with high levels of emotional and behavioural psychopathological symptomatology. That is, negative affect was positively related with symptomatology consistent with both internalising and externalising syndromes, though the results did show stronger relationships to internalizing (emotional) symptomatology. Similar results from Prior, Sanson, Smart, and Oberklaid (1999) in an Australian sample of 186 eleven and twelve year olds, showed high levels of negative affect was associated with actual psychological disorder, with almost half with high trait negative affect meeting the diagnostic criteria for a DSM-defined disorder.

Negative Affect and Internalising Disorders

Specific associations have been made between negative affect and specific internalising disorders, in populations from infant to adolescent, and into adulthood. Kochanska (1995) examined the relationship between negative affective temperament and emerging internalising symptomatology in 103 26-to-41-month-old toddlers, investigating the relationship in the context of several naturalistic and laboratory studies, as well as through the use of parental reports. Kochanska found universal support for the temperamental construct playing a significant, independent role in the development of internalisation.

Similarly, using a preschool aged sample of 104 children aged 21 months to six years of age, Shamir-Essakow, Ungerer, and Rapee (2005) assessed the relationship

between anxiety disorders and behavioural inhibition, a construct that, as described earlier, conceptually overlaps with negative affect. Their results indicated that behavioural inhibition was significantly associated with the development of childhood anxiety, and notably, was independent from a range of other variables such as mother child-attachment. Similarly, Laredo et al. (2007) examined the association between various temperamental dimensions and anxiety problems in 38 Spanish preschool children aged three to six. The children were evaluated by both parents and psychologists, and irrespective of the informant, children with high trait levels of negative affect had more pronounced symptoms of anxiety disorders than children with low levels of the trait. These results were supported by Pahl, Barrett, and Gullo (2012), who found a similar relationship between behavioural inhibition and anxiety using a sample of 236 Australian four-to- six year olds. Indeed research has generally indicated that children with high levels of behavioural inhibition, and thus negative affect, are at an at increased risk for all specific anxiety disorders, especially social anxiety (e.g. Biederman et al., 2001; Schwartz, Snidman, & Kagan, 1999) and the phobic disorders (e.g. Gladstone, Parker, Mitchell, Wilhelm, & Malhi, 2005). Such relationships have also been noted in adults. Using a community sample of over 300 adults in the Baltimore Epidemiologic Catchment Area Follow-Up Study, Bienvenu et al. (2001) found that high levels of neuroticism (again a construct related to negative affect) were significantly associated with increased lifetime diagnoses of social phobia, agoraphobia, panic disorder. Indeed, generally all continuous measures of anxiety symptoms have shown consistent positive correlations with negative affect/neuroticism (e.g. Watson, Gamez, & Simms, 2005).

Positive correlations have also been noted between negative affect/neuroticism and continuous measures of depressive symptomatology and the mood disorders (e.g. Saklofske, Kelly, & Janzen, 1995). Indeed the literature supporting a link between negative affect (and its related constructs) and mood disorders, especially depression is as wide ranging as that for anxiety disorders. West et al. (2008) investigated the 'difficult temperament' construct in 25 infants with paediatric bipolar disorder, and 25 healthy children. Consistent with the study hypothesis, infants with paediatric bipolar disorder experienced significantly more characteristics of 'difficult temperament' in infancy and toddlerhood than healthy controls. This lead West et al. (2008) to conclude that premorbid characteristics of the 'difficult temperament', such as difficulty with emotional regulation (a key component of negative affect), might be a marker for underlying dysfunction in affective processes that significantly increase risk for a mood disorder (see also Hirshfeld-Becker, Biederman, Calltharp, Rosenbaum, & Rosenbaum, 2003).

Anthony, Lonigan, Hooe, and Phillips (2002) found a similar relationship between negative affect and depression, in a community sample of nearly 300 ten-to-17-year-old youth. The participants completed questionnaires of temperament and measures of internalising symptoms. Using factor analysis, Anthony et al. (2002) showed that aspects of negative affect and overall negative temperament had strong associations with depressive symptoms, as well as with anxiety symptomatology. Trull and Sher (1994) also reported that university students with a lifetime diagnosis of major depression had generally higher levels of neuroticism than students without any diagnosis. Similarly, the previously cited study by Bienvenu et al. (2001), found that

high levels of neuroticism were significantly associated with increased lifetime diagnoses of major depression.

Limitations of Research Investigating Negative Affect and Internalising Disorders

One major problem with the research outlined above is that it is almost exclusively cross-sectional and or correlational in design, which, as Compas, Connor-Smith, and Jaser (2004) have noted, means the relationship between temperament and symptoms of depression may be open to temporal and methodological confounds. However, there is a small body of research, mostly longitudinal or prospective in nature that indicates these limitations do not create artificial relationships. Prospective or longitudinal studies have consistently shown a strong link between negative affect and internalising psychopathology. Caspi, Henry, McGee, Moffitt, and Silva (1995) assessed temperament dimensions when children were three and five years of age and showed that temperament dimensions analogous to negative affect positively predicted parent- and teacher-rated internalizing symptoms for the children as they progressed through childhood. Caspi, Moffitt, Newman, and Silva (1996) extended this study, demonstrating starkly the relationship between negative affect/behavioural inhibition and depressive symptomatology. In a longitudinal epidemiological study, 3-year-old children were classified into high and low negative affect/inhibition groups based on examiner observations of their behaviour. These participants were then reassessed for DSM-III-R disorders at 21 years of age, and individuals showing high trait levels of negative affect at three years of age had significantly more depressive symptoms and elevated rates of mood disorders as adults, than those low in trait negative affect at three years of age.

Another issue with early research on links between negative affect and the internalising disorders, was the limited focus on the possibility of differential gender effects. However, a study by Oldehinkel, Hartman, De Winter, Veenstra, and Ormel (2004) demonstrates a degree of gender universality in the association. They showed that negative affect was strongly related to anxiety, but also noted strong relationships between negative affect and both depression and somatic complaints in preadolescents. However, the key finding of this study was that while there were sex differences in the distribution of problem behaviours, the associations between negative affect and psychopathology were comparable for both genders.

Negative Affect and Externalising Disorders

Commensurate with the research cited in the previous section, specific associations have been noted between negative affect and externalising disorders. While the body of research is substantially smaller than for the internalising disorders, the link is fairly well established. One of the most cogent studies of this relationship was a longitudinal study by Gjone and Stevenson (1997). They investigated the prospective relationship between various temperament traits and general externalising psychopathology, in a study of over 700 Norwegian same-sex twins. Using various measures of temperament, and the CBCL as a measure of psychopathology, they tested the children biennially from seven until 17 years of age. They showed that characteristics reflecting negative emotionality predicted all externalising behaviours measured by the CBCL (attention problems, delinquent behaviour, and aggressive behaviour), as well as anxious and depressed syndromes, at all stages of childhood and into adolescence. Olson, Bates, Sandy, and Schilling (2002) also showed similar results using measurements of 'difficult temperament', noting that negative affective

characteristics showed a prospective relationship with externalizing pathology in early childhood. This study was an extension of Olson, Bates, Sandy, and Lanthier (2000) which had earlier demonstrated the same relationship for adolescents. In a study cited previously with regard to paediatric bipolar disorder, West et al. (2008), investigating the 'difficult temperament' construct in 25 infants with ADHD and 25 healthy children, found that children with ADHD had more characteristics of 'difficult temperament' in infancy and toddlerhood than healthy controls. While it must be noted that the children with ADHD generally rated lower on measures of 'difficultness' than children with bipolar disorder, the ratings were still substantively higher than for healthy control children, and the results still indicate that negative affect is a risk factor in the development of ADHD.

There is also a body of research providing evidence that high rates of negative affect are associated with, as well as predictive of, conduct problems (Lahey & Waldman, 2003). Research by Caspi et al. (1994) using around 500 boys from the Pittsburgh Youth Study measured a range of temperamental traits at age ten, and then correlated this with measures of conduct disordered behaviour (referred to as 'delinquency') at age 12 to 13, with measures completed by both the participants, their parents, and their teachers. Irrespective of the informant, consistent robust correlations between negative affect and conduct problems were found. Similar results have been found by Eisenberg et al. (1996). Research has also shown that both adolescent boys and girls who score higher on indexes of 'difficult temperament' have higher risk for developing conduct problems (e.g. Giancola, Mezzich, & Tarter, 1998; Kingston & Prior, 1995).

Additionally, studies have found that negative affect characteristics measured in childhood, show links with substance use and abuse disorders in adolescence (Masse & Tremblay, 1997) and adulthood (Cloninger, Sigvardsson, & Bohman, 1988). Similarly, children who have more permissive attitudes to substance use tend to score higher on neuroticism inventories than those who have less permissive views on substance use (Francis, 1996). While the externalising behaviours are often thought to be ADHD, conduct disorder and oppositional defiant disorder, some researchers consider substance-related disorders to be part of the externalising disorders because of their inherently behavioural characteristics (Teesson et al., 2003). Thus overall, there are clear links between negative affect and externalising psychopathology.

Negative Affect as a Risk Factor for Comorbidity

The previous section demonstrated that negative affect has been implicated as a risk factor for the development of psychopathological disorders. More interestingly in terms of the idea of a psychopathological liability, it has also been implicated in the development of comorbidity itself.

Homotypic Comorbidity & The Tripartite Model

Probably the most notable consideration of negative affect as a cause of potential homotypic comorbidities has been from the Tripartite Model of depression and anxiety. Clark and Watson (1991) formulated this theory as an attempt to explain the common links, and commonly found comorbidity, between depression and anxiety. Their model rests on three constructs, negative affect, positive affect and physiological hyperarousal. Only positive and negative affect are applicable to personality/temperament constructs, with positive affect reflecting "pleasurable

engagement with the environment" (Watson, Clark, & Carey, 1988, p. 347). In their model, low positive affect is a marker for depression and physiological hyperarousal is predicted to be specific to anxiety disorders only, but importantly, negative affect is the factor that is seen as common to both depression and anxiety (Clark & Watson, 1991). The Tripartite Model was initially developed and tested with adults, and support for the assumptions regarding positive affect and physiological hyperarousal constructs have been mixed, leading to significant revisions to the model, and also new iterations (e.g. revised integrative hierarchical model of depression and anxiety, Mineka et al., 1998). There has however, been consistent empirical support for the role of negative affect as a common link between depression and anxiety.

Watson et al. (1988) examined the overlap of anxiety and depressive disorders in adults and found that they could be discriminated easily. Only individuals with a depressive disorder reported low levels of positive affect, but both individuals with an anxiety disorder or a depressive disorder reported high levels of negative affect. Similarly, Brown, Chorpita, and Barlow (1998) showed consistent associations between negative affect and DSM–IV anxiety disorders and DSM–IV depressive disorders, in a sample of over 300 outpatients. However, later research by Weinstock and Whisman (2006) provides key evidence for negative affect and its role in the model. They tested a revised version of the model using data from the National Comorbidity Survey (see Kessler et al., 1994). Results showed high negative affect was consistent to the depressive and anxiety disorders, but notably, levels were substantially elevated in individuals with comorbid depression and anxiety.

The Tripartite Model has also been tested in children, though less frequently. Nonetheless, there has also been consistent support for the role of negative affect in the model using child and adolescent samples. In one of the earliest studies to examine the Tripartite Model in children, Joiner Jr., Catanzaro, and Laurent (1996) investigated the relationship between anxiety, depression and negative affect in 116 child and adolescent psychiatric inpatients, aged eight to 16. Using factor analysis, they showed a three-factor model of negative affect, depression and anxiety fit the observed data well, a finding consistent with the tripartite model. However, even stronger support for the tripartite model has been gained from research using stronger statistical methodologies such as SEM. Using SEM and confirmatory factor analysis, Lonigan, Phillips, and Hooe (2003) tested the tripartite model in a longitudinal study of 270 fourth to eleventh grade children. They showed that the negative affect was consistently related to symptoms of both depression and anxiety, and provided significant support for the role of negative affect in the development of symptoms of anxiety and depression.

Negative Affect and Heterotypic Comorbidity

Research has also clearly demonstrated that negative affect explains a large proportion of the heterotypic comorbidity between internalising and externalising disorders. Khan, Jacobson, Gardner, Prescott, and Kendler (2005) investigated the proportion of comorbidity explained by various personality dimensions, including negative affect, using SEM techniques in data from over 7000 participants from the population-based Virginia Twin Registry. Neuroticism accounted for 20 to 45 percent of the comorbidity within internalising disorders and 10 to 12 percent of the comorbidity within externalising disorders. However, neuroticism accounted for by

far the highest proportion of heterotypic comorbidity, accounting for between 19 and 88 percent of the variance between internalising and externalising disorders. On average, neuroticism accounted for over one quarter of the comorbidity among the disorders included in the study, leading Khan et al. (2005) to suggest that neuroticism (negative affect) was a key general underlying vulnerability factor for psychopathology and comorbidities.

Similar results supporting the role of negative affect in heterotypic comorbidity have been noted in child studies. Keiley et al. (2003) investigated common risk factors underlying co-occurring internalising and externalising syndromes, as assessed by the CBCL, using longitudinal data from 585 children from the American Child Development Project (see Dodge et al., 1994). They found that children with high levels of 'difficultness' (i.e. difficult temperament), were rated by mothers as having higher rates of comorbid internalising and externalising syndromes, leading the authors to conclude that negative emotionality may be a core part of the overlap between externalizing and internalizing symptoms as perceived by mothers. Similarly, Rhee et al. (2007) examined the degree to which the temperament characteristics measured before the age of three could explain covariation between CBCL assessed internalizing and externalizing behaviour at age four to 12 years. Using SEM to undertake genetic analyses on data from 225 monozygotic and 185 dizygotic twin pairs in the Colorado Longitudinal Twin Study, they showed that for both sexes, negative affect explained substantial and significant proportions of the heterotypic CBCL covariation. In summary, there is consistent and compelling evidence that negative affect plays a significant role in the development of comorbidity (Muris & Ollendick, 2005).

Mechanisms of Risk from Negative Affect

There is general consensus about the relationship between negative affect and the development of comorbidity within the literature. However, there is still substantial disagreement within the literature regarding the mechanisms of effect. Certainly temperament may influence key developmental experiences and interactions with the environment, and indeed at the core of most explanations is the idea that psychopathology occurs in individuals within the context of premorbid temperament/personality. However, part of the problem with attempting to understand the mechanisms of the risk effect of negative affect, is that there is a notable degree of overlap between measures of temperament and measures of psychopathological symptoms (Sanson, Prior, & Kyrios, 1990), which in part lead to the potential for conceptual and methodological problems distinguishing between temperament and psychopathology (Lengua, West, & Sandler, 1998). As a result, numerous models have been proposed to explain the relationship between temperament/personality and psychopathology. Four models in particular have gained acceptance as potential explanations for the temperament/personality–psychopathology relationship: the complication/scar model, the spectrum model, the pathoplasty/exacerbation model, and the vulnerability/predisposition model (Tackett, 2006).

According to the complication, or scar model, the development of psychopathology changes an individual's premorbid temperament/personality, such that the psychopathology may increase or decrease the level of a trait, relative to the premorbid level (Krueger & Tackett, 2003). This model is not particularly applicable to the idea of a psychopathological liability because, in part the direction of causality

is reversed. The spectrum model does not view personality and psychopathology as distinct entities, but rather as lying on a continuum or spectrum (or continua or spectra) such that the relationship between personality and psychopathology is dimensional, ranging from subclinical to clinical (Tackett, 2006). The pathoplasty/exacerbation model hypothesises that an individual's pre-existing personality characteristics may influence the manifestation of a DSM Axis I disorder, in course, severity, presentation, or prognosis (Krueger & Tackett, 2003). In the context of risk factor research, and the idea of a common psychopathological liability underlying psychopathology, the final model may be the most applicable. The vulnerability/predisposition model proposes that certain personality traits may place an individual at greater risk to develop a particular form of psychopathology (Tackett, 2006).

Tests of these four models have been undertaken in the child literature with reference to individual disorders. Some prospective longitudinal studies provide evidence for vulnerability/ predisposition and spectrum models, though testing the complication/scar and pathoplasty/exacerbation models is less common. However, there is little consensus regarding which model may explain the link most effectively (for comprehensive reviews see Krueger & Tackett, 2003; Widiger, Verheul, & van den Brink, 1999), though it should be noted that the models are not all mutually exclusive, and one discrete model may not provide a comprehensive answer (Tackett, 2006).

Summary

There is a clear, substantial, and consistent evidence base for a link between negative affect and childhood psychopathology as well as with comorbidity itself. It must be noted that the use of the 'difficult temperament' or behavioural inhibition in the research construct do create some lack of clarity with regard to the link between negative affect and psychopathology. This is because these concepts, while reflecting aspects of negative affect, are superordinate constructs encompassing many different aspects of the temperament construct in addition to negative affect. As a result, the degree to which dimensions or combinations of these dimensions are important for predicting risk for conduct problems is not entirely clear. However, the fact that there is general consistency in the results of studies using all three constructs (negative affect, 'difficult temperament' and behavioural inhibition), strongly suggests that it is negative affect that is key to understanding the link. As a result, negative affect must be considered as part of any common risk factor, or psychopathological liability, in any multivariate comorbidity models, such as being presented in this thesis.

Parental Psychopathology as a Parental Risk Factor for the Child

When looking at parental risk factors for the development of child psychopathology, parental psychopathology is one of the best known and most researched of such risk factors (Mäntymaa et al., 2012). Parental psychopathology is a particularly relevant candidate when considering constructs that may comprise a general liability factor in multivariate comorbidity, because research has consistently demonstrated that if a child has a parent, especially a mother, with psychopathology, their risk of developing their own psychopathology, and indeed comorbidities is substantially heightened.

Parental Psychopathology as a Risk Factor for Psychopathology

Link with Internalising Disorders

There is a wide body of research literature indicating a link between the presence of parental internalising psychopathology, especially the mood disorders, and persistent harmful effects on a child's cognitive and socio-emotional development (Cummings & Davies, 1994). In terms of development of psychopathology, children of depressed parents have consistently been found to be more likely to be depressed themselves (Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). For example, Singh et al. (2011) using a Children of Twins (CoT) design, examined a high-risk sample of 2555 children exposed to 1296 parents who were diagnosed with major depressive disorder (MDD) and who were twins from Australian Twin Register (ATR). The CoT design in this study allowed clear delineation of environmental and genetic factors that influence the parents and offspring, as well as delineation of any environmental confounds that twins share. The study found support for a causal association between parental and offspring MDD, most cogently demonstrated from the comparisons of offspring of monozygotic (MZ) twins discordant for MDD, who are as genetically related as half siblings. In such cases offspring of MZ twins exposed to parental depression more frequently met criteria for MDD than their cousins who were not exposed to parental depression, even after controlling covariates such as spousal depression (Singh et al., 2011). These results are similar to results found over the preceding 20 years (e.g. Orvaschel, Walsh-Allis, & Ye, 1988; Williamson et al., 1995). Associations have also been noted in other internalising disorders, with recent meta-analyses indicating that having a parent with a bipolar disorder increases a child's risk of developing bipolar disorder up to five-fold

(Hodgins, Faucher, Zarac, & Ellenbogen, 2002). Similarly children of parents with anxiety disorders have an elevated rate of anxiety disorders (Beidel & Turner, 1997).

Link with Externalising Disorders

The link between parental psychopathology and subsequent child externalising disorders, especially ADHD, is also well established. More than half of all parents with ADHD will have a child with ADHD (M. Weiss, Hechtman, & Weiss, 2000), with Faraone, Biederman, Mennin, Gershon, and Tsuang (1996) noting many of these families have more than one child with ADHD (see also Biederman et al., 1992; Biederman et al., 1998). Using 2040 families of twins from the Australian Twin ADHD Project, N. C. Martin, Levy, Pieka, and Hay (2006) showed, using univariate and bivariate genetic models, that ADHD showed high genetic heritability of .5 or greater in all subtypes, matching previous research in both westernised cultures (e.g. Levy, Hay, McStephen, Wood, & Waldman, 1997; Nadder, Silberg, Eaves, Maes, & Meyer, 1998; Sherman, McGue, & Iacono, 1997) and less-westernised cultures such as India (e.g. Latha, Nair, & Bhat, 2012).

Parental Psychopathology as a Broadband Risk

There is a body of research evidence that makes parental psychopathology particularly relevant when considering risk factors that may form a general liability factor. Considerable empirical evidence suggests that parental psychopathology may not be a disorder specific risk factor; that is the presence of disorder X in a parent may not just increase the risk for the child developing disorder X, but may also increase the risk of a child developing any psychopathological disorder. Tambs et al. (2009), interviewed 2801 young-adult Norwegian twins to investigate the degree to

which genetic risk factors were shared across anxiety disorder, rather than unique to individual anxiety disorders. They showed that the latent genetic liability to all anxiety disorders was substantially higher than the liability to the individual anxiety disorders. Similarly meta-analytic and systematic reviews (DelBello & Geller, 2001; Lapalme, Hodgins, & LaRoche, 1997) show that children with parents diagnosed with bipolar spectrum disorders, are not only at increased risk of developing bipolar disorder, but also are at increased risk of developing other mood disorders independent of the bipolar spectrum, such as major depressive disorder and dysthymia.

The disorder non-specificity of risk may not merely be limited to disorders within one class (e.g. mood or anxiety). Orvaschel et al. (1988) examined the prevalence of psychopathology in children of parents with and without major depression. Rates of all psychopathology in the children of depressed parents were consistently higher than those for the control group, with significant differences noted in the affective, anxiety and ADHD disorder classes. Similarly, the earlier cited Singh et al. (2011) twin study, while definitively finding that having a parent with MDD was associated with heightened hazard ratios for the child in developing depression, also noted that having a parent with MDD significantly and substantially increased the risk for the child developing conduct disorder.

Goodman et al. (2011) note that in general, the relationship between parental depression and internalizing problems is not significantly stronger than the relationship between maternal depression and externalizing problems. In a meta-analysis of the effect of parental depression, Connell and Goodman (2002) reported

weighted mean *r*-type effect sizes of .18 and .14 for maternal and paternal depression respectively for offspring internalizing problems and .17 and .16 respectively for offspring externalizing problems, demonstrating that the risk for psychopathology is not to be considered disorder specific. As such research can investigate parental psychopathology as a risk factor, using broad measures of psychopathology, such as the Hopkins Symptom Checklist (HSCL: Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974a; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974b), rather than more specific measures.

Equivalence of Maternal and Paternal Psychopathology as a Risk Factor

The majority of early studies investigating parental psychopathology as a risk factor, tended to investigate only maternal psychopathology, giving scant regard to paternal psychopathology as a risk factor (Marmorstein, Malone, & Iacono, 2004). However, while the evidence for paternal psychopathology is more limited and less generalisable, most results suggest the presence of disorders in mothers and fathers present equal risks for offspring. Indeed recent meta-analyses that were cited in the previous paragraphs (Connell & Goodman, 2002; Kane & Garber, 2004) have shown that the effect sizes for maternal and paternal psychopathology as a risk are equivalent. There are two main exceptions to this equivalence noted by Connell and Goodman (2002). Firstly, depression in mothers was found to be more closely related to children's internalizing problems than depression in fathers, though no difference was noted for child externalizing problems. Secondly, alcoholism and substance abuse disorders in mothers was more closely related to child externalizing problems than were such disorders in fathers, though no difference was noted for child internalizing problems. However, in both cases, there was still a significant and

substantial relationship between paternal psychopathology in either parent, and child behaviour problems. It should also be noted that there are some indicators that maternal and paternal psychopathology are in some ways interactive. Research by Brennan, Hammen, Katz, and Brocque (2002) demonstrated that maternal and paternal depression had an additive effect on the risk for child externalizing disorders, and had an interactive effect on the risk for child mood disorders. Thus when considering risk, such interactive/additive effects may be considered where possible.

Parental Psychopathology and Comorbidity

Unfortunately, there is comparatively limited research investigating parental psychopathology as a risk factor for comorbidity per se. However the research available generally supports the idea of parental psychopathology as a comorbidity risk factor. Probably the most informative study was undertaken by Kopp and Beauchaine (2007), who investigated comorbid conduct problems and depression by examining the relationship between parental psychopathology and children's diagnosis in 180 families with an eight–12-year-old child diagnosed with conduct problem, depression, both conditions comorbidly, or neither condition. The results of their study were mixed, but did suggest that comorbid internalizing and externalizing symptomatology is most likely to be a product of high rates of psychopathology, and indeed comorbidity in parents.

Mechanisms of Risk from Parental Psychopathology

There are many proposed mechanisms for how parental psychopathology affects children, with multiple mechanisms acting alone or in combination, potentially

underlying the association between parental and child psychopathology (Farah et al., 2008). Goodman and Gotlib (1999) proposed four potential mechanisms through which parental psychopathology can lead to maladaptive developmental outcomes for children. While these mechanisms were initially developed to explain the links between maternal depression and childhood psychopathology, they are considered applicable to a wider range of parental, especially maternal, psychopathology (Connell & Goodman, 2002).

Genetic Transmission

When considering parental psychopathology, the most commonly investigated mechanism of transmission, and the first mechanism proposed by Goodman and Gotlib (1999), is genetic transmission. There is a wide body of empirical research indicating that a child with a parent, especially a mother, with psychopathology, has heightened risk of developing psychopathology. As an example, behaviour genetic studies estimate that up to half of anxiety related behaviours, such as behavioural inhibition, anxiety sensitivity, or fear of negative evaluation, is inherited (Bögels & Brechman-Toussaint, 2006). Given that both parents contribute 50 percent of their children's genes, it could be thought that psychopathology in the child would be equally associated with psychopathology in either mothers or fathers. However, this prediction of equal influence assumes that disorders are equally heritable in either sex, and while this may be true for some disorders, it is not universal (e.g. alcohol disorders; see Cloninger, Sigvardsson, & Bohman, 1996). Reviews have also shown that there is great variability in the nature and degree of heritability of psychological problems across disorders (Rutter, Silberg, O'Connor, & Simonoff, 1999a, 1999b). As outlined earlier, the relationship between maternal depression and internalizing

problems is not significantly stronger than the relation between maternal depression and externalizing problems (Goodman et al., 2011), indicating that transmission may not be disorder specific; in itself suggestive of the presence of a common risk factor for comorbidity.

Additionally, while the evidence for a substantive genetic component is undeniable, the great degree of variability within and across disorders, combined with the notable proportion of variance left unexplained after considering genetic factors, means that genetic transmission is not the entire picture when it comes to looking at the mechanisms of transmission. Indeed, evidence that treating maternal psychopathology, in the absence of child specific treatments, could also improve childhood psychopathology (Weissman et al., 2006), led to the view that there were equally important environmental factors at play (Pilowsky, Wickramaratne, Nomura, & Weissman, 2006), which relate to the next three mechanisms proposed by Goodman and Gotlib (1999).

Dysfunctional Neuroregulatory Mechanism Development

The second proposed mechanism of transmission is through the development of dysfunctional neuroregulatory mechanisms. This is most commonly associated with maternal dysfunction, and is based on complications during the offspring's prenatal development that are a direct result of parental psychopathology. Research has linked a variety of forms of maternal psychopathology to poor prenatal care, and adverse prenatal conditions, such as foetal exposure to neuroendocrine dysfunction in the case of maternal depression, or exposure to toxic substances as in the case of substance abuse disorders (Connell & Goodman, 2002; Goodman & Gotlib, 1999).

Further evidence for dysfunctional neuroregulatory mechanisms comes from animal studies which show that poor maternal care has been shown to have enduring negative effects on hippocampal development in rats (Liu, Diorio, Day, Francis, & Meaney, 2000), with some similar findings noted in primates (Kozorovitskiy et al., 2005; Parker, Buckmaster, Sundlass, Schatzberg, & Lyons, 2006).

Exposure to Maladaptive Behaviour

The third proposed mechanism of transmission is through exposure to maladaptive affect, and behaviour. Here parental psychopathology is no longer viewed as a biological risk factor, but rather an environmental risk factor, with the child learning the maladaptive behaviours, affective disturbances, and cognitive distortions that are associated with the particular parental psychopathology. For example, meta-analyses suggest that mothers with depression have greater disengagement, greater negative behaviour, and low levels of positivity in interactions with their children (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007; Kochanska, 1991). These problems can manifest in many ways, including an inability to be nurturing, difficulty showing firm and consistent discipline, and tendencies to 'give in' to child tantrums creating unhelpful negative reinforcement (Marchand & Hock, 1998). Given that mothers are overwhelmingly the parent most responsible for child-rearing activities, it is considered more likely that children are exposed to more issues associated with psychopathology in mothers than in fathers. If quantity of exposure is the critical factor, maternal psychopathology in general should be more closely associated with internalizing and externalizing problems in children than paternal psychopathology (Connell & Goodman, 2002), though such views are not necessarily universal (Lamb, 1997).

Contextual Stressors

The final mechanism proposed to explain the association between parental and child psychopathology relates to contextual stressors. The argument in this case is that psychopathology in a parent or parents creates context specific stress within the family unit, and that children may develop emotional and behavioural problems as a result of maladaptive attempts to cope with those stressors. Indeed such a view has varied empirical support as there is a great deal of research linking psychopathology in adults with sources of contextual stress, including familial disruption, marital discord, and economic pressure (Connell & Goodman, 2002: see also next section on family functioning).

Summary

In summary, it is evident that parental psychopathology is a particularly relevant candidate when considering constructs that may comprise a general liability factor in a multivariate model of comorbidity. While it must be acknowledged that not all children of parents with psychopathology go on to develop psychopathology of their own, indicating that any relationship is probabilistic and not deterministic (Suveg, Shaffer, Morelen, & Thomassin, 2011), there is still substantial evidence for a link between parental and childhood psychopathology. Such a link is evident across the broader spectrum of childhood psychopathology, and in some evidence suggests links to comorbidity in itself. Certainly the fact that parental psychopathology may not be a disorder specific risk factor, but may increase the risk of a child developing any psychopathological disorder, independent of the type of parental disorder, makes this construct worthy of close consideration as a construct that may comprise a general liability factor as proposed in this thesis.

Family Functioning as an Environmental Risk Factor for the Child

In terms of more pure environmental risk factors for childhood psychopathology, there has been a considerable focus on the effect of a poor familial environment – the environment in which a child is raised – in the development of psychopathology. Indeed the literature investigating the relationship between family functioning and psychopathology has grown substantially in the last two decades (Naghavi, 2011), though more frequently in terms of association with the development of individual disorders than in terms of association with comorbidity. Nonetheless poor family functioning is a prime candidate for association with a common risk/liability factor because of its universal association as a risk factor for the development of a range of disorders or syndromes across the spectrum of psychopathology.

Family Functioning and the McMaster Model

Despite the increase in research on familial environments, defining family functioning is not a straight-forward proposition, in part because of the numerous definitions available. However for the purposes of this discussion, family functioning refers to nature in which actions of the family are performed, the social and structural properties of the global family environment that underlie such actions, and the patterns of the interconnecting relationships between that family system's members (Alderfer et al., 2008; Lesser, 1985; Lewandowski, Palermo, Stinson, Handley, & Chambers, 2010). Such a definition is broad and includes marital/dyadic relationships, the nature of parent-child affective relationships, and the nature of parenting and parental control of children, as all of these components have been implicated to influence child adjustment (Frick, 1994; Lengua, Wolchik, Sandler, & West, 2000; Loeber & Stouthamer-Loeber, 1986).

The McMaster Model of Family Functioning

While there are many models proposed to conceptualise family systems and family functioning, one of the most frequently used and most well validated models is the McMaster Model of Family Functioning (Epstein, Bishop, & Baldwin, 1978). The McMaster Model uses a systems based approach which sees the family as containing systems within systems, in a manner outlined in the above definition (Epstein, Bishop, Ryan, Miller, & Keitner, 1993). Developed in the late 1950s, it was slowly refined over the next twenty years, until the classic model was described in the late 1970s by Epstein et al. (1978). While there have continued to be some minor revisions, the model remains largely unchanged to this day.

The McMaster model does not cover every aspects of family functioning but, rather usefully in a discussion on risk factors, focusses on those dimensions of functioning which are seen as having most impact on the emotional and physical health problems of members of the family system (Epstein et al., 1978; Epstein, Ryan, Bishop, Miller, & Keitner, 2003; Miller, Ryan, Keitner, Bishop, & Epstein, 2000). The model focuses on six dimensions; Problem Solving, Communication, Roles, Affective Responsiveness, Affective Involvement, and Behavioural Control. The McMaster model does not see any dimension as more important in developing good family functioning. Rather, each dimension is seen to contribute equally to the functioning of a family system (Epstein et al., 1978; Epstein et al., 1993; Epstein et al., 2003). Within the McMaster model, healthy family functioning is conceptualised as involving clear communication, well defined-roles, cohesive structure and good affect regulation, whereas poor family functioning is conceptualised as being highly conflictual, with disorganised structure, and poor affective and behavioural control

(Alderfer et al., 2008; Epstein et al., 2003; Herzer et al., 2010; Lewandowski et al., 2010; Miller et al., 2000). The McMaster Model has been used widely in both research and clinical contexts (Herzer et al., 2010), in part because specific measures have been developed to assess family functioning based on this model. Probably the most widely used of these instruments is the Family Assessment Device (FAD: Epstein, Baldwin, & Bishop, 1983). The FAD is used to assess overall health/pathology of the family environment across the six McMaster dimensions, plus a General Functioning scale, which is a measure of global functioning. Indeed it is this General Functioning subscale that is often used in research assessing family functioning as a risk factor in psychopathological development, having been validated as a standalone brief measure (Ridenour, Daley, & Reich, 1999).

Poor General Family Functioning as a Risk Factor for Psychopathology

Risk for the Development of Internalising Disorders

There is considerable research evidence that poor family functioning, as measured by the general functioning scale of the FAD, is associated with a wide range of childhood psychopathology, both internalising and externalising. There is substantial literature linking familial dysfunction to the development of depression. Stein et al. (2000) investigated family functioning in three groups; families where one child had a diagnosis of depression, families with a child at high-risk of depression, and a control group. Maternal FAD General Functioning scores indicated significantly better functioning in families without a child with depression, than both families with a depressed child, and families with a child at high-risk of depression. Paternal FAD general functioning ratings were similar, with families with a child with depression showing significantly poorer general functioning than all other groups. Similar

results were found by Dietz et al. (2008), who showed increased family discord in families with currently depressed children when compared to both high-risk and control groups. Interestingly, they found some prodromal features of family discord in families with children at high-risk of depression, with family functioning significant worse than the control group, but better than families with currently depressed children.

There is also a substantial literature linking familial dysfunction to anxiety disorders (Bögels & Brechman-Toussaint, 2006). Families showing extremes of cohesion (indicative of over or under involvement), one of the six functioning aspects of the McMaster model, and which is viewed as demonstrating poor family functioning, has repeatedly been associated with child social anxiety (Peleg-Popko & Dar, 2001). Similarly, poor family communication and lack of family encouragement of autonomy have been related to child anxiety (Peleg-Popko, 2002). Further to this, high levels of familial disengagement and negative conflict have also been related to parent report of child anxiety, depression and aggression (Katz & Low, 2004).

Risk for the Development of Externalising Disorders

There is also a body of evidence about the relationship between poor general family functioning and the externalising disorders, especially ADHD. Cussen, Sciberras, Ukoumunne, and Efron (2012), using an Australian community-based sample of around two hundred primary caregivers, examined the relationship attention-deficit/hyperactivity disorder (ADHD) symptomatology and family functioning. Parents of children screening positive for ADHD reported poorer family quality of life, and decreased levels of family activities than parents of children without

ADHD. These results match similar findings by Kaplan, Crawford, Fisher, and

Dewey (1998), who found that parents of children with ADHD reported more

difficulties in general family functioning on the FAD than did normal controls.

Family Functioning as a Risk Factor for Comorbidity

Research has also demonstrated that poor family functioning is associated with

comorbidity itself. Burt et al. (2003) investigated the effect of family functioning, as

determined by high levels of parent-child conflict, was associated with the

comorbidity among the externalising disorders. Using same-sex twin pairs, they

examined genetic and environmental contributions to the relationship between

conflict and the covariation among the externalising disorders using structural

equation modelling (SEM). They compared that model, with separate models

examining the relationship between parent-child conflict and each disorder

individually. They found that the model examining covariation among all disorders

was a better fit than models looking at the effect of conflict disorder by disorder.

Furthermore, parent-child conflict accounted for a third of the covariation among the

disorders, though mediated by other genetic and environmental factors, suggesting

comorbidity among these disorders partially reflects poor family environment.

Effects of family functioning on comorbidity have also been shown in cross-cultural

samples, with Kiliç and Şener (2005), using a Turkish sample, showing that families

of children with comorbid ADHD and CD/ODD had significantly poorer functioning

than families of children with ADHD only.

Mechanisms of Risk from Poor General Family Functioning

There has been considerable debate as to whether poor family interactions precede childhood psychopathology, or whether it is a consequence of childhood psychopathology. As demonstrated from the research cited above, most research looking at family functioning in psychopathology is cross-sectional and thus the question of directionality is unclear. However, research by Dietz et al. (2008) in depression, suggests poor family interactions are likely bidirectional and may precede the onset of major depressive disorder because these relationships show no change when in remission. This led Dietz et al. (2008) to conclude that it is most likely that poor family interactions serve as risk factors for recurrent depression in youths, rather than being a consequence, though such a conclusion is not definitive.

The process through which poor family functioning contributes to childhood disorders is also not clear, in part because of the difficulty in studying families with young children who show poor functioning (Dickstein, Andre, Sameroff, Seifer, & Schiller, 1999). Emery (1989) outlined several main processes through which marital turmoil may contribute to childhood disorders. Some of these are applicable to family functioning more generally, because families with poor family functioning are often characterised by marital turmoil (Epstein et al., 2003). Emery (1989) suggested that marital (and familial) turmoil may contribute to childhood disorders by providing children with ineffective models of communication, conflict resolution and problem-solving, which leads to poorer coping behaviours in the child. Secondly, marital turmoil may disrupt bonds between parent and child, and thirdly poor family environments may serve as a general stressor, threatening the child's sense of security (Emery, 1989).

Certainly there is some evidence supporting these possibilities. Work in severe childhood/adolescent depression and suicidality has revealed that overall family dysfunction is generally poorer in families where a child has issues with suicidality, but that there are specific problems with communication and problem-solving (Prinstein, Boegers, Spirito, Little, & Grapentine, 2000). King, Hill, Naylor, Evans, and Shain (1993) noted in research on suicidality, that FAD subscales measuring communication and problem-solving, could differentiate suicidal from non-suicidal adolescents (see also Wagner, 1997). This suggests that poor behavioural models from which to develop effective communication and problem-solving may be a risk factor for children.

It must be noted however, that the effect of a poor family environment may be interactive/additive in nature rather than direct. Certainly, the interactive effect with parental psychopathology has long been noted. Family functioning is often regarded as poorer where there is a depressed parent (especially a depressed mother), with more negativity and verbally averse behaviour in parent-child interactions, and poorer affective engagement (McMahon & Wells, 1998), in turn leading to increased risks of psychopathology. Such interactive/additive effects are certainly worth considering and will be discussed further in the next section.

Summary

Overall there is substantial evidence for a link between family functioning and childhood psychopathology across the broad spectrum of childhood emotional and behavioural problems, as well as some links to comorbidity itself. The research regarding the directionality of the effect (comorbidity causes dysfunction or vice

versa) is limited, in part because of the difficulty in researching families with poor functioning, though it may be interactive in its effect with other risk factors. Nonetheless poor family functioning is a notable, consistent and easily ascertained marker of potential childhood psychopathology.

The Need for a Multivariate Approach to Consideration of Risk Factors

Despite the evidence presented in the previous sections, the study of risk factors for psychopathology is not without limitations. One of the major limitations is that while there is ample research demonstrating correlates of either internalizing or externalizing problems and indeed comorbidity, a sizeable proportion of this literature tends to focus on a single risk factor construct in isolation. That is, the focus of much research is on how a particular trait/risk factor is related to a particular form of psychopathology or bivariate comorbidity; an issue especially true for temperament, but also for family functioning and parental psychopathology (Wolff & Ollendick, 2006). Indeed, in regards to this limitation, there is much similarity between the problems identified in the comorbidity literature and this issue with risk factor research.

Additive and Interactive Effects of Risk Factors

A focus on singular risks in isolation is problematic, as it does not allow examination of how risks may operate together in terms of placing a child at risk for psychopathology. Indeed Frick et al. (1992) argue that independent examination of risk factors creates an initial confound in research because of their

interconnectedness. This is especially true, given the current view in developmental psychopathology is that parental, child and family risks are likely to work in a transactional and or reciprocal manner (Cicchetti & Toth, 2009), and not in isolation (Young Mun, Fitzgerald, von Eye, Puttler, & Zucker, 2001). Yet despite this prevailing view, there has been critique of much research because it "has focused on isolated variables, ignoring possible additive influences" (Atzaba-Poria, Pike, & Deater-Deckard, 2004, p. 707).

Additive and Cumulative Effects of Risk Factors

It is likely that the presence of multiple risk variables operating simultaneously will lead to a greater probability of the development (and maintenance) of psychopathology, than if only a single risk factor is present (Nelson et al., 2007). This is reflected in the results of several studies that repeatedly show that the more risk factors present, the more likely psychopathology is likely to develop in the child. For example, Atzaba-Poria et al. (2004) in a cross cultural study of 59 English origin and 66 Indian origin children in metropolitan England, showed that independent of ethnicity, the more accumulated risk factors a child had, the higher CBCL behavioural problems that they displayed. This indicates that the level of cumulative risk is vital in understanding psychopathology, regardless of the specific type of risk (see also Appleyard et al., 2005; Shaw, Vondra, Hommerding, Keenan, & Dunn, 1994). Indeed many generic models of risk/protective factors, such as the diathesis-stress model, model such additive effects, with the diathesis-stress model at its simplest hypothesising that environmental risk interacts with genetic predisposition leading to psychological problems in children and adolescents (see Ingram & Luxton, 2005 for a review).

Overlapping Effects of Risk Factors

Another reason for investigating multiple risk factors simultaneously is that there may be overlapping effects of risk factors. That is two risk factors may not separately account for independent variance in risk, but rather share variance in explaining psychopathology. Thus two (or more) risk factors may explain the same proportion of risk variance, with one variable having a causal primacy, or neither variable having a causal primacy but with both variables acting reciprocally (Velders et al., 2011). As an example, using a population based study with over 2500 participants, Velders et al. (2011) investigated the effect of parental depressive symptoms on the risk for childhood psychopathology. They found that while parental depression increased the risk of child emotional and behavioural problems, the increase was almost entirely explained by parenting and family variables. As a result of this and similar studies, it has been suggested that parental psychopathology and family functioning have reciprocal effects, without a causal primacy (Rehman, Gollan, & Mortimer, 2008). Indeed the relationship between parental and child psychopathology has long been known to be confounded by family discord (Rutter & Quinton, 1984), because parental psychopathology in itself is associated with worse family functioning (Hughes, Hedtke, & Kendall, 2008; Pilowsky et al., 2006). Thus debate remains as to the extent to which these risk factors independently contribute to child problem behaviour (Velders et al., 2011), and as such separate analysis of them may lead to spurious conclusions with regard to their effect; yet another reason why multiple risk factors should be considered simultaneously.

Interactions Between Risk Factors

More probably however, it is possible that there may be interactions between risk factors (Nelson et al., 2007). Indeed, models that take into account both the additive and interactive effects risk factors such as family functioning and temperament have long been advocated (Sanson & Rothbart, 1995). To illustrate how an interaction may operate, consider the role of family functioning and temperament. While both family functioning and temperament would be expected to directly predict child adjustment problems, for some children, the effect of a poor family environment would be expected to exacerbate their risk for psychopathology (Lengua et al., 2000). That is, it may be predicted that a child with high trait negative affect may be likely to develop psychopathology, but that this risk may be mitigated – that is they may be less likely to develop psychopathology - if the family environment they are exposed to functions well. However, a child with high trait negative affect who is raised in a poorly functioning family environment, may be placed at an exponential risk for developing psychopathology.

Certainly the research evidence has demonstrated such interactions between risk factors do exist. Morris et al. (2002) found a temperament-by-family functioning interaction, showing an interaction between irritability, a component of negative affect, and poor parenting. Children with high trait irritability, who experienced poor parenting, had significantly greater levels of externalizing behaviour than children with high trait irritability that did not live in environment with poor family parenting. This matched a previous study by Sanson, Oberklaid, Pedlow, and Prior (1991) who found that while children with a 'difficult temperament' had only a slightly raised incidence of adjustment problems relative to others, when 'difficult temperament' occurred together with poor family functioning/parenting, the level of risk for

problems increased substantially. Thus the role of negative affective temperament may not lead to psychopathology itself; rather it may do so in conjunction with particular environments (Oldehinkel, Veenstra, Ormel, de Winter, & Verhulst, 2006; Veenstra, Lindenberg, Oldehinkel, De Winter, & Ormel, 2006). Similarly, research has noted multiplicative effects between temperament and parental psychopathology. Wong, Zucker, Puttler, and Fitzgerald (1999), using multiple regression showed aspects of child temperament related to negative affect was a significant predictor of externalizing behaviour, but that this association was significantly stronger in the presence of high parental psychopathology.

Family functioning by parental psychopathology interactions have also been noted. Knappe, Beesdo, et al. (2009) investigated the link between various risk factors and the development of social phobia in a community sample of 1395 adolescents and their parents followed for over a decade. They found that dysfunctional family functioning characteristics were associated with higher rates of social phobia and greater persistence in the disorder. However, this effect was particularly pronounced when there was parental psychopathology, though subsequent research (Knappe, Lieb, et al., 2009) suggests that only particular aspects of family functioning may be at play. Children in these families are not only at an increased risk of psychopathology because they have a parent with psychological problems, but also due to an increased likelihood of exposure to marital conflict and poor family functioning that is consistently found to result from parental psychopathology (Hughes & Gullone, 2008; Kazdin & Kolko, 1986).

Complex Interactions Between Risk Factors

Interactions may not always be multiplicative in nature, but may be very complex. Crawford et al. (2011), using a sample of 65 three to five year old children, found that negative affect and family functioning had significant direct effects on childhood internalizing symptoms, with the role of parental psychopathology, in this case measured using pre-correlates of pathology, mediated by family functioning in the prediction internalizing symptoms. More generally, Burt et al. (2003) showed conflictual relationships between parents and children (related to poor family functioning) appears to act as a common vulnerability that increases risk for multiple childhood disorders, but that the association was mediated by other genetic and environmental factors. Given there is the probability of additive and interactive effects of risk factors in the development of psychopathology, there is a need to employ a multivariate perspective of the risk-psychopathology relationship in children. Indeed this is the same as the need to employ multivariate techniques when considering comorbidity. Such multivariate perspectives of the risk-psychopathology relationship will provide a more comprehensive understanding of these relationships (Tackett, 2006).

General and Domain-Specific Risk Factors

Just as much research has tended to examine risk factors in isolation, most have examined risk factors in the development of individual disorders, or either just internalizing or just externalizing problems in isolation. While this is eminently understandable, it is a serious shortcoming in the research literature, given that comorbidity is such an issue in psychopathology research. It is very possible that certain risk factors may be related to co-developing problems and comorbidity,

whereas other factors may be specific to only one domain of problem behaviours (Wolff & Ollendick, 2006).

Some research has demonstrated correlates of internalizing or externalizing problems in the same study, but has done so through separate analyses (e.g. Buist, Deković, Meeus, & van Aken, 2004; Leve, Kim, & Pears, 2005). Only a limited number of studies have investigated how risk factors might differentially impact upon internalizing and externalizing domains (e.g. Mesman & Koot, 2000; Weiss et al., 1998), and only a couple of studies have looked at how risk factors may be related to the broader spectrum of childhood psychopathology, and indeed comorbidity (e.g. Lee & Bukowski, 2012; Mäntymaa et al., 2012). This paucity of research on risk factors for comorbidity, only allows speculation as to how risk factors affect comorbidity (Wolff & Ollendick, 2006), and further research in this area is required. Such issues will be addressed in this thesis, with a multivariate model of comorbidity being proposed, and then once validated, the impact on this model of a multivariate array of risk factors and risk factor interactions will be investigated.

Summary

The previous section has clearly demonstrated that the aetiology of psychopathology is a complex, multifactorial process (Ollendick & Hersen, 1999), with additive and interactive effects of risk factors in the development of psychopathology, not just possible, but probable. As a result, a multivariate perspective of the risk-psychopathology relationship in children is necessary (Tackett, 2006), in a manner similar to the need for a multivariate perspective in comorbidity research. This certainly allows for a more comprehensive understanding of the risk-

psychopathology relationship. However, to take a multivariate approach to this topic, consideration must be given to methodology used.

Methodology for Analysing the Effects of Risk Factors

The majority of research conducted on risk factors to date has been of two types; ANOVA-type and correlational. The first subset of research uses what is often termed 'main-effect models' (Crawford et al., 2011), and utilises group based research. Such research often uses dichotomous groups (e.g. high/low levels of risk trait), examining the direct impact of risk factors on child psychopathology. Unfortunately this type of research is often limited because it tends to assess risk factors in isolation (Crawford et al., 2011; Vasey & Dadds, 2001). The second type of research, and generally the more widely used, has been purely correlational, commonly using multiple regression models. Such models do allow for the assessment of multiple risk factors simultaneously. However, Tackett (2006) argues that such correlational research also has limitations, as it seeks to identify relationships between risk factors and psychopathology, but does not necessarily provide a basis for understanding the relationships.

Use of Sophisticated Psychometric Techniques: Structural Equation Modelling
In an attempt to overcome the limitations of the ANOVA-type and correlational approaches previously outlined, there has been a recent growth in the use of Structural Equation Modelling (SEM) techniques (e.g Crawford et al., 2011; Lee & Bukowski, 2012), which overcome these limitations. Using SEM can provide an easy way to assess the impact of multiple risk factors as SEM has the flexibility of multiple regression based models. Additive effects can be modelled simply by

regressing risk factors onto a base model simultaneously. Similarly, interactive effects can be modelled by regressing interaction terms for all combinations of risk factors onto a base model. This can be done easily in most SEM programs (e.g. MPlus, Muthen & Muthen, 2010) by programming the multiplicative term (i.e. risk factor A $\times$ risk factor B).

The use of SEM can also provide a basis for understanding the relationships between risk factors and disorders, an issue previously identified as a major shortcoming of current research (Tackett, 2006). Firstly, use of SEM allows models of disorders and groups of disorders which have been validated (e.g. Tripartite Model) to be specified. Then once a base model has been specified, SEM uniquely allows the impact of multiple risk factors to be assessed. Importantly, this impact may not just be on the model as a whole, but on individual aspects of the model, which would allowing a deeper understanding of how the risk factors may uniquely impact different aspects of a disorder. Such modelling would easily allow for the assessment of which risk factors may be related comorbidity, and which may be specific to only one domain of problem behaviours, further overcoming problems identified previously (Wolff & Ollendick, 2006). In the case of a multivariate model of comorbidity assuming a common psychopathological liability, such as outlined in Chapter 1, and as will be proposed in the two subsequent chapters of this thesis, SEM would allow a base model of comorbidity to be specified, and then allow easy assessment of which risk factors are associated with the common factor, and which may only be related to specific domains of disorders.

Indeed that is the aim of the third study in this thesis (Chapter 5). Based on the results of Studies 1 and 2, which will specify a unique multivariate model of comorbidity, the effect of risk factors will be assessed, both additively and in interaction, on various aspects of the model, allowing an understanding of the interplay between risk factors and comorbidity. Such research would be unique, in part because it overcomes some of the major limitations currently identified in the research literature. This would also clearly provide a more systemic and integrative approach to understanding childhood disorders and their causes; an approach long advocated in the field of developmental psychopathology (Cicchetti & Cohen, 1995).

Chapter 3 - Study 1: Testing for a Common Factor Underlying Childhood Psychopathology, Using the BiFactor Model

Background

Since the publication of Feinstein's classic paper (Feinstein, 1970) on the concept of comorbidity, comorbidity has been the subject of much debate within the clinical psychology research literature (see Spitzer, 1994). As outlined in Chapter 1, a general conceptualisation of disorders as discrete entities and part of a simple structure, may not be the most appropriate conceptualisation of childhood psychopathology. Angold et al. (1999) argue that substantive causes must be sought, because none of the possible methodological or nososlogical explanations provided for comorbidity explain the entire phenomenon, and understanding of these issues is key to continued development of diagnosis and treatment of persons with psychological illness. This is especially important in childhood psychopathology, because understanding the aetiology of childhood comorbidity is vital for appropriate treatment and management, and for the development of preventative intervention (Angold et al., 1999; Caron & Rutter, 1991).

As outlined in Chapter 1, the most notable deficit with current research into comorbidity to date has been that it has been almost exclusively bivariate, investigating individual disorder pairs and attempting to provide explanations about the cause of the comorbidity between that pair of disorders. While bivariate modelling research has utility, it is clear that the phenomenon of comorbidity is

definitively multivariate in nature; a concept Krueger and Markon (2006) refer to as "multimorbidity". That is, comorbidity occurs at above chance levels between all disorders across the broad spectrum of psychopathology, rather than just between certain specific disorder pairs (Batstra et al., 2002). Thus if the phenomenon of comorbidity is multivariate in nature, there is a need to move beyond bivariate models of comorbidity, and develop multivariate models to provide substantive explanations.

Towards A Common Factor Model

In Chapter 1, it was noted that the most promising substantive explanation for comorbidity in the bivariate domain were models indicating comorbidity is caused by shared underlying causal risk factors between individual pairs of disorders. However, the most supported of this type of models, such as the Neale and Kendler (1995) 'Correlated Risk' model, is problematic if extended into the multivariate domain, because of a lack of parsimony. One interesting, alternative model of shared underlying causal risk factors that is considerably more parsimonious when considering multivariate comorbidity proposes a single common factor underlies all psychopathology. This model, which was conceptualised among others by Klein and Riso (1993), and operationalised by Neale and Kendler (1995), suggests comorbidity between psychological disorders/syndromes may be influenced by a higher-order factor (Lilienfeld, 2003), which for the purposes of this thesis is referred to as a psychopathological liability. This model hypothesises that comorbidity occurs because the comorbid disorders are alternate manifestations of a 'single' common liability, which is a multifactorial combination of heritable and environmental causes, or risk factors. While there has been little support for this single common

factor model in bivariate research (Neale & Kendler, 1995; Rhee et al., 2008), given it is considerably more parsimonious than other models of shared risk when transferred to the multivariate domains, it is worth further consideration in multivariate analysis.

The Current Study

This current study aims to test a common factor model based on the common liability model proposed by Klein and Riso (1993), and operationalised by Neale and Kendler (1995), and discussed in Chapter 1. This model will be tested across the breadth of childhood psychopathology and comorbidity (i.e.: multivariately), using Structural Equation Modelling (SEM) techniques, which as discussed in Chapter 1, is one of the most appropriate techniques for testing multivariate models of comorbidity (Krueger & Markon, 2006). This will be undertaken using models not previously considered in this comorbidity research, for reasons discussed presently.

Problems with Testing Bivariate Models in the Multivariate Domain: A Need to Develop Hybird Models

Krueger and Markon (2006) argue that when engaging in multivariate modelling of comorbidity new or hybrid models must be considered, because there are issues that cannot be considered or specified when simply investigating bivariate pairs of disorders. When considering a common factor model as implied by the common liability model, transference to a multivariate model – that is beyond a single disorder pair - may not necessarily be as straightforward as specifying more paths for the greater number of disorders. It is most likely that other theoretical extensions to the model must be considered during the extension to multivariate comorbidity. As a

result a hybrid of the basic common factor model may need to be considered and specified. One extension to the basic common factor that must be considered, is the concept of domain specific factors, specifically regarding internalising and externalising domains.

Domain-Specific Factors: Internalising and Externalising Factors

The possibility of domain-specific factors was outlined extensively in Chapter 2 in the context of risk factors. However, domain-specific factors must also be considered in the context of psychopathology (Wolff & Ollendick, 2006). The idea of a common factor model is derived from the idea of a 'single' common liability; a multifactorial combination of heritable and environmental causes, or risk factors. However, studies that have investigated how risk factors differentially impact the developmental trajectory of the internalising and externalising domains disorders (e.g. Buist et al., 2004; Leve et al., 2005), have consistently indicated that there may be both common and domain-specific features to the development of childhood psychopathology. That is, it appears that there may be potential risk factors that affect comorbidity as a whole, but other risk factors may only be specific to one domain of problem behaviour. Indeed given that there is significant support for the existence of the internalising and externalising domains, both in the DSM categorical system (Kendler, Prescott, Myers, & Neale, 2003), and more definitively in empirically validated syndromes (Achenbach et al., 2008), it makes sense to consider the internalising and externalising factors as the specific domains in any common factor model. Thus any model testing for a common factor should consider both a factor common to all disorders, and the potential for two domain-specific factors for the internalising and externalising domains, because of the consistent validation of these

domains. While the precise nature of any such model is open to debate, the bifactor

model (see Reise, Morizot, & Hays, 2007), may be the most appropriate model to

consider.

The Bifactor Model

Figure 3.1 shows a conceptual path diagram of the basic bifactor model, with, in this

case, three orthogonal latent factors on the right hand side of the diagram and one

latent factor on the left.
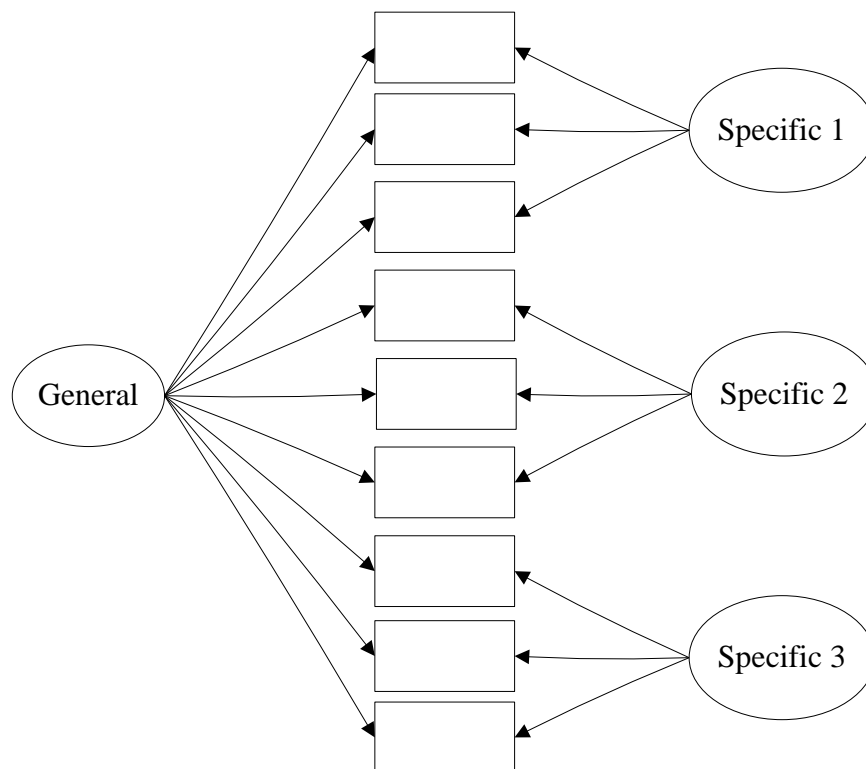
Figure 3.1. Conceptual path diagram of the bifactor model. The rectangular boxes

represent indicator variables.

The key of the bifactor model is the general or 'common' factor (the 'general' factor,

shown on left in Figure 3.1) on which all items load. The general factor explains the

covariance across all items. Figure 3.1 also shows the presence of specific factors (labelled "Specific" 1-3), upon which independent subsets of items load. An item only loads on one specific factor. The specific factors explain unique variance of the items within the specific factor subset, after accounting for the general factor.

Assumptions of the Bifactor Model and Applicability to Comorbidity Research

A bifactor models is considered appropriate for investigation when a proposed theoretical model has three component assumptions (Chen, West, & Sousa, 2006). The first assumption of the bifactor model is that there is a general factor hypothesised to account for the commonality of the items. Such a general factor could be seen to be analogous to common factor implied by common psychopathological liability model. While the proposed common factor from such a liability model is in fact hypothesised to be a multifactorial combination of genetic and environmental risks, this does not preclude use of the bifactor model. This is because the multifactorial combination is still hypothesised to produce a single global 'risk' outcome.

The second assumption of the bifactor model is that there are multiple domain specific factors, each of which is hypothesised to account for the unique influence of the specific domain over and above the general factor (Chen et al., 2006). If one considers psychological disorders, then it is clear that the 'Internalising' and 'Externalising' domains, which have empirical support in the literature, would meet the requirement of being specific factors in a bifactor model. As such this assumption of the bifactor model can be clearly met.

Given the arguments of Wolff and Ollendick (2006) outlined earlier, that consideration of specific as well as global factors is warranted within a model of comorbidity, the third assumption of the bifactor model is also met. This assumption is that there is specific interest in domain specific factors as well as the common factor (Chen et al., 2006). Such an assumption is inherent to the base bifactor model, because no covariance path is modelled between specific factors, and thus the relations among the general and domain specific factors are assumed to be orthogonal. This ensures that the specific factors only model the contribution that is completely separate to that of the general factor (Chen et al., 2006; Reise, 2012). Modelling such a covariance path contaminates the measurement of the domain-specific factors, and the estimation of any associations that are specific to these domain-specific factors (Reise et al., 2007). Such considerations would be vital if attempting to investigate and *differentiate* risk factors that affect comorbidity as a whole and risk factors specific to only one domain of problem behaviour.

Bifactor versus Second-Order Models

The last two assumptions of the bifactor model separate this model from the commonly used second-order models such as that shown in Figure 3.2. Within most fields, second-order models are more familiar as they have been more widely applied in a wider variety of substantive areas than have bifactor models (Reise, 2012). Second-order models are applicable when lower-order factors are substantially correlated with each other, but unlike the bifactor model, they are applicable when a higher-order factor is hypothesised to account for the relationship among the lower-order factors (Chen et al., 2006). Thus, the structure of second-order models do not allow study the role of domain specific factors that are independent of the general

factor; a unique advantage of the bifactor model over traditional second-order

models.
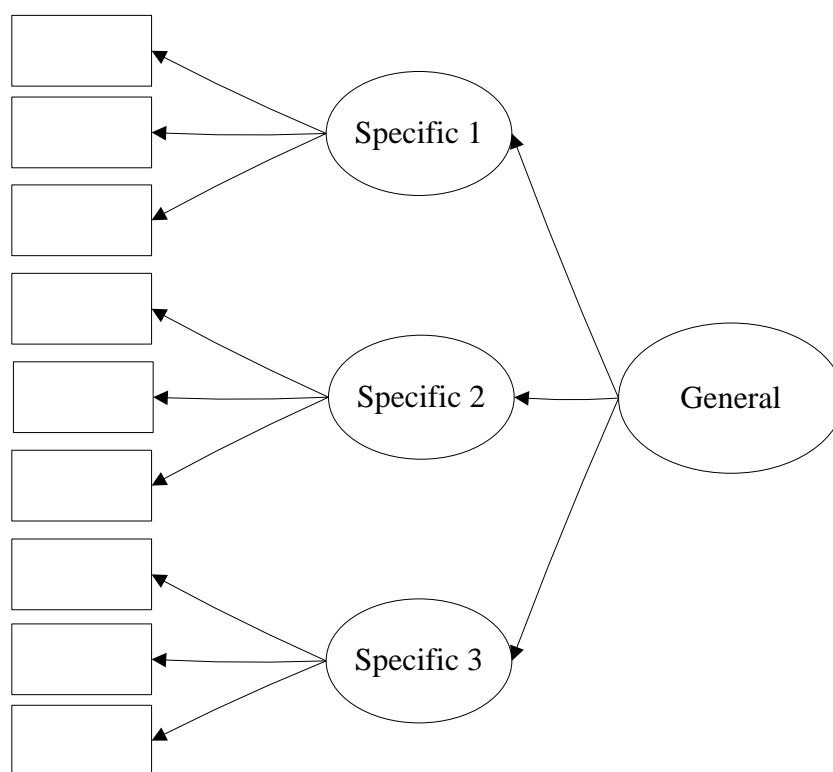
Figure 3.2. Conceptual path diagram of second-order models. The rectangular boxes

represent indicator variables.

Such a bifactor model as shown in Figure 3.1 must also be delineated from other

'bifactor' models such as the Schmid-Leiman bifactor model (Schmid & Leiman,

1957). This model has been a dominant approach within *exploratory* bifactor

modelling (Reise, 2012). This exploratory model allows each item to load on the

general factor and *all* specific factors (Mulaik & Quartetti, 1997; Wolff & Preising,

2005), unlike the confirmatory model which allows an item to load on only one

single specific factor (Reise, 2012). The Schmid-Leiman bifactor model also

constrains the loadings on the specific factor, as they are restricted to be proportional to the loadings on the general factor (Yung, Thissen, & McLeod, 1999); a restriction not specified in the confirmatory model.

Use of the Bifactor Model in Psychology and Health Research

Despite being originally described over 80 years ago (Holzinger & Harman, 1938; Holzinger & Swineford, 1937), the bifactor model has not been considered in the context of a multivariate model of comorbidity, and indeed has yet to be considered widely in psychopathology (Reise, 2012). Despite the lack of use within comorbidity and general psychopathology research, the bifactor model has a long history within certain fields of psychology and within the broader health research fields. In psychology, the bifactor model has been primarily used in the field of intelligence research (e.g. Gustafsson & Balke, 1993; Luo, Petrill, & Thompson, 1994), largely in relation to the Spearman (1927) conception of the '*g*', or general intelligence and domain specific intelligences. In health research, there has been increasing attention to the role of the bifactor model in health research in the past decade (see Reise et al., 2007). Recent research (e.g. Martel, von Eye, & Nigg, 2010; Toplak et al., 2009) has used the bifactor model to investigate the structural properties of ADHD symptoms with positive results, leading to breakthroughs in the conceptualisation and understanding of the disorder. Similarly, the bifactor model has been used to investigate the tripartite model of depression (Xie et al., 2012), and in health measurement modelling (e.g. Reininghaus, McCabe, Burns, Croudace, & Priebe, 2011). Indeed Reninghaus et al. (2011) argue that the bifactor model can complement and extend traditional dimensionality investigations, making it ideal for consideration in comorbidity research.

A Bifactor Model of Psychopathology

The consideration of a bifactor model in comorbidity research would be novel, but covers key structural aspects of psychopathology. The bifactor model would allow consideration of a common factor, but also allow independent consideration of the factors affecting the specific domains of internalising and externalising. Furthermore, if the bifactor model was seen to be an appropriate model for understanding psychological comorbidity/co-occurrence, it would provide clear support for a common factor and allow investigation of whether this common factor is in fact a psychopathological liability, as suggested by the Neale and Kendler (1995) models. This is because associations between the general/common factor and risk factors believed to be key in the development of comorbidity could be tested. If significant associations were found then it could be inferred that this common factor is indeed a liability factor. Furthermore, the use of a bifactor model would allow independent assessment of risk factors that were specific to certain classes of disorders, rather than comorbidity as a whole. This would overcome a serious shortcoming in risk factor research identified by Wolff and Ollendick (2006; see Chapter 2); that there is a lack of independent consideration of general and domain-specific risk factors.

The aim of this study is to investigate if support can be provided for a common factor model underlying multivariate psychopathology, using the bifactor model. However, as emphasised in Chapter 1, substantive differences do exist between statistically derived syndromes and DSM-IV-TR disorders, and thus the bifactor model would need to be considered and assessed against each diagnostic system separately. While there are many differing statistically derived dimensional systems, and many different methods of assessing DSM disorders, two of the most common will be used

in this study; the Achenbach System of Empirically Based Assessment as a statistically validated dimensional system and the Anxiety Disorders Interview Schedule as a method of assessing DSM-categorical psychopathology.

Dimensional Assessment of Psychopathology: The Achenbach System of Empirically Based Assessment

One of the most commonly used questionnaire systems providing a statistically validated dimensional system is the Achenbach System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001). The ASEBA was designed to measure problem behaviours in children and adolescents (Achenbach et al., 2008), and contains many versions, the most notable being the school-age assessment battery for ages six to 18. The ASEBA for school-age assessment includes the Child Behavior Checklist/6-18 (CBCL, completed by parents), the Teacher Report Form (TRF, completed by teachers) and the Youth Self-Report (YSR, completed by adolescents). These three checklists have comparable items, and they all have scales for the same eight statistically derived narrow-band syndromes: Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, and Aggressive Behavior. These syndromes were identified from both exploratory and confirmatory factor analyses (EFA & CFA) of ratings from both community and clinically referred child samples. Second-order factor analyses of these syndromes produce two higher-order factors, also referred to as broad-band syndromes, though some syndromes show a degree of cross-loading. The Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaints syndromes loaded highly on one factor constituting a higher order factor labelled 'Internalizing' and cover symptomatology similarly to the DSM-IV-TR

internalising disorders. The Rule-Breaking Behavior and Aggressive Behavior syndromes loaded highly on a second factor constituting a higher order factor labelled 'Externalizing', and cover symptomatology similarly to the DSM-IV-TR externalising disorders. The mean factor loadings for Thought Problems and Social Problems were about equal on both the Internalizing and Externalizing factors, and are termed 'mixed' syndromes.

However, despite the Attention Problems syndrome having a mean loading of .55 on Externalizing and .25 on Internalizing, it was designated to a mixed syndrome within the ASEBA scale. The reasoning for this was based on the difference between loadings for Attention Problems being substantially lower than the differences loadings for the two Externalising syndromes (see Achenbach & Rescorla, 2001, p.94). However as loading for Attention Problems is higher on Externalizing, relative to Internalizing, there is both a statistical and substantive argument that would suggest that it is better regarded as an Externalizing rather than a mixed syndrome. Firstly, from a statistical viewpoint, while the choice of threshold for a meaningful loading is often arbitrary, authors such as Gorsuch (1983) will suggest a loading of .32 is the minimum for a statistically meaningful loading. This is because a loading of .32 indicates that there is at 10 percent shared variance between the variable and the factor. Thus the loading of .25 for the Attention Problems factor can be argued to be below the cut-off for what can be considered a meaningful loading. Secondly, from a substantive viewpoint, the behaviours measured by the Attention Problems scale (poor attention, overactivity and impulsivity) are generally considered as externalizing behaviours (Döpfner et al., 2009; Kendall et al., 2001;

Yang et al., 2001). For these reasons, for the purposes of this study Attention

Problems was considered as an Externalizing syndrome.

ASEBA also have DSM-Oriented scales, which were developed from a top down

approach aimed at covering common childhood mental disorders found in the DSM-

IV-TR (Achenbach & Rescorla, 2001; Spatola et al., 2007). These scales, developed

first for the current version of the ASEBA scales, comprise only a subset of items

drawn from the ASEBA scales. Twenty-two experts from 16 cultures identified only

those items which were seen as being very consistent with DSM-IV diagnostic

categories, with only those items rated as consistent by at least 64 percent of raters

used for six separate scales (Achenbach et al., 2008; Spatola et al., 2007). These

DSM-Oriented Scales are designated as Affective Problems, Anxiety Problems,

Somatic Problems, Attention Deficit/Hyperactivity Problems, Oppositional Defiant

Problems, and Conduct Problems.

While these DSM-Oriented scales do not have the same empirical support as the

syndrome scales, they are nonetheless provided for use in a clinical perspective

(Achenbach et al., 2008; Achenbach & Rescorla, 2001). The content validity of

particular ASEBA items in relation to DSM-IV has also been supported by the fact

that the DSM-oriented scales are very consistent with DSM-IV diagnostic categories

and diagnoses (Achenbach & Rescorla, 2001; Krol, De Bruyn, Coolen, & van Aarle,

2006). Whilst no higher order scores are derived from these scales (Achenbach &

Rescorla, 2001), these six scales again cover both internalising and externalising

behaviours and indeed can be conceptualised in a structure similar to the empirically

derived Syndrome scales: Affective Problems, Anxiety Problems, and Somatic

Problems as internalising disorders and Attention Deficit/Hyperactivity Problems,

Oppositional Defiant Problems, and Conduct Problems as externalising disorders.

Indeed the DSM-Oriented scales do show some moderate, though not strong,

correlations with their equivalent empirically derived Syndrome Scales (Achenbach

& Rescorla, 2001) which would further validate such a structure. This would also

indicate that the DSM-Oriented scales are in fact partly dimensional in nature, and

perhaps reflect a blending of DSM categories, with dimensional measurement.

ASEBA Reference Group

Both the ASEBA Syndrome and DSM-Oriented Scales were validated using a

community based epidemiological sample, often referred to as the ASEBA Reference

Group (Achenbach & Rescorla, 2001). This sample contained around five thousand

individuals derived mostly from the United States of America, but also from England

and Australia. While individual data for this group is not available, correlation

matrices and sample measures of variability and central tendency are available. As

such, this information can be inputted into most SEM programs, such as MPlus

(Muthen & Muthen, 2010), and SEM models, such as the bifactor model proposed

here, can be assessed against this correlation matrix. This can allow an assessment of

the bifactor model for a community sample, rather than just using a clinical sample,

for reasons outlined in Chapter 1. The study presented in this thesis will undertake

such an analysis, to allow the assessment of the common factor in a bifactor model in

both clinical and community samples.

DSM Categorical Assessment of Psychopathology: Anxiety Disorders Interview Schedule

When diagnosing DSM disorders, especially in children, structured and semi-structured interview schedules are commonly used in order to improve diagnostic reliability. While there are a vast range of interview schedules, the Anxiety Disorders Interview Schedule (ADIS: Silverman & Albano, 1996) is one of the most commonly used in research and treatment outcome studies involving children (Lyneham, Abbott, & Rapee, 2007). One of the key advantages of the ADIS is that it is available in both Child and Parent Versions (ADIS-IV-C and ADIS-IV-P), allowing parallel and combined diagnosis (Silverman & Albano, 1996). The ADIS-IV-C/P are semi-structured interviews that were designed specifically for the assessment and diagnosis of anxiety disorders in children. The ADIS-IV-C/P allows categorization of close to the full spectrum range of anxiety disorders; social phobia, specific phobia, separation anxiety disorder, panic disorder, agoraphobia, post-traumatic stress disorder, generalised anxiety disorder, and obsessive-compulsive disorder. However, it can be used to diagnose the other major childhood disorders, including the depressive disorders (dysthymia, major depression), Attention Deficit/Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD). Thus the ADIS-IV-C/P covers the full range of common internalising and externalising disorders (Silverman & Albano, 1996; Silverman, Saavedra, & Pina, 2001).

To obtain a diagnosis, the total number of question responses where an indication of applicability has been made is calculated and then compared to the DSM-IV criteria requirements to determine whether the total number of symptoms endorsed is

sufficient to meet the number of symptoms required to meet DSM-IV criteria. If

sufficient criteria are met, then the child and/or parent is asked whether those

symptoms, taken together, lead to significant clinical interference or impairment.

Interference or impairment is regarded as occurring if the symptoms are interfering

with the child's schooling, family life, interaction with peers, or if they are causing

internal distress in the child. Impairment ratings are made by the children and parents

using a nine-point scale with an impairment rating greater than four warranting a

final diagnosis (Albano & Silverman, 1996). The ADIS-IV shows excellent construct

validity (e.g. Langer, Wood, Bergman, & Piacentini, 2010; Wood, Piacentini,

Bergman, McCracken, & Barrios, 2002), with diagnoses for all disorders being

reliably differentiated using the ADIS-C/P interview schedule (Silverman & Nelles,

1988; Silverman et al., 2001)

Aim of This Study

The primary aim of this study was to examine if there was support for a common

factor underlying child psychopathology, as implied by the common

psychopathological liability model outlined in Chapter 1. To test support for the

common factor, childhood behaviour problems, both at a DSM diagnosis level, and

at the level of empirically validated syndromes, were fitted to a bifactor model.

Specifically the intent was to assess the fit to a bifactor model of data from the

ASEBA CBCL Syndrome Scales, and from the ASEBA CBCL DSM-Oriented

Scales, as well as from diagnosis gained from a DSM-IV based diagnostic system;

specifically the parent version of the ADIS-IV. Scores/correlation matrices were

derived from a clinical sample of children and adolescents referred to Royal

Children's Hospital in Melbourne (Australia). Data from the ASEBA reference group

was also used to test the applicability of a common factor for a community based epidemiological sample. As this was an exploratory study, no specific hypotheses were made, though support for the bifactor model was predicted.

Method

Participants

Clinical Sample

The participants of what shall hereafter be referred to as the clinical sample, were 974 parents and their children referred to the Academic Child Psychiatry Unit (ACPU) of the Royal Children's Hospital, Melbourne, between 2001 and 2009. The ACPU is an out-patient psychiatric unit that provides services for children and adolescents with behavioural, emotional and learning problems. Children are referred to ACPU for psychological evaluation and management, with referrals generally sourced from other medical services, schools, and social and welfare organizations. The children were aged between 8 and 18 ($M = 11.07$ years, $SD = 3.08$ years), with 719 boys ($M = 10.76$ years, $SD = 3.03$) and 255 girls ($M = 10.76$ years, $SD = 3.03$). Approximately 46% of the sample was currently residing with both birth parents, with 32.7% residing in single-birth-parent households. A further 10.5% of participants were residing with at least one birth parent and a stepmother/father, with the remainder in other parenting situations including foster and adoptive care.

The mean age of child fathers was 42.96 years (range 26-69, $SD=7.44$ years), with the mean age of child mothers being 40.02 years (range 25-61, $SD=6.58$ years). The majority of fathers were employed (65.9%), with only 8% unemployed and 2.7 % in

home duties. Similarly, nearly half of all mothers (46.7%) had employment, with 35.5% in home duties, and 3.2% unemployed. In terms of parental education, 54.6% of fathers and 53.1% of mothers had completed High School certificate (or equivalent) education. The median income of family units was $30000 to $40000 per year, and approximately 63% of the sample was receiving some form of governmental assistance.

Approximately 21.6% of boys and 24.3% of girls in the sample were currently receiving medication as a treatment for a psychological disorder at the time of testing. Table 3.1 presents sample characteristics by age category and gender. As can be seen there were more children aged 6-11, and there were comparatively few above 15 years of age.

Table 3.1

Sex Breakdown of Age and Medication Status of the Clinical Sample.

Age	Males		Females		All Participants		Grand Total
	Not Medicated	Medicated	Not Medicated	Medicated	Not Medicated	Medicated	
6-11	330	76	92	15	422	91	513
12-18	207	79	94	47	301	126	427
All Participants	537	155	186	62	723	217	

Table 3.2 presents diagnostic characteristics for the sample, split by sex, based on information from the ADIS-IV-P (Silverman & Albano, 1996).

Table 3.2

Sex Breakdown of Diagnosis Category for the Sample, Based on the Results of the

ADIS-IV-P

Diagnosis	Males	Female	Total
One Diagnosis			
D	4	3	7
A	22	18	40
H	46	10	56
C	13	10	23
Two Diagnoses			
D/A	16	24	40
D/C	6	1	7
D/H	5	1	6
A/C	18	10	28
A/H	59	15	74
H/C	88	10	98
Three Diagnoses			
D/A/C	32	13	45
D/A/H	21	15	36
D/H/C	25	3	28
A/H/C	158	31	189
Four Diagnoses			
D/A/H/C	170	82	252
No Diagnosis	32	6	38

Note: A = Anxiety Disorder, D = Depressive Disorder, ADHD = Attention
Deficit/Hyperactivity Disorder, CDOD = Conduct/Oppositional-Defiant Disorder.

It should be noted that for ease of understanding, the wide range of ADIS-P

diagnoses have been collapsed into four main categories: Anxiety Disorders (e.g.:

social/specific phobia, panic disorder, agoraphobia, generalised anxiety disorder,

separation anxiety disorder), Depressive Disorders (e.g.: dysthymia, major

depressive disorder), Attention Deficit/Hyperactivity Disorder (any subtype) and

Conduct/Oppositional-Defiant Disorder. As can be seen from Table 3.2, this clinical

sample shows high degrees of comorbidity. Tables 3.3 and 3.4 presents mean *T*-

scores for the CBCL Syndrome and DSM-Oriented Scales respectively, as well as

the numbers within each of the CBCL categories of clinical severity.

Table 3.3

Clinical Sample Mean T-scores on the ASEBA Child-Behavior Checklist/6-18

Syndrome Scales with Numbers Within Each of the CBCL Categories of Clinical

Severity.

	Descriptives		N per Category		
	Mean	SD	Normal Range 50-64	Borderline Range 65-69	Clinical Range ≥70
Anxious/Depressed	66.95*	11.33	407	183	378
Withdrawn/Depressed	66.17*	10.67	435	194	339
Somatic Complaints	62.86	9.79	580	124	264
Social Problems	67.55*	9.92	360	215	393
Attention Problems	69.94*	11.58	328	226	414
Rule Breaking Behaviour	67.17*	9.45	371	175	422
Aggressive Behaviour	72.70**	12.64	260	146	562

Note: * within the Borderline range; ** within the Clinical range

All participants had CBCL Syndrome Scales available, but for various logistical reasons DSM-Oriented scales were only available for 752 participants. As shown in the first two columns of Table 3.3, the mean *T* scores for participants involved were in the Borderline clinical range for all syndromes, except Somatic Complaints which was in the normal range and Aggressive Behaviour in the Clinical range. However, as can be seen in the last three columns, there were substantial numbers of individuals who were in the normal range, indicating the sample contained a wide cross section of clinical severity.

Table 3.4

Mean T-scores of Participants on the ASEBA Child Behavior Checklist/6-18 DSM-Oriented Scales with Numbers Within Each of the CBCL Categories of Clinical Severity.

	Descriptives		N per Category		
	Mean	SD	Normal Range 50-64	Borderline Range 65-69	Clinical Range ≥70
Affective Problems	68.55*	9.84	245	131	386
Anxiety Problems	65.35*	9.11	312	109	341
Somatic Problems	61.49	10.16	487	115	160
Attention Deficit/ Hyperactivity Problems	66.48*	8.81	284	178	300
Oppositional Defiant Problems	67.83*	9.19	247	126	389
Conduct Problems	69.97*	11.43	224	129	409

Note: * within the Borderline range; ** within the Clinical range

Similarly, in Table 3.4, the mean *T* scores for participants involved were in the

Borderline clinical range for all categories, except Somatic Problems which was in

the normal range and Aggressive Behaviour in the Clinical range. However, again

there was a wide cross section of clinical severity.

ASEBA Reference Group

Full details about the Reference Group are available from the ASEBA Manual with

(Achenbach & Rescorla, 2001). In brief, the sample consisted of 4994 children and

adolescents aged from 6-18, derived from 40 US states and the District of Columbia,

1 state of Australia, and England. The sample consisted of 3098 boys and 1896 girls.

Approximately 65 percent of respondents were mothers, with only 10 percent being

fathers. Ethnicity was representative of a U.S. population.

Materials

Child Behavior Checklist (CBCL/6-18: Achenbach & Rescorla, 2001)

The primary measure was the Child Behavior Checklist (designated as the CBCL/6–

18) from the Achenbach System of Empirically Based Assessment (ASEBA:

Achenbach & Rescorla, 2001). The CBCL/6-18 is a questionnaire for parental

completion, and has 113 items used to rate children between 6 and 18 years of age.

Parents indicate the degree or frequency of each behaviour described in the item

within the previous 6 months, on a scale of 0 (not true), 1 (somewhat or sometimes

true) or 2 (very true or often true).

The CBCL/6-18, has scales for eight empirically and statistically validated

syndromes: Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social

Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, and

Aggressive Behavior. Second order factor analyses of the ASEBA syndromes show

two high order factors, with the Anxious/Depressed, Withdrawn/Depressed and

Somatic Complaints syndromes forming the Internalizing Problems factor, and the

Rule-Breaking Behavior and Aggressive Behavior forming the Externalizing

Problems factor. The sum of the eight syndrome scores, plus a further 17 items

which do not form part of any of the eight syndrome scales, provide a Total Score

measure of global problems.

For the purposes of this study, the Thought Problems syndrome from the CBCL

Syndrome Scales was not used on theoretical grounds, because childhood

psychopathology is generally operationalised in terms of emotional, behavioural and

social problems (Mash & Barkley, 2003), and thought problems traverse the range of

psychopathological disorders. Similarly, for reasons outlined earlier in the chapter,

the Attention Problems Syndrome from the CBCL Syndrome Scales was considered

an Externalising problem, despite being designated a mixed syndrome within the

ASEBA scales (Achenbach & Rescorla, 2001).

The ASEBA scales also have DSM-Oriented scales, which were developed from a

top down approach. The DSM-oriented scales comprise items identified by experts

from 16 cultures as being very consistent with DSM-IV diagnostic categories

(Achenbach & Rescorla, 2001). These DSM-Oriented Scales are; Affective

Problems, Anxiety Problems, Somatic Problems, Attention Deficit/Hyperactivity

Problems, Oppositional Defiant Problems, and Conduct Problems. No higher order

scores are derived from these scales.

CBCL Syndrome Scale and DSM-Oriented Scale scores are derived by summing the responses of the items in the respective scales, and then converting these raw scores to T scores. Externalizing, Internalizing and Total scores for the CBCL are computed in a similar manner. For the Syndrome and DSM-Oriented Scales, T scores above 70 are considered to be in the clinical range, while T scores between 65 and 70 are considered to be in the borderline clinical range. For the Externalizing, Internalizing and Total scores, T scores above 63 are considered clinical, while T scores between 60 and 63 are considered borderline clinical.

The CBCL/6-18 has sound psychometric properties, which are summarised in Achenbach and Rescorla (2001, 2007), and Achenbach et al. (2008). The averaged alpha values for the ASEBA scales across the original US sample were .96 for Total Problem Scale, .92 for Internalizing Problem and Externalizing Problem Scales, and .82 across the syndrome scales. The DSM-Oriented scales also show sound psychometric properties, with averaged alpha values across the original US sample being .81. The ASEBA shows reasonable equivalence across gender and age, with any differences between age/gender groups being of negligible effect sizes of ($\leq$ .01), though girls generally score higher on Internalising problems and lower on Externalising problems than boys (Rescorla et al., 2007). The ASEBA scales have also been demonstrated to have cross-cultural validity. While some significant differences between cultures have been found, the relative sizes of these differences are not particularly large, with small effect sizes ranging from .03 to .14 (Rescorla et al., 2007). The averaged alphas for the ASEBA scales from 33 societies were .94 for Total Problems, .87 for Internalizing and Externalizing, and .76 for syndromes. The DSM-Oriented scales also show sound cross-cultural psychometric properties, with

averaged alpha values of .74 cross-culturally from 33 societies 14 (Achenbach & Rescorla, 2007; Rescorla et al., 2007).

Although Achenbach and Rescorla (2001) suggest that it is preferable to use raw scores for statistical analyses, this data was not available from all files. For this reason, the *T*-scores were used in the current study. However, using data from those participants where scale score information was available, parallel testing using these scale scores was conducted, and no interpretable differences were noted.

Anxiety Disorders Interview Schedule for Children-Parent Version (ADIS-IV-P: Silverman & Albano, 1996)

Clinical diagnosis was made using the parent version of the ADIS-IV-P. The diagnoses reported earlier as well as that used in the analyses in the study were derived from this schedule. The ADIS-IV-P is a semi-structured interview, based on the DSM-IV diagnostic system (American Psychiatric Association, 1994). The ADIS-IV-P was designed primarily to facilitate the diagnosis of the major childhood anxiety disorders; social phobia, specific phobia, separation anxiety disorder, panic disorder, agoraphobia, post-traumatic stress disorder, generalised anxiety disorder, and obsessive-compulsive disorder. However, the parent version can also be used for diagnosing the other major childhood disorders, including the depressive disorders (dysthymia, major depression), ADHD, ODD and CD. The ADIS-IV-P guideline for diagnosis is that the child be given diagnosis of all disorders where diagnostic criteria are met, and as a result, does not take into account the hierarchical exclusionary rules in DSM-IV. The scores of ADIS-IV-P have sound psychometric properties (Silverman et al., 2001). Test-retest reliability for the ADIS-IV-P scores over a 7-to-

14-day interval has shown good to excellent reliability. Kappa values for ADIS-IV-P interviews with parents are generally good, ranging from 0.65 to 1.00 (Silverman et al., 2001).

The dysthymia and major depression diagnoses were collapsed into a single entity for the purposes of this study. There were two main reasons for this. The first reason is statistical in nature, and due to the extremely high correlation between the Dysthymia and Major Depression diagnoses, which raises the problem of multicolinearity. This high correlation is in part due the nature of ADIS-IV guideline for diagnosis which is that the child be given diagnosis of all disorders meeting the diagnostic criteria. The children who met the diagnosis for depression often met the criteria for Dysthymia. The second reason for treating these diagnoses as a single entity is substantive. Considerable research has shown that there is often poor discriminant validity for Dysthymia and Major Depression in children (Goodman, Schwab-Stone, Lahey, Shaffer, & Jensen, 2000). This poor differentiation goes beyond diagnostics, with children and adolescents often showing equivalence in impairment and competence (Garrison, Addy, Jackson, McKeown, & Waller, 1992; Goodman et al., 2000; Lewinsohn, Rohde, Seeley, & Hops, 1991), and thus combining these diagnoses into a single entity is justifiable.

Procedure

Children and parents participated in separate interview and testing sessions at the Royal Children Hospital, Melbourne. In all cases, parental consent forms were completed prior to the assessment. Data collected covered a comprehensive medical, neurological, educational, psychological and familial and social assessment of the

child and their family. Psychological data were collected by research assistants

(RAs), who were post-graduate students in clinical psychology courses, approved by

the Australian Psychological Society (APS). Standard procedures, as described in the

manual, were used for administration and scoring the CBCL, and ADIS-IV-P Parent

Version. Where necessary, researchers read the items to participants who then

completed their responses. Approximately 95% of the parent ADIS-IV interviews

involved mothers only, and the rest involved fathers only or both fathers and mothers

together. Clinical diagnosis was determined by two consultant child and adolescent

psychiatrists who independently reviewed these data. The inter-rater reliability for

diagnoses of the two psychiatrists was high (kappa = .90).

Data Analysis

In this study, the data was analysed using MPlus Version 6.1 (Muthen & Muthen,

2010).

CBCL Syndrome and DSM-Oriented Scales – Clinical Sample

The CBCL Syndrome Scale and DSM-Oriented *T*-scores for the clinical sample were

fitted to the bifactor model using the robust maximum likelihood (MLM) method of

estimation. The models tested are shown graphically in Figures 3.3 and 3.4. The

CBCL Syndrome Scale of social problems only loaded onto the common factor, and

did not load onto either of the specific factors. This was because this syndrome is

designated a 'mixed' syndrome, showing equal loadings on both the Internalizing

and Externalizing factors in the initial validation of the ASEBA scales (Achenbach &

Rescorla, 2001).
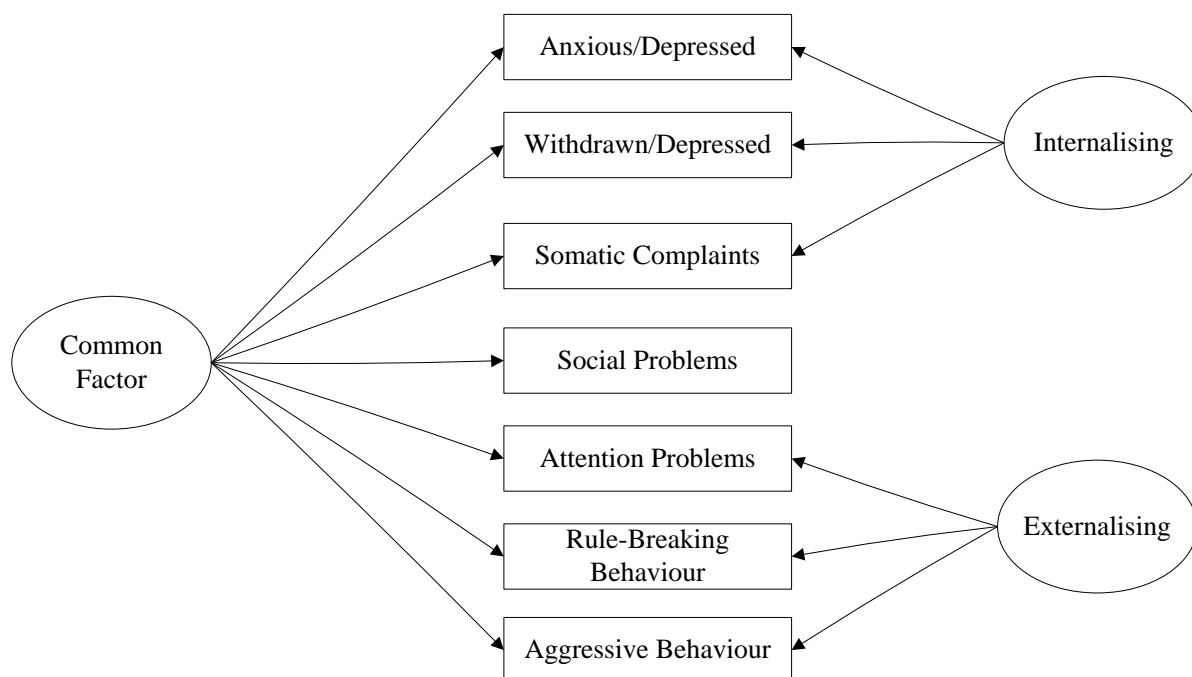
Figure 3.3. The tested bifactor model for the CBCL Syndrome Scales
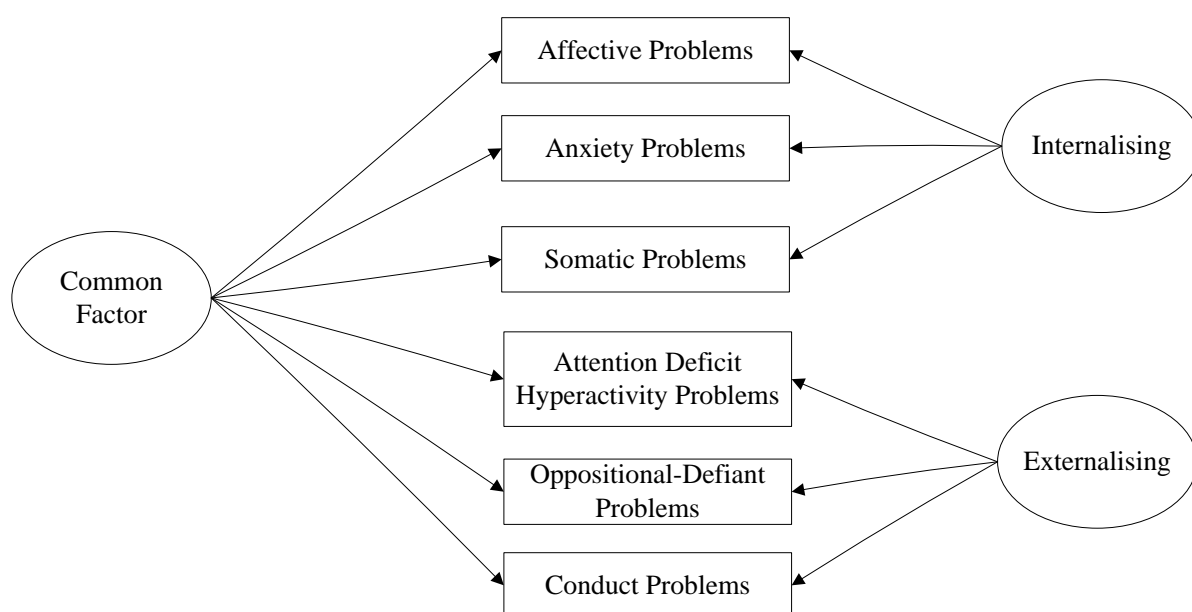
Figure 3.4. The tested bifactor model for the CBCL DSM-Oriented Scales

No covariance path was specified between the internalising and externalising factors

for both models, despite research evidence that these two factors may be correlated

(e.g. Krueger, 1999). This path is not modelled, as discussed earlier in the chapter,

because the bifactor model in its true form assumes that relations among the general

and domain specific factors are orthogonal. Were a covariance to be modelled, the

measurement of the domain specific factors becomes contaminated (Reise et al.,

2007), and not modelling this path ensures that domain specific factors measure only

contribution over and above that of the general factor (Chen et al., 2006).

CBCL Syndrome and DSM-Oriented Scales – ASEBA Reference Group

The correlation matrix for both the CBCL Syndrome and DSM-Oriented scales from

the ASEBA Reference Group was directly inputted into MPlus. These correlation

matrices, reproduced from Appendix 2 in the ASEBA manual (Achenbach &

Rescorla, 2001), are shown in truncated form in Tables 3.5 (Syndrome Scales) and

3.6 (DSM-Oriented Scales). The models specified were exactly the same as those

used for the clinical sample.

Table 3.5

Pearson Product-Moment Correlations for The Child Behavior Checklist (CBCL)

Syndrome Scales For the ASEBA Reference Group

	Anxious/ Depressed	Withdrawn/ Depressed	Somatic Complaints	Social Problems	Attention Problems	Rule- Breaking Behaviour
Withdrawn/ Depressed	.52					
Somatic Complaints	.38	.36				
Social Problems	.54	.47	.37			
Attention Problems	.39	.40	.31	.55		
Rule-Breaking Behaviour	.30	.33	.27	.45	.54	
Aggressive Behaviour	.47	.38	.35	.63	.61	.67

Note: All correlations were $p<.01$. Table is truncated and reproduced from Appendix E, Achenbach and Rescorla (2001, p. 230)

Table 3.6

Pearson Product-Moment Correlations for The Child Behavior Checklist (CBCL)

DSM-Oriented Scales For the ASEBA Reference Group

	Affective Problems	Anxiety Problems	Somatic Problems	Attention Deficit/ Hyperactivity Problems	Oppositional Defiant Problems
Anxiety Problems	.51				
Somatic Problems	.32	.25			
Attention Deficit/ Hyperactivity Problems	.43	.37	.21		
Oppositional Defiant Problems	.43	.33	.33	.59	
Conduct Problems	.43	.38	.19	.57	.67

Note: All correlations were $p<.01$ Table is truncated and reproduced from Appendix E, Achenbach and Rescorla (2001, p. 230)

ADIS-IV-P

The ADIS-IV-P data was dichotomous in nature (diagnosis present or diagnosis absent) and thus the bifactor model was fitted to the data using the robust weighted least squares (WLSMV) method of estimation which is more appropriate than MLM for categorical data (Brown, 2006). The ADIS-IV-P bifactor model is shown graphically in Figure 3.5. Again, as for the CBCL models, there is no covariance path between the internalising and externalising factors.
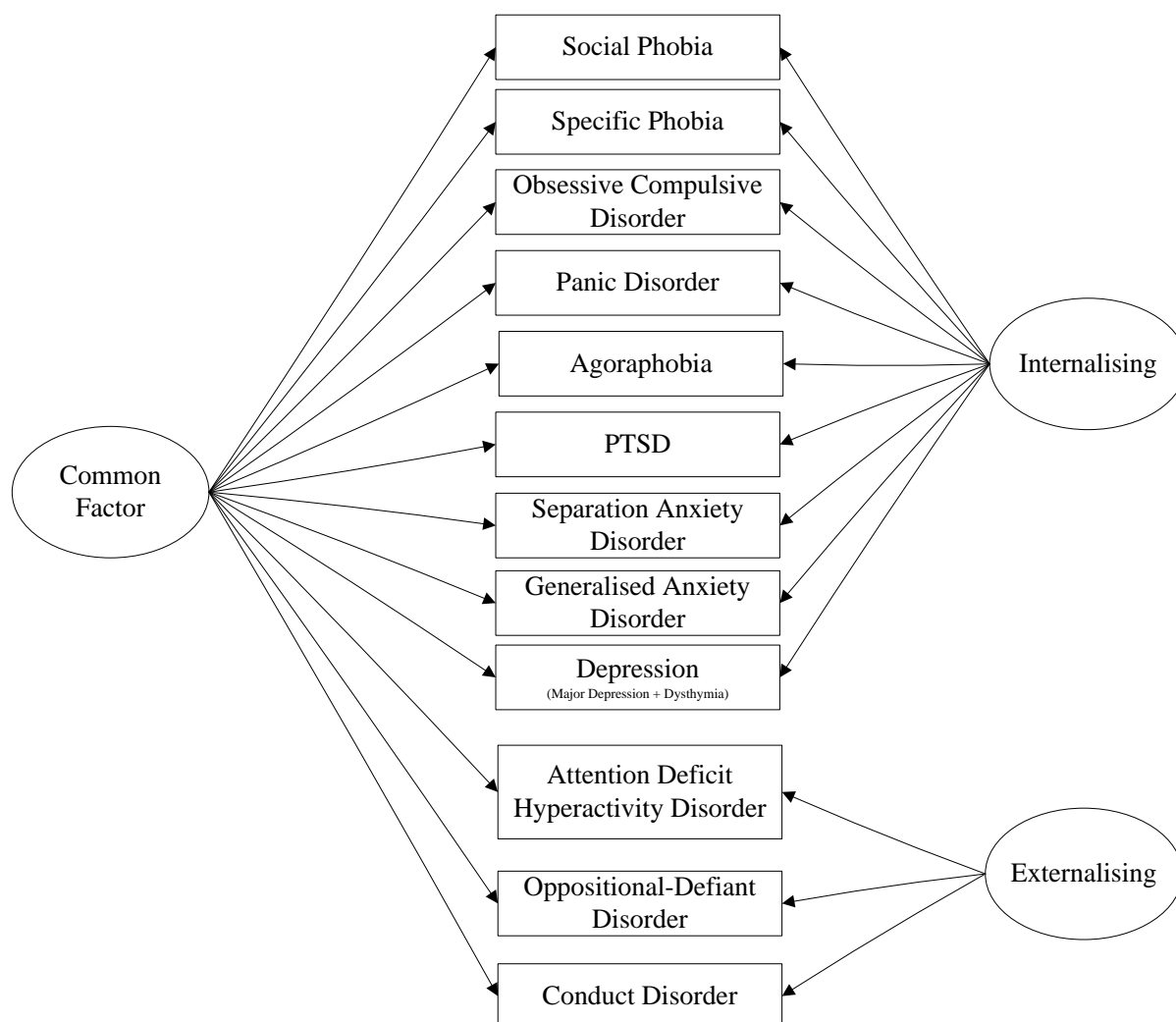
Figure 3.5. The tested bifactor model for the ADIS-IV-P.

Assessing Model Fit

The appropriateness of the models was determined by the fit indices derived from the

MPlus program. While MPlus calculates the χ^2 likelihood ratio test statistic, this

statistic is affected substantially by sample size, and as such almost any model will

be rejected when the sample size is large (Brown, 2006). In view of this,

approximate fit indices were used to evaluate the models. MPlus provides

approximate (or practical) fit indexes for the Tucker-Lewis Index (TLI), the

comparative fit index (CFI), the root mean squared error of approximation

(RMSEA), and the standard root mean square residual (SRMR).

TLI (Tucker & Lewis, 1973) is an index that measures the fit of the model against

the independence model; a model specifying no covariance between variables (i.e. all

variables are assumed to be uncorrelated). TLI is recommended because it has

features that penalise models for adding parameters that do not markedly improve

model fit (Brown, 2006). CFI (Bentler, 1990) similarly measures the fit of the model

against the independence model, but unlike TLI is based on the non-centrality

parameter (Brown, 2006). Unlike traditional null hypothesis significance testing, the

χ^2 goodness-of-fit test within SEM modelling is based around the desire of retaining

(or at least not rejecting) the null hypothesis (H_o) that there is a no difference

between predicted model and the observed data. Given this, it is statistically argued

that one should be testing to reject the alternative hypothesis (H_a), rather than testing

to reject the H_o. Such a test uses the non-central chi-square distribution created under

the case when H_a is assumed to be true in the population (i.e., the specified model is

incorrect in the population), with the non-centrality parameter derived from this

distribution. The non-centrality parameter estimate is calculated by subtracting the

model degrees of freedom from the chi-square value. It is argued that TLI (Hu &

Bentler, 1998; 1999) and CFI (Gerbing & Anderson, 1992) are both relatively

insensitive to sample size but sensitive to model misspecification, and as such are

reliable fit indices. For the CFI and TLI, values of .95 or above are taken as

indicating good model-data fit, with values above .90 indicative of acceptable fit

(Brown, 2006; Hooper, Coughlan & Mullen, 2008; Hu & Bentler, 1999).

RMSEA (Steiger & Lind, 1980) is a population based index, which, like CFI, relies

on the non-centrality parameter. The RMSEA indicates how well the model, with

unknown but optimally chosen parameter estimates, would fit the population

covariance matrix (Byrne, 1998). Thus RMSEA is regarded as assessing the extent to

which there is misfit in the proposed model (Brown, 2006; Chen, Curran, Bollen,

Kirby & Paxton, 2008; Steiger, 1998). The guidelines suggested by Hu and Bentler

(1998) are that RMSEA values close to 0.06 or below be taken as good fit. However,

Browne and Cudeck (1993) have suggested that RMSEA values from .06 to .08 can

be inferred as moderate fit, and 0.08 to .10 as marginal fit. SRMR (Jöreskog &

Sörbom, 1981) is an absolute fit index, in that it does not take into account model fit

relative to the independence model. Rather SRMR is conceptualised as an average

discrepancy between the correlations in the sample correlation matrix and the

correlations reproduced by the model (Brown, 2006). Hu and Bentler (1998) suggest

SRMR values of .08 or less taken as indication of good fit.

Results

CBCL Syndrome Scales – Clinical Sample

The goodness-of-fit values for the bifactor model of CBCL Syndrome Scales for the

clinical sample was χ^2 (8, N= 974) = 43.94, $p < .0001$; CFI = .987; TLI = .965;

RMSEA = .068; SRMR = .021. These findings indicate good support for the bifactor

model, with three of the four fit indices showing good fit and RMSEA showing

acceptable fit. Table 3.7 shows the standardised loading (STDYX) for each

syndrome on both the general and specific factors.

Table 3.7

Factor Loadings of the Bifactor Model for the CBCL Syndrome Scales for the

Clinical Sample

Syndrome	General Common Factor	Specific	
		Internalising	Externalising
Anxious/Depressed	.542	.706	
Withdrawn/Depressed	.467	.419	
Somatic Complaints	.392	.444	
Social Problems	.931		
Attention Problems	.602		.171
Rule-Breaking Behaviour	.428		.828
Aggressive Behaviour	.597		.619

Note. All factor loadings were significant ($p < .05$).

All syndromes loaded significantly on the general factor with all standardised

loadings above .392, indicating that all syndromes show substantive loadings on the

general factor, as per the guidelines of Gorsuch (1983), who suggests that a loading

of .32 (i.e. 10 percent shared variance between the variable and the factor) is the

minimum for a loading to be considered as meaningful. Two of the six syndromes

(Withdrawn/Depressed and Attention Problems) showed higher loadings on the

general factor than their respective specific factors, with a further two (Aggressive

Behavior and Somatic Complaints) having comparable loadings on each factor. Rule-

Breaking Behavior and Anxiety Problems showed higher specific factor loadings

than the general factor.

CBCL Syndrome Scales - ASEBA Reference Group

The goodness-of-fit values for the bifactor model for CBCL Reference Group

Syndrome Scales was χ^2 (8) = 83.63, $p < .0001$; CFI = .985; TLI = .960; RMSEA

= .073; SRMR = .021. These findings indicate good support for the bifactor model,

with three of the four fit indices showing good fit and RMSEA showing acceptable

fit. Table 3.8 shows the standardised loading (STDYX) for each syndrome on both

the general and specific factors.

Table 3.8

Factor Loadings of the Bifactor Model for the CBCL Syndrome Scales for the

ASEBA Reference Group

Syndrome	General	Specific	
	Common Factor	Internalising	Externalising
Anxious/Depressed	.546	.707	
Withdrawn/Depressed	.466	.418	
Somatic Complaints	.394	.446	
Social Problems	.932		
Attention Problems	.601		.174
Rule-Breaking Behaviour	.428		.811
Aggressive Behaviour	.599		.631

Note. All factor loadings were significant ($p < .05$).

All syndromes loaded significantly on the general factor with all standardised loadings above .428, indicating that all syndromes show substantive loadings on the general factor. Indeed the results and loading for the reference group are almost identical to the clinical sample and provide support for the clinical sample results.

CBCL DSM-Oriented Scales – Clinical Sample

The goodness-of-fit values for the bifactor model of CBCL DSM-Oriented Scales was χ^2 (4, N= 762) = 38.17, $p < .0001$; CFI = .980; TLI = .926; RMSEA = .106; SRMR = .023. These findings indicate moderate support for the bifactor model, with 2 of the four fit indices showing good fit, and TLI and RMSEA showing borderline poor fit. Table 3.9 shows the standardised loading (STDYX) for each syndrome on both the general and specific factors.

Table 3.9

Factor Loadings of the Bifactor Model for the CBCL DSM-Oriented Scales

Syndrome	General Common Factor	Specific Internalising	Externalising
Affective Problems	.676	.545	
Anxiety Problems	.513	.453	
Somatic Problems	.287	.528	
Attention Deficit/ Hyperactivity Problems	.423		.505
Oppositional Defiant Problems	.423		.760
Conduct Problems	.388		.776

Note. All factor loadings were significant ($p < .05$).

CBCL DSM-Oriented Scales – ASEBA Reference Group

All syndromes loaded significantly on the general factor with all standardised

loadings above .28, indicating that all syndromes show relatively substantive

loadings on the general factor. Two of the six scales (Anxiety and Affective

Problems) showed higher loadings on the general factor than their respective specific

factors, but all the others showed substantially higher loadings on the specific over

general factors.

The goodness-of-fit values for the bifactor model for CBCL DSM-Oriented Scales

was χ^2 (4) = 106.69, p < .0001; CFI = .973; TLI = .898; RMSEA = .121; SRMR

= .025. These findings indicate only marginal fit support for the bifactor model, with

2 of the four fit indices showing good fit, TLI acceptable fit, and RMSEA showing

borderline poor fit. Table 3.10 shows the standardised loading (STDYX) for each

syndrome on both the general and specific factors. All syndromes loaded

significantly on the general factor with all standardised loadings above .28,

indicating that all syndromes show substantive loadings on the general factor. Once

again, results and loadings for the reference group are almost identical to the clinical

sample and provide support for the clinical sample results.

Table 3.10

Factor Loadings of the Bifactor Model for the CBCL DSM-Oriented Scales for the

ASEBA Reference Group

	General	Specific	
Syndrome	Common Factor	Internalising	Externalising
Affective Problems	.679	.542	
Anxiety Problems	.518	.440	
Somatic Problems	.289	.523	
Attention Deficit/ Hyperactivity Problems	.411		.516
Oppositional Defiant Problems	.410		.758
Conduct Problems	.377		.785

Note. All factor loadings were significant ($p < .05$).

ADIS-IV-P

The goodness-of-fit values for the bifactor model for ADIS-IV-P was *WLSMVχ^2* (42,

N= 974) = 117.07, $p < .0001$; CFI = .957; TLI = .933; RMSEA = .043; SRMR

= .073. These findings indicate good support for the bifactor model, with 3 of the

four fit indices showing good fit and TLI showing acceptable fit. Table 3.11 shows

the standardised loading (STDYX) for each syndrome on both the general and

specific factors.

Table 3.11

Factor Loadings of the Bifactor Model for the ADIS-IV-P

Diagnosis	General Common Factor	Specific	
		Internalising	Externalising
Social Phobia	.463	.392	
Specific Phobia	.322	.365	
Panic Disorder	.354	.432	
Agoraphobia	.226	.770	
Post-Traumatic Stress Disorder	.697	.262	
Generalised Anxiety Disorder	.502	.414	
Separation Anxiety Disorder	.450	.531	
Obsessive-Compulsive Disorder	.314	.492	
Depression[#]	.886	.111	
Conduct Disorder	.278		.861
Oppositional Defiant Disorder	.387		.835
Attention Deficit/Hyperactivity Disorder	.134		.536

Note. All factor loadings were significant ($p < .05$).
Depression = Dysthymia + Major Depression

All disorders loaded significantly on the general factor, with most standardised

loadings above .3, indicating that most syndromes show substantive loadings on the

general factor, though the low loading of ADHD on the general factor is not

particularly supportive. However, four of the categories showed higher loadings on

the general factor than their respective specific factors, with a further three

(Aggressive Behavior and Somatic Complaints) having comparable loadings on each

factor. Rule-Breaking Behavior and Anxiety Problems showed higher specific factor loadings than the general factor.

Discussion

The primary aim of this study was to examine if there was support for a common factor underlying child psychopathology, as implied by the common psychopathological liability model outlined in Chapter 1. This was tested both at a DSM diagnosis level and at the level of empirically validated syndromes, using the bifactor model. In general, the findings in this study showed acceptable to good support for a common factor as modelled in the bifactor model, for all three measures used in the study.

CBCL Syndrome Scales

The results from this study demonstrate clear support for the bifactor model, and hence a common factor, for the CBCL Syndrome Scales. For the clinical sample, the model showed good fit, and for all syndromes in the CBCL there were significant, salient and substantive loadings on the general factor. Similar results were obtained for the ASEBA reference group. Indeed the results for both the clinical and ASEBA sample are highly comparable in terms of fit and factor loadings, indicating that the bifactor model is an appropriate model for this measure.

ADIS-IV-P

The results for the ADIS-IV-P show adequate support for the bifactor model, with acceptable fit, though three indicators from the ADIS-IV-P, the ADHD, Conduct

Disorder and Agoraphobia diagnoses, failed to showing a salient loading as defined by Gorsuch (1983). However, the loading is very close to that cut-off value for Conduct Disorder and Agoraphobia, and all of the loadings were still statistically significant. As such the results provide some support for a conceptualisation of a common factor for the ADIS-IV-P within the bifactor model.

CBCL DSM-Oriented Scales

There are some issues with the fit for the CBCL DSM-Oriented scales. While there was a good fit and salient and substantive loadings for the DSM-Oriented scales within the clinical sample, for the reference group, two of the four fit indices showed borderline poor fit. Overall, this indicates that the bifactor model as proposed may not be an entirely appropriate fit for the DSM-Oriented scales. However, the most likely reason for this result, in comparison to the excellent fit for the CBCL Syndrome Scales, is related to the initial construction of these scales. The DSM-Oriented scales were developed from a top down approach, comprising items identified by experts from 16 cultures as being very consistent with DSM-IV diagnostic categories (Achenbach et al., 2008; Achenbach & Rescorla, 2001). These scales have little empirical validation, and have not been subject to any meaningful exploratory or confirmatory factor analysis. As such, fitting any model to these scores is likely to lead to poor fit, and thus it is somewhat unsurprising that there was only marginal fit for the reference group sample.

Despite the issues surrounding fit for the ASEBA Reference Group, there is support of the concept of a common factor underlying psychopathology from the DSM-oriented scales. All of the DSM-oriented scales for both the clinical sample and the

ASEBA Reference Group show significant loadings to the general factor, with only the Somatic Concerns showing borderline salient loading to the general factor. Even if for some scales the loadings are lower for the general factor than for the specific factors, it is clear that there is some limited overall support for a conceptualisation of a common factor for the DSM-Oriented scales within the bifactor model.

Overall Findings

Taken together, the findings of the results from the clinical sample and the ASEBA reference group generally support the conceptualisation of a common factor underlying childhood psychopathology within a bifactor model, especially within the dimensional CBCL Syndrome Scales. Furthermore, given there is some support for the bifactor model of psychopathology within a DSM-based diagnostic categories, there appears to be a degree of universality for such a bifactor model, and a common factor underlying psychopathology.

Need for Further Validation of a Common Factor

Support for a common factor is not definitive however. This is because despite substantive loadings to the general factor by almost all disorders/syndromes, many had equivalent or higher loadings on the specific internalising or externalising factors. There are many potential reasons for this. It may be that some disorders/syndromes are impacted more by class specific factors than general factors. Given that it is argued that the common factor is potentially a multifactorial combination of genetic and environmental risks - a psychopathological liability - it may be that some risk factors which form part of this multifactorial combination, could be risk factors for the common factor, but potentially protective factors for the

domain-specific factors. This would make sense in light of the some definitions of a common liability which implies that person A manifests disorder X while person B manifests disorder Y because differences in the multifactorial combination of genetic and environmental risk factors (Neale & Kendler, 1995; Rhee et al., 2008). However, it is clear that further testing is needed to confirm support for the idea of a common factor. This will be the aim of the next study in this thesis, which will use an alternative SEM model to demonstrate support for the common factor.

Limitations of this Study

One potential criticism of the proposed model is that it does not split the internalising factor into sub-factors for the anxious-misery (also called distress) disorders and fear disorders. Certainly various research has supported such a split (e.g. Kendler et al., 2003). Kendler et al. noted that research has indicated that such a split can be argued on the basis of quantitative genetic studies. However, it should be noted that such a split has not always been supported by literature (e.g. Kessler et al., 2011; Krueger et al., 1998; Krueger & Finger, 2001), especially when using clinical samples, and thus it is felt that there is sufficient justification to model internalising disorders as a unitary dimension.

Conclusion

In conclusion, the findings of the results from the clinical sample for both dimensional and categorical classification systems, and the results using ASEBA reference group for dimensional classification, generally support the conceptualisation of a common factor underlying childhood psychopathology, within

a bifactor model structure. However, it is clear that further testing of this concept is
needed to provide support for the idea of a common liability factor.

Chapter 4 - Study 2: Testing for a Common Factor Underlying Childhood Psychopathology, Using the Circumplex

In the previous chapter, the idea was proposed that a common factor may underlie child psychopathology, as implied by the common psychopathological liability model outlined in Chapter 1. Such a common factor would, if empirically validated, provide a clearer explanation of the comorbidity between psychological disorders/syndromes. Study 1 tested for the common factor using the bifactor model. This model was tested in both a clinical sample, and using an epidemiological sample in the form of the ASEBA Reference Group. The results showed mostly good fit to the bifactor model, and support for a common factor. The common factor within the bifactor model was saturated with substantive loadings for nearly all conditions across both DSM-based categorical diagnoses and for empirically validated syndromes in the form of the Child Behavior Checklist (CBCL) from Achenbach System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001).

An Alternative Test for the Common Factor

Support for the common/general factor could not be considered definitive however, despite substantive loadings to the general factor by almost all disorders/syndromes. This was because many disorders/syndromes had equivalent or higher loadings on the specific internalising and externalising factors. While some potential explanations for this were proposed that do indicate that support for a common factor

is not invalidated, it was clear that further testing for the common factor is required. While further testing with the bifactor model on other measures and using other samples is a possibility, alternative psychometric and structural equation modelling (SEM) techniques can provide another avenue for understanding the idea of the common liability. One particular method which is appropriate for investigating the possibility of a common liability factor is the circumplex. Proposed initially by Guttman (1954), it has been widely used in psychometrics, and in psychological research, especially in the area of personality. However the circumplex has not been used widely within clinical psychological research. The circumplex has several assumptions but one is most notable when considering the idea of the common liability factor. The circumplex assumes that the nature of relationships among constructs can best be described as an ordering of variables along the circumference of a circle, which implies that the constructs being mapped bear some relation to each other (Fabrigar, Visser, & Browne, 1997). If a circumplex is a good fit, it means that there is some meaningful shared relationship between all the constructs assessed. Therefore if a representation of broad childhood psychopathology could be mapped on a circumplex, support for the general factor demonstrated in the bifactor model could be inferred. Thus the primary aim of this study was to further examine if there was support for a common factor underlying child psychopathology using an alternative model, the circumplex.

The Nature of the Circumplex

The circumplex model was most clearly outlined by Guttman (1954) who described the circumplex as a "system of variables which has a circular law of order" (p. 325), where variables would be of equal 'complexity' but would differ among themselves

in the kind of content they define. Circumplex models have been shown to be relevant for psychological domains, including personality and temperament, affective states, and interpersonal interactions (e.g. Becker & Krug, 1964; Di Blas, 2007; Markey, 2006; Plutchik & Conte, 1997; Schaefer, 1961; Wiggins & Trapnell, 1996; Wiggins, Trapnell, & Phillips, 1988). However, the relevance of a circumplex model for child behaviour problems has not yet been fully tested.

Core Conceptual Properties of the Circumplex

Conceptualising the relations of a set of variables for a domain as a circumplex implies three core conceptual properties (Fabrigar et al., 1997). First, the set of variables for the domain are interrelated. As noted, the existing data for the CBCL show positive associations for the eight empirically validated syndromes and for the six DSM-Oriented categories (Achenbach & Rescorla, 2001). Similarly, there are high levels of correlation among DSM categories as measured by the ADIS-IV-Parent (Angold et al., 1999; Lilienfeld, 2003; Silverman & Albano, 1996).

The second core conceptual property of the circumplex is that the variables for the domain are best represented by two major dimensions. Consistent with this, there is now general acceptance that child behaviour problems in general, and specifically both the ASEBA syndromes and DSM-categories of childhood behaviour, are viewed as representing the basic dimensions of Externalizing and Internalizing behaviour problems (Achenbach et al., 2008; Achenbach & Rescorla, 2001; Angold et al., 1999; Lilienfeld, 2003). Indeed support for this is also derived from the results from Study 1, as there were significant and substantive loadings on the domain

specific factors of externalising and internalising within a bifactor model, for all measures assessed.

The final core conceptual property of the circumplex is one that is unique to the circumplex structure, and this is the property most apposite when considering the idea of the common shared liability. This key property is that the variables within the assessed domain can be described in terms of an ordering along the circumference of a circle, with the direction and strength of the relationships between the variables related to the distance between the variables on the circumference. In other words the more closely related two variables within the domain are, the closer together they will be on the circumplex perimeter; the less associated two variables within the domain are, the further they will be apart.

An additional assumption of the circumplex is sometimes considered and that is that the variables are equally spaced along the circumference of the circle. However, Fabrigar et al. (1997) have pointed out this is not mandatory for the circumplex structure and is made more for theoretical reasons. There are no theoretical grounds to suggest such an assumption would hold true for ASEBA syndromes or the ADIS-IV-P DSM-categories of childhood behaviour, and given the views of Fabrigar et al. (1997) this assumption is not further considered.

Conceptual Links Between the Circumplex Model and the Bifactor Model

The nature of the circumplex shares some key conceptual similarities with the bifactor model examined in the previous chapter; similarities which make the circumplex an appropriate model to provide additional support for the proposed bifactor model. As outlined in Chapter 3, a bifactor model contains specific factors

on which independent subsets of items load. These specific factors explain unique variance of the items within the specific factor subset. The bifactor model of childhood psychopathology proposed in Chapter 3 contains two such specific factors; an Internalising Behaviour factor, and an Externalising Behaviour factor. The presence of two such specific factors in the proposed bifactor model matches the key conceptual property of the circumplex in that the variables for the domain are best represented by two major dimensions. Similarly the circumplex assumes that the nature of relationships among constructs as ordered along the circumference of a circle implies that the constructs being mapped bear some relation to each other (Fabrigar et al. 1997). This closely resembles the concept of a general/common factor of the bifactor model which is defined as representing the variance/relationships that are shared across all items (Reise, Morizot, & Hays, 2007). Thus it is clear that there are certain conceptual similarites between the proposed bifactor model, and the circumplex.

Overall, it is quite clear that the nature of childhood psychopathology, whether at a categorical or dimensional level, meet the basic principles and assumptions of the circumplex. Were good fit to the circumplex to be achieved, it would provide strong support to the idea of a common shared relationship between all the behaviour problems assessed, from which support for the general factor as modelled and conceptualised in the bifactor model can be made.

Testing the Circumplex

Browne (1992); (Fabrigar et al., 1997) developed a non-standard covariance structure model procedure, called the circular stochastic process model with Fourier series

(CSPMF), for testing circumplex models. Like all covariance structure models, CSPMF assumes that the total variance of an observed score is comprised of the common score variance (variance attributed to the underlying latent factor) and unique score variance (which also includes error variance). Also, when one variable (based on its common score) is used as a reference point on the perimeter of a circle, the other variables (based on their common scores) can be located on the circumference of a circle in terms of polar angles from the reference variable. A further assumption of the model is that the angle of separation of two common score variables is related to the correlation between them. Specifically, the correlation between common score variables is an inverse function of their angular distance.

Browne (1995) developed software called CIRCUM which executes the CSPMF. CIRCUM evaluates the extent to which the Pearson product-moment inter-correlation matrix of observed scores fits the circumplex model. CIRCUM provides estimates of the communality of each measured variable, the polar angles of common score variables, the minimum common score correlation, and an estimate of the mathematical relationship between the distances on the circle and correlations among common scores, called the correlation function. The communality of each measured variable is the proportion of variance estimated to represent common variance. The polar angle indicates where the variables are located on the circumference, relative to the variable used as the reference point. The correlation function represents the relationship of the angles of separation between common score variables to the correlations between common score variables. Theoretically, when the correlations are all positive, as in the case of the childhood behaviour problems, the polar angle between two common score variables at 180°, called the minimum correlation

function, will be 0. Thus, the correlations of the other variables with the reference

variable (which is set at 0) will decrease moving (either clockwise or counter-

clockwise) from the reference variable to the point located at 180°. They will then

show increases moving from 180° back to the reference variable.

Aims of the Study

The aim of this study is to assess the fit to the circumplex of childhood

psychopathology, both in terms of empirically validated syndromes and DSM-based

diagnostic categories, using the same data used in Study 1. This was done by

subjecting both the clinical sample and ASEBA Reference Group data, from the

CBCL Syndrome and DSM-Oriented Scales, to the CSPMF procedure in CIRCUM.

Additionally, the clinical sample data from the Anxiety Disorders Interview

Schedule-Parent Version (ADIS-IV-P) used in Study 1 was also subjected to the

CSPMF procedure implement in CIRCUM. While this is an exploratory study,

support for the circumplex was hypothesised. Based on the mean factor loadings of

the eight syndromes on the Internalizing and Externalizing factors (Achenbach &

Rescorla, 2001), it could be speculated that the distance of the syndromes on the

circumference, if starting from Aggressive Behaviour, will be: Aggressive Behaviour

→ Rule-Breaking Behavior → Attention Problems → Social Problems → Somatic

Complaints → Withdrawn/Depressed → Anxious/Depressed, with the distance

between Aggression and two depression scales being the largest. Similarly for the

ASEBA Syndrome scales one would predict a structure, beginning with Oppositional

Defiant Problems to be: Oppositional Defiant Problems → Conduct Problems →

Attention Deficit/ Hyperactivity Problems → Somatic Problems → Anxiety

Problems → Affective Problems. Predicting a structure for the ADIS-IV-P is slightly

more difficult as clear factor loadings from a factor analysis are not available.
Furthermore, it is difficult to predict the precise placement of the eight different
anxiety disorders on the circumplex. However, if those disorders were grouped
together, then based on the predictions for the ASEBA Syndrome scales, we might
predict a structure, beginning with Oppositional Defiant Disorder to be: Oppositional
Defiant Disorder → Conduct Disorder → ADHD →Depressive Disorders →
Anxiety Disorders.

Method

Participants

Clinical Sample

The participants were the same 968 parents and their children referred to the
Department of Academic Child Psychiatry of the Royal Children's Hospital,
Melbourne, between 2001 and 2009 that were used in Study 1. From this sample,
CBCL Syndrome scores were available from all participants, and DSM-Oriented
scores were available from 762 participants.

ASEBA Reference Group

Details about the ASEBA Reference Group are outlined in Study 1.

Materials

The primary measures were the same as for Study 1 on the circumplex model; the
Child Behaviour Checklist (designated as the CBCL/6–18) from the Achenbach
System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001)
and the Anxiety Disorders Interview Schedule-Parent Version (ADIS-IV-P:

Silverman & Albano, 1996). Details on this measure are shown in the materials

section of Study 1.

Again, as per the previous study on the bifactor model, CBCL *T*-scores from the

sample were used in the current study as the raw were not available from all files.

Also as per Study 1, the Thought Problems syndrome from the CBCL Syndrome

Scales was not used on theoretical grounds. Similarly, the ADIS-IV-P diagnoses of

Dysthymia and Major Depression diagnoses were collapsed into a single entity titled

Depression.

Procedure

The data collection procedures were the same as those in Study 1.

Data Analysis

For the clinical sample, the 7 x 7 Pearson product-moment intercorrelation matrix for

CBCL Syndrome Scales (minus Thought Problems), the 6 x 6 Pearson product-

moment intercorrelation matrix for the CBCL DSM-Oriented Scale and the 12 x 12

Pearson product-moment intercorrelation matrix for the ADIS-IV-P were calculated

using SPSS Version 20. Technically, given the nature of the ADIS-IV, which

provides dichotomous categories (diagnosis present or diagnosis absent), a phi

coefficient matrix would be appropriate. However, a Pearson correlation coefficient

estimated for two binary variables will return the phi coefficient, and thus the use of

a Pearson's product-moment intercorrelation matrix is appropriate (Guilford, 1936).

Model Estimation

The correlation matrices for both the CBCL Syndrome and DSM-Oriented scales

from the ASEBA reference group were directly inputted into CIRCUM (Browne,

1995). The correlation matrices for the ASEBA reference group, reproduced from

Appendix 2 in the ASEBA manual (Achenbach & Rescorla, 2001), are shown in

Chapter 3, in Tables 3.5 (Syndrome Scales) and 3.6 DSM-Oriented Scales. The

maximum likelihood (ML) method of estimation was used to fit the model, as this

was the only appropriate estimator available in CIRCUM. CIRCUM, which executes

the circular stochastic process model with Fourier series (CSPMF), evaluates the

extent to which the intercorrelation matrix of observed scores fits the circumplex.

The Rule-Breaking Behaviour Syndrome subscale and the Conduct Problems DSM-

Oriented scale were designated as the reference variable for the ASEBA analyses,

and Conduct Disorder diagnosis was designated as the reference variable for the

ADIS-IV-P analysis. This means that CIRCUM set the location of the respective

scale score to 0° and estimated the location of the other subscales/diagnoses with

reference to this subscale.

The fit of the model was examined by consecutively increasing the number of free

parameters, beginning with one parameter and retaining the solution with the lowest

number of free parameters, beyond which there was no further improvement in

model fit. For all analyses, only one parameter had to be freed to attain parsimonious

solutions. No constraints were placed on the locations of the syndrome scores, the

communalities, and the minimum common score correlation. CIRCUM allows these

additional constraints to be placed on the model, but they are not necessary for a

circumplex (Fabrigar et al., 1997). There was one boundary parameter for the

Syndrome Scale analysis, and none in the DSM-Oriented Scale analysis or the ADIS-IV-P analysis. In CIRCUM, a boundary parameter is an estimate of communality that is approximately equal to one (Browne, 1992). Having one or two boundary parameters is common and need not be a source of concern (Fabrigar et al., 1997).

Assessing Model Fit

CIRCUM calculates the χ^2 likelihood ratio test statistic, but this statistic is affected substantially by sample size, and as such almost any model will be rejected when the sample size is large. In view of this, practical fit indices were used to ascertain model fit. CIRCUM provides an estimate of the root mean square error of approximation (RMSEA), which was described in Chapter 3. CIRCUM also provides the residual matrix, and the maximum likelihood discrepancy function. These can be used for independent computation of the standard root mean square residual (SRMR), and the goodness of fit index (GFI). The formula used for calculating SRMR, which was described in Chapter 3, was that suggested by Brown (2006) as shown in Equation 4.1. The GFI (Jöreskog & Sörbom, 1981) was developed as an alternative to the chi-square test and shows how closely the model comes to replicating the observed covariance matrix (Diamantopoulos & Siguaw, 2000). The formula for calculating GFI was that suggested by MacCallum and Hong (1997) as shown in Equation 4.2.

$$SRMR = \sqrt{\frac{\text{sum of the square of each residual in the correlation matrix}}{\text{number of elements in the correlation matrix}}} \qquad (4.1)$$

$$GFI = \frac{\text{number of observed scores}}{\text{number of observed scores} + 2(\text{maximum likelihood discrepancy function})} \qquad (4.2)$$

Hu and Bentler (1998) outline guidelines for determining good fit, with SRMR values of .08 or less taken as indication of good fit. With regard to RMSEA, Hu and Bentler (1998) argue that RMSEA values of .06 or less can be taken as indicative of good fit. However, Browne and Cudeck (1993) have suggested that RMSEA values less than .08 can be inferred as reasonable fit and it is this upper limit that is used here. For the GFI, Hu and Bentler (1998) outline that values of .90 or greater are indicative of good fit. Thus an acceptable fit was considered to have been achieved if both RSMEA and SRMR values were $\leq$ .08, and the GFI value was $\geq$ .90.

Results

CBCL Syndrome Scales – Clinical Sample

Pearson product-moment correlations for the CBCL Syndrome Scales in the clinical sample were calculated and are shown in Table 4.1. The correlation matrix shown in Table 4.1 was inputted into CIRCUM to assess the CBCL Syndrome scales against the circumplex. The analysis revealed a significant chi-square, χ^2 (7) = 43.59, p < .001, but as indicated previously, this statistic is affected substantially by sample size, and thus this fit index was not used to assess fit. CIRCUM indicated RMSEA = .07 (90% CI = 0.05 - 0.10), demonstrating an acceptable fit to the circumplex. Other fit indices were calculated as per Equations 4.1 and 4.2, and indicated SRMR = .04 and GFI = .99, demonstrating a good fit to the circumplex. Taken together, these values indicate good fit for the circumplex model of CBCL syndrome scales.

Table 4.1

Pearson Product-Moment Correlations for The Child Behavior Checklist (CBCL)

Syndrome Scales For N=974 Parent Reports in the Clinical Sample

	Anxious/ Depressed	Withdrawn /Depressed	Somatic Complaints	Social Problems	Attention Problems	Rule- Breaking Behaviour
Withdrawn/ Depressed	.55**					
Somatic Complaints	.53**	.37**				
Social Problems	.51**	.43**	.37**			
Attention Problems	.29**	.32**	.26**	.56**		
Rule-Breaking Behaviour	.16**	.17**	.12**	.41**	.40**	
Aggressive Behaviour	.33**	.27**	.18**	.56**	.47**	.77**

Note: * p< 0.05 (2-tailed), ** p< 0.01 (2-tailed)

Table 4.2 shows the communality indices for the different syndromes of the CBCL.

As shown in Table 4.2, the communality indices, which represent the correlations

between measured and common score variables, were all relatively high, ranging

from .58 to 1.00, thereby indicating that all syndromes had relatively high levels of

the common factor.

Table 4.2

Communality Index and 95% Confidence Interval (CI) for Community Indices and Polar Angles and 95% Confidence Interval (CI) for Polar Angles for the Child Behavior Checklist Syndrome Scales for the Clinical Sample

Syndrome	Communality Index		Polar Angle	
	Estimate	95% CI	Estimate	95% CI
Rule Breaking Behaviour	.77	.74 - .80	0º	**
Aggressive Behaviour	1.00*		7º	1º - 14º
Anxious/Depressed	.95	.73 - .99	172º	160º -183º
Somatic Complaints	.58	.52 - .63	200º	179º - 221º
Withdrawn/Depressed	.62	.57 - .66	210º	190º - 229º
Social Problems	.86	.82 - .90	271º	260º - 281º
Attention Problems	.66	.61 - .70	287º	276º - 299º

Note:* Indicates on a boundary parameter. No 95% CIs can be calculated for boundary   parameters. ** Designated as the reference variable for the analyses

The correlation function, shown in Figure 4.1, satisfied the requirement of monotonic decrement from 0° to 180°, and the minimum common score correlation was 0.33. A key finding was the first Beta value. This Beta value, which provides an estimate of the proportion of variability explained by the shared/common factor, was .66, indicating 66% of the variance can be accounted for by the common factor. This provides strong support for the hypothesised common factor for the CBCL Syndrome Scales, within a circumplex model.
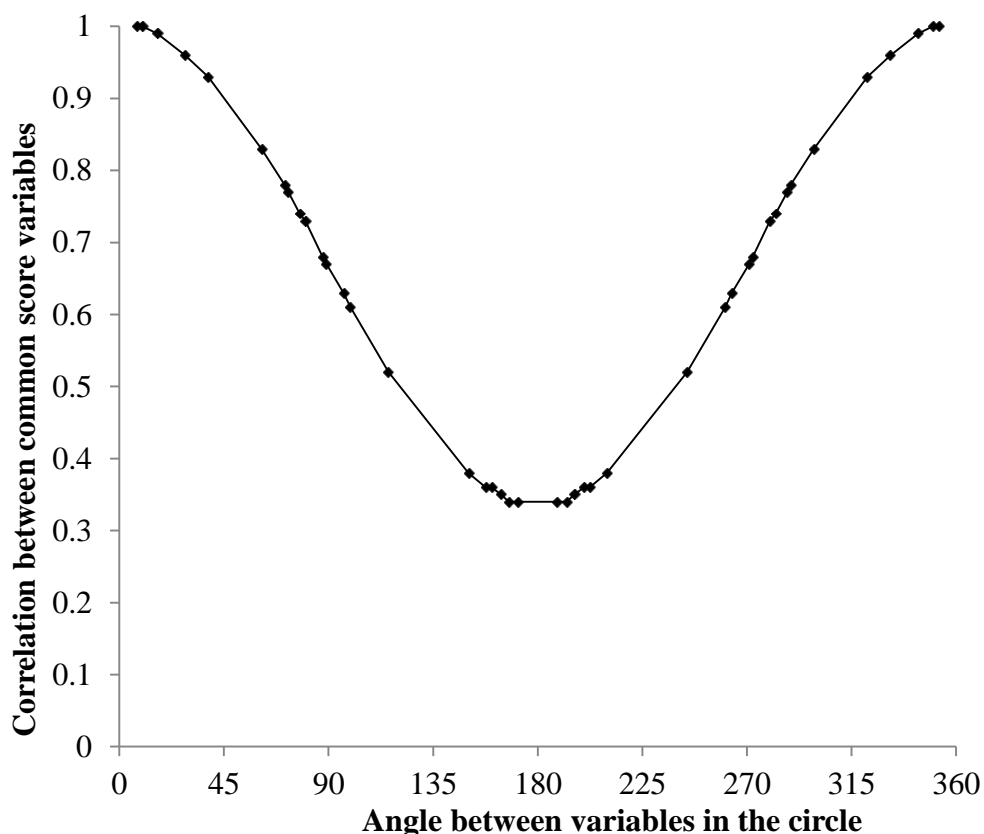
Figure 4.1. Correlation function for the CBCL Syndrome Scales for the clinical

sample

Table 4.2 also includes the point estimates of the polar angles, and their 95%

confidence intervals, for the different syndromes of the CBCL. These estimates are

also depicted graphically in Figure 4.2. As can be seen in the table and figure, for the

CBCL, the space occupied by the polar angles for the externalizing syndromes of

Aggressive Behaviour, Rule-Breaking Behaviour and Attention Problems were

within 80°, with the angle between Aggressive and Rule-Breaking Behaviours being

only 7°. Social problems was closer to the externalizing syndromes on the

circumplex, than the internalizing syndromes. The three internalizing syndromes of

Anxious/Depressed, Withdrawn/Depressed and Somatic Concerns were within a 38°

angle, and were roughly on the opposite end of the circumference to the externalising

syndromes. The total space occupied by all syndromes was 195° (in an anticlockwise direction).
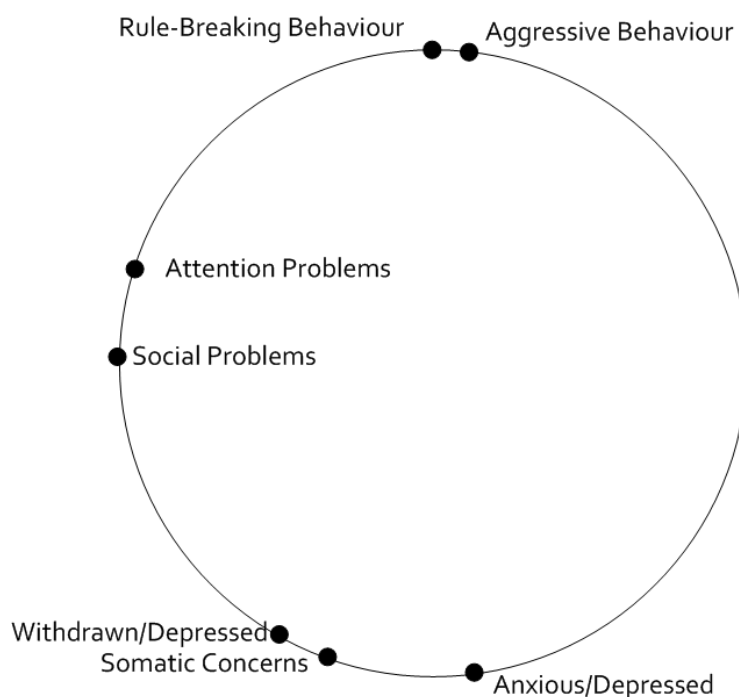
Figure 4.2. Polar angle estimates of the CBCL Syndrome Scales for the clinical sample

CBCL DSM-Oriented Scales - Clinical Sample

Pearson product-moment correlations for the CBCL DSM-Oriented Scale were calculated and are shown in Table 4.3. The correlation matrix shown in Table 4.3 was inputted into CIRCUM to assess the DSM-Oriented scales against the circumplex. The analysis revealed a significant chi-square, χ^2 (3, *N*= 974) = 17.87, *p* < .001, but as indicated previously, this fit index was not used to assess fit. CIRCUM indicated RMSEA = .08 (90% CI = 0.05 - 0.12), demonstrating marginally acceptable fit to the circumplex. Other fit indices were calculated as per Equations 4.1 and 4.2, and indicated SRMR = .02 and GFI = .99, demonstrating a good fit to

the circumplex. Taken together, these values indicate good fit for the circumplex

model of CBCL DSM-Oriented scales.

Table 4.3

Pearson Product-Moment Correlations for the Child Behavior Checklist (CBCL)

DSM-Oriented Scales For N=752 Parent Reports in the Clinical Sample

	Affective Problems	Anxiety Problems	Somatic Problems	Attention Deficit/ Hyperactivity Problems	Oppositional Defiant Problems
Anxiety Problems	.59**				
Somatic Problems	.48**	.39**			
Attention Deficit/ Hyperactivity Problems	.28**	.26**	.17**		
Oppositional Defiant Problems	.29**	.19**	.08*	.56**	
Conduct Problems	.28**	.12**	.08*	.56**	.75**

Note: * p< 0.05 (2-tailed), ** p< 0.01 (2-tailed)

Table 4.4 shows the communality indices for the different DSM-Oriented factors of

the CBCL. The communality indices, ranging from .54 to .95, indicated that all

syndromes had relatively high levels of the common factor. The correlation function,

shown in Figure 4.3, satisfied the requirement of monotonic decrement from 0° to

180°, with a minimum common score correlation of .29. The First Beta value

was .64, indicating approximately 64% of the variance can be accounted for by the

common factor. This provides strong support for the hypothesised common factor for

the DSM-Oriented scales within the circumplex model.

Table 4.4

Communality Index and 95% Confidence Interval (CI) for Community Indices and

Polar Angles and 95% Confidence Interval (CI) for Polar Angles for the Child

Behavior Checklist DSM-Oriented Scale

Syndrome	Communality Index		Polar Angle	
	Estimate	95% CI	Estimate	95% CI
Conduct Problems	.88	.84 - .92	0º	*
Affective Problems	.95	.62 – 1.00	145º	120º - 170º
Somatic Concerns	.54	.47 - .60	174º	122º - 226º
Anxiety Problems	.67	.61 - .73	181º	130º - 232º
Attention Deficit/ Hyperactivity Problems	.80	.57 - .93	294º	263º - 326º
Oppositional Defiant Problems	.86	.81 - .89	354º	344º - 4º

Note:*Designated as the reference variable for the analyses
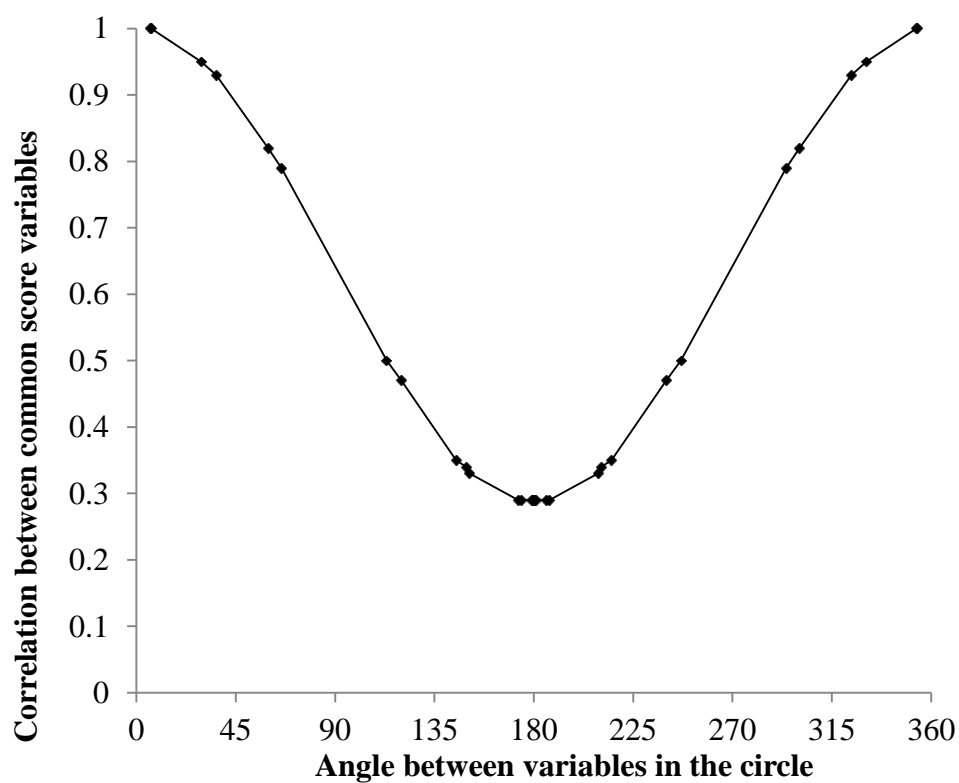
Figure 4.3. Correlation function for the CBCL DSM-Oriented Scales in the clinical

sample.

Table 4.4 also includes the point estimates of the polar angles, and their 95%

confidence intervals, for the different DSM-Oriented scales of the CBCL. These

estimates are also depicted graphically in Figure 4.4. As can be seen in the table and

figure, the space occupied by the polar angles for the externalizing syndromes of

Conduct, Attention Deficit Hyperactivity and Oppositional-Defiant Problems, were

within 66°, with the angle between Conduct and Oppositional-Defiant Problems

being only 6°. The three internalizing syndromes of Affective, Anxiety and Somatic

Problems, were within 36° of space and were roughly on the opposite end of the

circumference to the externalising syndromes. The total space occupied by all

syndromes was 215° (in an anticlockwise direction).
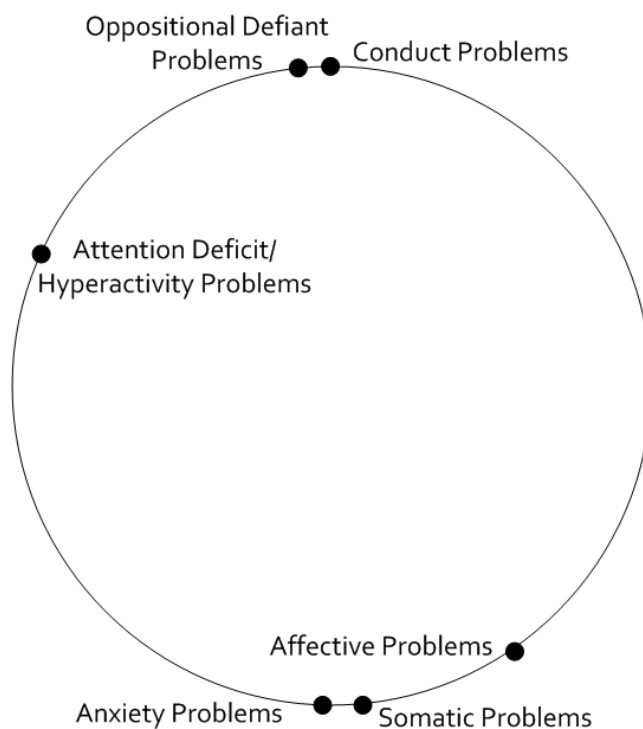
Figure 4.4. Polar angle estimates of the CBCL DSM-Oriented Scales in the clinical

sample

CBCL Syndrome and DSM-Oriented Scales - ASEBA Reference Group

The correlation matrix for both the CBCL Syndrome and DSM-Oriented scales from the ASEBA Reference Group (Achenbach & Rescorla, 2001) was inputted into CIRCUM. The fit indices for the Syndrome scales were, $\chi^2 (7) = 15.21$, $p < .001$, RMSEA = .04 (90% CI = 0.01 - 0.07), SRMR = .02 and GFI = .99, demonstrating a good fit to the circumplex. The fit indices for the DSM-Oriented scales were, $\chi^2 (3) = 3.13$, $p < .001$, RMSEA = .01 (90% CI = 0 - 0.06), SRMR = .01, and GFI = .99, demonstrating a good fit to the circumplex.

Table 4.5 shows the communality indices for the Syndrome and DSM-Oriented Scales. The communality indices, were all relatively high, with most above .70, and all but the Somatic Problems from the DSM-Oriented Scales showing communality above .50, thereby indicating that there were acceptably high levels of the common factor for each subscale in the reference sample. There was one boundary item for the Syndrome scales and none for the DSM-Oriented scales, which were within acceptable tolerances (Fabrigar et al., 1997).

Table 4.5

Communality Index and 95% Confidence Interval (CI) for Communality Indices for the Child Behavior Checklist (CBCL) Syndrome and DSM-Syndrome Scales from the ASEBA Scale Reference Sample

Syndrome	Syndrome Scales		Syndrome	DSM-Oriented Scales	
	Communality	95% CI		Communality	95% CI
Rule Breaking Behaviour	1.00*		Conduct Problems	.83	.75 - .90
Withdrawn/ Depressed	.75	.66 - .82	Affective Problems	.95	.06 – 1.00
Anxious/ Depressed	.73	.67 - .78	Somatic Problems	.38	.31 - .46
Somatic Complaints	.51	.45 - .57	Anxiety Problems	.61	.53 - .68
Social Problems	.79	.75 - .82	Attention Deficit/ Hyperactivity Problems	.75	.70 - .80
Attention Problems	.72	.68 - .76			
Aggressive Behaviour	.86	.83 - .88	Oppositional Defiant Problems	.82	.77 - .85

Note:* Indicates on a boundary parameter. No 95% CIs can be calculated for boundary parameters.

The correlation functions for both scales, which are shown in Figure 4.5, satisfied the requirement of monotonic decrement from 0° to 180°. The minimum common score correlation for the Syndrome Scales was .43 and .54 for the DSM-Oriented scales. These figures are higher than ideal, though given the model fitted all other requirements of the circumplex, this high score alone does not invalidate the model (Fabrigar et al., 1997).
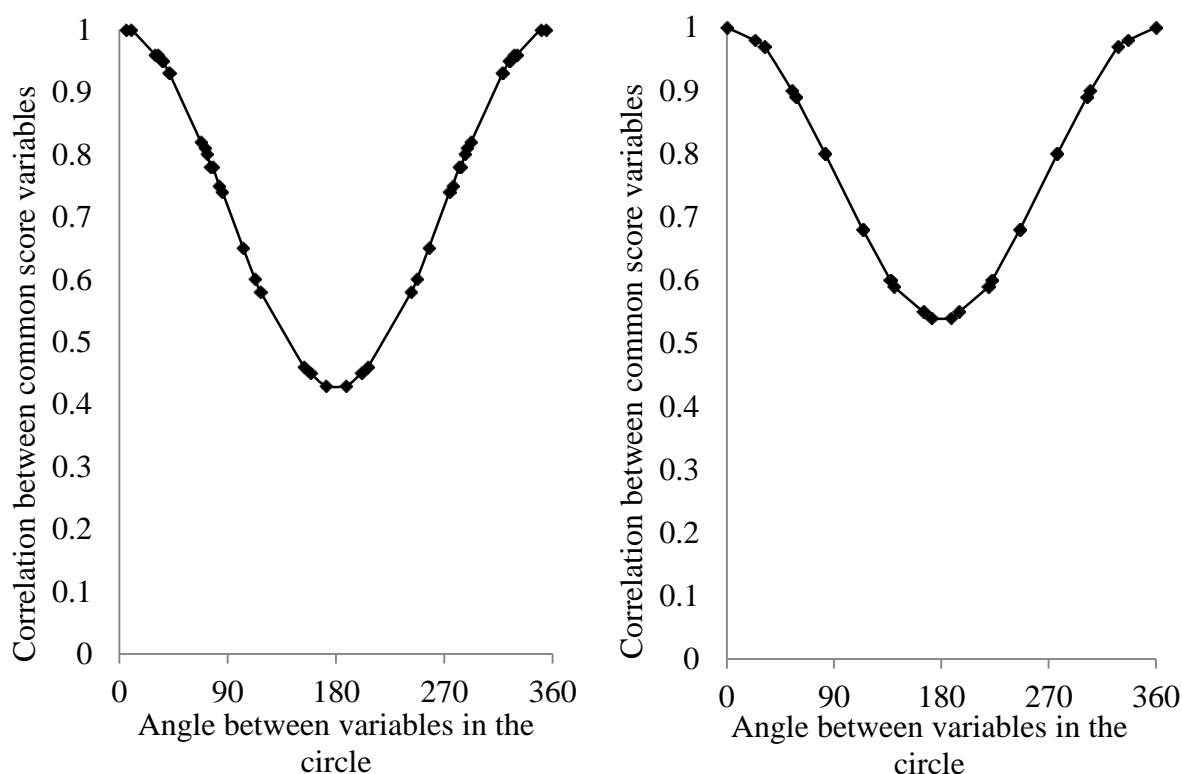
Figure 4.5. Correlation functions for the CBCL Syndrome Scales (left) and the

CBCL DSM-Oriented scales (right) for the ASEBA Scale Reference sample

The first Beta values were .71 for the Syndrome scales and .77 for the DSM-Oriented

scales, indicating 71% of the variance in Syndrome scales, and 77% of the variance

in the DSM-Oriented scales, can be accounted for by the common factor. This

provides strong support for the hypothesised common factor within the reference

sample. Taken together, these results are consistent with the assumptions for a

circumplex model, and provide strong support for a common factor for the CBCL

Syndrome Scales and DSM-Oriented scales within a normative sample.

Table 4.6 shows the point estimates of the polar angles and their 95% confidence

intervals for both the CBCL Syndrome and DSM-Oriented scales, with Figure 4.6

graphically depicting these polar angles.

Table 4.6

Polar Angles and 95% Confidence Interval (CI) for Polar Angles for the ASEBA Reference Sample, Child Behavior Checklist (CBCL) Syndrome Scale and DSM-Oriented Scale

	Syndrome Scales			DSM-Oriented Scales	
Syndrome	Polar Angle	95% CI	Syndrome	Polar Angle	95% CI
Rule Breaking Behaviour	0º	*	Conduct Problems	0º	*
Withdrawn/ Depressed	171º	158º - 185º	Affective Problems	165º	141º - 189º
Anxious/ Depressed	201º	182º - 221º	Somatic Problems	223º	138º - 308º
Somatic Complaints	207º	182º - 231º	Anxiety Problems	223º	143º - 302º
Social Problems	243º	229º - 256º	Attention Deficit Hyperactivity Problems	305º	268º - 342º
Attention Problems	274º	263º - 286º			
Aggressive Behaviour	284º	275º - 294º	Oppositional Defiant Problem	337º	299º - 15º

Note: *Designated as the reference variable for the analyses

As can be seen in the table and figure, for the CBCL Syndrome scales, the space occupied by the polar angles for the externalizing syndromes of Aggressive Behaviour, Rule-Breaking Behaviour and Attention Problems were within 86º, a similar distance to that found for the clinical sample. However, the distance between Aggressive and Rule-Breaking Behaviours was 76º rather than 7º for the clinical sample. The Social Problems factor was evenly positioned between the externalizing syndromes and the internalizing syndromes. The three internalizing syndromes of

Anxious/Depressed, Withdrawn/Depressed and Somatic Concerns were close to each other within a 38° angle, and were roughly on the opposite end of the circumference to the externalising syndromes. The total space occupied by all syndromes was 189° (in an anticlockwise direction).
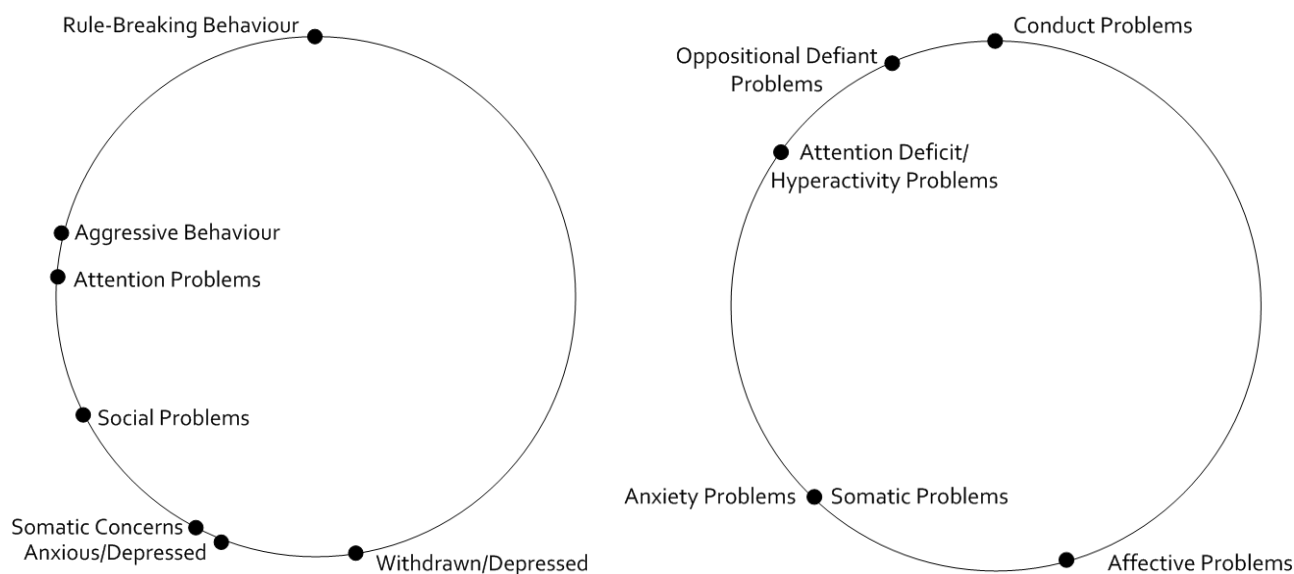

Figure 4.6. Polar angle estimates of the CBCL Syndrome Scales (left) and the DSM-Oriented scales.

For the DSM-Oriented scale, the externalizing syndromes of Conduct, Attention Deficit Hyperactivity and Oppositional-Defiant Problems, were evenly spaced within a 55° angle space. The three internalizing syndromes of Affective, Anxiety and Somatic Problems, were within 58° of space and were roughly on the opposite end of the circumference to the externalising syndromes. However, the angle for somatic and anxiety problems occupied the same angle, which is a slightly unusual finding. However, given that all of the Anxiety Disorders within DSM-IV-TR have many somatic-related symptoms as part of their diagnostic criteria, the close relationship between these two clusters is theoretically plausible (American Psychiatric

Association, 2000). The total space occupied by all syndromes was 195° (in an

anticlockwise direction).

ADIS-IV-P

Pearson product-moment correlations for the ADIS-IV-P were calculated and are

shown in Table 4.7.

Table 4.7

Pearson Product-Moment Correlations for ADIS-IV-P for N=974 Parent Reports in

the Clinical Sample

	DEP#	PTSD	SOC	SPE	PD	AGO	GAD	SAD	OCD	CD	ODD
PTSD	.19**										
SOC	.21**	.17**									
SPE	.12**	.16**	.19**								
PD	.15**	.14**	.12**	.12**							
AGO	.04	.17**	.21**	.16**	.17**						
GAD	.36**	.24**	.28**	.23**	.22**	.17**					
SAD	.17**	.21**	.25**	.18**	.18**	.16**	.31**				
OCD	.10**	.08*	.16**	.16**	.15**	.20**	.21**	.16**			
CD	.12**	.13**	.13**	.05	.03	.02	.04	.12**	.01		
ODD	.19*	.07*	.10**	.06	.03	.01	.09**	.12**	.09**	.49**	
ADHD	.01	.08*	.12**	.08*	.02	.07*	.04	.05	.06	.25**	.29**

Note: *p< 0.05 (2-tailed); ** p< 0.01 (2-tailed)
DEP = Depression, PTSD = Post-Traumatic Stress Disorder, SOC = Social Phobia, SPE = Specific Phobia, PD = Panic Disorder, AGO = Agoraphobia, GAD = Generalised Anxiety Disorder, SAD = Seperation Anxiety Disorder, OCD = Obsessive-Compulsive Disorder, CD = Conduct Disorder, ODD = Oppositional Defiant Disorder, ADHD = Attention Deficit/Hyperactivity Disorder
# Depression = Dysthymia + Major Depression

The correlation matrix was inputted into CIRCUM to assess the DSM-Oriented scales against the circumplex. The analysis revealed a significant chi-square, χ^2 (42, $N= 974) = 74.48$, $p < .001$, but as indicated previously, this fit index was not used to assess fit. CIRCUM indicated RMSEA = .03 (90% CI = 0.02 - 0.4), demonstrating excellent fit to the circumplex. Other fit indices were calculated as per Equations 4.1 and 4.2, and indicated SRMR = .01 and GFI = .99, demonstrating an excellent fit to the circumplex. Taken together, these values indicate good fit for the circumplex model of ADIS-IV-P diagnostic categories.

Table 4.8 shows the communality indices for the different ADIS-IV-P diagnostic categories. As shown, the communality indices, ranged from .34 to .79, with an average communality of .5, indicated that all syndromes had relatively substantive levels of the common factor.

Table 4.8

Communality Index and 95% Confidence Interval (CI) for Community Indices and

Polar Angles and 95% Confidence Interval (CI) for Polar Angles for the ADIS-IV-P

in the Clinical Sample

Diagnosis	Communality Index		Polar Angle	
	Estimate	95% CI	Estimate	95% CI
CD	.63	.55 - .70	0º	*
ODD	.79	.70 - .87	14º	359º - 28º
DEP#	.64	.52 - .76	130º	113º - 147º
GAD	.66	.60 - .73	180º	155º - 204º
PD	.34	.28 - .42	203º	170º - 235º
PTSD	.40	.33 - .47	214º	186º - 241º
SAD	.50	.44 - .57	224º	202º - 247º
SOC	.49	.43 - .56	226º	203º - 248º
SPE	.40	.33 - .47	226º	201º - 252º
OCD	.35	.29 - .43	227º	200º - 255º
AGO	.38	.31 - .45	234º	209º - 260º
ADHD	.42	.35 - .49	330º	308º - 352º

Note:*Designated as the reference variable for the analyses
DEP = Depression, PTSD = Post-Traumatic Stress Disorder, SOC = Social Phobia,
SPE = Specific Phobia, PD = Panic Disorder, AGO = Agoraphobia, GAD =
Generalised Anxiety Disorder, SAD = Seperation Anxiety Disorder, OCD =
Obsessive-Compulsive Disorder, CD = Conduct Disorder, ODD = Oppositional
Defiant Disorder, ADHD = Attention Deficit/Hyperactivity Disorder
Depression = Dysthymia + Major Depression

The correlation function, shown in Figure 4.7, satisfied the requirement of monotonic

decrement from 0° to 180°, with a minimum common score correlation of .14. The

First Beta value was .57, indicating approximately 57% of the variance can be

accounted for by the common factor. This provides strong support for the

hypothesised common factor for the DSM-Oriented scales within the circumplex.
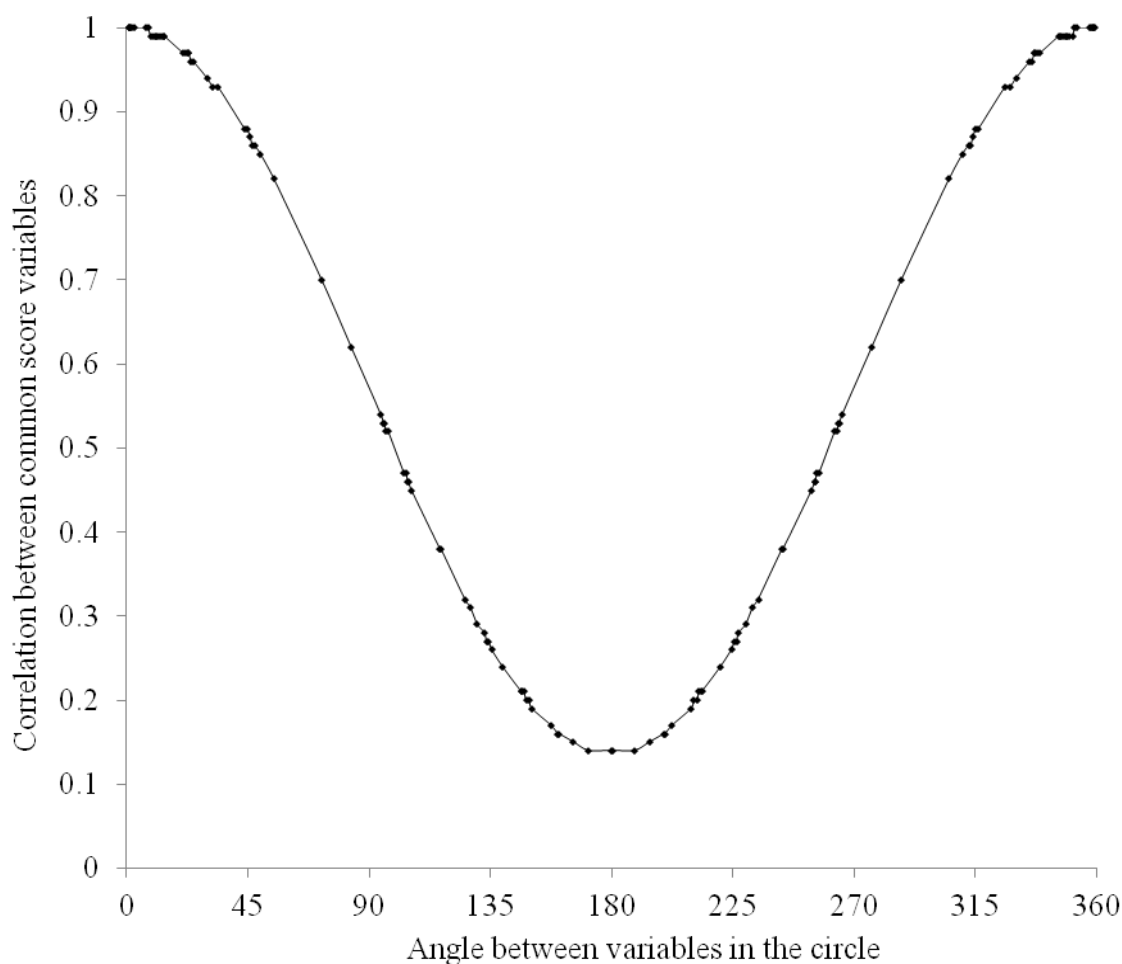
Figure 4.7. Correlation function for the ADIS-IV-P in the clinical sample

Table 4.8 also includes the point estimates of the polar angles, and their 95%

confidence intervals, for the different ADIS-IV-P. These estimates are also depicted

graphically in Figure 4.8.
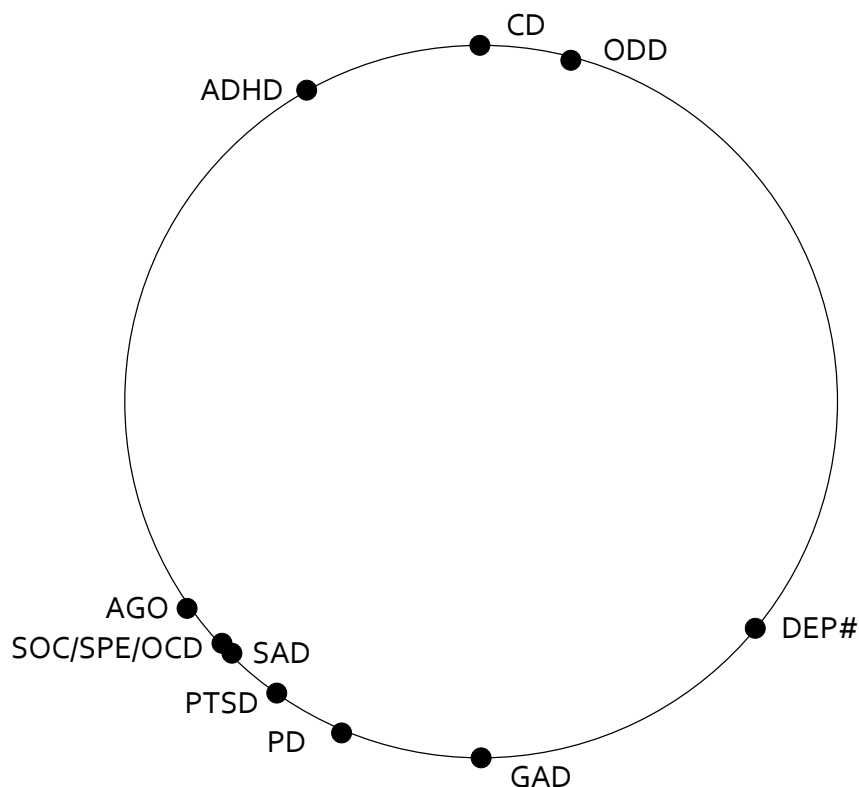
Figure 4.8. Polar angle estimates of the ADIS-IV-P in the clinical sample

Note: DEP[#] = Depression, PTSD = Post-Traumatic Stress Disorder, SOC = Social Phobia, SPE = Specific Phobia, PD = Panic Disorder, AGO = Agoraphobia, GAD = Generalised Anxiety Disorder, SAD = Seperation Anxiety Disorder, OCD = Obsessive-Compulsive Disorder, CD = Conduct Disorder, ODD = Oppositional Defiant Disorder, ADHD = Attention Deficit/Hyperactivity Disorder
[#]Depression = Dysthymia + Major Depression

As can be seen in the table and figure, the space occupied by the polar angles for the externalizing syndromes of CD, ADHD and ODD, were within 44°, with the angle between CD and ODD being only 14°. The internalizing disorders were roughly on the opposite end of the circumference to the externalising syndromes within 104° of space. The anxiety disorders were within 54°. One unusual result was the four anxiety disorders which occupied three degrees of space, including social and specific phobia which share the same space. Given that all were Anxiety Disorders within DSM-IV-TR, the close relationship between these disorders is theoretically plausible. Indeed the difficulty separating the phobia disorders is not without support

within the clinical literature (American Psychiatric Association, 2000), in part because of their high comorbidity (Angold et al., 1999). The total space occupied by all syndromes was 264° (in a clockwise direction).

Discussion

The primary aim of this study was to provide further support for underlying child psychopathology, using the circumplex. This was an extension of Study 1, which showed support for a common factor, as implied by the common liability model outlined in Chapter 1, using the bifactor model. Within the clinical sample, fit indices generally confirmed a circumplex structure, for all scales used. In support for the common factor, there was good fit to the circumplex for the CBCL Syndrome and DSM-Oriented scales, with the ADIS-IV-P showing excellent fit to the circumplex. For the CBCL, the communality indices for the different syndromes/categories were high, and for the ADIS-IV-P while lower than for the two CBCL scales, still relatively high, averaging around .5. This indicated that all syndromes/diagnostic categories had substantive high levels of loadings on their respective common factors. These findings were fully supported by the results from the ASEBA Reference group sample, which showed excellent fit to the circumplex.

One of the very noticeable findings, were the very high First Beta values for all analyses. This Beta value provides an indication of the proportion of total variance that is accounted for by the common factor. For the clinical sample, the Beta values exceeded .6 for the ASEBA scales, and .5 for the ADIS-IV-P. The results from the ASEBA reference group, an epidemiological sample, were even more supportive of a common factor, as Beta values exceeded .7. Taken together this indicates that for all

measures and all samples, more than half of the total variance in childhood

psychopathology can be accounted for by the common factor.

Examination of the polar angles across the measures showed consistent overall trends

with regards to the way syndromes/diagnostic categories are located along the

circumplex circumference. In all instances the externalizing syndromes were next to

each other, the internalizing syndromes were next to each other, and the externalizing

syndromes internalizing syndromes were on opposite ends on the circumplex. While

there was substantial variability across the scales with regard to the specific locations

of various syndromes/disorders around the circumference, this is due to the fact that

they assess childhood psychological problems in substantively different ways.

The correlation functions for all analyses satisfied the requirement of monotonic

decrement from 0° to 180°, an impressive finding given no measurement tool was

guided by a circumplex perspective in development. However, it needs to be noted

that the findings show that the minimum correlation functions for the clinical sample

were around .3 for the CBCL scales in the clinical sample, and .14 for the ADIS-P.

For the ASEBA reference group, these minimum correlation functions were

around .45. A predicted minimum correlation for positively correlated variables in a

circumplex, as in the current analyses, is zero. However, Fabrigar et al. (1997) noted

that this theoretical value of 0 is rarely obtained with a non-simulated data set.

CIRCUM controls for random measurement error in the observed scores, but cannot

control biases resulting from systematic measurement errors, such as common

method variance, which can inflate the associations between scores. This is

particularly relevant for childhood behaviour ratings completed by parents, such as

for the CBCL, as previous studies have shown that such scores have high common method variance (Gomez, Burns, Walsh, & Hafetz, 2005; Hartung, McCarthy, Milich, & Martin, 2005; Servera, Lorenzo-Seva, Cardo, Rodríguez-Fornells, & Burns, 2010). As such the minimum correlation function values are unlikely to reach that theoretical minimum of 0. Indeed Fabrigar et al. (1997) notes that minimum correlation functions of the size found in this study do not invalidate a circumplex interpretation provided other assumptions are met. As all other assumptions of the circumplex have been met, one can consider that the models still retain validity.

Lack of Discrimination Between Syndromes/Disorders

One potential issue in the results is the marked lack of separation between some of the syndromes/disorders. For the CBCL DSM-Oriented Scales, Anxiety and Somatic Complaints had negligible separation for the clinical sample, and indeed had no separation in the ASEBA Reference group. For the ADIS-IV-P there was a substantial lack of separation among anxiety disorders, and a shared space for Specific and Social Phobias. A number of explanations can be offered for the syndromes/diagnoses that showed negligible separation. One possibility is that they may be measuring similar behaviours. This may certainly be true for the shared positioning of the Phobia disorders in the ADIS-IV-P; while they are considered separable disorders from a DSM-IV perspective, the cognitive underlying the fear responses in these disorders are very similar (Hofmann, 2008). Similarly, for the negligible separation between Anxiety and Somatic Complaints on the CBCL DSM scales the fact that Anxiety Disorders within DSM-IV-TR have many somatic-related symptoms, means the close relationship is a theoretically plausible explanation (American Psychiatric Association, 2000; Bulbena & Pailhez, 2011; Haug,

Mykletun, & Dahl, 2004; Wilhelm & Roth, 2001; World Health Organization, 1993).

An alternate explanation is that, although they may be measuring different

behaviours, respondents may have difficulty differentiating between them. However,

whichever explanation is accurate, neither provides an invalidation of the circumplex

structure for child behaviour problems, and thus the results can still be considered as

support for a common factor.

The strong support for the circumplex within childhood psychopathology, provides

clear support for the common factor proposed in the bifactor model, given the

conceptual similarities identified. Indeed in combination with results from modelling

using the bifactor structure, it can be argued that there is clear substantive evidence

for the existence of a shared relationship between all childhood psychopathology,

such as proposed in the 'Alternate Forms' model of Neale and Kendler (1995), and

as outlined previously in Chapter 1 and 3. Support for this is strengthened by two

factors. Firstly, there is support for the common factor irrespective of diagnostic

systems, with results for DSM-based diagnostic categories, and empirically validated

syndromes being equally strong. Secondly, support for a common factor was equal

for both a clinical sample and an epidemiological sample, in the form of the ASEBA

Reference Group. Thus the support for a common factor, within the context of

multivariate analysis of childhood psychopathology and comorbidity, appears to be

universal.

Future Directions

Given the strong support for a common factor underlying childhood comorbidity, the

next issue to be considered is determining the nature of this common factor. The

basis for the common factor model was drawn from the idea of a *psychopathological liability*, similar to that proposed in the 'Alternate Forms' model of Neale and Kendler (1995) as outlined previously in Chapter 1. The psychopathological liability is conceptualised as being derived from a multifactorial combination of heritable and environmental causes, and implies a continuous spectrum of liability to disorders. In this model comorbidity occurs because the comorbid disorders are alternate manifestations of a single psychopathological liability spectrum, each with a differing threshold on this spectrum. Thus if a person reaches the threshold for multiple disorders, then they may manifest them separately and comorbidly.

If this common factor is indeed the 'psychopathological liability' as proposed in this model, then one would expect that the common factor would show significant and substantive associations with a range of genetic and environmental factors that are believed to be related to the development of comorbidity. Some possibilities include the constructs outlined in Chapter 2, including temperament constructs such as negative emotionality, parental psychopathology and poor family functioning. Modelling the association of these constructs with the common factor as modelled by the circumplex would be difficult, in part because of a lack of available programs to undertake such analyses. However, the bifactor model has been demonstrated as an appropriate model for understanding a common factor within childhood psychopathology and comorbidity, and use of the bifactor model more easily allows assessment of any potential associations of risk factors with the common factor. Modelling of this can be undertaken with Multivariate Latent Regression procedures, using the bifactor model as a base model. This is the aim of the following study in this thesis.

Conclusion

In conclusion, the present results demonstrate that child behaviour problems conform to a circumplex structure, both in terms of DSM-based diagnostic entities, and empirically validated syndromes. The fit to a circumplex, in combination with results from modelling using the bifactor structure in Study 1, provides support for the presence of a common factor underlying childhood psychopathology. However, further modelling is required to ascertain exactly what construct or constructs comprise this liability factor.

Chapter 5 - Study 3: Examination of Risk Factors Associated with the Common Liability Factor

The results of Study 1, with a bifactor model showing substantive loadings to a general factor for all modelled syndromes/diagnoses, combined with the results of Study 2 with the circumplex model, especially through high first beta values, show strong support for a common factor across all psychopathology. Given the strong support for a common factor, the nature of the common factor requires further evaluation. The basis for the common factor model was drawn from the idea of a psychopathological liability (Klein & Riso, 1993; Neale & Kendler, 1995); a multifactorial combination of heritable and environmental risk factors. If this common factor is indeed the psychopathological liability as proposed in this model, then one would expect that the common factor would show significant and substantive direct and interactive associations with a range of genetic and environmental factors that are believed to be related to the development of comorbidity. The aim of this study is to assess if risk factors, both individually and in interaction, show associations with the general and specific factors of the bifactor models of psychopathology, as would be expected if this general factor is a liability factor.

What Risk Factors Could Form the Liability Factor

Determining the constructs which may form part of any liability factor, and thus show associations with the common factor in the bifactor model, is open to question and theoretical debate. However, as reviewed in Chapter 2 of this thesis, three key

psychological constructs – the temperamental construct of negative affect, parental

psychopathology and familial functioning – should be considered to have

associations with the common factor, as they would be considered prime candidates

to form part of a multifactorial liability to psychopathology and comorbidity. These

three constructs cover each of the three broad domains of psychopathological risk

factor identified by Crawford et al. (2011), and thus form a broad cross section to

investigate. A full review of these risk factors is covered in Chapter 2, though a brief

review of each is provided presently.

Negative Affect

One key temperamental dimension that is part of almost all conceptualisations, and

which has been implicated in the development of psychopathology, is negative

affect. Negative affect, also referred to as negative emotionality, negative affectivity,

and neuroticism (Rothbart et al., 2000), was defined by Watson and Clark (1984) as a

pervasive disposition to experience unpleasant affective states such as guilt, anxiety

and irritability (see also Tellegen, 1982).

Research has consistently demonstrated that the temperament dimension of

negatively affect or its equivalent is involved in the aetiology and development of

child psychopathology (Lonigan & Phillips, 2001), and it has been proposed that

internalising and externalising behaviour share this factor in common (Lahey &

Waldman, 2003; Lilienfeld, 2003). It must be noted that research on the link between

negative affect and psychopathology lacks some clarity, because a large degree of the

research investigates superordinate constructs like 'difficult temperament' or

behavioural inhibition. These superordinate concepts, while reflecting aspects of

negative affect, are constructs encompassing many other different aspects of the temperament construct in addition to negative affect (De Pauw & Mervielde, 2010; Kagan et al., 1984). However, there is general consistency in the results of studies using both the negative affect construct and its superordinates, and this is that negative affect plays a significant role in the development of psychopathology and comorbidity (Muris & Ollendick, 2005). As a result, negative affect must be considered as part of psychopathological liability factor.

Parental Psychopathology

Research has consistently demonstrated that if a child has a parent with psychopathology, especially a mother, their risk of developing their own psychopathology is substantially heightened. There are many proposed mechanisms for how parental psychopathology affects children, including through genetic transmission, exposure to the parent's maladaptive affect, and environmental and contextual stressors associated with the parental illness (Connell & Goodman, 2002). As outlined in Chapter 2, much research has demonstrated a link between parental and child psychopathology, and though the research base investigating parental psychopathology as a risk factor for comorbidity is more limited, the available research generally supports the idea of parental psychopathology as a risk factor for comorbidity. This indicates that parental psychopathology is a particularly relevant candidate when considering constructs that may comprise a general liability factor.

Family Functioning

Family functioning is loosely defined as the nature in which functions of the family are performed, and the patterns of the relationships which connect members of a

family system (Lesser, 1985). Poor family functioning has been consistently investigated in relation to development of childhood psychopathology and comorbidity (Dietz et al., 2008), suggesting that family functioning is also a relevant candidate when considering constructs that may comprise a general liability factor.

Modelling the Associations Between Risk Factors and the General Factor

Modelling the associations between risk factors and the common factor is easily done using Structural Equation Modelling (SEM) procedures; specifically Multivariate Latent Regression (MVLR) procedures (see Bentler, 1980). Modelling relationships like these would be difficult using the circumplex, in part because of a lack of available programs to undertake such analyses, but also because modelling risk factors with the cirucmplex would not allow a clear assessment of global and domain specific risk factors such as those allowed by the bifactor model, and to be discussed presently. Such modelling is easily undertaken using the bifactor model using any SEM program, with the latent factors from the bifactor models for the CBCL and the ADIS-IV-P regressed onto either latent or observed scores for any risk factors.

Interactions Between Risk Factor Variables

As indicated in Chapter 2 however, the need to consider interactions between risk factor is also necessary. Much research has tended to examine risk factors in isolation (Wolff & Ollendick, 2006), which does not allow examination of the transactive fashion in which they work (Frick et al., 1992). SEM techniques can help with modelling such additive and interactive effects between risk factors, with programs such as MPlus (Muthen & Muthen, 2010) allowing very complex interactions between variables to be modelled. In this case, scores from assessment

tools for risk factors, such as those measuring negative affect, parental

psychopathology and family functioning, could be modelled to show additive and

interactive effects in any and all possible combinations of these factors. Interactive

effects can be modelled by regressing the bifactor model latent factors onto

interaction terms. This can be done easily in programs like MPlus by programming

the multiplicative term (i.e. risk factor A × risk factor B). Overall, SEM modelling

can allow both an assessment of the independent associations of a risk factor on the

specific and general factors from the bifactor model, but also the unique effect of risk

factor interactions.

Assessment of Global and Domain-Specific Risk

Just as much research has tended to examine risk factors in isolation, much research

has tended to examine risk factors in the development of either internalizing or

externalizing problems, but fewer studies have looked at how risk factors may be

related to the broader spectrum of childhood psychopathology, and indeed

comorbidity (Lee & Bukowski, 2012; Mäntymaa et al., 2012). As outlined in Chapter

2, some research has demonstrated correlates of internalizing or externalizing

problems in the same study, but has done so through separate analyses (e.g. Buist et

al., 2004; Leve et al., 2005), but only a limited number of studies have investigated

how risk factors might differentially impact upon internalizing and externalizing

domains (e.g. Mesman & Koot, 2000; Weiss et al., 1998). One great advantage of

assessing risk factors within a bifactor model was directly outlined in Study 1. The

bifactor model directly allows for the consideration of the risk factors that affect the

broad spectrum of psychopathology through assessing the association of the risk

factors on the general factor, as well as consideration of how risk factors may be

differentially associated with the internalising and externalising domains (Chen et al., 2006; Reise et al., 2007). The use of a bifactor structure in this context overcomes the limitations of precious research into the effect of risk factors.

Aim of This Study

The aim of this study is to assess the way negative affect, parental psychopathology and family functioning, and interactions between these variables, are associated with the general and specific factor of the bifactor models of psychopathology, as indexed in the CBCL Syndrome Scales[1], and the disorders of the ADIS-IV-P. For the purposes of this study negative affect was measured using an unpublished measure of trait child temperament, parent psychopathology was measured using the Hopkins Symptom Checklist (HSCL: Derogatis et al., 1974a; Derogatis et al., 1974b), and family functioning assessed by the widely used Family Assessment Device (FAD: Epstein et al., 1983). Should significant associations between risk factors/risk factor interactions and the common psychopathology factor be found, then it would imply that the common factor is most likely a liability factor as proposed by Klein and Riso (1993) and Neale and Kendler (1995) in their common liability factor models. Given that this study is using a new baseline model of psychopathology (the bifactor model), no specific hypotheses about the effect of the risk factors, and risk factor interactions is made. However, given the overwhelming evidence regarding the impact of negative affect on psychopathology, it is expected that this construct will universally associate with the model.

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[1] It should be noted that there was an attempt to model the effect of risk factors on the CBCL DSM-Oriented scales. However, any attempts to model the associations between the CBCL DSM-Oriented scales bifactor model and the risk factors led to model non-convergence, despite many iterations of the model. As a DSM-style approach was covered by the ADIS-IV-P (albeit a categorical system), this failure to find convergence, while inconvenient, was not considered to be overly problematic.

Method

Participants

The participants were the same clinical sample used for studies 1 and 2.

Materials

Measures of Psychopathology

The primary measures, the Child Behaviour Checklist (designated as the CBCL/6–18) from the Achenbach System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001) and the Anxiety Disorders Interview Schedule for Children-Parent Version (ADIS-IV-P: Silverman & Albano, 1996), were the same as in Study 1 and 2. Details on this measure are shown in the materials section of Study 1. Again, as per the previous studies, CBCL *T*-scores from the sample were used in the current study.

Academic Child Psychiatry Unit-Infant Temperament Questionnaire (ACPU-ITQ)

Negative affect was measured using an unpublished measures of trait child temperament; the Academic Child Psychiatry Unit-Infant Temperament Questionnaire (ACPU-ITQ). This scale has nine items, one for each of the Thomas and Chess (1977) temperament constructs (activity, rhythmicity, approach /withdrawal, adaptability, responsiveness, intensity of reaction, quality of mood, distractability, attention span/persistence). The ACPU-ITQ was developed as a short assessment of temperament in the course of the the global assessments conducted by the Academic Child Psychiatry Unit. Each item is rated on a 7-pt Likert Scale. The item related to Thomas and Chess' quality of mood was specifically used as a measure of negative affect, with higher scores indicating higher levels of negative affect. The ACPU-ITQ has reasonable psychometric properties, and has been

validated in three specific child populations; healthy control (N=35), children with

ADHD (N=50), and children with depressive disorders (N=30). Internal consistency

is moderate with Cronbach's alphas ranging from .71 to .87, and a one-week test-

retest reliability of .87. Concurrent validity was determined with Rothbart

temperament scales (Rothbart et al., 2001).

Hopkins Symptom Checklist (HSCL)

Parent psychopathology was measured using the Hopkins Symptom Checklist

(HSCL: Derogatis et al., 1974a; Derogatis et al., 1974b). The HSCL is a widely used

measure which comprises 58 items which are representative of common

symptomatology observed among outpatients, and measures general mental health

and or psychological distress. The HSCL measures current psychiatric symptoms on

a 4-point Likert scale from not at all to extremely, and assess how much a

problem/symptom has bothered them during the previous seven days. The HSCL was

initially developed to measure functioning across five domains (somatisation,

obsessive-compulsive, interpersonal sensitivity, depression, anxiety). However,

research suggests that the 58-item HSCL is best used to measure general distress

(Green, Walkey, Taylor, & McCormick, 1989), and thus the sum score of the HSCL

was used, as has been similarly done in other studies (e.g. Bollerslev et al., 2005;

Hser et al., 2006). Thus the range of scores was 58 to 232, with higher scores

indicating poorer mental health. The validity and reliability of the HSCL has been

consistently demonstrated (Green et al., 1989).

Family Assessment Device (FAD: Epstein et al., 1983)

Family functioning was measured using the Family Assessment Device (FAD:

Epstein et al., 1983). The FAD is a widely used 60 item scale assessing overall

health/pathology of the family environment across seven domains including

Communication, Problem Solving, Roles, Affective Responsiveness & Involvement,

Behavior Control, and General Functioning. These seven domains have been

validated in normal and clinical samples (e.g. Kabacoff, Miller, Bishop, Epstein, &

Keitner, 1990), though they have been shown through confirmatory factor analysis

(CFA) to fall into two main higher order factors, collaboration and commitment,

which can also be used as subscales (Ridenour et al., 1999). Each item is rated on a

4-point Likert Scale from strongly agree to strongly disagree. For the purposes of this

study, the General Functioning subscale of the FAD was used to provide an overall

assessment of family functioning. The General Functioning subscale, which contains

12 items, has been independently validated as a brief method of assessing overall

family functioning (Ridenour et al., 1999). General Functioning, evaluates the global

functioning of the family for all previously outlined domains (Epstein et al., 1983;

Stein et al., 2000).

The psychometric validity and reliability of the FAD have been demonstrated with

Cronbach's alphas on the scales in the initial development ranging from .72 to .92

(Epstein et al., 1983; Halvorsen, 1991), with the General Functioning subscale

showing $\alpha = .92$. Sound one-week test–retest reliability has been demonstrated with

ranges from .66 to .76 (Epstein et al., 1983; Miller, Epstein, & Bishop, 1985), and

correlations with social desirability measures are low (Prinstein et al., 2000).

Subsequent research reviewed in Epstein et al. (2003) and Miller et al. (2000) have

demonstrated equivalent reliability and validity. The clinical utility has been

documented in several studies, with Miller, Epstein, Bishop, and Keitner (1985) noting that high FAD scores (representing poorer functioning) were strongly related to clinician ratings of poor or unhealthy family functioning. Similar demonstrations of predictive validity regarding the ability to differentiate between clinical and non-clinical families has been demonstrated (Keitner, Miller, & Ryan, 1996; Tutty, 1995), and is outlined further in Epstein et al. (2003) and Miller et al. (2000). The FAD has shown cross-cultural applicability across a wide range of cultures (Herzer et al., 2010).

Procedure

The data collection procedures were the same as those in studies 1 and 2.

Data Analysis

The data was analysed using MPlus Version 6.1 (Muthen & Muthen, 2010). Using a multivariate latent regression technique the association between the risk factors and the common, internalising and externalising latent factors from the bifactor model tested in Study 1. Initial modelling of risk factors included demographic variables such as sex and age as covariates. However, in all cases these demographic variables did not show significant relationships with the bifactor model latent factors. Modelling without these covariates did not substantively alter the model, and thus in the models reported here, these demographics variables were excluded from the analysis. In the first analysis, the latent factors from the bifactor models for the CBCL Syndrome Scales were regressed onto the observed scores for three risk factors; negative affective temperament as measured by the ACPU-ITQ, parental psychopathology as measured by the sum of the HSCL, and family functioning as

measured by the FAD. The model tested for the CBCL Syndrome Scales is shown graphically in Figure 5.1.
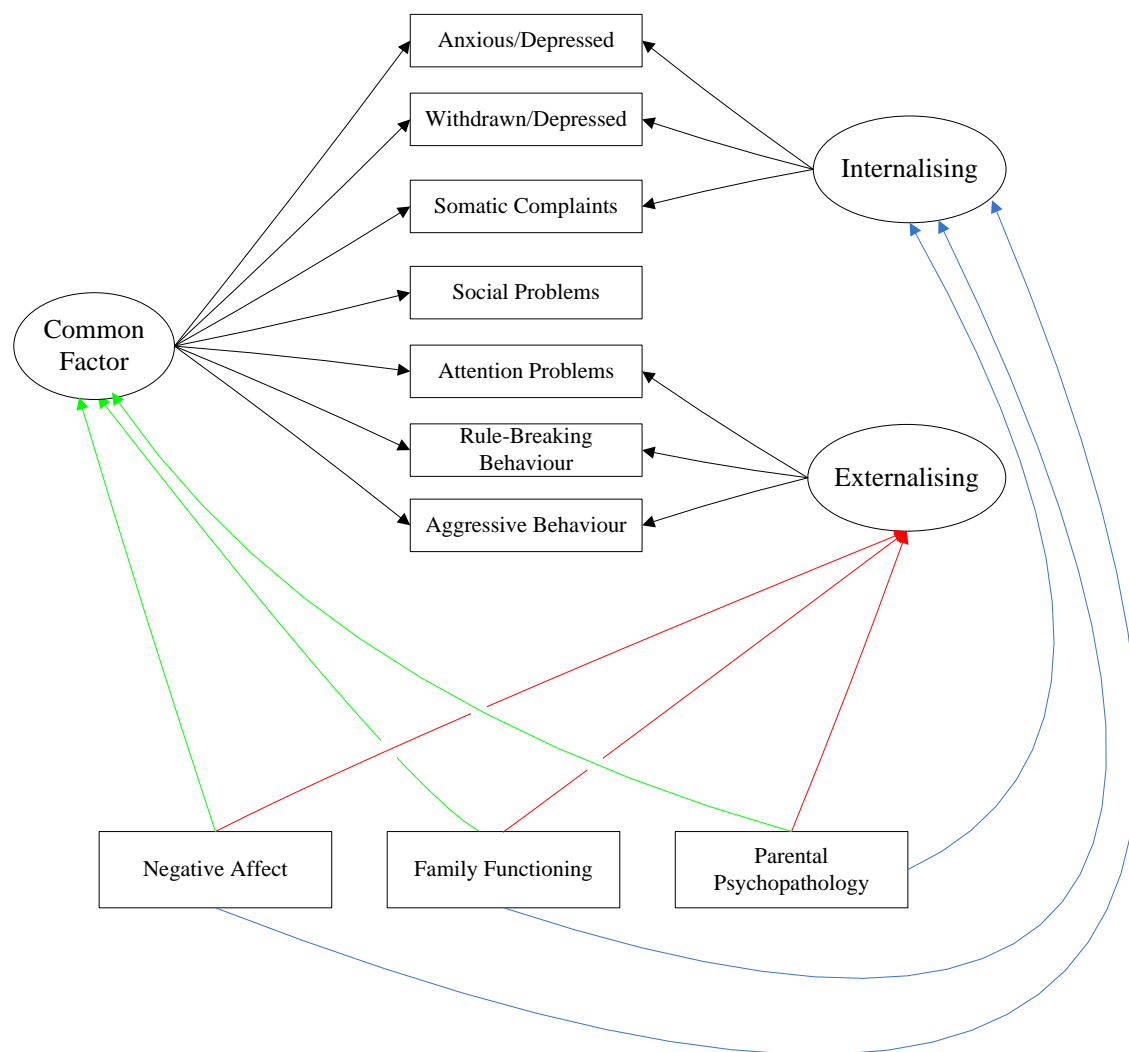
Figure 5.1. The tested path model of risk factor associations with the bifactor model for the CBCL Syndrome Scales. The red lines are regression paths from each risk factor to the externalising latent factor. The blue lines are regression paths from each risk factor to the internalising latent factor. The red lines are regression paths from each risk factor to the common factor.

In the second analysis, to assess the interactive effects of risk factors, the latent factors from the bifactor models for the CBCL Syndrome Scales were regressed onto the observed scores for the risk factors as well as every interactive combination of the risk factors, using MPlus syntax commands. This model is shown graphically in Figure 5.2. The same procedure was then repeated for the bifactor model of the ADIS-IV-P. The first analysis regressed the latent factors from the bifactor models onto the observed scores for three risk factors, as shown graphically in Figure 5.3. The second analysis assessed the interactive effects of risk factors, by regressing the bifactor model latent factors onto the observed scores for the risk factors as well as every interactive combination of the risk factors as shown graphically in Figure 5.4.

The method of estimation for the CBCL Syndrome Scales was the robust maximum likelihood (MLM), while the method of estimation for the ADIS-IV-Parent was the robust weighted least squares (WLSMV) method of estimation for the reasons previously outlined in Study 1. While the χ^2 likelihood ratio test statistic, root mean squared error of approximation (RMSEA), the standard root mean square residual (SRMR), the comparative fit index (CFI), and the Tucker-Lewis Index (TLI) are reported, the nature of model fit was not important to the aims of this study. Rather for this study, the key components of the analyses were the regression paths between the risk factors (and risk factor interactions), and the internalising, externalising and common latent factors that underlie the base bifactor model. Thus it is the significance or otherwise of these paths that is the focus of reporting. However the assessments of fit made use the criteria outlined in Chapter 3.
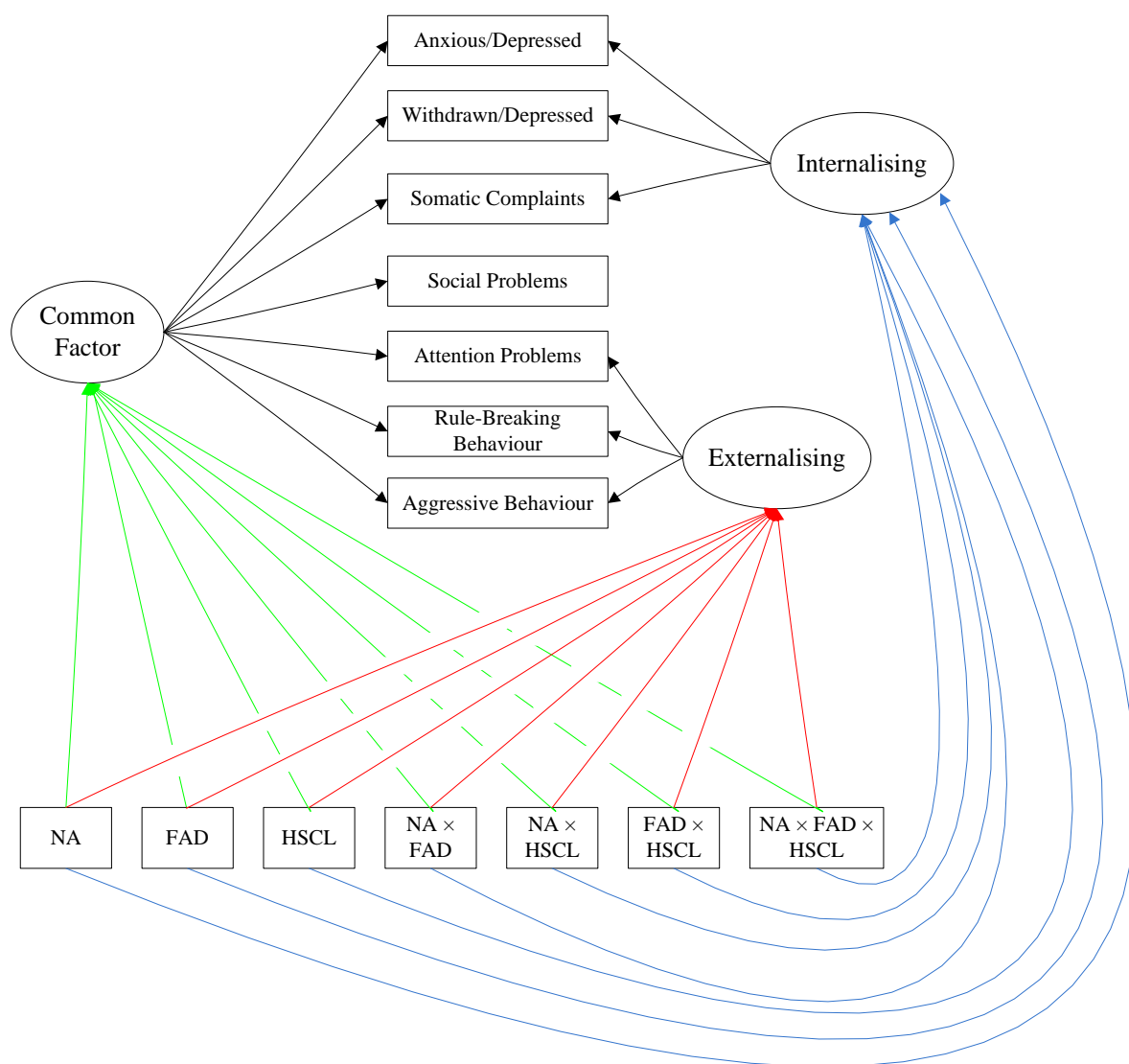
Figure 5.2. The second tested model for the CBCL Syndrome Scales involving risk factors and interaction terms between risk factors. The red lines are regression paths from each risk factor to the externalising latent factor. The blue lines are regression paths from each risk factor to the internalising latent factor. The red lines are regression paths from each risk factor to the common factor. NA refers to negative affect, FAD refers to family functioning, and HSCL refers to parental psychopathology as measured by the Hopkins Symptom Checklist.
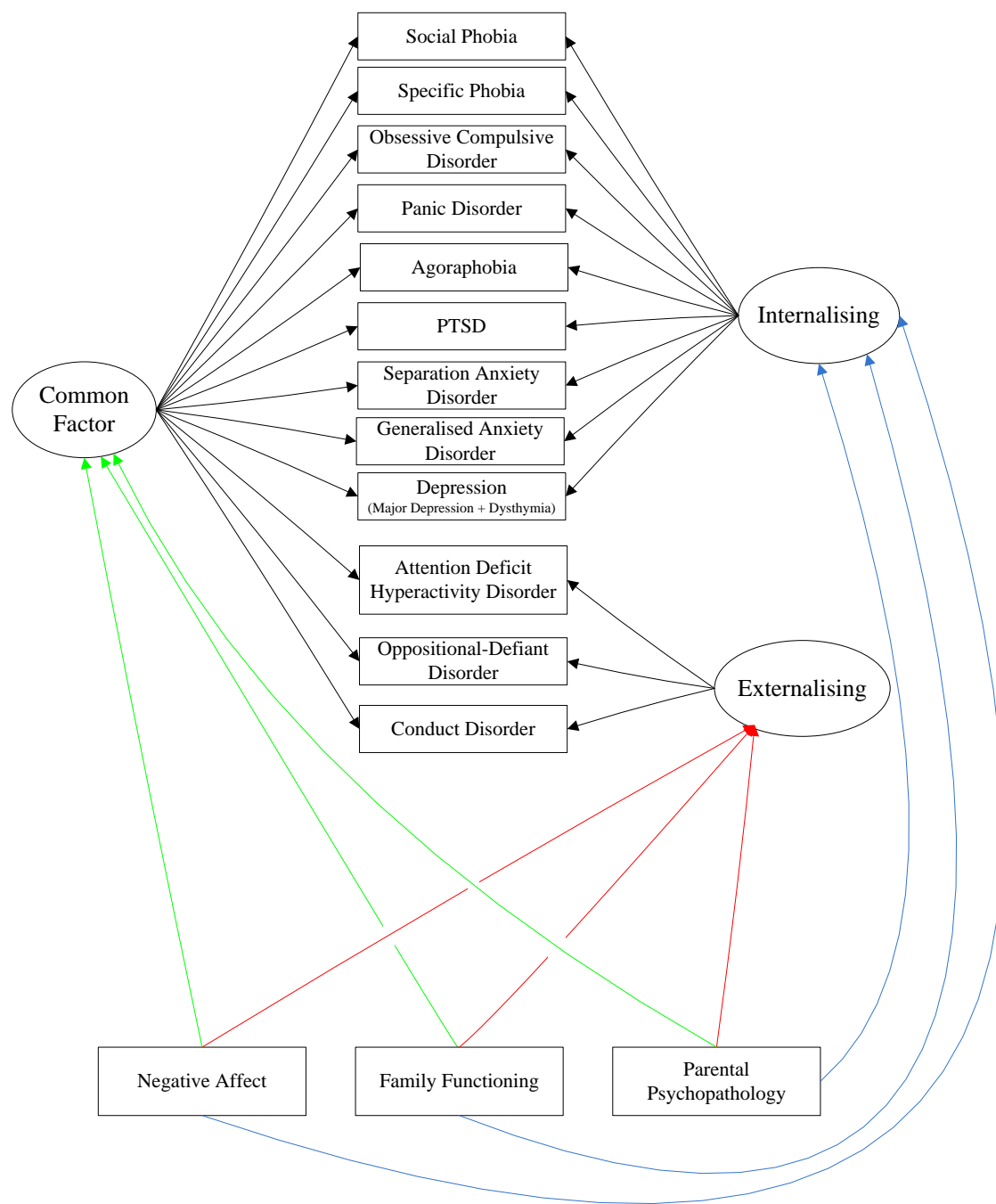
Figure 5.3. The first tested path model of risk factor associations with the bifactor model for the ADIS-IV-P Scales. The red lines are regression paths from each risk factor to the externalising latent factor. The blue lines are regression paths from each risk factor to the internalising latent factor. The red lines are regression paths from each risk factor to the common factor.
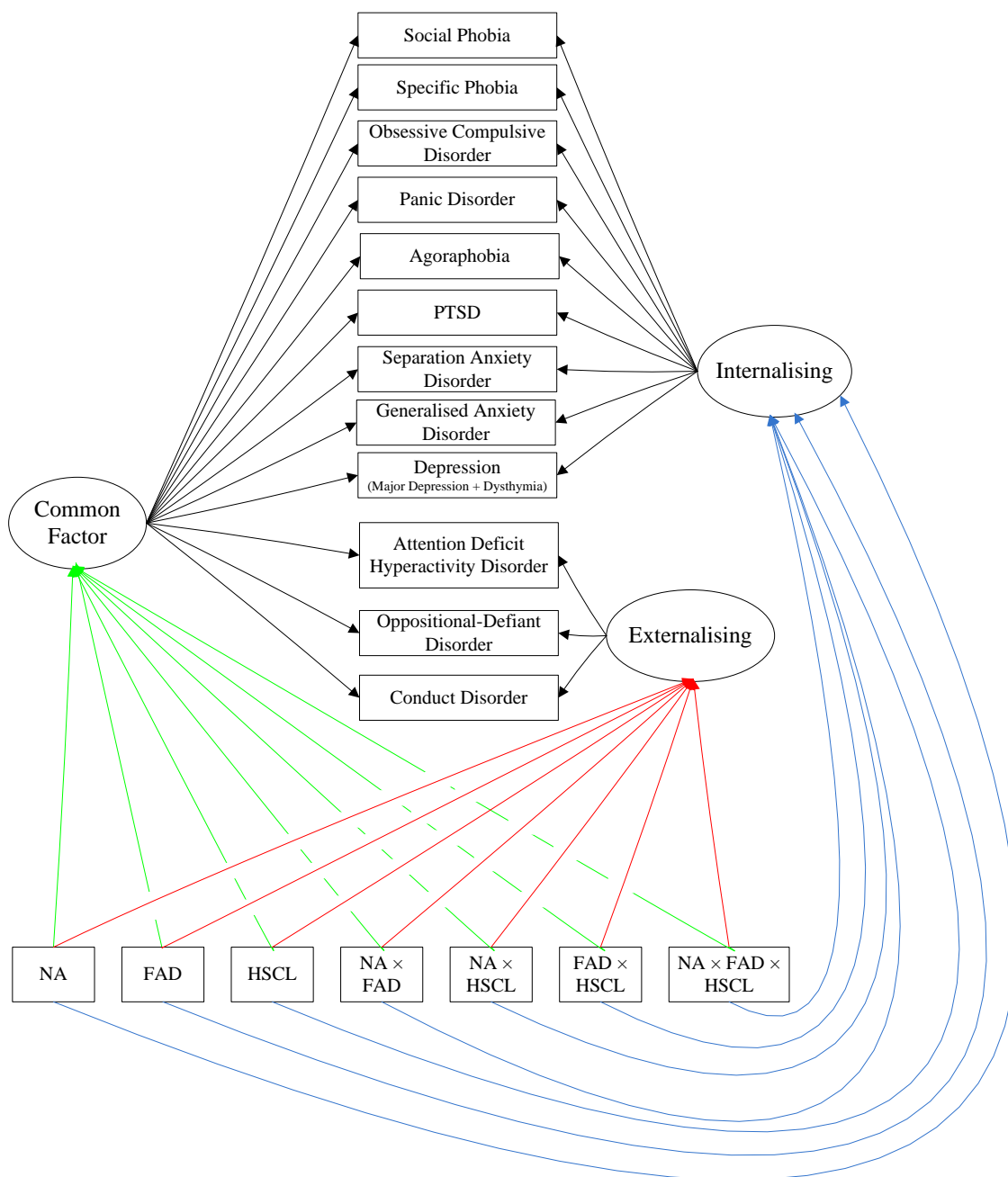
Figure 5.4. The second tested model for the ADIS-IV-P Scales involving risk factors and interaction terms between risk factors. The red lines are regression paths from each risk factor to the externalising latent factor. The blue lines are regression paths from each risk factor to the internalising latent factor. The red lines are regression paths from each risk factor to the common factor. NA refers to negative affect, FAD refers to family functioning, and HSCL refers to parental psychopathology as measured by the Hopkins Symptom Checklist.

Results

Results for the CBCL Syndrome Scales

Risk Factors Alone

The first model which was tested was the model shown in Figure 5.1, with the latent factors from the bifactor model for the CBCL Syndrome Scales regressed onto the observed scores for the three risk factors. The goodness-of-fit values for this model was χ^2 (20, N= 974) = 112.51, $p < .0001$; CFI = .967; TLI = .935; RMSEA = .072; SRMR = .031. Though assessing fit is not the key aspect of this analysis, the model showed good to acceptable fit. Table 5.1 shows the unstandardised and standardised regression path weights (STDYX) of each risk factor on the internalising, externalising and common latent factors.

As can be seen in Table 5.1, negative affect showed significant paths to all latent factors, as did the HSCL sum as a measure of parental psychopathology. The measure of family functioning, loaded significantly only on the externalising behaviour latent factor.

Table 5.1

Standardised and Unstandardised Regression Path Weights (STDYX) for Each Risk Factor on the Internalising, Externalising and Common Latent Factors from the Bifactor Model of the CBCL Syndrome Scale.

Risk Factor	b	Std. Error	β
Association with Internalising			
NA	1.09*	0.45	.10
FAD	-0.03	0.06	-.02
HSCL	0.09*	0.01	.34
Association with Externalising			
NA	1.07*	0.38	.10
FAD	0.26*	0.05	.20
HSCL	0.03*	0.01	.13
Association with Common Factor			
NA	0.43*	0.17	.09
FAD	-0.01	0.02	-.02
HSCL	0.04*	0.01	.29

Note: * p<.05. b = standardised coefficient. β = standardised/Beta coefficients. NA refers to negative affect. FAD refers to family functioning. HSCL refers to parental psychopathology.

Risk Factors and Interactions

The next model tested, shown in Figure 5.2, regressed the latent factors from the bifactor model for the CBCL Syndrome Scales onto the observed scores for the three risk factors, and specified interaction terms between the risk factors. The goodness-of-fit values for this model was χ^2 (20, N= 974) = 112.51, $p < .0001$; CFI = .967; TLI = .935; RMSEA = .072; SRMR = .031. Though assessing fit is not a key aspect of this analysis, the model showed good fit. Table 5.2 shows the unstandardised and standardised regression path weights (STDYX) of each risk factor and risk factor interaction on the internalising, externalising and common latent factors.

Table 5.2

Standardised and Unstandardised Regression Path Weights (STDYX) for Each Risk Factor and Risk Factor Interactions, on the Internalising, Externalising and Common Latent Factors from the Bifactor Model of the CBCL Syndrome Scale.

Risk Factor	b	Std. Error	β
Association with Internalising			
NA	1.14*	0.47	.11
FAD	-0.02	0.06	-.02
HSCL	0.09*	0.01	.35
NA×FAD	0.64	0.10	.03
NA×HSCL	-0.001	0.02	-.002
FAD×HSCL	-0.002	0.002	-.04
NA×HSCL×FAD	-0.001	0.003	-.02
Association with Externalising			
NA	0.85*	0.39	.08
FAD	0.25*	0.05	.19
HSCL	0.03*	0.01	.11
NA×FAD	0.01	0.07	.01
NA×HSCL	-0.01	0.001	-.04
FAD×HSCL	0.003*	0.001	.07
NA×HSCL×FAD	0.01*	0.002	.08
Association with Common Factor			
NA	0.49*	0.18	.10
FAD	-0.10	0.02	-.01
HSCL	0.03*	0.01	.31
NA×FAD	-0.01	0.04	-.07
NA×HSCL	-0.001	0.01	-.01
FAD×HSCL	-0.001	0.001	-.02
NA×HSCL×FAD	-0.001	0.001	-.02

Note: * p<.05. b = standardised coefficient. β = standardised/Beta coefficients. NA refers to negative affect. FAD refers to family functioning. HSCL refers to parental psychopathology.

As can be seen in Table 5.2, negative affect showed significant paths to all latent factor, as did the HSCL sum as a measure of parental psychopathology. The measure of family functioning, loaded significantly only on the externalising behaviour latent factor. In terms of interactions, no risk factor interactions loaded on the common or internalising factors, but the interaction of parental psychopathology and family functioning, and the three-way interaction of all risk factors, showed significant paths to the externalising factor.

Results for the ADIS-IV-P

Risk Factors Alone

The next model which was tested was the model shown in Figure 5.3, with the latent factors from the bifactor model for the ADIS-IV-P regressed onto the observed scores for the three risk factors. The goodness-of-fit values for this model was χ^2 (54, N= 974) = 103.93, p < .0001; CFI = .961; TLI = .954; RMSEA = .032. Though assessing fit is not the key aspect of this analysis, the model showed good to acceptable fit. Table 5.3 shows the unstandardised and standardised regression path weights (STDYX) of each risk factor on the internalising, externalising and common latent factors. As can be seen in Table 5.3, negative affect showed significant paths to externalising and common factor, with the HSCL score showing a significant path to the externalising factor. The measure of family functioning only loaded significantly on the externalising behaviour latent factor, and negatively on the internalising factor.

Table 5.3

Standardised and Unstandardised Regression Path Weights (STDYX) for Each Risk

Factor on the Internalising, Externalising and Common Latent Factors from the

Bifactor Model of the ADIS-IV-P.

Risk Factor	b	Std. Error	β
Association with Internalising			
NA	0.01	0.04	.02
FAD	0.02*	0.01	-.21
HSCL	0.002	0.002	.15
Association with Externalising			
NA	0.13*	0.06	.10
FAD	0.03*	0.01	.18
HSCL	0.003*	0.001	.11
Association with Common Factor			
NA	0.09*	0.04	.13
FAD	0.01	0.01	-.08
HSCL	0.003	0.002	.18

Note: * p<.05. b = standardised coefficient. β = standardised/Beta
coefficients. NA refers to negative affect. FAD refers to family
functioning. HSCL refers to parental psychopathology.

Risk Factors and Interactions

The final model tested, shown in Figure 5.4, regressed the latent factors from the

bifactor model for the CBCL Syndrome Scales onto the observed scores for the three

risk factors, and specified interaction terms between the risk factors. The goodness-

of-fit values for this model was χ^2 (20, N= 974) = 112.51, $p < .0001$; CFI = .967; TLI

= .935; RMSEA = .072; SRMR = .031. Though assessing fit is not key to the

analysis, the model showed good fit. Table 5.4 shows the unstandardised and

standardised regression path weights (STDYX) of each risk factor and risk factor

interaction on the internalising, externalising and common latent factors.

Table 5.4

Standardised and Unstandardised Regression Path Weights (STDYX) for Each Risk

Factor and Risk Factor Interactions, on the Internalising, Externalising and

Common Latent Factors from the Bifactor Model of the ADIS-IV-P.

Risk Factor	b	Std. Error	β
Association with Internalising			
NA	0.08*	0.04	.10
FAD	-0.01	0.01	-.08
HSCL	0.01*	0.001	.24
NA×FAD	-0.001	0.009	-.003
NA×HSCL	0.001	0.001	.01
FAD×HSCL	-0.001	0.001	-.09
NA×HSCL×FAD	0.001	0.001	.16
Association with Externalising			
NA	0.01	0.05	.02
FAD	-0.01	0.01	-.13
HSCL	0.003	0.002	.18
NA×FAD	-0.02	0.02	-.20
NA×HSCL	0.004	0.004	.23
FAD×HSCL	-0.001	0.001	-.06
NA×HSCL×FAD	-0.002	0.02	-.54
Association with Common Factor			
NA	0.05	0.02	.12
FAD	0.01*	0.004	.26
HSCL	0.001	0.001	.05
NA×FAD	0.001	0.006	.01
NA×HSCL	-0.001	0.001	-.11
FAD×HSCL	0.001	0.001	.06
NA×HSCL×FAD	0.001	0.004	.27

Note: * $p<.05$. b = standardised coefficient. β = standardised/Beta
coefficients. NA refers to negative affect. FAD refers to family
functioning. HSCL refers to parental psychopathology.

As can be seen in Table 5.4, negative affect showed significant paths to the internalising factor, as did the HSCL sum as a measure of parental psychopathology. No risk factor showed a significant path with the externalising factor, and only family functioning had a significant path with the common factor. In terms of interactions, no risk factor interactions loaded significantly on any latent factor.

Discussion

The aim of this study is to assess the way negative affect, parental psychopathology and family functioning, and interactions between these variables, are associated with the general and specific factors of the bifactor model of psychopathology, as indexed in the CBCL Syndrome Scales, and the disorders of the ADIS-IV-P. Were such associations to be noted, then it could be inferred that the likelihood is that the common factor, at least in part, is a liability factor, or a psychopathological liability factor, as proposed within the models of Klein and Riso (1993) and Neale and Kendler (1995). It was generally predicted that negative affect would universally associate with the latent variables within the bifactor model. The results generally supported this prediction for the CBCL Syndrome Scale based bifactor model of psychopathology, with significant associations with parental psychopathology also found. However, results are not as clear for the ADIS-IV-P.

Results for the CBCL Scales

For the CBCL Syndrome Scales, when assessing the individual contribution of risk factors (without interaction terms), negative affect and parental psychopathology measures showed significant regression paths both to the common factor, but also to the internalising and externalising factors of the bifactor model. This is consistent

with previous research outlined in Chapter 2, which has shown that high negative affect is associated with both internalising and externalising behaviours, as well as with comorbidity. Similarly, parental psychopathology, as a genetic and environmental risk factor, is associated with an increased risk of a broad range of psychopathology and comorbidity. Familial functioning was only found to be associated with externalising behaviour within the model. This certainly matches literature showing that poor family functioning is associated with the externalising behaviours, especially ADHD. However, the failure to find an association with internalising disorders is somewhat unexpected. This may in part be due to the fact that the Family Assessment Device General Functioning Scale, whilst well validated as a measure, is a broadband measure of family functioning. Research investigating poor family functioning, as outlined in Chapter 2, has been both broad- and narrowband, and it may be more useful to consider family functioning in a narrowband manner in future, looking factors such as parent-child communication, parent-child or parent-parent conflict. These factors are indicative of poor family functioning, but may not be picked up adequately by the general scale of the FAD. Individual measures of these were not available within the context of the study. Alternatively, family functioning may simply not be a reliable risk predictor of the general factor within the context of the bifactor model of psychopathology. It may be that family functioning may be a more accurate predictor of child psychopathology on the individual disorder level, rather than the broader dimensional level. This explanation is plausible, given no link was found between family functioning and the general liability factor. However, the failure to find associations between the FAD and the latent factors may be due to the variance it shares with parental psychopathology. As noted in Chapter 2, the interactive effect between family

functioning and parental psychopathology has long been noted, with family functioning often worse in families where a parent has psychopathology (McMahon & Wells, 1998). As a result, measures of family functioning and parental psychopathology may share considerable overlap in variance, leading to only one showing significant associations when both are simultaneously examined.

When interactive associations were considered with the CBCL Syndrome Scales, the only additional significant associations found were interactions between family functioning and parental psychopathology, and a three way interaction (family functioning and parental psychopathology, negative affect) showing associations with the externalising latent factor. Again these interactions are not without support in the literature, but more interactive effects, especially with the common factor, might have been expected. However, again the broadband nature of both the FAD and the HSCL (the total sum was used) may mean that there is a slight lack of specificity which may prevent the finding of interactive effects. Nonetheless, negative affect and parental psychopathology measures still showed significant regression paths to the common factor indicative of their relative importance to the common factor. Similarly the results do demonstrate that risk factors do not operate in isolation, and as such multivariate modelling of risk factors is imperative.

Results for the ADIS-IV-P

For the ADIS-IV-P, when assessing the individual contribution of risk factors, the results were very mixed, in part because of the paucity of associations detected. Negative affect showed significant associations with the externalising and common latent factors. However, in a finding definitely not predicted, negative affect showed

no association with the internalising factor. Parental psychopathology showed a significant regression path only with externalising behaviours, and family functioning with internalising and externalising latent factors but, as for the CBCL, not the general liability factor. When interactive associations were considered, the results showed substantial changes. No interactions were found to associate with any of the latent factors. However, negative affect and parental psychopathology now only showed significant regression paths with the internalising latent factor, and family functioning only showed association with the common factor. These results are, on the surface, somewhat confusing, and could indicate a lack of utility for the model, at least in the context of the ADIS-IV-P. However it is the variability between the two analyses, combined with the nature of the measurement tools used, that gives some insight into the results.

Categorical Measurement of ADIS-IV-P versus Dimensional Measurement of Risk
The ADIS-IV-P is a categorical diagnostic tool which does not take into account degrees of severity, unlike the CBCL Syndrome Scales which are dimensional, and take into account degrees of severity (Edelbrock & Costello, 1988; Gould et al., 1993; Kamphaus et al., 2006). That is, the ADIS-IV-P only indicates whether a diagnosis is present or absent; not how 'severe' any impairment is. In contrast, while two individuals may be within the 'Clinical' range on the CBCL Syndrome Scales, as determined by the T-score of over 70, the nature of the scale allows an idea of how severe the problems are within the 'clinical range'. For example, a child with an individual CBCL Syndrome Scale T-score of 70 can be usually regarded as less impaired than a child with a T-score of 90 on the same scale. Risk factors, by their very nature are not categorically present or absent; they are present to degrees of

severity (Kazdin et al., 1997). The degree or severity is considered to be directly related to risk, with the greater the degree/severity, the greater the risk. All the scales used to measure the risk factors in this study (FAD, HSCL, ACPU-ITQ), and indeed most other studies investigating risk factors, use scales to measure risk factors that are dimensional in nature. Thus there is a mismatch between the nature of the risk factor measurement and the measurement of psychopathology within the ADIS-IV-P which may undermine attempts to link the two systems, and hence may explain the somewhat unusual results for the ADIS-IV-P.

Rutter and Sroufe (2000) argue that because categorical classification does not account for marginally functional behaviour phenomena, such as the transition between normality and psychopathology, the variables that affect these transitions (i.e. the risk factors) cannot be easily investigated in the context of categorical systems. Thus the scarcity of associations between risk factors and the bifactor model latent factors for the ADIS-IV-P may be a function of the measurement mismatch, rather than a lack of utility of the model per se.

Evidence for the Common Factor Being a Liability Factor

Nonetheless, this study, especially in light of the results relating to the CBCL syndrome scales, has demonstrated that the risk factors that would be expected to form a liability factor do show associations with the common factor in the bifactor model of psychopathology. Given such associations, there is tentative, though not definitive evidence that the common factor, at least in part, is a liability factor, or a psychopathological liability factor, as proposed within the models of Klein and Riso (1993) and Neale and Kendler (1995). Similarly, given that negative affect and

parental psychopathology show substantial associations with both the general liability factor and with the internalising and externalising dimensions, the results implicate negative affect and parental psychopathology as two important risk factors in the aetiology, development, and maintenance of a broad spectrum of psychopathology, but also more crucially, as key risk factors to the development of *multivariate comorbidity*.

The proposed *psychopathological liability* underpinning the bifactor model as outlined in previous chapters, suggests person A manifests disorder X while person B manifests disorder Y because of chance or differences in their combination of genetic and environmental risk factors. Linking the results of this study to this theoretical model, it could be argued that the presence of high levels of negative affect and parental psychopathology, may lead to the development of psychopathology, with the differences in the degree of severity of these two factors, in combination with family functioning (and potentially other factors which may not have been assessed in this study) leading to differing outcomes in terms of specific disorders that may be manifested. However, such conclusions are tentative, and will need further replication.

Limitations

It should be noted that there are a plethora of possible risk factors that may impact on either the general or specific factors of the bifactor model. The present study only examined the risk factors of parental psychopathology, negative affect and familial functioning, as these factors are among the most highly implicated in the development of psychopathology. Ideally a wider range of risk factors would be

investigated in future research. In this study, the limitation in the number of risk factors assessed was in part a product of the information that is collected by the source agency, the Academic Child Psychiatry Unit. However, as outlined in Chapter 2, these three psychological constructs cover the domains of risk identified by Crawford et al. (2011) - child traits and behaviours, maternal (parental) traits and behaviours, and environmental risk - and should be considered a reasonable cross-section of potential risk factors.

Summary and Conclusion

In conclusion, this study, especially in light of the results relating to the CBCL syndrome scales, has demonstrated that the risk factors of negative affect and parental psychopathology appear to be key risk factors within the bifactor model of psychopathology, and thus appear to be major risk factors in the development of comorbidity. The fact that there is broad linkage with all latent factors of the bifactor model suggest that these risk factors may be key in the aetiology, development, and maintenance, of the broad spectrum of psychopathology and comorbidity. Importantly the associations of risk factors with the common factor of the bifactor model, suggest that this common factor may be a psychopathological liability factor. The implications of this study will be further expanded in the next chapter, which will provide a general discussion of the three studies in this thesis, and outline the implications of this study within the context of the results of Studies 1 and 2.

Chapter 6 - General Discussion

The overall aim of this thesis was to propose a general factor to capture the common variance for the major internalizing and externalizing childhood syndromes and disorders. In this context, through three empirical studies, this thesis evaluated the plausibility of such a common factor in two studies, and potential risk factors that may be associated with such a common factor in the third study. This chapter will present a summary of the results of the three studies presented in Chapters 3, 4 and 5, followed by a general discussion, including the implications of the thesis findings. Also covered are the limitations of the thesis findings and suggestions for future research.

Summary of Results

Study 1

The first study examined if the broad spectrum of childhood psychopathology could be characterised by a bifactor model. The bifactor model allowed for the modelling of a common factor as implied by the common liability models, but also allowed for consideration of domain specific factors that are necessary to consider in the context of multivariate psychopathology; in this case the well validated internalising and externalising domains of child psychopathology. The bifactor model was tested from clinical data from 974 parents and their children referred to the Royal Children's Hospital, Melbourne. Data from three measures was used: the Syndrome and DSM-Oriented scales from the Child Behavior Checklist (CBCL), and DSM diagnoses derived from the Anxiety Disorders Interview Schedule-Parent Version (ADIS-IV-P). The model was also tested on the Achenbach System of Empirically Based

Assessment (ASEBA: Achenbach & Rescorla, 2001) Reference Group data, which is the epidemiological sample used to validate the CBCL. The results for the clinical sample showed good to excellent fit for all three measures, with significant loadings on the general factor for all indicators on all scales, and salient loadings (>.32; Gorsuch, 1983) on the general factor for the entire CBCL Syndrome and DSM-Oriented Scales. For the ADIS-IV-P, three indicators were below the Gorsuch cut-off for loading salience, though only the ADHD diagnosis was substantially below this, and all indicators still loaded significantly. For the ASEBA Reference Group sample there was excellent fit for the Syndrome Scales, though there was only marginally acceptable fit for the DSM-Oriented Scales.

Study 2

Study 2 extended Study 1 and investigated whether the major childhood syndromes/disorders conform to a circumplex structure, which would support the presence of a common factor underlying these syndromes/disorders. The circumplex was tested using the same data and same measures as Study 1. Results for the clinic-referred sample demonstrated that child psychopathology conformed to a circumplex structure for the clinical sample for all measures, with between 57 and 66 percent of variance accounted for by the common factor. For the ASEBA reference group, the circumplex structure fit well for both the Syndrome scales and the DSM-Oriented scales, with 71 and 77 percent of variance accounted for by the common factor respectively. This provided strong support for the presence of the hypothesised common factor.

Study 3

Study 3 aimed to investigate the nature of this hypothesised common factor, and investigate whether three constructs – negative affective temperament, parental psychopathology and familial functioning – may be associated with this common factor. If the common factor showed association with these risk factors, thought to be key in the development of multivariate comorbidity, then it could be inferred that the likelihood is that the common factor is a psychopathological liability factor as proposed by Klein and Riso (1993) and Neale and Kendler (1995). Thus the association of those three constructs with the general and domain-specific risk factors of the bifactor model was investigated. The results for the CBCL Syndrome Scales demonstrated that negative affect and parental psychopathology, both individually and in interaction, had significant relationships with this common factor. The results for the ADIS-IV-P were very mixed, in part because of the paucity of associations detected. However, the lack of associations may be a function of measurement mismatches between the categorical nature of measurement underpinning the ADIS-IV and the dimensional measurement of risk factors. This is because categorical classification as per the ADIS-IV does not account for the transition between the normal and the psychopathological, and as such it is difficult to investigate the way in which risk factors, which are dimensional in nature, affect this transition (Rutter & Sroufe, 2000).

Implications of the Results of the Thesis Studies

Evidence for a Common Factor Underlying Psychopathology

Taken together, the findings for Studies 1 and 2 indicate that there is clear support for a common factor that underlies the broad range of syndromes/diagnostic

categories that comprise childhood psychopathology. This support is irrespective of

the classification systems used, as the results were substantively identical for both an

empirically validated dimensional system, based on the ASEBA scales (Achenbach

& Rescorla, 2001), and a DSM-based categorical system, based on diagnoses from

the ADIS-IV-P (Silverman & Albano, 1996). Previous research investigating models

of a common liability factor, such as the 'Alternate Forms' model (Klein & Riso,

1993; Neale & Kendler, 1995), have not shown support for a common factor,

generally finding them to be poorly fitting (e.g. Neale & Kendler, 1995; Rhee et al.,

2008). The question then arises; why do the results of this study show support for a

common factor, when previous studies have not shown such support? There are two

plausible explanations for these differences.

The first potential explanation is that the studies in this thesis tested *multivariate*

models of comorbidity rather than the bivariate models. Evidence presented

throughout this thesis indicates comorbidity is a multivariate phenomenon (see

Angold et al., 1999), and it is therefore reasonable to expect that any theory

addressing the genesis/aetiology of comorbidity should be applicable when assessed

multivariately. Any model proposing a common factor/liability to explain

multivariate comorbidity may not be truly testable in the bivariate domain (Krueger

& Markon, 2006). This is because within a bivariate domain, any model would

therefore potentially ignore a wide range of potential covariates (i.e. the other

disorders), and thus not show appropriate fit. Thus the poor fit in previous research

into common factor models, may be an artefact of the fact it was bivariate in nature,

and a resultant inability to accurately assess the phenomenon. The second

explanation for the difference between this research and past research on common

factor models derives directly from the first explanation. Because Studies 1 and 2 examined psychopathology and comorbidity from a multivariate perspective, the studies presented in this thesis were able to present sophisticated and novel methods/models (i.e. the bifactor models and circumplex) to investigate comorbidity; a direct contrast to prior proposed models, and a direction advocated by Pesenti-Gritti et al. (2008) amongst others. The common factor modelled by the circumplex and bifactor models is not the same as hypothesised in the 'Alternate Forms' model of Neale and Kendler (1995). For example, the bifactor model results from Study 1 show that there exists both a common or general factor, as well as domain specific factors underlying the development of psychopathology. This in fact means that the models presented in this thesis are quite different to the standard common factor models, and may be considered in some way as hybridised versions of the common factor models like the 'Alternate Forms' and 'Correlated Risks' models (Klein & Riso, 1993; Neale & Kendler, 1995) which were outlined in Chapter 1. Hence support for a common factor in this study, in contrast to previous studies, is likely to be directly attributable to the uniqueness of the model(s) and conceptualisations of a common factor that have been presented in this thesis.

The Common Factor As A 'Psychopathological Liability'

Given statistical support for a common factor in the context of a multivariate analysis, using multiple methodologies, it is conceivable that this common factor is in fact a psychopathological liability similar to that conceptualised in the 'Alternate Forms' model of Neale and Kendler (1995) and based on the work of Klein and Riso (1993). Such a conclusion is supported by the results of Study 3, which showed that the common factor had significant associations with risk factors and risk factor

interactions thought to be key in the development of multivariate comorbidity. Specifically the common factor showed significant associations with negative affect and parental psychopathology, both individually and in interaction. The fact that the common factor showed association with risk factors allows some inference that the common factor is either a factorial combination of risk factors, and hence a psychopathological liability, or at least is in some way associated with a liability factor. While such an inference is speculative, the logic is supported by the results, and is consistent with the structure of proposed common factor models (Lilienfeld, 2003). Nonetheless, given the support for a common factor which is potentially a psychopathological liability, there are resultant significant implications for the understanding of childhood psychopathology and comorbidity, in theory and aetiology, nosology, and indeed clinical practice. These implications will be outlined in the subsequent sections.

Implications of a Common Factor for Psychological Theory and Clinical Practice

Implications of a Common Factor for Nosology

A common factor or psychopathological liability has crucial implications for classifications systems within psychology, and suggests that some evolution in psychopathological nosology is warranted. As explained in Chapter 1, and outlined throughout the thesis, psychopathology has been conceptualised in terms of putatively distinct categories, and clinical psychological nosology is dominated by putatively categorical systems; most notably the American Psychiatric Association's Diagnostic and Statistical Manual, now in its text-revised fourth edition (DSM-IV-TR: American Psychiatric Association, 2000), and the World Health Organisation's International Classification of Diseases, now in its tenth revision (ICD-10: World

Health Organization, 1993). Both the DSM and ICD are fundamentally categorical systems, which allow only indication of the presence or absence of the disorder, and do not allow consideration of the severity of a disorder, or the number of symptoms met (Ferdinand et al., 2004).

While a common liability factor may lend itself to arguments around minimal thresholds for psychopathology (Rhee et al., 2008), it does not lend itself to a purely categorical diagnostic system. This is because risk factors, by their very nature are not categorically present or absent; they are present to degrees of severity (Kazdin et al., 1997), with greater the degree of severity, the greater the risk. Indeed the mismatch between categorical diagnostic systems and the dimensionality of risk factors was raised in Study 3, with mismatches potentially creating the problematic results for the categorical ADIS-IV-P. A mismatch between aetiological phenomena and their outcome in terms of nosology is thus problematic.

There have been attempts within the DSM to introduce elements of dimensional approaches toward the release of DSM-5 (American Psychiatric Association, 2013) in mid-2013. However, such dimensional approaches have mainly been considered for inclusion within adult disorders, and even then, mostly within the Axis II Personality Disorders as a measure of the degree to which a patient matches an archetype (Skodol et al., 2011). Other dimensionality may be included before the final release of DSM-5, though it is unlikely to be widely used. It is also unclear whether the eleventh revision of the World Health Organisation's International Classification of Diseases (ICD-11), due to be released in 2015, will also begin to embrace integrative dimensional models, though the initial revision guidelines are

suggestive of this (World Health Organization, 2013). There have long been arguments for adding elements of dimensionality within the DSM system, and the argument for such has been especially true for childhood psychopathology (Rutter, 2003). The argument in part is to account for the multiplicity in sources of variance for children, such as gender, informant and age-related differences (Hudziak et al., 1998). The results of this thesis would suggest that such approaches are warranted, and the move towards greater dimensionality within the DSM as a whole, appropriate.

Given the support in the empirical studies of this thesis for a common underlying factor, perhaps a further conceptual change in the way diagnosis of child disorders is conceptualised may be worthy of consideration. While adding dimensional elements to the extant diagnostic categories is a modest proposal (Brown & Barlow, 2005), there have also been arguments for the inclusion of higher-order dimensions in the nosology (Brown & Barlow, 2009). Such higher-order models, sometimes referred to as meta-structures, are argued to easily allow an understanding of comorbidity as a natural function of shared underlying risk, and would emphasise the interplay between disorders and risks within an individual (Andrews et al., 2009; Krueger & Markon, 2011). The results of this thesis certainly suggest that inclusion of higher-order dimensions such as internalising and externalising would be appropriate, and address the overlap that exists among the current DSM-IV disorders. Indeed this would make the diagnostic system more sensitive to potential secondary disorders, as per the arguments of Brown and Barlow (2009). However, in terms of higher order factors, based on the support for a general factor, an updated nosology could consider a general higher-order dimension of 'general psychopathological dysfunction' (or

similar). Based on the bifactor model support for the 'internalising' and 'externalising' domains, such a nosology may then consider specifiers for this global diagnostic category, such as 'predominately internalising', 'predominately externalising', and 'mixed' types. Such a system could then be supplemented by dimensional rating of specific behaviours that would be derived from the current specific diagnostic entities. A system structured like this would be a conceptual break from the DSMs current model, and of course would have to demonstrate clinical utility (see Kraemer, 2007 for a review on why such systems may be problematic). Indeed, too strong a focus on higher-order dimensions could lead to a nosological system becoming overly reductionistic (Brown & Barlow, 2009), and lead to a disregard of the substantial variability of psychopathology. However, it should be noted that such a system is not that far removed from systems currently used by clinicians in diagnosis and assessment when they use instruments such as the ASEBA scales (Achenbach & Rescorla, 2001), the Conners' (Conners, 2008a, 2008b), or the BASC (Reynolds & Kamphaus, 2005). While this theoretical debate needs to be considered further, along with further validation of the models presented in this thesis, there is a clear need to continue the theoretical debate regarding the structure and functioning of psychopathological nosology systems.

Implications of a Common Factor for Clinical Practice

There are also significant implications of a common factor for clinical practice. Achenbach (1995) raised relevant questions regarding the treatment of comorbid disorders. These questions relate to whether one disorder should be treated first, whether the comorbid disorders should be treated separately, or whether there should be a single treatment for both disorders. Based on the results of the studies in this

thesis, a common factor would imply that a single treatment for both disorders may be more efficacious, given a single liability distribution underlying the two disorders. Indeed such approaches are already being undertaken in the current push towards what is referred to as 'transdiagnostic' models of treatment.

Transdiagnostic models have their origins in research on treatment of the emotional (anxiety and mood) disorders (Barlow, Allen, & Choate, 2004). In effect it is based upon the notion that there are unifying principles to comorbid disorders, as evidenced by the comorbidity in itself (Moses & Barlow, 2006), that should be the principle focus of any treatment, rather than focussing on the individual disorder (Craske, 2012). Transdiagnostic models of treatment are becoming increasingly prominent (Nolen-Hoeksema & Watkins, 2011) because they are designed with the idea that many patients referred for therapy have comorbid presentations – a clinical reality as discussed in Chapter 1 – rather than the traditional view of therapy as disorder-specific (Mansell, Harvey, Watkins, & Shafran, 2009). These approaches de-emphasise differences among disorders (Brown & Barlow, 2009), and propose that understanding and treating psychological disorders is enhanced by focusing on common and unified factors across disorders (Mansell et al., 2009). Thus transdiagnostic therapies address core properties rather than narrowly construed disorder specific features (Barlow et al., 2004). While currently the focus of transdiagnostic treatment has been around emotional (mood and anxiety) disorders in adults (Brown & Barlow, 2009), recent efforts have been made to translate this approach to treatments for children/adolescents (Chu, 2012).

Support for a common factor as shown in this thesis thus provides substantive support for a transdiagnostic approach. This is because a common factor emphasises the commonality that exists among (comorbid) disorders. From that it follows logically that treatment/intervention efforts will almost certainly be more successful when successfully targeting those components that are relevant to all disorders within a presentation. While currently the focus of transdiagnostic treatment has been around emotional (mood and anxiety) disorders, efforts have been made to conceptualise how to translate this process into treatments for the broader range of psychopathology (Chu, 2012; Racer & Dishion, 2012); efforts which the results of this thesis would strongly support. Based on this thesis and the results of Study 3, treatment focussed specifically on negative affective temperament would be hypothesised to be important in such a transdiagnostic process. Such a suggestion is not entirely novel, because negative affect, and its related personality trait of neuroticism, has been argued to be the most crucial factor in mental health (Lahey, 2009). Similarly, the results of Study 3 may also suggest some treatment focus in treatment of children with psychopathology should actually focus on ensuring that the child's parents receive appropriate interventions for any psychopathology they are experiencing.

While it may seem daunting for a clinician to specify broad liability phenomena as the target for prevention rather than specific manifestations in the form of specific psychopathology, such fears are unwarranted (Krueger & Markon, 2011). Indeed it must be remembered that current evidence suggest that these liability phenomena are the building blocks of the manifestation of psychopathology (Kazdin et al., 1997), and may in fact be the true targets of the interventions as they currently stand (Nolen-

Hoeksema & Watkins, 2011). A change to a more global approach is thus not a major paradigm shift, but rather an acknowledgement of the clinical reality. Drawing from these implications of a common factor regarding treatment, there are resultant implications for assessment in clinical practice. Given that the bifactor model supports the presence of global and domain-specific factors, consideration in clinical practice must be given to these factors, in terms of screening for the potential presence of psychopathology. Knowledge of the factors common to all psychopathology (i.e. the risk factor components of the common factor/liability) means that specific questioning around these factors can be undertaken (Moses & Barlow, 2006). Indeed in clinical practice, the idea of domain-specific risk factors may be just as vital as knowledge about the global common risk factor. This is because the domain-specific risk factors may be able to be used as clinical markers in diagnostic decision making, whereas the presence of global risk factors only suggests the diagnostic possibility of psychopathology in general. Indeed Tackett (2006) acknowledged that in order to utilise information about risk factors to develop targeted clinical assessments and interventions, it will be necessary to understand which risks relate to psychopathology on a broad level as well as on a narrow level.

Implications of a Common Factor for Theory on Aetiology/Development
A common factor underlying all psychopathology in childhood can theoretically provide a clear explanation of the high, above chance levels of comorbidity between childhood disorders and syndromes, as well as how psychopathology may manifest differently. Indeed if this common factor is liability, as defined by Neale and Kendler (1995) then it provides a clear model for psychopathological development, in contrast to current conceptualisations. The results of the bifactor model presented in

Study 1, in conjunction with models of liability factors (Klein & Riso, 1993; Neale & Kendler, 1995), provides a basis for the explanation proposed here.

A common factor, if it is a common liability, suggests that all manifestations of childhood psychological illness may, at least to some degree, have some common geneses (Klein & Riso, 1993; Neale & Kendler, 1995; Rhee et al., 2004; Rhee et al., 2008). This common genesis would lend itself to the idea that multiple manifestations of psychopathology could occur, in part because the common liability is conceptualised to be multifactorial in nature. However, because the bifactor model suggests both global and domain-specific factors underlie psychopathology, as per the assumptions of the bifactor model (Chen et al., 2006), it is likely that different manifestations of psychopathology are because of differences in the combinations of global and domain-specific factors. Thus, while the development of psychopathology in general is dependent upon a common liability, the specificity of that psychopathology (i.e. which specific disorder/s or syndrome/s) would be an interactive effect between the global and domain specific risks present within the individual. Given the conceptualisation of the liability as being comprised of a multifactorial combination of risks, it may also be suggested that some risk factors which form part of this multifactorial combination could be global risk factors, but also domain-specific protective factors (Mesman & Koot, 2000), which would add some complexity to the model. Further testing is certainly needed to clarify the nature of the interplay, but such explanations are consistent with the arguments of Wolff and Ollendick (2006) among others who have clearly advocated for consideration of factors that may have specific aspects on single domains, rather than merely focussing on global risk factors.

The idea of a common genesis for all psychopathology would certainly mark a paradigm shift in understanding of the aetiology and development of psychopathology. However, such a paradigm shift is most probably necessary, if a comprehensive theoretical explanation for comorbidity is to be developed. Indeed, the evidence for the need for a paradigm shift may come from the fact that until recently, comorbidity was usually being investigated bivariately rather than multivariately, despite clear evidence for it being a multivariate concept (Angold et al., 1999; Lilienfeld, 2003). Thus the move towards a multivariate conceptualisation of comorbidity also necessitates consideration to changes in conceptualisations of the aetiology of psychopathology as well (Krueger & Markon, 2006; Krueger et al., 2005), and such a paradigm shift is eminently arguable.

A Need for a Multivariate Perspective of Comorbidity

Finally, this thesis has clearly demonstrated the need to consider comorbidity and risk factors from a multivariate perspective. While this has been emphasised throughout the thesis thus far, results do emphasise the need for a greater focus on considering comorbidity, and indeed risk factors, from multivariate perspectives. Bivariate modelling research can provide a more detailed understanding of individual fundamental elements of comorbidity and or risk factors (Krueger & Markon, 2006; Wolff & Ollendick, 2006) and thus still has an important place within the literature. However, on its own, bivariate research may lead to spurious conclusions, largely because of the failure to consider other potential covariates. Furthermore, as seen in this thesis, multivariate models, especially hybrids or those derived from novel techniques such as the bifactor model, provide unique insights into comorbidity (Krueger & Markon, 2006). This thesis has demonstrated that a common liability to

all psychopathology, viewed from a unique perspective, in the form of the bifactor models and circumplex, is a potentially suitable model for understanding the nature of psychopathology and comorbidity. Such models have largely been regarded as sub-optimal for explaining the comorbidity construct at a bivariate level (Neale & Kendler, 1995; Rhee et al., 2008). However, using a multivariate perspective has allowed the reconsideration of such models with positive results. While it must be acknowledged that there has been a recent increased emphasis on multivariate research, such research must continue to be pursued more vigorously.

Limitations of this Thesis

The results of this research must be considered with regard to the potential limitations of the studies. This section will discuss these potential limitations across all studies and their potential impact upon the results/conclusions drawn. Limitations which impact upon individual studies will also be discussed.

Sample, Data and Design Limitations

Use of Clinical Samples

One immediate limitation apparent in this thesis is the use of a hospital referred clinical sample. While many studies do use clinical samples, and indeed such research often provides cogent support for theory, it is imperative that the limitations of using clinical populations are considered. Sampling bias is problematic within clinical samples (Kendall et al., 2001), and as such, they cannot be considered representative of the general population with the disorder. This is because the rate of individuals presenting to clinical settings tends to be greater than in general population samples, they usually have greater symptom severity, and/or come from

families more burdened by their children's problems (Angold et al., 1999). This can

result in incorrect estimates of strength of association between disorder, though

Angold et al. (1999) point out that it may just as likely underestimate strength of

association as overestimate. Other issues, including issues of method variance as

outlined in Chapter 1 can also be an issue. In addition to this overarching issue, the

fact that this clinical data was collected from a single source clinic adds further

potential sampling bias. While data collection from a single source is quite common,

it does create a potential sampling bias as certain members of the population are

potentially underrepresented or overrepresented relative to others in the population

(Cohen & Cohen, 1984).

Thus it is generally considered to be advantageous to use samples from the general

population to produce findings of greater generalisability than that garnered from

studies of clinical samples (Caron & Rutter, 1991; Essau, 2003). It must be

emphasised though that the limitations of using clinical samples, and clinical samples

from a single source, have been ameliorated for the base models proposed in Studies

1 and 2. This is because the bifactor and circumplex models were tested using the

ASEBA reference group; an epidemiological or community sample (Achenbach &

Rescorla, 2001). However, Study 3 only used a clinical sample and as a result, the

generalisability may be limited. Despite this limitation, the use of clinical samples

does not completely undermine the utility of Study 3. Kendler (2004) argues that

clinical samples are required to clarify the factors relating to the aetiology of

comorbidity, and or when trying to identify potential risk factors for

psychopathology. In part this is because clinical populations are again necessary to

ensure that there is definitive clinical utility in research (Krueger & Markon, 2006).

However, most importantly, Rhee and colleagues (Rhee, Hewitt, Corley, & Stallings, 2003; Rhee, Hewitt, Corley, Willcutt, & Pennington, 2005; Rhee et al., 2004) have concluded that it is often difficult to test models of comorbidity/co-occurrence when the prevalence of one or both of the disorders is low (as it is in community samples), or when correlations between liabilities are small, in part because of the low levels of the desired trait. Lower prevalence is more common in community samples. Therefore it is clear that the use of clinical samples in Study 3 far from invalidates the research finding, especially given the use of epidemiological samples to validate the base models in Studies 1 and 2. However, further validation with community based samples would be recommended.

Use of CBCL T-Scores

One minor limitation noted in the study was the use of CBCL *T*-scores for both the Syndrome and DSM-Oriented scales, rather than raw scores. Achenbach and Rescorla (2001) suggest that it is preferable to use raw scores for statistical analyses. This is because *T* scores truncate the lower end of the scales, and raw scores may allow for the full range of variation to be taken into account. However, scaled score data was not available from all files used in the sample, and for this reason, the *T* scores were used in the current study. Using data from those participants where scale score information was available, parallel testing using these scale scores was conducted, and no interpretable differences were noted. As a result, it appears this limitation may have had no appreciable impact on the results. Indeed this potential for lack of impact is acknowledged by Achenbach and Rescorla (2001).

Use of Cross-Sectional Designs

The cross-sectional nature of the data used in all studies reported in this thesis, makes statements regarding causality only tentative in nature. Cross-sectional data makes establishing causality difficult, in part because there is at least a potential to confound state, trait and scar effects (Khan et al., 2005). However, given the prospective nature of the research in testing new models, the cost and difficulty in gaining longitudinal data is not justifiable. However, future research investigating the common factor model of psychopathology should consider the use of longitudinal data, or alternatively, use lifetime diagnosis (or trait-based measures) to allow more causal effects to be inferred.

Response Bias and Common Method Variance

Psychopathology and risk factors were measured with rating scales that were completed by one parent source. There is therefore potential for response bias due to common method variance. Response bias in this context means that two individuals may give different subjective ratings about the same individual's psychopathology. It has been shown, for example, that mothers and teachers have biases that can produce differing conclusions about psychopathology (Martin, Scourfield, & McGuffin, 2002). Thus the use of only one source of information in the three studies, in this case questionnaires usually completed by the mother of the child, can create a common method bias. Common method bias is where two unrelated variables may be linked simply because they were collected using the same method (Lindell & Whitney, 2001; Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). While this does not invalidate the research findings (see Doty & Glick, 1998), its impact must be

noted (Conway & Lance, 2010), and the conclusions viewed somewhat more tentatively.

As outlined in Chapter 1 however, the effects of method covariance, while present, are minimal enough to indicate that the results of this finding are not invalidated by this potential confound (Keiley et al., 2003). Similarly, the use of multiple sources – often the most common method used to counteract this bias – may itself be problematic (Jarrett & Ollendick, 2008), in part because there are often low correlations between different informants' ratings of child symptomatology (e.g. Achenbach, McConaughy, & Howell, 1987), and thus it can be difficult to obtain convergence across multiple informants (Edelbrock & Costello, 1988).

Conceptual Limitations

Limited Number of Risk Factors Investigated

A number of common genetic, biological, physiological, psychological, environmental, and social factors may be implicated in the development of comorbidity. While a plethora of possibilities exists, the present thesis selectively examined negative affect, parental psychopathology, and family functioning. As such, it was not intended to be an exhaustive review of all of the aforementioned possibilities. Rather these factors were selected as they cover each of the risk factor categories outlined by Crawford et al. (2011); child traits and behaviours, maternal (parental) traits and behaviours, and environmental risk. Indeed as Chapter 2 outlined, the three constructs selected are probably the most investigated from each of those categories. It is also important to note that there are several other temperament constructs which could be included in such a model, though it is

negative affect which is generally viewed as a more diverse risk factor (Lahey & Waldman, 2003; Lilienfeld, 2003). It is also important to note that the selection of risk factors was also a product of the data collected by the source agency, and as such, it must be acknowledged that other risk factors may play a significant role in the psychopathological liability construct.

Overlap of Temperament and Measures of Psychopathology

Research has consistently demonstrated that temperament and symptom measures appear to have some overlap in content (e.g. Sanson et al., 1990). Lengua et al. (1998) noted that negative affect tended to show positive correlations to symptom measures, with especially consistent relationships between negative affect and the mood disorders, though Bates (1990) indicates that such associations may be expected (De Pauw & Mervielde, 2010). While the conceptual overlap does not invalidate the results, it does provide a potential confound in the associations between the two concepts that must be acknowledged.

Paternal Psychopathology

As previously indicated, the source of parental information in this study was most commonly the mother of the child. As a result the parental psychopathology ratings were almost universally maternal, leaving the effects of paternal psychopathology largely untested. While early risk factor research gave scant regard to paternal psychopathology as a risk factor (Marmorstein et al., 2004), the presence of disorders in mothers and fathers present equal risks for offspring, with effect sizes generally equivalent in magnitude (Connell & Goodman, 2002; Kane & Garber, 2004). Thus the inability to assess the effect of paternal psychopathology means that this study

cannot assess the full magnitude of the effect of parental psychopathology.

Nonetheless, given that this thesis showed the (primarily) maternal ratings were so

strongly related to child psychopathology, were future research to investigate both

maternal and paternal psychopathology in combination, the strength of any

association would be expected to increase (Brennan et al., 2002).

Confounding Effect of Parental Psychopathology and Familial Functioning

As part of the conceptualisation of risk factors, consideration should be given to the

fact that risk factors may not only influence child psychopathology, but may also

influence each other (Crawford et al., 2011). Dickstein et al. (1998) have noted that

these results indicate that maternal mental illness and family functioning are

negatively associated, especially when using nonspecific indicators such as the

Global Assessment of Functioning (GAF: American Psychiatric Association, 2000)

from the DSM, or the HSCL sum score as used in this study. As such, this presents a

partial confound when considering the additive/interactive effects of these two

constructs as risks for childhood psychopathology.

Future Directions

The findings of this thesis have implications for a number of directions in future

research.

Further Testing and Iterations of the Model

Given the infancy of the literature on multivariate models of comorbidity, and the

fact that both the circumplex and bifactor models presented in this thesis are new in

terms of use in studying comorbidity, more studies are needed to replicate these

findings. Extensions of the studies presented in this thesis would include the use of different informants. This is easily done for the ASEBA scales (Achenbach & Rescorla, 2001), through the use of the Teacher Report Form (TRF) and the Youth Self-Report (YSR). Similarly, the ADIS-IV (Silverman & Albano, 1996) has a Child version (ADIS-IV-C) which could be used. Were these models to be validated further through the use of multiple informants, then support for the circumplex and bifactor models, and hence a psychopathological liability, would be enhanced.

Future studies should also consider the use of dimensional psychometric measures other than that used in this thesis. While the ASEBA scales (Achenbach & Rescorla, 2001) are probably the most widely used measures, as well as arguably the broadest dimensional measure of psychopathology in children (Achenbach et al., 2008), they are far from the only dimensional measures available. Instruments such as the Conners Comprehensive Behavior Rating Scales (Conners CBRS: Conners, 2008b), and the Behavior Assessment System for Children scales (BASC-2: Reynolds & Kamphaus, 2005). Both the Conners CBRS and BASC-2 are widely used in research, and were the circumplex and bifactor models to show fit for these measures, it would clearly demonstrate the universality of the models.

Extension to Adult Populations

Serious consideration should also be given to testing the models presented in this thesis in adult populations. As outlined briefly in Chapter 1, comorbidity is as prevalent in adult populations as it is in child populations (e.g. Jacobi et al., 2004). For many years in the early study of psychopathology and comorbidity, there was a substantial disconnect between adult and child research (Rutter, 1996), though this

has ameliorated to some degree in the last decade or so. Indeed many theoretical models, such as the tripartite model of depression and anxiety (Clark & Watson, 1991) have been developed, tested, and validated in both adult and child populations. However, it must be noted that adult and child psychopathology differ in many ways (see Hudziak, Achenbach, Althoff, & Pine, 2007), and thus the model may not work within the adult domain. Nonetheless, a worthwhile extension of this thesis would be testing within the adult domain, to see if an integrative theory of comorbidity across the lifespan is plausible.

Use of More Advanced Psychometric and SEM Techniques

Once the circumplex and bifactor models of psychopathology have been validated, with replication, in both child and adult populations, consideration should be given to the use of advanced SEM-related procedures, such as Latent Growth Models (LGM), Latent Class Analysis (LCA), and Factor Mixture Models (FMM). These advanced models, which were briefly touched upon in Chapter 1, would provide further detail regarding the nature of the development of comorbidity and the nature of the psychopathological liability construct, temporal impacts of the model, and the applicability of the model for different groups (latent classes) of individuals.

Latent Growth Modelling (LGM), is a well validated technique for examining change across time, and is regarded as superior to many other methods of longitudinal data investigation (Byrne & Crombie, 2003). LGM would allow the investigation of inter-individual differences in the trajectories of the development of comorbidity, and then allow investigation of the specific and global risk factors that impact such trajectories. It may be, that different combinations of risk factors may

differentially affect individual trajectories of psychopathological development, and, were different trajectories noted, such information would allow a more targeted identification of areas for preventative intervention efforts.

Latent Class Analysis (LCA) would allow a more fine-grained analysis of the applicability of the circumplex and bifactor models to particular latent groups. LCA can model subtypes, or latent classes that exist within a sample population (Hagenaars & McCutcheon, 2002). LCA identifies clusters which group together cases (i.e. individuals) who share similar characteristics and or behaviours. LCA could therefore be used to identify different classes of individuals based on their levels of different forms of psychopathology. Such classes would be expected to be defined by the levels of domain-specific symptomatology. Based on the bifactor model proposed here, and prior research findings which showed that the specific domains of internalising and externalising behaviour are separable phenomenon (Krueger, 1999; Krueger et al., 1998; Krueger & Finger, 2001), it might be hypothesised that at least four latent classes (i.e. four different groups of individuals) may exist within a group of individuals assessed for psychopathology. These classes might be; low internalising/low externalising behaviour, high internalising/low externalising behaviour, low internalising/high externalising behaviour, and, high internalising/high externalising behaviour. Whatever classes were identified by such analyses, the applicability of the circumplex and bifactor models could then be assessed for each identified class/group, allowing a more detailed understanding of the nature of the psychopathological liability construct for different latent classes. LCA could also be used to create classes/groups based on the degree to which different risk factors are present, which would in turn allow an investigation of the

differing trajectories of psychopathology based in risk factors; a technique called
Growth Mixture Modelling (Jung & Wickrama, 2008).

An even more detailed understanding of the bifactor and circumplex models could be
ascertained by the use of an even more advanced and innovative technique called
Factor Mixture Models (FMM). One potential limiting assumption of LCA is that it
assumes individuals in a class do not differ systematically in terms of severity. This
means that LCA can model subtypes or classes, but cannot model different severities
within the latent classes. FMM (Muthen & Shedden, 1999) overcomes this limitation
by allowing the modelling of different severities within each class. The use of such
techniques would allow a very comprehensive understanding of the
psychopathological liability construct proposed in this thesis, and an understanding
of how different severities of the factors forming this construct may impact on
outcomes. Other non-SEM psychometric techniques such as Item Response Theory
(IRT) could also be implemented. IRT could allow investigation of the nature of the
common factor by exploring how each diagnosis maps onto the factor using
information derived from the test information function. It would presume that the
diagnoses would measure primarily the higher end of the factor (Krueger & Finger,
2001), but such information would be verifiable by such analysis.

Wider Ranges of Risk Factors

Finally, future research will need to investigate a wider array of potential risk factors.
The selection of risk factors in this thesis was a product of the data collected by the
source agency, and as noted other risk factors may play a significant role in the
psychopathological liability construct. Also, given the implications of the bifactor

model regarding the presence of global and domain-specific factors, consideration must be given to separation of these factors. Indeed the identification of the domain-specific risk factors may be just as vital as identification of the global risk factors. This is because the domain-specific risk factors may be able to be used as clinical markers in specific diagnoses, whereas the presence of global risk factors only suggests the diagnostic possibility of psychopathology in general.

Future research should also give consideration to potential mediator and moderator effects between risk factors underlying the psychopathological liability construct. Considerable research (e.g. Elgar et al., 2007; Grant et al., 2006; Lim, Wood, Miller, & Simmens, 2011; Patterson & Capaldi, 1990; Suveg et al., 2011; Vera, Granero, & Ezpeleta, 2012) has demonstrated clear mediation and moderation effects between risk factors and psychopathological outcomes. While modelling such effects may make the models more complex and less parsimonious, they may give insight into the workings of the global and domain-specific risk factors and the psychopathological liability construct.

Summary and Conclusion

The overall aim of this thesis was to propose a general factor to capture the common variance for the major internalizing and externalizing childhood syndromes and disorders. Study 1 demonstrated strong support for the presence of the hypothesised common psychopathological liability factor. This support was strengthened by the results of Study 2, but the results of Study 1 also demonstrated that domain-specific factors may also exist. Study 3 demonstrated that negative affect and parental psychopathology, both individually, and in interaction, appear to be key risk factors

associated with the common factor, suggesting this common factor may be a psychopathological liability factor.

Overall, results suggest that, in contrast to current conceptualisations of childhood psychopathology as discrete and distinguishable entities, there is a common liability to all psychopathology in childhood. Such a common liability helps explain the high level comorbidity of childhood disorders and syndromes, because the liability suggests that all manifestations of psychological illness may, at least in part, have some common geneses. The results here suggest that negative affect and parental psychopathology are key risk factors in understanding this common liability.

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