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Childhood Predictors of Personality Disorders

by

Johanna Koerting

Supervisors: Edmund Sonuga-Barke, Roelie Hempel, Thomas Lynch

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ABSTRACT

Personality disorders (PDs) are characterised by enduring patterns of inner experience and behaviour that deviate markedly from the expectation of the individual's culture, have their onset in adolescence or early adulthood, are pervasive, inflexible and stable over time, and can lead to serious distress and impairment in daily life and functioning (APA, 2013). Even though empirical research in this area is mostly lacking, prominent theoretical models of PD argue that individual child characteristics such as difficult temperament, and adverse environmental factors such as negative parenting behaviours, interact to increase a child's risk of developing personality pathology later in life.

This thesis examined childhood predictors of Personality Disorders (PDs). The aim was to investigate (1) Whether externalising and/or internalising childhood problems were predictive of personality pathology in early adulthood; (2) Whether the associations between childhood problems and personality pathology were moderated or mediated by negative parenting; and (3) Whether continuities in child psychopathology explained the associations between childhood problems and PDs.

In Chapter 1, an overview about what is currently known about the developmental pathways to PD was provided. Applying an interactive model, biological and environmental risk factors, as well as evidence looking at the interplay between these factors, was reviewed. Further, an overview about the risk markers for the development of PD, in the form of common childhood disorders (EXT and INT problems) was given. In Chapter 2, all published prospective longitudinal studies about the predictive validity of childhood externalising and internalising problems regarding PDs were collated and meta-

analysed. Chapter 3 outlined and justified the methodology applied across studies. In addition, the methodological challenges encountered when conducting this research were discussed, as well as the methods applied to overcome these challenges.

In Chapter 4, using a prospective longitudinal design, childhood problems were investigated as predictors of personality pathology in early adulthood. Childhood data was collected in 1990/1991, of three-year-old children and their families, where children were assessed for emotional and behavioural problems, namely hyperactivity, emotional problems, shyness and conduct problems. Both additive and interactive effects of baseline variables were assessed in relation to personality pathology in early adulthood, controlling for age, sex and socio-economic status (SES) at baseline. The results showed that externalising but not internalising problems were significantly predictive of personality pathology at follow-up. Only one additive effect and no interactive effects were found.

In Chapter 5, the effects of adverse parenting on the associations between childhood problems and PD were investigated. Specifically, we explored whether maternal and/or paternal lack of warmth and/or overcontrol as assessed by retrospective reports by the young persons, significantly influenced the relationship between childhood disorders and personality pathology. We found that paternal indifference and maternal overcontrol were predictive of adult PD; these negative parenting dimensions added to the effects of childhood problems detected in Chapter 4 in the prediction of PD. No moderation effects and few partial mediation effects were detected.

In Chapter 6, the effects of continuities of childhood psychopathology on PD were assessed. Specifically, it was investigated whether the effects of childhood problems on PD were mediated by homotypic or heterotypic continuities in psychopathology (i.e. continuity within the same (homotypic) or different (heterotypic) 'class' of disorder) which were assessed in early adulthood. Only homotypic continuities were

found: hyperactivity at age 3 was associated with adult Attention-Deficit Hyperactivity Disorder (ADHD), and conduct problems at age 3 were associated with later Oppositional Defiant Disorder (ODD). ADHD did not mediate the relationship between hyperactivity and adult PD, and ODD partially mediated the association between conduct problems and adult PD. In Chapter 7, the results of all previous chapters were summarised and discussed.

In sum, we found strong and robust associations between childhood externalising problems and PD; these were not influenced by negative parenting, and they were not mediated by continuation of symptoms into adulthood. Negative parenting, especially paternal indifference, additionally increased the risk for a PD, but parenting did not interact with childhood problems in the prediction of PD. Specifically, the following risk patterns were found: (1) childhood hyperactivity, conduct problems and paternal indifference predicted Borderline PD; (2) childhood conduct problems and maternal overcontrol predicted Antisocial PD; and (3) childhood hyperactivity predicted Avoidant PD. These results are in contrast to the consensus that child characteristics and environmental factors interact in the development of PD. Rather, the results would support a model of separate pathways leading to PD. Our findings have several implications for early intervention and prevention strategies. The findings were discussed in relation to current developmental theories of PD, as well as their implication for our understanding of developmental pathways for PD.

FACULTY OF SOCIAL AND HUMAN SCIENCES

Psychology

Thesis for the degree of Doctor of Philosophy

CHILDHOOD PREDICTORS OF PERSONALITY DISORDERS

Johanna Luise Koerting

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DECLARATION OF AUTHORSHIP

I, Johanna Luise Koerting, declare that this thesis and the work presented in it are my own and has been generated by me as the result of my own original research.

Childhood predictors of personality disorders

I confirm that:

1. **This work was done wholly or mainly while in candidature for a research degree at this University;**
2. **Where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;**
3. **Where I have consulted the published work of others, this is always clearly attributed;**
4. **Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;**
5. **I have acknowledged all main sources of help;**
6. **Where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;**
7. **None of this work has been published before submission.**

Signed:.....

Date: .....

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Definitions and Abbreviations

Abbreviation	Definition
ADHD	Attention Deficit Hyperactivity Disorder
ASPD	Antisocial Personality Disorder
AVPD	Avoidant Personality Disorder
BCL	The Behaviour Checklist
BPD	Borderline Personality Disorder
CBRS-S	Conners Behavioural Rating Scale – Self Report
CBT	Cognitive Behaviour Therapy
CD	Conduct Disorder
CI	Confidence Interval
CIC	Children in the Community Study
CP	Conduct Problems
CSA	Childhood sexual abuse
DSM	Diagnostic and Statistical Manual
DV	Dependent Variable
EAS	Emotionality, Activity, Sociability Scale
EP	Emotional Problems
EXT	Externalising
FFM	Five Factor Model
HYP	Hyperactivity
ICD	International Classification of Diseases
INT	Internalising
IV	Independent Variable
LCM	Least Control Model – meta-analytical model where effect size data for the association between childhood variables and outcome variables with the least amount of statistical control over any covariates was included

MAR	Missing at random
MCM	Most Control Model – meta-analytical model where effect size data for the association between childhood variables and outcome variables with the most amount of statistical control over any covariates was included
MNAR	Missing not at random
MOPS	Measure of Parental Style Questionnaire
NPD	Narcissistic Personality Disorder
OCPD	Obsessive-Compulsive Personality Disorder
ODD	Oppositional Defiant Disorder
OR	Odds Ratio
PD	Personality Disorder
PEDIA	Programme for Early Detection and Intervention for ADHD
PID-5	Personality Inventory for DSM-5
SES	Socio-economic status
SHY	Shyness
SP	Social Phobia
SPD	Schizotypal Personality Disorder
WWP	Werry-Weiss-Peter Activity Scale

Chapter 1: Literature Review – Childhood Predictors Of Personality Disorders

Objectives

This chapter will provide an overview about what is currently known about the developmental pathways to Personality Disorders (PDs). Applying an interactive model, biological and environmental risk factors, as well as evidence looking at the interplay between these factors, will be reviewed. Further, risk markers for the development of PD, in the form of common childhood disorders (externalising [EXT] and internalising [INT] problems) will be reviewed. Evidence will be discussed from both retrospective and prospective studies, focusing on prospective evidence where possible.

1.1 Introduction

According to the current Diagnostic and Statistical Manual (DSM-5; American Psychological Association (APA), 2013) personality disorders (PDs) are characterised by enduring patterns of inner experience and behaviour that deviate markedly from the expectation of the individual's culture. According to DSM-5, PDs have an onset in adolescence or early adulthood, are pervasive, inflexible and stable over time, and can lead to serious distress and impairment in daily life and functioning. Approximately 10-15% of the adult population are affected by a PD (APA, 2000; Grant et al., 2008; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Mattia & Zimmerman, 2001).

Besides functional impairment and emotional distress, personality pathology is also associated with significant financial costs to the healthcare system, social services and wider society. In England, the health and social care service costs of all people with PD who were in contact with their general practitioners were estimated at £704 million per year in 2008 (McCrone, Dhanasiri, & Patel, 2008). When productivity losses were included, the cost rose to £7.9 billion per year. Another study in the Netherlands that used data from health and social care contacts for people attending specialist PD services, reported that the cost of PDs was £11,126 per patient

(Soeteman, Hakkaart-van Roijen, Verheul, & Busschbach, 2008). However, these studies do not provide an estimate of the total economic burden, because they are limited to patients in contact with services and are therefore not representative of the PD population as a whole. Many of those with a PD are unknown to services (NIMH, 2003), reject treatment rather than seek it (Tyrer, Mitchard, Methuen, & Ranger, 2003), or have a different primary diagnosis (Ranger, Methuen, & Rutter, 2004). It is also unlikely that everyone with a PD is diagnosed (NICE, 2009b). Furthermore, these figures do not include costs to other service sectors, such as health and social services, or the criminal justice system. For instance, the average cost of a violent crime involving wounding in the UK is £19,000 per incident (Brand & Price, 2000), and studies have estimated that almost 50% of prisoners in the UK have a diagnosis of Antisocial PD. In the UK it costs around £65,000 to imprison a person, however this figure not include the additional costs of providing treatment for PD in prison (£36,000 per year) or the costs for additional security that might be necessary (NICE, 2009a). Finally, the estimates do not include indirect costs of PD to the economy more widely, e.g. through inability to work and premature death, so the true costs of PD are certainly substantially higher than the estimated figures.

1.1.1 Assessment of Personality Disorders

1.1.1.1 Assessment according to the Diagnostic and Statistical Manual of Mental Disorders

Prior to DSM-III, reliability of PD diagnosis was rather poor (Spitzer, Endicott, & Robins, 1975; Spitzer & Fleiss, 1974). Diagnoses were entirely based on clinicians' subjective judgements unrestricted by specific guidelines. With the publication of DSM-III in 1980, specific and explicit diagnostic criteria for mental disorders were first introduced. However, whilst standardised criteria enhanced diagnostic reliability of most other mental disorders, for PD reliability only improved marginally with interrater reliability values (kappas) for specific PDs ranging from .26 to .75 in field trials (Williams & Spitzer, 1980) and .01 (Schizoid PD) to .49 (Antisocial PD) in clinical practice (Mellsop, Varghese, & Joshua, 1982). Values for Cohen's Kappa range from 0 to 1, and > .70 is considered satisfactory. Another major critique was that the conceptualisation of PDs was more grounded in

theoretical perspectives of Work Group members rather than based on empirical research (Widiger, 2012). The process of revising for DSM-IV was therefore aimed at appraising the system introduced in DSM-III, and systematically and explicitly reviewing relevant empirical research. However, the content for PD stayed largely the same in DSM-IV and was again subject to a lot of criticism. The revisions for DSM-5 therefore focused on fundamentally revising the diagnostic system and introducing an entirely new model of classification. These revisions proved to be very controversial however (Blashfield & Reynolds, 2012; Tyrer, Crawford, & Mulder, 2011; Widiger, 2012) and were aborted at the last minute. As a consequence, the current classification system (DSM-5) is now more or less identical to DSM-IV and DSM-III.

Since the introduction of DSM-III PDs have been conceptualised using a polythetic-categorical approach, whereby a specified number of criteria have to be met in order to make a diagnosis. A total of ten different PDs are listed, classified into three separate clusters, based on descriptive similarities. Cluster A PDs are described as "odd or eccentric PDs" and include Paranoid PD, Schizoid PD and Schizotypal PD. Cluster B PDs are described as "dramatic, emotional, or erratic PDs" and include Antisocial PD, Borderline PD, Histrionic PD and Narcissistic PD. Cluster C PDs are described as "anxious or fearful PDs" and include Avoidant PD, Dependent PD and Obsessive-Compulsive PD. Cluster A and C PDs are generally associated with negative emotionality, anxiety or distress, i.e. with internalising symptomatology; Cluster B, on the other hand, includes problems with poor inhibitory control, inabilities to delay gratification, and impulsive/reckless behaviours linked to chaotic relationships and/or poor interpersonal functioning, i.e. symptomatology on the externalising dimension (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009).

1.1.1.2 Alternative Models of Assessment

There are several advantages of the categorical diagnostic system of the DSM (Frances, 1993; Gunderson, Links, & Reich, 1991; Millon & Davis, 1996). It is easy to use by clinicians who are required to make rapid diagnoses of large numbers of patients who they only see briefly. Furthermore, all current and prior diagnostic systems have been categorical, so the

typologies are historically well-established and serve as a reference for clinicians. However, the current categorical system has been widely criticised for a number of reasons (Blashfield & Reynolds, 2012; Tyrer et al., 2011; Widiger, 2012): (i) extensive co-occurrence among PDs – most individuals diagnosed with a PD meet criteria for more than one PD; (ii) extreme heterogeneity among patients receiving the same diagnosis; (iii) arbitrary thresholds of categorical diagnoses; (iv) temporal instability of diagnoses, inconsistent with the relative stability of personality traits and impairment in PD; (v) poor coverage of personality psychopathology by the specific PDs; and (vi) poor convergent validity across PD assessments.

As a consequence, alternative models of personality pathology have been discussed, including dimensional and hybrid models (Krueger, 2002b; McGlashan et al., 2005; Simonsen, 2010; Widiger & Clark, 2000; Widiger & Costa, 2002; Widiger & Simonsen, 2005). In addition, the usefulness of variable-centred versus person-centred approaches has been debated. Variable centred approaches focus on personality traits, on the relationship between these traits in populations, and on understanding how dimensions of personality variation are organized empirically. Person-centred approaches, on the other hand, focus on differences between individuals when examining relationships between variables. Person-centred researchers argue that personality traits should not be studied in isolation but instead focus should be on the constellation of traits that define each person, aiming to identify groups or subsets of individuals, i.e. "prototypes", who have similar configurations of traits and thus share the same basic personality structure (Block, 1971).

The most prominent person-centred model was originally developed by Block (1971) in which three personality prototypes were depicted, namely 'overcontrolled', 'undercontrolled' and 'resilient'. Overcontrol/undercontrol refers to a meta-dimension of impulse inhibition versus impulse expression; resiliency refers to a meta-dimension of the dynamic, flexible capacity to modify one's level of control in response to contextual demands. These prototypes were independently developed by other researchers who found similar constructs (Asendorpf, Borkenau, Ostendorf, & van Aken, 2001; Asendorpf, Denissen, & van Aken, 2008; Asendorpf & van Aken, 1999; Caspi, 2000; Caspi & Silva, 1995; Chapman & Goldberg, 2011; Eisenberg et al., 2000;

Markon, Krueger, & Watson, 2005; Meeus, Van de Schoot, Klimstra, & Branje, 2011; Robins, John, Caspi, Moffitt, & Stouthamer-Loeber, 1996). The construct validity of this broad person-centred personality perspective has received empirical support, both cross-culturally and longitudinally (Asendorpf & van Aken, 1999; Chapman & Goldberg, 2011).

Overcontrol and undercontrol largely parallel the well-established division in psychopathology between internalising and externalising disorders. Both adult (Krueger & Markon, 2006b) and child (Achenbach & Edelbrock, 1984) psychopathology can be organised into a hierarchy at the top of which are these two broad factors, i.e. externalising (EXT) and internalising (INT). In children, EXT problems are described as behaviours characterised by aggressiveness, difficulties with interpersonal relationships and rule breaking, as well as displays of irritability and belligerence (Achenbach & Edelbrock, 1978; Hinshaw, 1992). In adults, these problems manifest for example as antisocial behaviours, substance abuse, or alcohol problems (Krueger, Markon, Patrick, & Iacono, 2005). In contrast, child INT problems include social withdrawal, inhibition, shyness, feelings of worthlessness or inferiority, and dependency (Achenbach & Edelbrock, 1978; McCulloch, Wiggins, Joshi, & Sachdev, 2000). INT psychopathology in adults includes phenomena such as depressive symptoms, anxiety/fearfulness and phobias (Krueger & Markon, 2006b). Evidence based on adoption, family and twin studies suggests that there is a genetic basis to both EXT problems and INT problems (Bartels et al., 2004; Fanous, Gardner, Prescott, Cancro, & Kendler, 2002; Jang & Livesley, 1999; Kendler, Prescott, Myers, & Neale, 2003; Markon, Krueger, Bouchard, & Gottesman, 2002; Roberts & Kendler, 1999). Research indicates moderate to strong continuities in EXT and INT behaviours from early to middle childhood through adolescence and into adulthood (Ferdinand & Verhulst, 1995; Fergusson, 1998). The link between undercontrol/overcontrol and EXT/INT can also be seen in longitudinal studies. Undercontrolled children have been shown to be more likely to develop EXT disorders (Eisenberg et al., 2000; Kendler et al., 2003; Krueger, 1999), whereas overcontrolled children are more likely to develop INT disorders and become socially isolated adults (Asendorpf et al., 2008; Caspi, 2000; Chapman & Goldberg, 2011; Eisenberg et al., 2000; Robins et al., 1996).

1.1.1.2.1 Variable Centred Approaches To Personality And Personality Pathology

Continuous models make classifications by locating individuals among graded dimensions. Several approaches to dimensional personality pathology have been proposed, but the view that the majority of PD researchers agree with (Bernstein, Iscan, & Maser, 2007) is to conceptualise personality pathology as extreme and/or maladaptive variants on a continuum of normal personality traits. This approach largely examines how models of normal personality can be used as a method for conceptualising PD (Saulsman & Page, 2004). The most widely used model of normal adult personality is the Five Factor Model (FFM; Costa & McCrae, 1992), represented by Neuroticism, Extraversion, Openness to Experience, Agreeableness and Conscientiousness. Evidence suggests that PD can indeed be conceptualised as maladaptive variants of the FFM, for example through the results of a meta-analysis by Saulsman & Page (2004). Similarly, Clark (2007) asserted that the FFM can be accepted as representing the higher-order structure of both normal and abnormal personality traits; and Costa and Widiger (2002) reviewed the results of over 50 studies and supported the notion that PDs can be captured in terms of the domains and facets of the FFM.

The consensus that PD can be conceptualised as extreme variants of the FFM was reflected in alterations suggested for DSM-5: to apply a dimensional model with five domains (Negative Affectivity, Detachment, Antagonism, Disinhibition and Psychoticism) that closely align with the dimensions of the FFM. Only a subset of the 10 DSM-IV PDs were suggested to be retained in DSM-5, namely Borderline PD, Narcissistic PD, Schizotypal PD, Avoidant PD and Obsessive-Compulsive PD, as a set of PD types. Antisocial PD was suggested to be combined with psychopathy to create an Antisocial PD/psychopathy type. In line with this new model, an assessment instrument operationalising the new model was created - The Personality Inventory for DSM-5 (PID-5) (Krueger, Derringer, Markon, Watson, & Skodol, 2012).

However, the revisions for PD in DSM-5 were heavily criticised – the criteria for deletion were not explicit and the final selection appeared arbitrary to

experts (Livesley, 2010). Interestingly, suggestions for revisions for the International Classification of Diseases 11 (ICD-11) have also included deletion of specific PDs, however, these are not identical to the PDs that were suggested for deletion from the DSM: for example, Schizoid PD was to be retained and Borderline PD to be deleted (Widiger, 2012). As the plans for revising the classification system of PD were aborted, this alternative model was moved to section III of DSM-5, to be further researched, and the DSM-IV conceptualisation of PD was transferred more or less verbatim to DSM-5.

1.1.1.2.2 Hierarchical Organisation of Traits

Most competing variable centred models are multidimensional models where traits are organised in a hierarchical structure. At the bottom of hierarchical structures are first-order constructs, or facets, that reflect relatively specific behavioural, cognitive or emotional tendencies. These first-order constructs show patterns of covariation that compose second-order, or higher-order, dimensions. Further, there is evidence that, at an even higher level, these higher-order traits show reliable patterns of co-variation, forming "metatraits". Thus, traits build a hierarchical structure that ranges from lower-order traits to higher-order traits to metatraits (De Young, 2006; Digman, 1997; Markon et al., 2005). There is increasing evidence suggesting that most variable centred personality models can be readily integrated within a common hierarchical structure. For instance, Widiger & Simonsen (2005), based on a thorough review of proposals for dimensional personality assessments in the empirical literature, provide a model that maps most of these proposals onto the five broad traits of Antagonism (Agreeableness), Constraint (Conscientiousness), Emotional Instability (Neuroticism), Extraversion, and Unconventionality (Openness to Experience). At the highest level of the hierarchy they place the "metatraits" EXT and INT. Evidence generally supports this notion of this common latent structure across personality inventories (Clark & Livesley, 2002; Markon et al., 2005), which has also been shown to integrate abnormal personality (Markon et al., 2005).

1.1.2 Temperament

Personality (and personality pathology) is strongly related to temperament. Temperament has been defined as "constitutionally based individual differences in emotional, motor and attentional reactivity and self-regulation" (Rothbart & Bates, 1998; p. 109). From early infancy, children show considerable variability in their reactions to the environment, which, together with the mechanisms that regulate them, constitute the child's temperament (Rothbart, 2007). Temperamental characteristics are believed to demonstrate consistency across situations, as well as relative stability over time. As in the area of personality, models of temperament vary; however, three robust dimensions have been found in each big model of temperament, namely Negative Affect, Positive Affect and Effortful Control/Constraint (Anthony, Lonigan, Hooe, & Phillips, 2002; Clark, 2005). As such, two of these temperament dimensions refer to affect, whereas the third refers to regulation. These three robust dimensions resemble dimensions in the big personality models, where Negative Affect is linked to Neuroticism, Positive Affect to Extraversion, and Effortful Control to Conscientiousness (Anthony et al., 2002). The similarity between conceptualisations of personality and temperament is striking (Eisenberg et al., 2000; Krueger & Tackett, 2003; Shiner & Caspi, 2003). An important distinction is that temperament is assumed to have some biologically based substrate, whereas the role of biology is less central to most conceptualisations of personality (Eisenberg et al., 2000).

It is argued that certain disorders are more likely to develop in individuals who are more extreme on relevant temperament dimensions than others, especially in the face of environmental stressors (Clark, 2005). Negative affectivity is associated with a broad range of psychopathology (Krueger, Caspi, Moffitt, Silva, & McGee, 1996; Ormel et al., 2005; Watson & Clark, 1984) whereas low effortful control is associated with EXT disorders (Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Lynam, Leukefeld, & Clayton, 2003; Ormel et al., 2005). Effortful control also moderates the effects of negative affectivity on problems; highly negative children will be less likely to show problems when they have higher levels of effortful control (Rothbart & Bates, 2006; Rothbart & Posner, 2006).

1.1.3 Developmental Pathways to Personality Disorder

Historically, it was believed that PD does not manifest, and should not be diagnosed, in children or adolescents (APA, 2000) because their personalities were not fully integrated and due to a concern that this could lead to a possible stigmatisation (Cicchetti & Crick, 2009). Because personality pathology has often been viewed as being unmodifiable and resistant to intervention, it was thought that diagnosing a child with a PD could lead to a lifelong categorisation of dysfunction. It is becoming increasingly clear, however, that PD symptom constellations identified in adulthood have their origins in childhood (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Cohen, Chen, Kasen, & Brook, 2006; Johnson, First, et al., 2005; Kernberg, Weiner, & Bardenstein, 2000; Mervielde, De Clercq, De Fruyt, & Van Leeuwen, 2005; Shiner, 2007; Westen & Chang, 2000) and that PD prevalence rates in adolescents are comparable to those in adults (Shiner, 2009). Unfortunately, far less is known about the developmental pathways leading to PD than is known about the developmental pathways leading to other major psychological disorders (Shiner, 2009). The precursors of PD have received relatively little attention (De Clercq & De Fruyt, 2007; De Clercq, De Fruyt, & Widiger, 2009) and even though authors of texts and chapters on PD do refer to childhood antecedents (Cohen, 2008; Johnson, First, et al., 2005), this literature is more based on clinical experiences and theoretical expectations than empirical research.

Instrumental to understanding the developmental pathway to any disorder are prospective longitudinal studies. Retrospectively gathered data are often distorted by memory and reporting biases (Maughan & Rutter, 1997). For instance, adults in emotional distress may be more ready and willing to report earlier childhood adversity; those who are functioning relatively well often underreport it instead (Maughan, Pickles, & Quinton, 1995; Robins et al., 1985) which may artificially increase the association between early adversity and adult outcomes. This issue is particularly pertinent in studies about PD due to a tendency of patients with PD to misinterpret or misreport past experiences with family members (Bailey & Shriver, 1999). To date, however, prospective research on childhood precursors and pathways to PD has been relatively sparse (Crick, Murray-Close, & Woods, 2005). Therefore,

much of what is currently known about precursors to PD is based on cross-sectional data and retrospective recall of childhood events.

1.1.3.1 Interactional/Transactional Models of the Development of Personality Disorders

Most theoretical models of the developmental pathways to PD are interactional models that are based on the notion that individual vulnerabilities and environmental risk factors interact throughout life to influence a child's development, beginning as early as prenatally (Crick et al., 2005; Power, 2013). Interactional models emphasise that the interaction of pre-existing individual vulnerabilities and environmental stressors lead to disorder. They tend to focus on how the environment impacts individuals but are relatively silent about how individuals affect their environments (Power, 2013). Slightly different versions of interactional models are transactional models, such as the influential biosocial model of Borderline PD by Linehan (1993). They are similar to interactional models in that they assume that an interaction between the individual and the environment influences the child's development. However, they differ slightly in that they have a particular focus on bidirectional or reciprocal effects (Belsky, 1984; Sameroff, 2009). That is, transactional models assume that the child is not only influenced by, but also influences the environment which in turn has an effect on his/her development.

1.1.3.2 Early Identification of Risk Markers and Early Intervention of Personality Disorders

If precursors for the development of PDs can be identified during childhood, then treatment approaches aimed at early identification and prevention can be implemented, as has been proposed in other areas such as Attention Deficit Hyperactivity Disorder (ADHD) (Sonuga-Barke, Koerting, Smith, McCann, & Thompson, 2011). Some specific PDs such as Borderline PD have been suggested to be "leading candidates" for developing such programmes. For instance, Chanen & McClutcheon (2013) argue that Borderline PD is common in clinical practice, among the most functionally disabling of all mental disorders, often associated with help-seeking and it has been shown to respond to intervention even in those with established

disorders. However, the more behaviour patterns are established the more difficult they become to treat (Burke, Loeber, & Lahey, 2007; Linehan, 1993). Thus, earlier identification of vulnerability may be necessary to prevent the significant costs to individuals, their family members, and society (Crowell, Beauchaine, & Linehan, 2009).

It is not clear, however, what treatment or prevention approaches might be most appropriate or where they might be best targeted. It has been suggested that stand-alone universal prevention approaches may not be feasible due to the generally low prevalence of PDs, and it would be unclear as to what form of intervention would be most effective (Chanen & McCutcheon, 2013). Similarly, selective prevention approaches where those at high risk for PDs could be targeted would be impractical: many of the known risk factors are environmental, and those risk factors most strongly associated with development of PD (e.g. abuse/neglect) are commonly associated with outcomes other than PD, too (multifinality) (Cicchetti & Toth, 2009). Instead, the most optimal prevention method was suggested to be 'indicated prevention' where individuals displaying risk markers of the disorder are targeted (Chanen & McCutcheon, 2013). Risk markers such as typical childhood disorders (e.g. EXT and INT psychopathology) could be regarded as targets for indicated prevention of PD. As such, identification of risk markers in children is useful for two reasons: (i) Early signs and symptoms in children could be identified and directly targeted and (ii) The risk markers themselves could become target of interventions. Further, whilst standardised prevention/early intervention programmes have not yet been implemented specifically for PD, some early interventions, such as the High/Scope Perry Preschool Program, which was originally developed for boosting children's IQ, have been highly recommended in a study explicitly investigating the economic cost of severe antisocial behaviour in children (Romeo, Knapp, & Scott, 2006).

1.2 Biological Risk Factors

Evidence for biological risk factors comes two sources: (i) From studies directly investigating heritability rates of specific PDs and (ii) Indirectly

through investigating heritability of latent vulnerabilities in the form of "difficult" temperament dispositions. Both will be discussed below.

1.2.1 Genetic Risk/Heritability of Specific Personality Disorders

Several studies using twin data have demonstrated a significant heritability of specific PDs. Behavioural genetic designs typically estimate the influence of additive genetic influences (A), environmental influences shared in common (C), and nonshared environmental influences including error (E) on the variance and covariance between variables. Additive genetic effects represent the extent to which genotypes "breed true" from parent to offspring. Shared environmental influences distinguish the general environment of one family from another and influence all children within a family to the same degree (Rowe, 1994). Nonshared environmental factors (Hetherington, Reiss, & Plomin, 1994) include events that have differential effects on individual family members. E is not estimated directly but constitutes the residual variance after the effects of genetic and shared environmental effects have been removed. Different models are then tested to explore the effects of A, C and E by removing (i) The effects of genetic variance, (ii) The effects of shared environmental variance and, (iii) The effects of both genetic and shared environmental variance (E only model).

Torgersen et al. (2000) studied heritability of PD in a sample of adult monozygotic and dizygotic twins, and their results strongly suggest a genetic basis for all PDs. The best-fitting models had a heritability of 60% for PDs generally, and 37% for Cluster A, 60% for Cluster B, and 62% for Cluster C. Among the specific PDs, heritability was high for most PDs: 79% for Narcissistic PD, 78% for Obsessive-Compulsive PD, 69% for Borderline PD, 67% for Histrionic PD, 61% for Schizotypal PD and 57% for Dependent PD. Only for Schizoid PD, Paranoid PD and Avoidant PD, heritability rates were rather low (28% to 29%). Dependent PD was the only specific PD where a non-genetic model was not rejected, even though a model including genetics was a better fit. Another study (Coolidge, Thede, & Jang, 2001) tested heritability rates of DSM-IV PD features, as assessed through parent reports, in a sample of monozygotic and dizygotic child and adolescent twin pairs. Heritability estimates were high for all PDs, ranging from 50% (Paranoid PD) to 81% (Dependent PD and Schizotypal PD), supporting the

view that PDs are influenced by genes. However, as their sample included children from the age of 4, it is questionable whether their study did, in fact, measure personality pathology. A third study about heritability of PDs (Kendler, Gatz, Gardner, & Pedersen, 2006; Reichborn-Kjennerud et al., 2007; Torgersen et al., 2008) used an unselected community sample of twins. The best-fitting models included genetic and unique environmental factors and no sex or shared environmental effects. Heritability rates were much lower than those reported in the other studies and ranged from 21% to 28% for Cluster A PDs (Kendler et al., 2006), from 24% to 38% for Cluster B PDs (Torgersen et al., 2008), and from 27% to 35% for Cluster C PDs (Reichborn-Kjennerud et al., 2007). In a more recent study, Torgersen et al. (2012) argued that these differences in results may be related to interviewer bias. In their study, they used both clinical interview and questionnaire data for assessing Cluster B PDs in a sample of 2,800 twins. Their results showed that heritability assessed by interview was around .30, and around .40-.50 when assessed by questionnaire. Thus, divergences in heritability estimates may be related to assessment methods used.

Thus, there is evidence that PDs are influenced by genes and that they are at least party heritable. However, there is no consistency across studies as to the level of heritability. Obsessive-Compulsive PD, for instance, had one of the highest heritability estimates (77%) in two studies (Coolidge et al., 2001; Torgersen et al., 2000), but the lowest heritability of the three Cluster C disorders (27%) in a third study (Reichborn-Kjennerud et al., 2007). Similarly, whilst Narcissistic PD had the highest heritability estimate (79%) of all PDs in one study (Torgersen et al., 2000), it had the lowest estimate (24%) of all Cluster B PDs in another (Torgersen et al., 2008). This variation in effect sizes may be due to differences in sample size and populations studied. In sum, whilst heritability estimates vary across studies and specific PDs, it seems clear that PDs are at least partly heritable, and that genetic influences do affect the development of PDs.

1.2.2 Genetic Risk/Heritability of Vulnerability to Personality Pathology

According to interactional models, PDs develop due to an interaction of genetic vulnerability and environmental stressors. For example, “difficult” temperamental traits have been argued to increase the risk for the

development of a PD when interacting with environmental risk factors (Beauchaine et al., 2009; Linehan, 1993; Zanarini et al., 1997). Temperament traits have been found to be genetically influenced, with a lot of research focusing on the broad temperament traits of impulsivity and negative affectivity/emotionality. For instance, one study demonstrated that the core symptoms of Borderline PD, i.e. impulsivity and affective instability, were substantially heritable (Torgersen, 1984). Similarly, other research (Coccaro, Bergeman, & McClearn, 1993) demonstrated the heritability of impulsive aggression in a twin study, with heritability rates of 41%, and Krueger et al. (2002a) found impulsivity to be highly heritable (around 80%). Another study (Silverman et al., 1991) investigated whether affective and impulsive traits were more prominent in first degree relatives of probands with Borderline PD than in relatives of probands with other PDs or with schizophrenia. They found that, compared to the two other groups, relatives of Borderline PD patients had significantly higher levels of affectivity and impulsivity. These findings show that the latent vulnerability to develop the disorder, i.e. difficult temperament traits, is genetically influenced.

Longitudinal studies have investigated the predictive validity of impulsivity and affectivity/negative emotionality assessed in childhood with regard to personality pathology in adolescence/adulthood. Belsky et al. (2012), for example, looked at the effect of temperament on Borderline PD features in a sample of twins and their families. Offspring's lack of control, approach, inhibition and impulsivity was assessed at age 5, and Borderline PD features were assessed at age 12. The results showed that lower self-control and higher impulsivity were associated with Borderline PD features. Similarly, Carlson et al. (2009) investigated the effect of children's emotionality (assessed at 30 months) and emotional instability (assessed at age 12) on Borderline PD symptoms, assessed in adulthood. At both assessments, emotionality was associated with Borderline PD at outcome. Another study (Trentacosta & Shaw, 2008) investigated whether negative emotionality assessed in infancy predicted antisocial behaviour at 11/12 years in boys. They found this association to be significant. Taken together, these findings suggest that certain genetically influenced temperament traits, as assessed in childhood, are associated with PD in adulthood. However, these relationships were assessed univariately that is, the

relationship between temperament in childhood and PD in adolescence was investigated without taking into account the potential influence of other factors. Interactional models suggest that the interplay between temperament and environmental factors, such as parenting behaviours, influence the development of personality pathology. Therefore, whilst the results of the above studies do indicate a significant relationship between temperament and PD, one cannot infer whether these traits directly affect PD development, whether the effect can be explained by covariance with other factors, or whether it is only in interaction with environmental stressors, that they lead to a full diagnosis of the disorder.

1.3 Environmental risk factors for PD

Environmental risk factors for PD can be broadly grouped within the following interrelated concepts: (i) prenatal, perinatal and early postnatal risk factors; (ii) child maltreatment (abuse and neglect experiences); (iii) parent factors (negative parenting, insecure attachment, loss/early separation from parents, and parent psychopathology); and (iv) familial and socio-demographic adversity.

1.3.1 Prenatal, Perinatal and Early Postnatal Risk Factors

As suggested by Winsper, Wolke, and Lereya (2015), there are several potential mechanisms through which prenatal adversity may increase offspring vulnerability to the development of psychopathology. Firstly, prenatal adversity may permanently alter offspring organ structure and functioning, increasing the risk of mental illness in later life; i.e. there may be direct physiological effects on the foetus (Raikkonen & Pesonen, 2009; Schlotz & Phillips, 2009). Secondly, prenatal adversities may serve as markers for risk exposure in childhood. For example, prenatal anxiety and depression could portend maladaptive parenting in childhood (Lereya & Wolke, 2012). Thirdly, associations between prenatal adversities and later psychopathology could be partly attributable to continuing experience of the same risk during childhood, exposing the child to chronic stressors, increasing allostatic load and heightening the likelihood of mental illness (Hostinar & Gunnar, 2013).

Most of the evidence regarding the association between pre/peri/postnatal factors and personality pathology exists in the area of Cluster A PDs, in particular Schizotypal PD. This is mainly due to the link between Schizotypal PD and schizophrenia: Schizotypal PD is often viewed as a premorbid or prodromal stage, or an attenuated form, of schizophrenia. As such, it is regarded as a condition that can provide important insights into the origins of schizophrenia (Raine, 2006). Indeed, there is evidence that Schizotypal PD is genetically linked to the schizophrenia-spectrum; for instance, Schizotypal PD is elevated in the family members of schizophrenia patients (Siever & Davis, 2004) as well as in the relatives of those with Schizotypal PD and in the adopted offspring of mothers with schizophrenia-spectrum disorders (Battaglia, Bernardeschi, Franchini, Bellodi, & Smeraldi, 1995; Tienari et al., 2003). Based on evidence that prenatal, perinatal and early postnatal complications can increase the risk of offspring schizophrenia (Hultman, Sparen, Takei, Murray, & Cnattingius, 1999), these risk factors have also been studied in Schizotypal PD.

For example, prospective longitudinal studies show that exposure to influenza during the fifth (Venables, 1996) or sixth (Machón et al., 2002) month of pregnancy has been associated with Schizotypal PD symptoms, as has exposure to cold temperature during the second trimester (Venables, 1996), and prenatal malnutrition (Hoek, Brown, & Susser, 1998), as well as postnatal malnutrition (Venables, Raine, Dalais, Liu, & Mednick, 2005). Further, in a sample of undergraduates, high Schizotypal PD symptoms were significantly associated with retrospectively reported pregnancy and birth complications, in particular breathing problems or need for oxygen, artificial induction of labour, and breech birth (Bakan & Peterson, 1994). Another study showed that obstetric complications and low birth weight were associated with childhood premorbid Schizotypal PD and Schizoid PD traits in a retrospective study of adult psychosis patients (Foerster, Lewis, Owen, & Murray, 1991). Similarly, lower placental and birth weight, smaller head circumference at 12 months and lower gestational age were found to predict Schizotypal PD traits longitudinally (Lahti et al., 2009).

Cluster B and C PDs have not been as widely researched, but some evidence has been found that links these PDs with prenatal, perinatal and early postnatal complications. Regarding Cluster C, for instance, in a sample of

260 males and females detained in a maximum security hospital, it was found that Dependent PD and Avoidant PD were associated with perinatal complications (Coid, 1999). Regarding Cluster B, a longitudinal study showed that maternal substance use (smoking and/or drinking) during or shortly after pregnancy and low birth weight were related to adolescent antisocial behaviour (Bor, McGee, & Fagan, 2004). Further, malnutrition in the first or second, but not the third trimester of pregnancy, has been shown to be related to an increased risk for Antisocial PD (Neugebauer, Hoek, & Susser, 1999). Bandelow et al. (2005) demonstrated that Borderline PD patients retrospectively reported significantly higher rates (21.5%) of premature birth than a healthy control group. There were no significant differences between the two groups regarding other birth risk factors including age of mother or father over 35 years at childbirth, low birth weight, Caesarean section, or perinatal complications. Further, no significant effects were found after controlling for confounders (familial psychopathology, childhood sexual abuse, separation from parents and unfavourable parental rearing styles). However, another study investigating prenatal adversity as a potential risk factor for BPD showed different results (Schwarze et al., 2013). One hundred patients with a DSM-IV diagnosis of BPD were compared to 100 matched controls regarding the course of pregnancy, maternal stressors, birth complications and childhood trauma. This information was supplemented with information obtained from participants' mothers and from prenatal medical records. The results indicated that BPD patients were significantly more often exposed to adverse intrauterine conditions, such as tobacco exposure, medical complications, maternal traumatic stress, familial conflicts, low social support and partnership problems during pregnancy. Prenatal adversity accounted for 26% of the variance in BPD, the most important predictors being prenatal tobacco exposure and medical complications.

Similarly, recent data from the Avon Longitudinal Study of Parents and Children (ALSPAC) (Winsper et al., 2015) assessed associations between prenatal adversities (tobacco/alcohol consumption and depression/anxiety) and BPD at 11–12 years while controlling for relevant confounders. Exposure to the same risk factor during early childhood was controlled for, to assess whether prenatal adversity was an independent predictor of BPD

or a proxy for postnatal risk. (For example, when assessing associations between prenatal maternal depression and BPD, postnatal maternal depression was controlled for.) Maternal anxiety and depression, as well as alcohol and tobacco consumption was assessed during pregnancy. Postnatal risks, including maladaptive parenting and parent conflict, family adversity, maternal anxiety and depression and maternal alcohol and tobacco consumption, were assessed during early childhood. The results showed that all prenatal risk factors were significantly associated with later BPD. When controlling for sex, birth weight and postnatal exposure to anxiety and depression, maladaptive parenting and family adversity, prenatal anxiety and depression remained significantly associated with BPD.

1.3.2 Child Maltreatment

Child maltreatment is a summary category that includes several subcategories of abuse (emotional, physical, sexual) and neglect (e.g. physical, emotional). The effects of different types of childhood abuse/neglect are the most widely investigated environmental risk factor for PD. For instance, one study (Lobbestael, Arntz, & Bernstein, 2010) investigated the effects of different types of retrospectively reported abuse on all 10 PDs in a sample of psychiatric patients and controls. They found that sexual abuse was associated with Paranoid, Schizoid, Borderline, and Avoidant PD; physical abuse was associated with Antisocial PD; emotional abuse with Paranoid, Schizotypal, Borderline, and Cluster C PD; and emotional neglect with Histrionic and Borderline PD. Another study (Hengartner, Ajdacic-Gross, Rodgers, Müller, & Rössler, 2013), which examined the association between retrospectively reported child maltreatment in a general population-based community sample, found significant associations between abuse/neglect. However, different types of associations were observed: when investigated univariately, emotional abuse and neglect as well as physical abuse and neglect were significantly related to all 10 PD dimensions. These associations were also investigated multivariately, by adjusting all predictors for each other (i.e. other types of abuse/neglect and other variables such as poverty or parental divorce and substance abuse) and accounting for covariance between all PDs. Multivariately, emotional abuse was by far the strongest predictor, yielding

significant associations with most PD dimensions. Sexual abuse showed no practical significance due to the low effect sizes.

Longitudinal evidence comes from the Children in the Community (CIC) Study, which is the largest and best known prospective longitudinal study about PD, carried out by Patricia Cohen, Thomas Crawford, Stephanie Kasen and others. The CIC sample is a cohort of children originally aged 1-10 selected in 1975 from randomly sampled family households (N=976). Interviews covered a wide array of issues related to offspring well-being including the child's health, temperament, attitudes, behaviour, relationship with parents, and social environment, including parental problems, and significant events such as extended separations, divorce, and deaths of family members. The sample was assessed for PD symptoms at offspring mean ages 16, 22 and 33. Regarding childhood maltreatment, findings showed that documented childhood maltreatment was associated with increased risk for Antisocial PD, Borderline PD, Dependent PD, Narcissistic PD and Paranoid PD after controlling for offspring age, parental education, and parental psychiatric disorders (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Johnson, Cohen, Smailes, et al., 2001). Antisocial PD, Borderline PD and Narcissistic PD remained significantly associated with documented childhood maltreatment after controlling for symptoms of other PDs. Specific types of abuse were also investigated with relation to specific types of PD. Documented physical abuse was associated with elevated symptom levels of Antisocial PD, Borderline PD, Dependent PD and Schizoid PD after age, parental education, and parental psychiatric disorders were controlled statistically. Antisocial PD remained associated with physical abuse after symptoms of other PDs were controlled for. Documented sexual abuse was associated with elevated symptom levels of Borderline PD after offspring age and parental psychiatric disorders were controlled for. Documented childhood neglect was associated with elevated symptom levels of Antisocial PD, Avoidant PD, Borderline PD, Dependent PD, Narcissistic PD, Paranoid PD, and Schizotypal PD after controlling for offspring age, parental education, and parental psychiatric disorders. Antisocial PD, Avoidant PD, Borderline PD and Narcissistic PD remained significantly associated with documented neglect after co-occurring PD symptoms were controlled statistically. Johnson and colleagues (Johnson et

al., 1999; Johnson, Cohen, Smailes, et al., 2001) also investigated these associations between retrospectively recalled childhood abuse/neglect and PDs. Interestingly, the results looked quite different, with very little overlap between official records and retrospective reports of abuse/neglect. Out of 639 families, 31 had officially recorded maltreatment histories, and 81 self-reported child maltreatment. Only eight cases of maltreatment were identified through both official records and self-reports (agreement of kappa=0.11).

1.3.2.1 Child maltreatment and Borderline PD

Most of the research regarding child maltreatment has been conducted in the area of Borderline PD. Cross-sectional studies show that 30% to 90% of Borderline PD patients retrospectively report childhood abuse (Ball & Links, 2009; Bornovalova, Gratz, Delany-Brumsey, Paulson, & Lejuez, 2006; Carlson et al., 2009; Zanarini et al., 2000). Borderline PD patients report higher rates of both childhood abuse (Herman, Perry, & Van der Kolk, 1989; Soloff, Lynch, & Kelly, 2002; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989) and childhood neglect (Johnson, Cohen, et al., 2000; Zanarini et al., 1989) than individuals with other PDs (Zanarini et al., 1989) or other Axis I psychiatric disorders (Ogata et al., 1990). The association between childhood abuse and Borderline PD has been documented in a variety of samples, e.g. psychiatric inpatients (Bradley, Jenei, & Westen, 2005), psychiatric outpatients (Golier et al., 2003), urban drug users (Bornovalova et al., 2006), and community adolescents (Rogosch & Cicchetti, 2005). A recent study examined whether retrospectively reported childhood emotional abuse was uniquely associated with BPD when controlling for other forms of childhood abuse in a sample of undergraduates (Kuo, Khoury, Metcalfe, Fitzpatrick, & Goodwill, 2015). Results indicated that frequency of childhood emotional abuse (but not sexual or physical abuse) was uniquely associated with BPD feature severity. In addition, there was an indirect relationship between childhood emotional abuse and BPD features via difficulties with emotion regulation. The authors conclude that, of the different forms of childhood abuse, emotional abuse specifically, may have a developmental role in BPD pathology. However, most of the above evidence is based on retrospective reports of childhood abuse, limiting the

findings due to known issues with retrospective recall bias (Maughan et al., 1995; Maughan & Rutter, 1997; Robins et al., 1985).

Some studies have looked at the effect of childhood maltreatment on Borderline PD prospectively. For example, Helgeland et al. (2004) compared developmental antecedents in Borderline PD patients to non-Borderline controls on the basis of medical records compiled in childhood. They found that both abuse and neglect were significantly more common in Borderline PD than non-Borderline PD individuals. No conclusions can be drawn with regard to specific types of abuse and neglect and the development of Borderline PD, however, as they did not test for specific types of abuse/neglect. There is some evidence (Carlson et al., 2009) that early childhood maltreatment (assessed at 12-18 months) as well as physical and sexual abuse (but not neglect) assessed in later childhood and adolescence correlated with adult Borderline PD. As these relationships were not assessed multivariately, however, no conclusions could be drawn from the results from this study about influences of potential confounds. Belsky et al. (2012) investigated the effect of physical maltreatment on Borderline PD symptoms in 12 year old twins and found a significant association, which remained significant in a twin difference design, controlling for unmeasured family-level confounds: compared to non-maltreated co-twins, maltreated twins exhibited more Borderline PD symptoms. Nevertheless, as this study was based on children at outcome, it remains to be seen whether similar effects would be found in a sample of adults.

The childhood abuse type that has been most widely researched and most often been reported as a pathogenic factor for Borderline PD is childhood sexual abuse (CSA). CSA has been reported to be especially prevalent among adult patients with Borderline PD: 40% to 76% of Borderline PD patients report CSA, significantly higher than among groups of related disorders (Zanarini et al., 2000). One study found that significantly more Borderline PD individuals than depressed individuals retrospectively reported histories of CSA and physical abuse, with CSA emerging as the only significant predictor when controlling for other types of abuse, family environment and comorbid depression (Weaver & Clum, 1993). Another study demonstrated that it was particularly on-going CSA that was related to Borderline PD symptomatology in a clinical population (Silk, Lee, Hill, & Lohr, 1995).

Sansone et al. (2006) showed that even though a range of individual forms of trauma (e.g. witnessing violence, physical neglect, emotional abuse) were associated with Borderline PD, multivariately, they found that only CSA was an independent predictor of Borderline PD. Finally, it was also found that Borderline PD was associated with retrospectively reported sexual abuse, above and beyond the effects of co-occurring childhood maltreatment, perceived parenting style, Axis I symptoms, and non-Borderline PD criteria (Hernandez, Arntz, Gaviria, Labad, & Gutiérrez-Zotes, 2012).

However, even though there is a consensus among PD experts, and most of the evidence points towards a particularly strong association between CSA and Borderline PD, other studies have not confirmed this. For example, Zanarini, et al. (2000) found that Borderline PD patients retrospectively reported significantly more verbal, emotional and physical, but not sexual, abuse from both parents than patients with other PDs. Similarly, results from a prospective longitudinal study with a sample of individuals who had experienced CSA before age 11 (as evidenced through court case records) demonstrated that the risk for Borderline PD was not increased compared to controls (Widom, Czaja, & Paris, 2009). Rather, it was primarily physical abuse and neglect that increased the risk for Borderline PD. An additional study suggested that, whilst data shows that CSA is an important risk factor for Borderline PD, it is by no means necessary, nor sufficient, to develop the disorder: in a sample of depressed patients, 8% of participants who reported no abuse were diagnosed with Borderline PD; conversely, 67% of patients with severe abuse did not develop Borderline PD (Joyce et al., 2003). Fossati et al. (1999) meta-analysed the findings of studies on the effects of CSA on adult Borderline PD and reported only moderate pooled effect sizes which, they argued, did not support the theoretical formulations considering CSA as a major psychological risk factor or a causal antecedent of Borderline PD. Finally, Bornovalova et al. (2013) used a longitudinal twin design to examine the causal association between sexual, emotional, and physical abuse in childhood and BPD traits at age 24 using a discordant twin design and biometric modelling. Additionally, they examined the mediating and moderating effects of symptoms of childhood EXT and INT disorders on the link between childhood abuse and BPD. Although childhood abuse, BPD traits, and INT and EXT symptoms were all correlated,

the discordant twin analyses and biometric modelling showed little to no evidence that was consistent with a causal effect of childhood abuse on BPD traits. Instead, their results indicated that the association between childhood abuse and BPD traits stemmed from common genetic influences that overlapped with INT and EXT disorders. These findings are inconsistent with the widely held assumption that childhood abuse causes BPD; the authors suggest that BPD traits in adulthood are better accounted for by heritable vulnerabilities to INT and EXT disorders.

In general, the above evidence suggests that childhood maltreatment is a risk factor for the development of PD. However, evidence is diverse, with relationships between specific types of abuse/neglect and specific PDs not always consistent, studies supporting different relationships between PD and types of childhood maltreatment and different interactions with moderators. Finding clear and unambiguous antecedents for the overly specific PD categories is elusive because the impact of environmental factors such as parental neglect and emotional and sexual abuse are nonspecific and hence relate to several PDs. Further, the results are difficult to integrate due to the differences in types of adverse experiences assessed, instruments used, samples examined, and co-occurring variables controlled. Moreover, different forms of childhood maltreatment co-occur, and comorbidity among PDs is high. However, overall it seems clear that maltreatment is related to the development of PDs.

1.3.3 Parent Risk Factors

1.3.3.1 Negative parenting behaviours

When child maltreatment is noted, this is often only the tip of the iceberg and indexes pervasive difficulties in the family in caregiving and general parenting behaviours (Bradley et al., 2005; Fassler, Amodeo, Griffin, Clay, & Ellis, 2005). The family environment is generally considered to be one of the most important sources of socialisation for most children (Johnson, Cohen, et al., 2006). A large body of literature has identified the child's primary caregiver as the person to whom the child turns for comfort and regulation of stress (Sroufe, Carlson, Levy, & Egeland, 1999; Van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). Thus, the deficits that are evident among

individuals with PDs may result from problematic relationships with the parents, and evidence suggests that there is an association between unfavourable parenting and the development of PDs (Keinänen, Johnson, Richards, & Courtney, 2012).

In the extant research on parenting practices in relation to child behaviour there are several prominent theoretical models (see Power, 2013). Factor analytic studies obtained from psychometric assessment studies, and studies using independent observer ratings have identified two broad and universal dimensions of parenting (Grusec, Rudy, & Martini, 1997; Suchman, Rounsaville, DeCoste, & Luthar, 2007). These are parental control (overcontrol vs lack of control) and parental warmth (i.e. warmth/sensitivity vs lack of affection/indifference). Both these parenting dimensions have been found to have a strong influence on children's adjustment: for instance, research has revealed a link between low levels of parental warmth and externalising problems (Lee & Gotlib, 1991; Shaw et al., 1998). Low levels of warmth (e.g., lack of support or involvement) has been argued to interfere with a child's capacity to modulate and regulate arousal, and consequently, with a child's capability of considering the consequences of his/her actions (Chang, Olson, Sameroff, & Sexton, 2011; Eisenberg et al., 2005; McKee, Colletti, Rakow, Jones, & Forehand, 2008; Walton & Flouri, 2010). In addition, studies have reported significant associations between low levels of warmth and high levels of internalising problems in children (Garber, Robinson, & Valentiner, 1997; Hammen, Shih, & Brennan, 2004). It has been suggested that children learn to avoid the dysregulation that results from insensitive or unresponsive parenting (i.e., parenting characterised by a lack of warmth) by withdrawing (Tronick & Gianino, 1986). This internalising response may become the child's preferred coping strategy which in turn has been suggested to place the child at risk for developing a number of symptoms related to internalising disorders (Field, 1995).

There is some evidence for the detrimental effects of negative parenting on the development of PD. For example, results from the CIC study demonstrate that maladaptive parenting behaviours significantly increase the risk for PD in early adulthood independent of earlier childhood difficult behaviour and psychiatric disorder (Johnson, Cohen, et al., 2006; Johnson,

Cohen, Kasen, Smailes, & Brook, 2001). Specifically, problematic rearing styles (e.g. harsh punishment, low expression of affection) were significantly associated with adult PD, even after the influence of childhood behavioural and emotional problems and lifetime psychiatric disorders was taken into account. Further, risk for offspring PD increased steadily as a function of the number of problematic parenting behaviours. Specifically, aversive parental behaviour was associated with elevated risk for offspring Borderline PD, Paranoid PD and Schizotypal PD, and after controlling for the covariates with Borderline PD, Narcissistic PD, Paranoid PD, Schizotypal PD and Schizoid PD. Low parental affection or nurturing was associated with elevated risk for offspring Antisocial PD, Avoidant PD, Borderline PD, Paranoid PD, Schizoid PD and Schizotypal PD, and after controlling for covariates with Antisocial PD, Avoidant PD, Borderline PD, Dependent PD, Histrionic PD, Narcissistic PD, Paranoid PD, Schizoid PD and Schizotypal PD symptoms. The results indicate that certain types of parental child-rearing behaviours are associated with the development of PDs in adulthood, but no unique associations could be established.

The negative influence of maladaptive parenting has been most widely researched in the area of Borderline PD. Many PD experts have offered theories about how on-going deviations in parent-child interactions are likely to be associated with Borderline PD symptoms (Fonagy & Luyten, 2009; Fruzzetti, Shenk, & Hoffman, 2005; Linehan, 1993). One of the most influential theories by Linehan (1993) argues that 'emotionally invalidating' family environments during childhood, in combination with a difficult temperament disposition, may lead to chronic patterns of emotion dysregulation, including impulsive self-damaging behaviour, which can culminate in a diagnosis of Borderline PD in adulthood. This theory is generally supported by evidence: for instance, Zanarini, et al. (2000) compared patients with Borderline PD to patients with other PDs. They found that, consistent with the 'emotionally invalidating environment' theory, patients with Borderline PD reported significantly more often than the controls that both their parents denied their right for their own thoughts and feelings, that they did not protect their children, and treated them inconsistently. Schuppert et al. (2012) investigated differences in retrospectively reported parenting style in a group of referred adolescents

with Borderline PD features and healthy controls. The Borderline PD group reported significantly less emotional warmth, more rejection and more overprotection from their mothers than the control group. Hierarchical logistic regression revealed that some of these parental rearing styles, specifically less emotional warmth, and more overprotection, strongly differentiated between controls and adolescents with Borderline PD symptoms. Another study prospectively investigated the effects of negative parenting behaviours on Borderline PD symptoms in a community sample of over 6000 mothers and their 11 year old children (Winsper, Zanarini, & Wolke, 2012). Negative parenting behaviours such as hitting, resentment and hostility were significantly associated with Borderline PD symptoms. After controlling for confounders, suboptimal parenting led to higher odds for Borderline PD symptoms, and path analysis showed a direct relationship of suboptimal parenting on Borderline PD. However, some opposing evidence about the relationship between parenting and Borderline PD exists: one study examined the relationship between parenting style and Borderline PD criteria in 126 inpatient and outpatient females aged between 18 and 65 years (Hernandez et al., 2012). They found that, after controlling for childhood maltreatment, Axis I symptoms, and non-Borderline PD criteria, parenting style (overprotection and parental care) was not significantly associated with Borderline PD symptoms.

Whilst not many studies have been published that investigate the effect of maladaptive parenting on Antisocial PD, a large body of evidence exists about the association between parenting and Antisocial behaviour, conduct problems and delinquency assessed in adolescents/adults. As these behaviours are core aspects of a diagnosis of Antisocial PD, inferences regarding the association between negative parenting and Antisocial PD can be made. Evidence strongly suggests that maladaptive parenting affects the development of antisocial behaviour, and it is especially negative parenting in early childhood rather than later childhood that influences the development of antisocial behaviour. For instance, one study prospectively investigated the effects of maladaptive parenting assessed in early childhood, as well as at age 13, on Antisocial behaviour at age 16 (Aguilar, Sroufe, Egeland, & Carlson, 2000). It was found that early mother-child interactions, but not those at age 13 years, predicted antisocial behaviour at

age 16 years. Similarly, Trentacosta & Shaw (2008) prospectively investigated the effect of maladaptive parenting in early childhood on early adolescent antisocial behaviour in offspring of low-income mothers and their sons. They found that maternal hostile and controlling responses to toddler noncompliance predicted offspring self-reported antisocial behaviour at 11 years. One of the few studies using diagnostic criteria for Antisocial PD supported the association between negative parenting and antisocial behaviour (Horwitz, Widom, McLaughlin, & White, 2001). It was found that low parental affection/nurturing, assessed by self-report at age 16 years, was associated with Antisocial PD at age 22.

Even though there is some evidence for the notion that fathers play an important role in their children's social and behavioural development (Harper & McLanahan, 2004; King, 1994; Lamb, 1997; Patterson & Dishion, 1988), the majority of studies on the influence of parenting practices on young children's development have focused on mothers. The general assumption is that maternal negative parenting affects the development of the child more strongly than paternal negative parenting (Enns, Cox, & Clara, 2002; Kimbrel, Nelson-Gray, & Mitchell, 2007). However, even though fewer studies have investigated fathers' influence, research has revealed that negative parenting by both the mother and the father affect the child's outcome (Black, Dubowitz, & Starr, 1999; Kelley, Smith, Green, Berndt, & Rogers, 1998; Lamb, 1997; Tamis-LeMonda, Shannon, Cabrera, & Lamb, 2004). For instance, it has been shown that (lack of) parental warmth of both mothers and fathers was significantly related to the children's externalising and internalising problems after divorce (Sandler, Miles, Cookston, & Braver, 2008). Further, studies have demonstrated that parenting by the mother and by the father affect children differently. For instance, Cabrera et al. (2007) found that paternal (lack of) warmth/supportiveness influenced the child much more strongly than maternal lack of warmth, whereas maternal overcontrol/intrusiveness had much stronger effects on the child than paternal overcontrol/intrusiveness. However, research assessing the differential effects of negative parenting by the mother and by the father on PD is mostly lacking.

1.3.3.2 Insecure Attachment

It has been argued that maladaptive parenting behaviours negatively influence the development of personality pathology due to the attachment patterns the child learns from the interaction with the parents. Attachment theory (Bowlby, 1973) suggests that early life experiences with caregivers provide infants with an internal working model about relationships and interpersonal functioning. Attentive and nurturing caregivers provide the infant with the expectation that others are reliable, trustworthy and responsive. Abusive, neglectful or unresponsive caregiving may result in the expectation that others will not respond to or meet one's need for love and care. It is suggested that these early attachment styles affect the experience of interpersonal relationships in adulthood (Sroufe et al., 1999).

Attachment in infancy is assessed via the "strange situation" (Ainsworth, Blehar, Waters, & Wall, 1978) which is a well-known laboratory procedure that assesses how infants respond to separations from the mother, exposure to an adult stranger and reunions with the mother. Three variations of insecure attachment patterns have been identified (Ainsworth et al., 1978; Main & Solomon, 1986): Avoidant attachment, where infants are indifferent or ignore the return of the caregiver after separation; anxious/ambivalent attachment, where infants seek contact with the caregiver but fail to be soothed by him or her; and disorganised attachment, where infants lack a coherent pattern of responding to separation and reunion and display contradictory behaviour patterns, disorganisation, and disorientation. In adults, insecure attachment styles can be construed as individual differences on two orthogonal dimensions: anxiety, indicating the need for approval and the fear of rejection and abandonment; and avoidance, indicating avoidance of intimacy and discomfort with closeness and dependence on others (Brennan & Shaver, 1998). Different combinations of anxious and avoidant attachment styles classify different types of insecure attachment in adults, namely 1. Preoccupied attachment (high anxiety and low avoidance); 2. Dismissing attachment (low anxiety and high avoidance) and; 3. Fearful attachment (high anxiety and high avoidance) (Bartholomew & Horowitz, 1991). In addition, a fourth insecure attachment style is identified and assessed in adults in the Adult Attachment Interview,

namely unresolved/disorganised attachment (Main & Goldwyn, 1998) which shows trauma resulting from unresolved loss or abuse.

Insecure attachment styles have been suggested to provide a useful conceptual framework for understanding the interpersonal dysfunction that is salient in PDs (Bartholomew, Kwong, & Hart, 2001; Meyer & Pilkonis, 2005). Most of the evidence about attachment and PD comes from cross-sectional studies investigating current attachment patterns in adolescents or adults, arguing that current attachment style reflects attachment patterns learnt in childhood. For instance, one study demonstrated that individuals characterised by fearful attachment had the highest likelihood of at least one PD diagnosis, with a particular risk for Avoidant PD, as well as Borderline PD, Paranoid PD and Schizotypal PD symptoms (Brennan & Shaver, 1998). The association found between these PDs and attachment constructs fits well with attachment theory and emphasises how worries about close relationships are closely linked with emotional dysregulation: These specific PDs are associated with worries about abandonment (Borderline PD), rejection (Avoidant PD) or being harmed by others (Paranoid PD and Schizotypal PD). Some studies have found a specific relationship between preoccupied attachment and Avoidant PD and Dependent PD, and between dismissing attachment and Schizoid PD, Narcissistic PD, Antisocial PD and Paranoid PD (Fossati et al., 2003; Livesley, 1987; Livesley, Schroeder, & Jackson, 1990; West, Rose, & Sheldon-Keller, 1994).

In adolescent samples, whilst there was some overlap with adult samples, slightly different patterns have been observed. One study showed that adolescents with a dismissive attachment style were at a particularly elevated risk for Narcissistic PD and Antisocial PD, and adolescents with a preoccupied attachment style were more likely to have Histrionic PD, Borderline PD or Schizotypal PD (Rosenstein & Horowitz). Another study found that fearful attachment was associated with all PDs, particularly with Borderline PD; avoidant attachment was most strongly associated with Cluster A PDs; and anxious/ambivalent attachment was associated with Borderline PD, Histrionic PD and Dependent PD (Nakash-Eisikovits, Dutra, & Westen, 2002). Avoidant attachment was not associated with any form of PD. Crawford et al. (2006) assessed attachment in a community sample

during adolescence and adulthood and found that, across a 17-year interval, Cluster B and C symptoms were associated with elevated anxious attachment. Avoidant attachment in adolescence was positively associated with Cluster A symptoms and inversely associated with Cluster B and C symptoms in adulthood.

A lot of research in the area of attachment has focused specifically on Borderline PD. For instance, Fonagy et al. (1996) demonstrated that inpatients with a Borderline PD diagnosis were characterised more frequently by an insecure attachment style than matched controls; 92% of Borderline PD patients were assessed as having insecure attachment types, especially the preoccupied and unresolved types. Similarly, another study found that only 7% of the Borderline PD group had a secure attachment style, 20% were dismissing, 23% preoccupied, and 50% unresolved (Barone, 2003). In a further study, insecure attachment uniquely predicted Borderline PD, even after controlling for gender, childhood traumatic experience, and Axis I mental disorders (Nickell, Waudby, & Trull, 2002). Agrawal et al. (2004) systematically reviewed 13 studies about attachment patterns in Borderline PD in adulthood. Even though comparability was difficult due to a wide range of attachment style measures across studies, as well as differences in samples, comparison groups and types of relationships that were investigated (peer, parent, others), every study concluded that there is a strong association between Borderline PD and insecure attachment. The types of attachment found to be most characteristic of Borderline PD subjects were unresolved, preoccupied, and fearful.

Thus, available data supports the notion that insecure attachment is associated with PD, albeit with mixed results regarding any unique associations between particular attachment styles and specific PDs. Other studies have not supported this, however. For example, one study demonstrated that attachment security (assessed at 18 months) was unrelated to Antisocial PD features in adulthood (Shi, Bureau, Easterbrooks, Zhao, & Lyons-Ruth, 2012). Another study found that disorganised attachment at age 3 did not add to prediction of adult Borderline PD over and above the effects of other parenting variables (Carlson et al., 2009). Lyons-Ruth et al. (2013) prospectively investigated whether infant attachment security independently predicted Borderline PD symptoms in

adulthood or whether the effects could be explained by parenting or by abuse later in childhood. Interestingly, they found that whilst security of the infant's attachment behaviour did not predict Borderline PD symptoms, the mother's reaction to the child's attachment cues, specifically withdrawal, did predict Borderline PD. Maternal withdrawal behaviour to the infant's attachment cues emerged as the most important prospective predictor of later Borderline PD symptoms. This suggests that insecure attachment may be mostly linked to PD as a result of the maladaptive behaviours of the parent towards the child.

Taken together, evidence suggests that there are strong links between insecure attachment and PD, and attachment patterns provide useful conceptual frameworks for understanding the interpersonal dysfunction that is salient in PDs (Bartholomew et al., 2001; Fonagy & Bateman, 2005; Meyer & Pilkonis, 2005). However, these associations are rather unspecific as multiple types of attachment are linked with different PDs, and findings have not been replicated consistently. Moreover, even where links are better established as is the case with Borderline PD and preoccupied attachment, the claim that systematic relations have been found cannot be made, as this attachment style seems to be overrepresented also in other clinical groups, such as depressed patients (Van IJzendoorn & Bakermans-Kranenburg, 1996).

1.3.3.3 Loss/early separation from parents

Evidence suggests that early separation from caregivers may predict elevations in PD symptoms. Bowlby (Bowlby, 1969, 1973) argued that early separations are significant threats to emotional development and that extended separations undermine the emotional security infants or toddlers normally experience when they are closely attached to primary caregivers. Young children rely heavily on caregivers to be available, sensitive, and responsive to their needs, especially as their own coping resources are developmentally immature. As such, separations from a parent are not just alarming in early childhood; the distress is compounded when infants and toddlers have limited ability to modulate potentially overwhelming emotions on their own.

The effects of separation from parents on the development of adult PD have been investigated both cross-sectionally and longitudinally. For instance, one study examined the effects of separations from parents on PDs and tested whether early (before the age of five) or long separations were especially strongly linked to PD (Lahti et al., 2012). Overall, they found that separations significantly increased the risk of PDs. This effect was particularly strong in women overall, whereas men who experienced separations were at an increased risk of Cluster B PDs in particular. The effects of separations were especially characteristic of those separated before the age of five, while separation duration did not predict the risks for PDs.

Separation from parents in childhood was found to be related to specific PDs, mainly Borderline PD and Antisocial PD, presumably because research was carried out mostly about these two PDs. For example, one study compared traumatic childhood experiences in the psychiatric records of 751 females aged 16–45 with a discharge diagnosis of Borderline PD with those of women with other PDs (Laporte & Guttman, 1996). They found that the Borderline PD group experienced more losses than women with other PDs. Over 93% of the Borderline PD participants experienced at least one form of separation or abuse in childhood. Logistic regression demonstrated that a history of adoption was one of the most important risk factors for the development of Borderline PD. With regard to Antisocial PD, it was found that early separation from parents (being adopted or raised in foster care/by relatives or parental death) was specifically associated with Antisocial PD in a sample of 260 males and females detained in a maximum security hospital (Coid, 1999). On the other hand, another study that investigated whether retrospectively reported experiences of separation from parents differed in patients diagnosed with Avoidant PD and controls found no differences between groups (Arbel & Stravynski, 1991).

Other studies have provided evidence for a more complex relationship between the effects of early separations from parents and PD. For instance, the CIC study showed that, rather than separations as such, it was the reasons for early separation that played a significant role in the development of PD (Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009). Mothers provided data on early separations when children were on average

5 years old; Borderline PD was assessed 8 years later and at three subsequent data points over the next 20 years. Initially, it was found that children with early separations had significantly higher Borderline PD symptoms than those not separated. Furthermore, whilst for the whole sample Borderline PD symptoms declined with age, the level of Borderline PD symptoms declined less for those who had extended early separations. However, when comparing the effects of early separations due to different reasons, significant differences were found. Separations due to illness of mother or child did not predict Borderline PD, but separation due to reasons such as extended visits to a relative, or due to personal or professional reasons, predicted both higher Borderline PD symptoms as well as slower developmental declines in symptoms. Further, inconsistent parenting and maternal dissatisfaction with the child mediated the effect of early separation enough to reduce the estimated independent effect on mean symptoms to a level that was no longer statistically significant. Based on these findings, Crawford et al. (2009) argued that separation might be a long-term risk only insofar as it reflects a lack of maternal investment in caregiving, rather than a risk factor in itself. Another study compared Borderline PD patients with healthy controls with regard to traumatic life events during childhood (Bandelow et al., 2005). No significant differences between the groups were found with regard to separations from the mother, but the absence of the father was reported more often by the Borderline PD subjects. With regard to reasons for separations, absence due to war service or due to death of father had no effect on Borderline PD, but absence for other reasons was more frequent in the Borderline PD group than the control group.

1.3.3.4 Parent psychopathology

Another major risk factor for the development of PD is parental psychopathology. As discussed above, the risk of developing personality pathology is partly heritable. Further, genetically determined "difficult" temperament and environmental stressors, such as maladaptive parenting behaviour, interact and increase the risk of a PD. All of these risk factors may be increased in children of parents with psychiatric problems: 1. These children may have inherited the genetic disposition to psychiatric problems themselves. 2. They may have inherited temperament traits which make

them more vulnerable to environmental stressors; 3. Parent/s with psychiatric problems may be more prone to interact with the child in a way that might influence the development negatively. Evidence has shown, for instance, that parents with a diagnosis of Borderline PD tend to oscillate between extreme forms of angry hostility and passive aloofness in their interactions with their children (Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012). In addition, a community-based cross-sectional study which explored the relationship between parental PD and child maltreatment indicated that mothers with diagnosed BPD were more likely than those with sub-clinical BPD features and those with no significant features to have engaged in child maltreatment (Laulik, Allam, & Browne, 2014). In short, parent psychopathology may increase the chances of offspring psychopathology based on both genetic and environmental factors. Evidence from cross-sectional studies has identified parental psychopathology as a particularly strong risk factor for the development of PD. One study investigated the effects of family history of mental disorders in first degree relatives (depression, schizophrenia, alcoholism, learning disability or PD) on PD in a sample of 260 males and females detained in a maximum security hospital (Coid, 1999). They found that, overall, PD was associated with a family history of mental illness. Specifically, they demonstrated that Antisocial PD was associated with a family history of PD and that Borderline PD was associated with family history of depression; Schizoid PD, however, was negatively associated with a family history of mental disorder. Another study focused on the effects of maternal Axis I and II psychopathology (assessed through maternal self-report) in adolescents with Borderline PD features (Schuppert et al., 2012). They found that mothers in the Borderline PD group reported significantly more anxiety, depression and cluster C personality traits than mothers in the control group, and maternal psychopathology strongly differentiated controls from adolescents with Borderline PD symptoms. Bandelow et al. (2005) compared family histories of psychiatric disorders in Borderline PD patients and healthy controls. They found that Borderline PD patients reported significantly higher rates of psychiatric disorders in their families in general; anxiety disorders, depression, and suicidality (but not schizophrenia) in first degree relatives showed the largest differences from the control group. Similarly, another study showed that parental mental illness was a significant and unique

predictor of Borderline PD scores compared to controls (Trull, 2001). This association remained significant when controlling for the effects of childhood physical and sexual abuse and lifetime Axis I disorders.

These findings were supported through prospective longitudinal studies. For instance, results from the CIC study showed that paternal and maternal sociopathy were each independently related to later PD symptoms (Cohen, 1996). Similarly, Farrington et al. (2000) found that maternal psychopathology (assessed at offspring age 8-10) significantly increased the risk for the development of Antisocial PD in early and middle adulthood. Lahey et al. (2005) looked at the effects of parental Antisocial PD, assessed at offspring age 7-12, on Antisocial PD, assessed at offspring age 18/19. They found that maternal but not paternal Antisocial PD predicted Antisocial PD in offspring. In addition, Winsper et al. (2015) showed that, in a community study with over 6,000 mothers, both pre- and postnatal maternal anxiety and depression were associated with later BPD. In sum, the notion that parental psychopathology increases the risk for offspring PD was supported by results from both cross-sectional and longitudinal studies.

1.3.4 Family and Socioeconomic Adversity

Risk factors relating to the parents of course need to be regarded within the context of the wider family system and socioeconomic environment of the family. Stressors such as poverty, a non-intact and chaotic family structure, conflict and/or violence between parents, or parental separation/divorce, have been identified as a risk factor for the development of PD. For instance, one study compared retrospectively reported rates of family adversity in a case-control study with offender patients diagnosed with either schizophrenia or with a PD admitted to a high-security hospital (Gibbon, Ferriter, & Duggan, 2009). Compared with those with schizophrenia, patients with a PD had experienced higher rates of family criminality, parental separation, and multiple changes of caregiver and institutional care. Less than a third of the PD group had experienced childhood without a change in parenting. Another cross-sectional study investigated the effects of parental separation and parental conflict, and being raised in poverty, in a sample of 260 males and females detained in a

maximum security hospital, which they found to affect the development of Antisocial PD and Borderline PD (Coid, 1999). Bandelow et al. (2005) compared Borderline PD patients with a healthy control group with regard to retrospective reports of traumatic childhood events. They showed that patients reported significantly more often than controls that their parents had had marital discord or separations. In general, violence was reported significantly more often in the patients' families, especially more violence of the father against the mother (but not vice versa). However, when controlling for confounders (familial psychopathology, childhood sexual abuse, separation from parents and unfavourable parental rearing styles) these effects became non-significant.

Similarly, a prospective study investigated the influence of non-intact family structure and low parental education level in childhood (assessed at age 8) on Antisocial PD (Sourander et al., 2005). It was found that both factors, especially nonintact family structure, strongly increased the risk for Antisocial PD. Another study utilised data from an Australian longitudinal study to identify, amongst other factors, whether marital instability was an early risk factor for adolescent antisocial behaviour (Bor et al., 2004). Over 8000 participants were assessed based on maternal reports, child assessments and medical records, and adolescent antisocial behaviour was measured when children were 14 years old. They found that marital instability doubled or tripled the odds of antisocial behaviour.

In the CIC study, Cohen et al. (2008) studied the effects of socio-economic status (SES)-associated risks on the level of symptoms of Schizotypal PD and Borderline PD and compared these to the effects on depressive symptoms. They found that low family SES in childhood had modest but robust independent effects on both Borderline PD and Schizotypal PD in middle adulthood, despite substantial cumulative effects of trauma history, stressful recent life events, IQ, poor parenting, and comorbid symptoms. SES effects on depressive symptoms, on the other hand, were generally absent in this study. Parental conflict, however, did not predict Borderline PD longitudinally when investigated together with other variables such as child temperament, negative parenting and child maltreatment (Crawford et al., 2009).

A population based prospective longitudinal study tested the effects of several early adversity factors on the development of Borderline PD symptoms in late childhood (Winsper et al., 2012). Adversity was assessed through young maternal age, financial difficulties, problematic partner relationship, maternal affective disorder, substance abuse, or involvement in crime. It was found that family adversity had a direct significant impact on Borderline PD symptoms. They also found a dose–response effect with an increase in family adversity leading to increased odds of Borderline PD symptoms. Finally, Farrington et al. (2000) prospectively investigated the influence of psychosocial factors, assessed at age 8-10, on the development of Antisocial PD, assessed at age 18 and age 32. They found that socioeconomic factors (low income, poor housing, low social class and large family size) significantly increased the risk for Antisocial PD.

1.4 Interaction between Genetic and Environmental Influences

Thus far, this chapter presented evidence about genetic and environmental risk factors associated with the development of PD. Of course these factors are intertwined and can only be separated theoretically. Genes determine the extent to which individuals are sensitive to influences from the environment (Caspi et al., 2002), i.e. psychiatric problems are built on genetically determined latent vulnerabilities that interact with a variety of environmental factors and conditions (De Fruyt & De Clercq, 2012).

Evidence about the interaction of biological and environmental risk factors comes from the CIC study (Crawford et al., 2009) which showed that maternal reports of childhood temperament at age 9 predicted higher Borderline PD symptoms in adulthood when controlling for childhood trauma. Specifically, impulse aggression, as indexed by angry tantrums at age 9, and high emotionality, as indicated by frequent crying, mood reactivity, and demands for attention, predicted Borderline PD symptoms in adolescence and adulthood, supporting the notion that temperament in childhood is associated with adult PD. Further, temperament risks were mediated by inconsistent mothering and maternal dissatisfaction with the

child, supporting the notion that it is difficult temperament in combination with environmental stressors that increase the risk of a PD in adulthood.

Another study investigated cross-sectionally, whether the effect of impulsive traits on antisocial behaviour varied across neighbourhood context in a population-based sample of 85,000 schoolchildren aged 10–19 (Meier, Slutske, Arndt, & Cadoret, 2008). Results suggested a robust moderating effect of neighbourhood context on impulsivity risk for antisocial behaviour. Specifically, the relation between impulsivity and delinquency was greater in high risk neighbourhoods compared to low risk neighbourhoods. Finally, a recent review looking at gene-environment interaction studies in BPD showed that, even though almost all of the included studies suffered from methodological and statistical issues, the best evidence supported a gene-environment correlation (rGE) model, indicating that those at risk for BPD are also at increased risk for exposure to environments that may trigger BPD (Carpenter, Tomko, Trull, & Boomsma, 2013).

Thus, there is evidence that genes determine the extent to which individuals are sensitive to influences from the environment (Caspi et al., 2002). However, it has also been suggested that environmental events regulate gene transcription (Bagot & Meaney, 2010) and shape brain architecture and functioning. Further, there is evidence from twin research indicating that environmental variables are partly genetically determined (Vinkhuyzen, Van Der Sluis, De Geus, Boomsma, & Posthuma, 2010). In line with this is recent evidence indicating those factors that are considered "environmental" influences on PD can be explained by genetic influences. A recent study tested discordant twin and biometric models to evaluate the effects of genetic and environmental influences on the association between child abuse and adult BPD (Bornovalova et al., 2013). They used a large sample of twins followed longitudinally from age 11 to 24. They showed that monozygotic twins discordant for child abuse had similar levels of Borderline traits, while dizygotic twins discordant for child abuse differed significantly regarding their levels of Borderline traits. These results suggest that an association between child abuse and Borderline PD traits is likely mediated by common genetic factors. Further, their biometric analysis provided corroborating evidence for genetic mediation effects in the

association between child abuse and Borderline PD. Genetic effects accounted for a small but statistically significant amount of variance in child abuse and, though the genetic effect was modest, genetic factors accounted for most of the association between Borderline PD and abuse. They also tested the validity of diathesis-stress models by investigating whether any interactions with child EXT or INT problems existed. Finding stronger effects of maltreatment in children with EXT or INT symptomatology would have supported the validity of interactions between child dispositions and environmental stressors. However, they did not find any significant interactions, arguing against the validity of interaction models. The authors conclude that the results provide evidence that the association between exposure to traumatic events and Borderline PD may be best accounted for by common genetic influences rather than traumatic events causally influencing Borderline traits. They further argue that their data speaks against a causal influence of childhood abuse on Borderline PD, but rather that the observed associations can be explained genetically. These findings are in line with Vinkhuyzen et al.'s (2010) argument that environmental factors might be better described as "external factors that might be partly under genetic control" (p. 285).

1.5 Protective factors

The developmental pathways to PD, and the relationship between genetic vulnerability and adverse environmental experiences leading to PD, are complex. It seems clear that genetic vulnerability increases the likelihood that a PD will be diagnosed later in life and that, similarly, environmental stressors heighten the likelihood that personality pathology will develop. Epidemiologic studies indicate, however, that, for many these environmental stressors have a less direct impact on mental health outcomes later in life than might be expected (Rind, Tromovitch, & Bauserman, 1998). Further, only about half of the patients with PDs retain these diagnoses over follow-up periods ranging from 6 months to 15 years (McDavid & Pilkonis, 1996; Perry, Banon, & Ianni, 1999) and significant rates of improvement in PD symptoms over time have been found in patient (Grilo et al., 2004; Shea et al., 2002), non-patient (Lenzenweger, 1999), and community (Johnson, Cohen, et al., 2000) populations. These findings imply that protective

factors may exist that shield a child with genetic vulnerabilities from negative outcomes, or enable some to get better.

Protective factors can be broadly grouped as positive family factors (including parenting behaviours), social support, and individual child characteristics. Positive family factors and social support have been found to be associated with the development of adaptive traits which in turn are likely to mediate whether individuals adapt effectively to any experienced adversities (Garmezy, 1985; Shiner, 2000). Not much research has been carried out in the area of PD specifically, but evidence suggests that parental empathy, support and warmth helps children to cope effectively with many types of adversity (Cowen, 1994; Luthar & Zigler, 1991). Strong and supportive relationships with family members are associated with healthy interpersonal functioning (Werner & Smith, 1982). Social support outside the family has also been found to facilitate the development of adaptive personality traits (Garmezy, 1985). The presence of a mentor during adolescence has been found to be associated with improved academic achievement, attitudes about school, relationships with parents and peers, and self-esteem, and with reductions in aggressive behaviour and substance use (Wolkow & Ferguson, 2001; Zimmerman, Bingenheimer, & Notaro, 2002). These protective environmental factors of course interact with the temperamental dispositions of the child (Kendler, 1996), and some may benefit more from certain experiences than others. For example, individuals with externalising tendencies may need more parental supervision whereas internalising problems may need more parental warmth and support (Johnson, Bromley, & McGeoch, 2005). Individual characteristics that enable those who experience negative events to cope adaptively have also been identified, such as intelligence, optimism, self-confidence, self-efficacy, sociability, internal locus of control, and an active coping style (Cowan, Cohn, Cowan, & Pearson, 1996; Klohnen, 1996; Luthar & Zigler, 1991; Shiner, 2000; Werner & Smith, 1982).

Research about protective factors specifically for PD is limited. The CIC study (Johnson, Cohen, Kasen, et al., 2001) focused on specific parenting behaviours that may protect children from developing PD. This was investigated by testing the association of specific parenting behaviours of mothers and fathers with adaptive personality traits in adulthood, namely

confident optimism, insight and warmth, productive activity, and skilled expressiveness. Parenting behaviours assessed were affection, communication, time spent with child, praise and encouragement, parental role fulfilment, tendencies to speak kindly to the child, recreational activities with the child, tendencies to respond calmly to needs, attentiveness and dedication, encouragement of autonomy. They found that all types of maternal and most of the paternal parenting behaviours were associated with offspring's healthy personality in adulthood. These relationships remained significant when controlling for offspring's age and sex, behaviour problems, and parental mental health. Another more recent study examined whether education and coping strategies reduced the detrimental effects of childhood maltreatment on PD in a general population sample (Hengartner, Muller, Rodgers, Rossler, & Ajdacic, 2013). They found that low education was related to Antisocial, Borderline, Schizotypal and Histrionic PDs, whereas low emotion-focused coping was associated with Paranoid, Schizoid, Borderline, Avoidant and Obsessive-Compulsive PDs. Low problem-focused coping was related to Schizoid PD, and high problem-focused coping to Histrionic PD. High dysfunctional coping was significantly related to all 10 PDs. Obsessive-Compulsive PD scores were significantly lower in maltreated subjects with high emotion-focused coping. Antisocial, Borderline and Narcissistic PD scores were significantly higher in maltreated subjects with high dysfunctional coping. The authors concluded that education and adaptive coping may have a protective effect on PD symptomatology.

Skodol et al. (2007) investigated the effects of retrospectively reported positive experiences in childhood or adolescence (achievements, positive relationships with others, caretaker competencies) on remission from PD in a longitudinal study with adults diagnosed with a PD. They found that univariately, achievement (extracurricular activities, leadership, work and popularity) was related to remission of Avoidant PD, and positive relationships were related to remission from Avoidant PD, Borderline PD and Schizotypal PD. Caretaker competence was related to remission from Avoidant PD only. Achievement continued to predict remission from Avoidant PD and Schizotypal PD when the effects of other positive experiences, age, gender, and maltreatment experiences were controlled for.

No positive experiences were found to predict Borderline PD when controlling for confounders. The effects of positive experiences on the other PDs were not tested.

1.6 Risk markers

In order to develop and implement prevention and early intervention approaches it is not only important to identify early risk factors, but also risk markers. Whilst risk factors, as discussed above, are factors that independently or in combination increase an individual's risk of developing a certain disorder, risk markers are early warning signs that an individual may be on the pathway of developing a disorder. As such, these risk markers can be regarded as early symptoms of a later disorder and may be useful indicators for who might benefit from early intervention or prevention approaches. Further, these early symptoms may become targets for treatment themselves. Because the DSM-IV classification of disorders in childhood and adolescence was restricted to Axis I psychopathology, most of the childhood risk markers for PD are common Axis I childhood disorders such as Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD) (Dowson, Sussams, Grounds, & Taylor, 2001), mood disorders (Kasen et al., 2001), anxiety disorders (Bienvenu & Stein, 2003) and attention-deficit disorders (Young, Gudjonsson, Ball, & Lam, 2003). As such, these common childhood disorders may be the most useful predictors for PD currently available.

1.6.1 Externalising childhood problems

Research about child EXT problems and PDs has mostly been carried out in the area of Borderline PD and Antisocial PD where strong associations were consistently found. For instance, Burke & Stepp (2012) showed that ODD and ADHD, but not CD, predicted Borderline PD in a sample of men (age 24) first assessed at age 7-12, controlling for all other PDs, drug abuse and age. Similarly, another study examined the developmental links between childhood ADHD and ODD (assessed at ages 8 and 10), and Borderline PD (assessed at age 14) in a large sample of girls (Stepp, Burke, Hipwell, & Loeber, 2012). Using latent growth curve models, they found that higher

levels of both ADHD and ODD scores at age 8 uniquely predicted BPD symptoms at age 14. Further, increase in ODD severity from age 8–10, but not age 10–13, predicted Borderline PD symptoms at age 14. Conversely, for ADHD, increases in scores from age 10–13, but not 8–10, predicted Borderline symptoms at age 14. The authors argue that this suggests that for adolescent Borderline symptoms, difficulties with emotion regulation and relationships may precede problems with impulse control.

Similarly, strong associations between childhood EXT problems and Antisocial PD have been demonstrated. For example, in a large follow-up study of hyperactive boys and controls, ADHD was highly significantly related to Antisocial PD in young adulthood, middle adulthood and later adulthood (Klein et al., 2012; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993, 1998). Particularly strong links have been found between CD and Antisocial PD. For instance, one study showed that childhood CD significantly increased the risk for adult Antisocial PD (Odds Ratio (OR) = 4.3) (Copeland, Shanahan, Costello, & Angold, 2009). Interestingly, the OR was even higher (OR = 5.2) when controlling for other EXT and INT childhood disorders.

A recent study which was part of the Avon longitudinal study of parents and children (ALSPAC) investigated the effects of childhood conduct problems, assessed on six occasions between age 4 and 13, on a range of behaviour outcomes at age 18 in a community sample (Kretschmer et al., 2014). Whilst Antisocial PD was not directly assessed, the behaviour outcomes investigated are clearly related to ASPD, namely a range of externalising behaviours (e.g. substance use/abuse, self-reported offenses, criminal involvement, risky sexual behaviours and gambling). The results showed that individuals who displayed childhood to adolescence persistent conduct problems were at greater risk for almost all forms of later problems, compared to individuals who showed adolescent-onset conduct problems and individuals without childhood conduct problems.

Lahey et al. (2005) ran a prospective longitudinal study with young adult males, first assessed at age 7-12 as outpatients of a mental health clinic. Whilst they found robust linear associations between child CD symptoms and Antisocial PD, they showed that ADHD without CD did not predict

Antisocial PD, and ODD did not predict Antisocial PD when controlling for CD. Thus, whilst initially all EXT childhood problems were associated with Antisocial PD, they found a unique association with CD. This is perhaps not surprising considering that childhood CD is regarded as the 'childhood version' of Antisocial PD, and onset before age 15 is a diagnostic criterion for adult Antisocial PD in the DSM (APA, 2000). However, other studies have found that other EXT disorders also had independent associations with Antisocial PD. For example, one study compared different groups of adult males, based on childhood disorders assessed at age 8 (Sourander et al., 2007). They found that the group with children who only had conduct problems and no hyperactivity problems in childhood had an increased likelihood of developing Antisocial PD in adulthood (OR = 3.5). However, even though the OR was slightly lower (OR = 2.7), children who only displayed hyperactivity problems (and no conduct problems) in childhood also had an increased risk of developing Antisocial PD. Therefore, the links between specific childhood EXT problems and Antisocial PD still needs to be clarified.

The generally significant associations between childhood EXT problems and Cluster B PDs, particularly Antisocial PD and Borderline PD, are not unexpected considering that Cluster B is regarded as the 'dramatic-erratic' or 'undercontrolled' PD cluster with symptomatology on the externalising spectrum. These findings are in line with the view that "externalising" as a broad, higher-order psychopathology factor underpins the most commonly occurring EXT mental disorders and accounts for the covariance among childhood and adult EXT disorders (Krueger, 2002b; Krueger, McGue, & Iacono, 2001). As such, the association between EXT problems in childhood and Cluster B PD in adolescence or adulthood can be regarded as homotypic continuity. Whether any unique associations between specific childhood EXT problems and specific Cluster B PDs exist, still needs to be clarified.

1.6.2 Internalising childhood problems

Research about the association between childhood INT problems and PD is mostly lacking except for Antisocial PD and Borderline PD. In the area of Antisocial PD mixed results have been found, with some evidence supporting the notion that Antisocial PD is linked to childhood INT

problems, whereas other studies have found no such association. For example, one study found no significant relationship between childhood depressive symptoms (assessed at age 7-12) and Antisocial PD in young adult males (Lahey et al., 2005). Similarly, another study found that depression in children and adolescents (mean age 14.1 years) in psychiatric care did not predict Antisocial PD at mean age 30.5 (Ramklint, von Knorring, von Knorring, & Ekselius, 2003), and two further studies did not find significant associations between childhood depression or anxiety and Antisocial PD in young adults (Copeland et al., 2009; Diamantopoulou, Verhulst, & van der Ende, 2010). Some evidence suggests that it might be INT problems in combination with EXT problems in childhood, rather than INT problems alone, that increase an individual's risk to develop Antisocial PD. For instance, one study compared groups of young adult males based on their assessment of childhood problems at age 8 (Sourander et al., 2007). It was found that the group of children who had only INT problems were not at an increased risk to develop Antisocial PD. However, those children who had a combination of high INT and EXT problems had the highest risk for developing Antisocial PD in adulthood (OR=5.4) , even more so than individuals who had high CD (OR=3.5) or high hyperactivity problems (OR=2.7). Another study showed that depression in children and adolescents (mean age 14.1 years) in psychiatric care predicted Borderline PD at mean age 30.5, also after adjusting for sex, age, and other childhood disorders (Ramklint et al., 2003). One prospective longitudinal study (Belsky et al., 2012) showed that INT problems, assessed at age 5, were associated with higher INT BPD features at age 12. However, in a study with males only (Burke & Stepp, 2012) no associations between depression and anxiety (assessed in children aged 7-12) and Borderline PD in adulthood was found.

1.7 Chapter Summary

The aim of this chapter was to provide an overview about what is currently known about childhood predictors for the development of PD, summarising evidence from both retrospective and prospective studies. Generally, not much research has been carried out about childhood precursors to PD; especially prospective longitudinal research is rare. Available evidence is mostly about Antisocial PD and Borderline PD and any conclusions about

the effects of risk factors for the development of PD need to be drawn with caution, bearing in mind that most studies only focused on these two specific PDs.

The findings from those studies that are available do support the view that both biological and environmental factors exist that increase the risk for a child to develop personality pathology. Heritability estimates indicate that, even though effect sizes vary across studies and specific PDs, PDs are influenced by genes. Further, heritable "difficult" temperament (especially impulsiveness and negative affectivity/emotionality) may be latent factors predisposing individuals to develop a disorder when exposed to environmental stressors. Certain interrelated environmental stressors have been found to particularly increase the risk of a PD. These include child maltreatment, negative parent factors or general familial and socioeconomic adversity.

It seems clear that certain factors increase the risk for PD, but to date no specific links between any risk factors and any specific PDs have been confirmed. For instance, negative parenting variables have been identified as strong risk factors for the development of PD, but no links between specific parenting practices and specific PDs have been identified as of yet. In addition, the differential effects of negative parenting by the mother and father have not been explored. Established risk factors such as child abuse are non-specific and lead to a range of psychopathology (multifinality), and the same disorder can be caused by a variety of factors (equifinality). Further, whilst difficult temperament may increase proneness to psychopathology, these children may still have a healthy development in the presence of protective factors, such as social support or familial warmth. Similarly, environmental adversity such as child maltreatment may not necessarily lead to the development of a PD in children who have an "easier" temperamental disposition.

Evidence also suggests that common EXT and INT child problems are predictive of later personality pathology. At present, they may be the most useful risk markers in terms of identification of individuals who may be at risk of developing personality pathology, and they could also be directly targeted in early intervention approaches. However, whether any specific

associations between individual child EXT disorders and specific adolescent/adult PDs exist still needs to be explored, as the evidence to date is rather unspecific. In addition, assessing the unique predictive validity of specific child EXT problems by testing their association with specific PDs whilst controlling for other child disorders and PDs would be valuable, in particular for the development and implementation of early identification and preventive intervention.

Etiologic explanations of PD are shifting from single factor theories to multiple causal pathways (Paris, 2009). Some argue for the importance of disentangling the effects of specific adverse childhood conditions or events on PD (Bradley et al., 2005; Fergusson & Mullen, 1999). Others argue that attempts to disaggregate the effects of clustered adversities may offer relatively little insight into processes of risk and resilience (McLeod & Almazan, 2003). What is clear, however, is that much more research is needed in order to gain a better understanding of the pathways leading to personality pathology, particularly prospective longitudinal research.

1.8 Thesis Aims

In light of the evidence to date, the aim of this thesis was to investigate early childhood predictors in the form of childhood externalising and internalising problems in a prospective longitudinal study. Specific aims were:

(1) To investigate whether common externalising and internalising childhood problems predict personality pathology in adulthood.

(2) To assess whether any unique associations between specific childhood EXT/INT problems and specific PDs exist.

(3) To explore whether any combinations of significant childhood predictors show additive and/or interactive effects in the prediction of PD.

(4) To investigate whether negative parenting by both the mother and the father affect the development of personality pathology, and whether the effects differ for mothers and fathers.

(5) To test whether the effects of negative parenting by the mother and/or father add to, moderate or mediate the effects of child problems in the prediction of PD.

(6) To investigate whether any associations between childhood problems and PD can be explained by a continuation of childhood symptoms into adulthood (i.e. whether the associations between childhood problems and adult PD are mediated by adult co-occurring psychopathology).

Chapter 2: Childhood Externalising and Internalising problems Predict Personality Disorders – A Meta-Analysis of Prospective Longitudinal Studies

Chapter 2 Objectives

The first step in the process of developing an early intervention approach involves identifying individuals at risk for the development of the disorder. In the previous chapter, a broad overview about the risk factors and risk markers for the development of personality disorders (PDs) was provided. The most optimal prevention method has been suggested to be 'indicated prevention' where individuals displaying risk markers of the disorder are targeted (Chanen & McCutcheon, 2013). Risk markers such as typical externalising (EXT) or internalising (INT) childhood disorders could be regarded as targets for indicated prevention of PD. With this in mind, the focus of this chapter was on risk markers, manifested as common childhood EXT and INT disorders. The aim was to systematically collate and meta-analyse prospective longitudinal studies of the relationship between childhood EXT and INT problems and adolescent or adult PDs.

2.1 Introduction

Clinicians have long been hesitant to diagnose PDs prior to adulthood (Allertz & van Voorst, 2007; Chanen & McCutcheon, 2008). However, some have argued that PDs are as prevalent in adolescents as they are in adults (Grilo et al., 1998; Johnson, Cohen, et al., 2000; Westen, Shedler, Durrett, Glass, & Martens, 2003). Further, PD symptoms in adolescents show diagnostic continuity over time (Bernstein, Cohen, Skodol, & Bezirganian, 1996; Johnson et al., 1999) and often persist into young adulthood (Grilo, Walker, Becker, Edell, & McGlashan, 1997; Johnson et al., 1999; Johnson,

Cohen, et al., 2000). Some studies have demonstrated a stability of underlying PD dimensions in childhood (Crick et al., 2005; Stepp, Pilkonis, Hipwell, Loeber, & Stouthamer-Loeber, 2010), and the general consensus is that PD symptom constellations identified in adulthood have their origins in childhood (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Bromley, & Bornstein, 2006; Johnson, First, et al., 2005; Kernberg et al., 2000; Mervielde et al., 2005; Shiner, 2007; Westen & Chang, 2000). Further support comes from studies testing the longitudinal stability of personality prototypes: overcontrolled, undercontrolled and resilient. Overcontrol/undercontrol refers to a meta-dimension of impulse inhibition versus impulse expression; resiliency refers to a meta-dimension of the dynamic, flexible capacity to modify one's level of control in response to contextual demands (Asendorpf et al., 2001; Asendorpf et al., 2008; Asendorpf & van Aken, 1999; Block, 1971; Caspi, 2000; Caspi & Silva, 1995; Chapman & Goldberg, 2011; Eisenberg et al., 2000; Markon et al., 2005; Meeus et al., 2011; Robins et al., 1996).

Overcontrol and undercontrol largely parallel the well-established division between internalising and externalising disorders which have been identified both in children and adults (Achenbach & Edelbrock, 1984; Krueger & Markon, 2006a). EXT problems are characterised by "acting out" behaviours, such as aggressiveness, difficulties with interpersonal relationships, antisocial behaviours, substance/alcohol abuse (Achenbach & Edelbrock, 1978; Krueger et al., 2005). Conversely, INT problems are characterised by "acting in" problems, such as social withdrawal, inhibition, depression, anxiety/fearfulness and phobias (Achenbach & Edelbrock, 1978; Krueger & Markon, 2006a; McCulloch et al., 2000). Several large-scale studies about phenotypic and/or genetic structure examining comorbidity patterns consistently confirmed a hierarchical structure with EXT and INT at the top (Kendler et al., 2003; Krueger, 1999; Krueger et al., 1998; Vollebergh et al., 2001).

If precursors and risk factors for the development of PDs can be identified during childhood, then treatment approaches aimed at early identification and prevention can be implemented, as has been proposed in other areas such as ADHD (Sonuga-Barke et al., 2011). Some specific PDs such as Borderline PD have been suggested to be "leading candidates" for

developing such programmes. For instance, it has been argued that Borderline PD is common in clinical practice, among the most functionally disabling of all mental disorders, often associated with help-seeking and it has been shown to respond to intervention even in those with established disorders (Chanen & McCutcheon, 2013). Early interventions for antisocial behaviour have also been shown to be (cost) effective in the US (Schweinhart & Weikart, 1998), and have been highly recommended in a study explicitly investigating the economic cost of severe antisocial behaviour in children (Romeo et al., 2006).

Unfortunately, far less is known about the developmental pathways to PD than is known about those leading to other major psychological disorders (Shiner, 2009). The precursors to PD have received relatively little attention (De Clercq & De Fruyt, 2007; De Clercq et al., 2009) and even though authors of texts and chapters on PD do refer to childhood antecedents (Cohen, 2008; Johnson, First, et al., 2005), this literature is more based on clinical experiences and theoretical speculations rather than empirical research. Known antecedent risk factors include; (i) Genetic factors, with heritability estimates averaging around 40-50% (South, Reichborn-Kjennerud, Eaton, & Krueger, 2012); and (ii) Environmental risk factors, such as maltreatment (abuse and neglect) (Johnson et al., 1999; Johnson, Cohen, Smailes, et al., 2001; Lobbestael et al., 2010), negative parenting (Johnson, Cohen, et al., 2006), parental mental illness (Schuppert et al., 2012), or socioeconomic factors (Winsper et al., 2012). Studies have also identified a number of putative early markers of risk pathways in the form of common childhood problems such as externalising (e.g. disruptive) disorders and internalising problems (e.g. anxiety). Regrettably a large proportion of studies examining PD precursors are cross-sectional, and are based on retrospective assessments of childhood events, which might be distorted by recall bias or due to pre-existing childhood traits contributing to the onset of some types of childhood adversities (Johnson, First, et al., 2005; Mannuzza, Klein, & Moulton, 2002; Maughan & Rutter, 1997). This concern is particularly salient for the study of the childhood antecedents of PDs, as fundamental to their pathologies are distortions in the perceptions of themselves and other persons.

2.1.1 Aims and hypotheses

The first step in the process of developing an early intervention approach involves identifying individuals at risk for the development of PDs. If more was known about the behavioural precursors of PD these could help target and tailor interventions and provide clues about new potential targets for interventions. With this in mind the aim of this study was to systematically collate and meta-analyse prospective longitudinal studies of the relationship between childhood problems and adolescent or adult PDs. The focus was specifically on individual markers of risk manifested as common childhood disorders, such as externalising and internalising problems. Based on the premise of both homotypic and heterotypic continuity of symptomatology from childhood into adulthood, we predicted that both childhood EXT and INT problems would be associated with adolescent/adult PDs with symptomatology on both the EXT spectrum (i.e. Cluster B PDs) and on the INT spectrum (i.e. Cluster A and C PDs).

2.2 Method

A systematic literature search was conducted for papers published up to December 2014, searching four widely used computerised databases (PsychARTICLES, PsychINFO, MEDLINE, CINAHL). The search terms related to three main areas: 1. Childhood characteristics, i.e. childhood EXT problems such as Attention Deficit Hyperactivity Disorder (ADHD), Conduct Disorder (CD), and Oppositional Defiant Disorder (ODD), or more general EXT childhood problems such as aggression; and childhood INT problems such as anxiety or mood disorders; 2. Outcome variables, i.e. specific PDs and personality pathology, assessed both categorically and dimensionally, and PD clusters; 3. Prospective longitudinal methodology. See Appendix A.1 for the specific search terms and syntax used. In addition, Google/Google Scholar searches were performed. Included articles were hand-searched for relevant references and citations.

2.2.1 Inclusion/Exclusion Criteria

For the full inclusion/exclusion criterion set, see Table 1.

Study Design Only studies using a prospective longitudinal design were included. Studies that assessed childhood predictors retrospectively were excluded due to known issues with long-term recall of childhood events or childhood disorders (Mannuzza et al., 2002; Maughan & Rutter, 1997).

Baseline/Follow-up age There is a consensus that by adolescence a lot of maladaptive PD traits will already have been established. Data suggests that the prevalence of adolescent PDs is roughly equivalent to that observed amongst adults, and that PD symptoms in adolescents show diagnostic continuity over time (Bernstein et al., 1996; Johnson et al., 1999). Therefore, only studies that used children (aged up to 12 years) at baseline and adolescents or adults (aged 12 or above) at outcome were included, and studies that included adolescents at baseline were excluded. Because the chosen age ranges overlapped, a minimum follow-up period of three years was chosen as an inclusion criterion.

Baseline Variables Childhood variables were grouped as EXT problems and INT problems. EXT and INT problems have been suggested to be at the most general level of the hierarchical organisation of general psychopathology both in adults and in children (Achenbach & Edelbrock, 1984; Krueger & Markon, 2006b). In children, EXT problems are described as behaviours characterised by deficits in inhibition, include aggressiveness, difficulties with interpersonal relationships and rule breaking, as well as displays of irritability and belligerence (Achenbach & Edelbrock, 1978). Specific examples of EXT problems are Conduct Disorder (CD), Attention Deficit Hyperactivity Disorder (ADHD) or Oppositional Defiant Disorder (ODD). Conversely, INT problems include social withdrawal, inhibition, shyness, feelings of worthlessness or inferiority, and dependency (Achenbach & Edelbrock, 1978; McCulloch et al., 2000). INT psychopathology includes symptoms such as depression, anxiety/fearfulness and phobias (Krueger & Markon, 2006b).

Outcome Variables Outcome variables were also grouped according to their overarching EXT and INT classifications i.e. Cluster B (Antisocial PD, Borderline PD, Histrionic PD and Narcissistic PD) with symptomatology on the externalising spectrum, and Clusters A (Paranoid PD, Schizoid PD, Schizotypal PD) and C (Avoidant PD, Dependent PD, Obsessive-Compulsive

PD) with symptomatology on the internalising spectrum. In addition, studies that included data about psychopathy at outcome were included. Whilst psychopathy is not currently included in the DSM (APA, 2013) , it is regarded as a personality disorder. It is closely linked with Antisocial PD, with which it shares a lot of diagnostic overlap, and the two disorders stem from the same underlying construct first described by Cleckley (Cleckley, 1941, 1976). Further, they were going to be combined as a joint Antisocial PD/psychopathy diagnostic type in DSM-5, but because revisions were aborted, this combined type is now part of DSM-5 Appendix (APA, 2013). Therefore, studies that contained data about psychopathy were included and pooled with studies about Antisocial PD. Because the constructs of Antisocial PD and psychopathy are, despite substantial overlap, not entirely congruent, analyses were carried out both with and without inclusion of psychopathy data; wherever exclusion of psychopathy studies changed the results, these are presented separately.

Studies published prior to 1980 – the publication year of DSM-III – were not included because PD diagnosis prior to DSM-III was very unreliable (Spitzer et al., 1975; Spitzer & Fleiss, 1974), and diagnostic criteria diverge substantially from the current system, whereas DSM-III, DSM-IV and DSM-5 are mostly congruent. Studies using either categorical or dimensional assessments were considered for inclusion. However, these only qualified if they included data specifically about PD or personality pathology; papers assessing related concepts not specifically about PD were excluded. For instance, papers about antisocial behaviour, criminality and delinquency were not included unless participants were specifically assessed for Antisocial PD.

Table 1: Inclusion/Exclusion Criteria

Inclusion Criteria	Exclusion Criteria
• Studies written in English	
• Studies published 1980 (inclusive) onwards	
• Primary, original research published in peer reviewed journals	• Publication not peer-reviewed or not based on primary research (e.g. dissertations, book chapters, reviews etc.)
• Prospective longitudinal study design	• Study design not prospective longitudinal (e.g. retrospective or cross-sectional)
• Case-control or cohort studies	• Studies using only clinical samples, e.g. studies without a healthy control group
• Baseline age up to 12 years (inclusive)	
• Follow-up age at least 12 years (inclusive)	
• Minimum follow-up period of 3 years	
• Includes measures on child EXT/INT variables, emotionality or temperament at baseline	
• Includes data specifically on PD/personality pathology at outcome	• Outcome assessment not specifically about PD/personality pathology (e.g. antisocial behaviour rather than Antisocial PD)
• PD assessment based on DSM-III or ICD-9 criteria onwards	• PD assessment based on earlier versions than DSM-III or ICD-9

EXT – externalising; INT – internalising; PD – Personality Disorder

2.2.2 Study selection process

Firstly, titles and abstracts were screened for relevance. Any paper about the longitudinal relationship between childhood predictors and PD/personality pathology was considered relevant at this stage. To examine

reliability of decisions a random sample of 100 titles and abstracts was independently screened for relevance by an additional researcher, namely Dr Elizabeth Smith (ES), a Research Fellow in Developmental Psychopathology and Educational Psychologist; agreement about inclusion was high (kappa=.87). After this stage, full texts were obtained for the remaining papers and reviewed independently by JK and ES, applying the full inclusion criteria set. In order to minimise bias, ES was blind to the journal of the publication, the authors and their institution. Agreement about inclusion was high (kappa=.83). Any discrepancies were resolved by further review and discussion with Professor Edmund Sonuga-Barke who was the primary supervisor of this PhD and who was independent of the initial decisions.

2.2.3 Quality assessment

The study quality and/or internal validity of each included publication were evaluated according to a predetermined criterion set. Because no existing quality criterion set was suitable for the purposes of this research, a new set of quality criteria specifically relating to the quality of prospective longitudinal research was generated. Relevant areas for quality assessment were collated using other longitudinal quality assessment tools (e.g. The Newcastle-Ottawa Scale) and additional areas of importance were identified by JK. These themes were discussed and revised with the PhD supervisory team. In addition, Professor Barbara Maughan, an expert in childhood to adulthood longitudinal studies, provided advice. The final set of criteria (see Appendix A.2) rated study quality in the following areas: 1. Representativeness of sample; 2. Sample size; 3. Attrition rate; 4. Assessment of baseline variables; and 5. Assessment of outcome variables. Each paper received an overall quality score of 0-9, with studies scoring 6-9 points being rated as "high quality" and studies scoring five or below rated as "low quality". All quality assessments were completed independently by JK and ES, with disagreements settled by discussion.

2.2.4 Data extraction

Data relating to participant characteristics (gender, age at baseline, age at follow-up), baseline variables (EXT problems and INT problems) and

outcome variables (categorical and dimensional assessments of specific PDs and PD clusters) was extracted. Wherever possible, effect sizes, in the form of Odds Ratios (ORs) were calculated by JK, or converted from other statistics reported in papers (for details about conversions, see Appendix A.3). If this was not possible, effect sizes reported in papers were used. If neither was possible on the basis of the information in the paper, the lead author was contacted and asked to provide further information. If no additional necessary information from lead authors could be obtained, lower limit effect sizes were estimated using *p*-levels, as suggested by Rosenthal (1994): z-scores for *p*-levels were found through standard normal deviates and then transformed to effect sizes (see Appendix A.3). This was the case for one publication [XXVI]. One paper, for which further information could not be obtained from the lead author (Farrington, 2000), had to be excluded because confidence intervals were not given, effect sizes could not be estimated based on the data provided in the paper, and specific *p*-levels were not reported. Further, for two papers [X, XVI], although significant results were included in the meta-analyses, statistics for non-significant associations could not be obtained from the authors and are therefore not considered in the analyses.

2.2.5 Analyses

Several meta-analyses were performed, pooling effect sizes of studies containing data about the association between childhood variables and PDs in random effects models. Separate meta-analyses were carried out if at least three studies could be included. Childhood predictors and PDs were analysed twice – once in terms of their overarching categorisation and once in terms of the more specific designation: 1. For overarching categories of childhood predictors, these were grouped as EXT problems and INT problems. PDs were grouped as DSM Clusters (A, B, C). 2. For specific categories, childhood predictors were divided into specific EXT problems (ADHD, CD, ODD) and INT problems (anxiety, depression). PDs were split into specific PDs (Schizotypal PD, Schizoid PD, Paranoid PD, Antisocial PD/psychopathy, Borderline PD, Histrionic PD, Narcissistic PD, Obsessive-Compulsive PD, Dependent PD, Avoidant PD). Therefore, wherever possible,

separate meta-analyses were conducted for PD Clusters and specific PDs, both overarching and specific levels of EXT problems and INT problems.

Included papers often reported predictors or controlled for extraneous variables other than those of interest in the current analysis. In order to account for the impact of these variations, for each meta-analysis, effect sizes of included papers were pooled in two separate models: 1. The 'least control model' (LCM), where for every included paper, effect size data for the association between childhood variables and outcome variables with the *least* amount of statistical control over any covariates was included. Ideally, this was data without control over any covariates (i.e. univariate data). If papers reported multivariate datasets, the model with the least amount of control over any covariates was used. For instance, if several multiple regression models were reported, the data from the model with the lowest number of covariates was included. If only one set of data was reported, this was included, whether it was univariate or multivariate data. 2. The 'most control model' (MCM) included effect size data for the association between childhood variables and outcome variables with the *most* amount of statistical control over any covariates. For example, if a paper reported both univariate and multivariate models, multivariate statistics were included. If several multivariate models were reported, the model with the largest amount of control over any covariates was used (e.g. if several multiple regression models were reported, data from the model with the highest number of covariates was included). If only one set of data was reported, this was also included, whether it was univariate or multivariate data. For details about differences in covariates in both LCM and MCM, see Appendix A.4.

This approach was adopted to provide a sense of the range of possible effects within each study. Ideally, only multivariate studies including the same confounders would have been included in each MCM, in order to only pool comparable, homogenous studies. However, after inspection of available publications it transpired that, due to the variation of available studies, this approach was not feasible – in none of the available categories was it possible to group at least 3 papers, therefore this approach was dropped. An alternative option would have been to only include univariate data in the LCM and only multivariate data in the MCM; however, due to the

low number of published papers in some of the categories, some of the models would have had to be dropped due an insufficient amount of available studies. As a consequence, the above approach was adopted, where in all models, all available papers were included. LCM and MCM should not be regarded as two entirely different models, but rather as one model – the comparison is given to provide a sense of the range of possible effect sizes within each model.

The statistical stability of results was evaluated by removing each study individually and recalculating the pooled OR and 95% confidence interval (CI). Duplication of data was avoided by checking for overlapping samples; when multiple papers of the same dataset were published, the earliest publication of this data was included in the analysis (e.g. study X). For studies that reported several follow-ups of the same sample using the same outcome variable [XVII-XXXI], these were combined using mean values. For each study, if several ratings were given in the same category (e.g. both CD and ADHD assessed in the same sample), these ratings were first combined into a pooled rating (e.g. combined "EXT problems" rating) and then, wherever possible, analysed separately per subgroup (e.g. separate analyses for "CD as a predictor of Antisocial PD" and "ADHD as a predictor of Antisocial PD").

All analyses were performed using Comprehensive Meta-Analysis (Version 2.2064). Publication bias was assessed by Begg and Mazumdar's rank order correlations (Begg & Mazumdar, 1994). Because different characteristics between studies might contribute to variation in effect sizes, focusing only on overall pooled outcomes could be misleading, especially if included individual studies are not sufficiently homogenous. Random-effect models incorporate an estimate of between-study heterogeneity into the calculation of the common effect (Deeks, 2001). Because heterogeneity across study results was expected, effect sizes were pooled in random-effects models which produced pooled odds ratios (ORs) with 95% confidence intervals (CI).

Between-study heterogeneity was quantified with I^2 statistics and evaluated using the Cochran Q test (Q), where $p < .10$ indicated a high level of between-study heterogeneity (Higgins, Thompson, Deeks, & Altman, 2003). The I^2 statistic is the percentage of variability in effect estimates that is due

to heterogeneity rather than to sampling error where values of 25%, 50% and 75% indicate low, moderate and high levels of heterogeneity, respectively (Higgins & Thompson, 2002). To further explore the sources of any between-study heterogeneity, influence of moderator variables were carried out through unrestricted meta-regressions (Maximum Likelihood). Moderators were gender, age at baseline, age at follow-up, overall quality rating of paper, quality of baseline assessment and quality of follow-up assessment.

2.3 Results

2.3.1 Search results

Initial searches yielded 5,706 results of which 5,584 were excluded following review of title and abstract, and a further 97 were excluded following review of the full text (see Figure 1 for PRISMA flow chart). Twenty-eight papers were included, the majority of which contained data about Cluster B PDs, in particular Antisocial PD and Borderline PD (see Table 2). Assessment of publication bias using Begg and Mazumdar's rank order correlations was not significant for any of the analyses ($p<.05$, one-tailed), so the results are unlikely to be subject to publication bias. For full study characteristics of included papers, please see Table 2.

2.3.2 Meta-Analyses

Twenty-two separate meta-analyses were performed. Please see Figures 2-5 for overview forest plots, presenting summary graphs of pooled effect sizes. Overviews of significant and non-significant meta-analyses are provided in Table 3, and detailed forest plots for all meta-analyses are provided in Appendix A.5.

Table 2: Characteristics of included studies

Study (first-named author), location	N	Predictors							Personality Disorder at outcome	Baseline age in years	Follow-up age in years	Gender	Paper Quality
		EXT				*INT*							
		ADHD	*CD*	*ODD*	*Other*	*DEPR*	*ANX*	*Other*					
I – Belsky (2012), UK	2,141	XX			XXX			XXX	BPD	5	12	Mixed (49% male)	good
II – Carlson (2009), USA	162	X			XX				BPD	12	28	Mixed (50% male)	low
III – Copeland (2009), US	838	X	X	X			XX		ASPD	9-12	19-21	Mixed (exact proportions not given)	good
IV – Diamantopoulou (2010), NL	421	X		X		X			ASPD	6-8	20-22	Mixed (41% male)	good
V – Forsman (2007), SE	1855 (EXT) 1851 (ADHD)	X			X				Psychopathy	8-9	16-17	Mixed (49% male)	good
VI – Glenn (2007), MU	333 (LCM)						X	X	Psychopathy	3	28	Mixed (61%	good

	111 (MCM)											male)	
VII – Lahey (2005), US	163	X	X	X		X	X		ASPD	10.05	18.5	Male	good
VIII – Miller (2008), US	181	X							All PDs	9.21	18.41	Mixed (89% male)	good
IX – Shi (2012), US	56	X			XX				ASPD	5 / 7	19.9	Mixed (58.9% male)	low
X – Sourander (2005), FI	2,712	XX	XX			X	X		ASPD	8	20.5	Male	good
XI – Stepp (2012), US	1,233	X	X	X		X			BPD	8 / 8-12	14	Female	good
XII – Fergusson (2005), NZ	973		X						ASPD	7-9	21-25	Mixed (51% male)	good
XIII – Fischer (2002), US	210	X							ASPD BPD	7	20.8	Mixed (91% male)	Good
XIV – Natsuaki (2009), US	174				XX				PPD (Cluster A)	9-12	15.3	Mixed (60% male)	good
XV – Burke (2007), US	163	X	X	X		X	X		Psychopathy	7-12	18-19	Male	good

XVI – Schaeffer (2003), US	297				X				ASPD	6.2	19.5	Male	good
XVII – Moffitt (2002), NZ	458		X						ASPD	5-11	26	Male	good
XVIII – Caspi (1996), NZ	961				X			X	ASPD	3	21	Mixed (52% male)	good
XIX – Weiss (1985), CA	102	X							ASPD	6-12	25.1	Mixed (90% male)	low
XX – Claude (1995), CA	104	X							ASPD	7.3	19.7	Male	good
XXI – McMahon (2010), US	511 (LCM) 754 (MCM)		X						ASPD	10	19	Mixed (65% male)	low
XXII – Hellgren (1994), SE	56	X							ASPD BPD	7	16	Mixed (52% male)	low
XXIII – Bernstein (1996), US	641		X						Cluster A Cluster B Cluster C	5.5	15.9	Mixed (49% male)	good
XXV – Bornovalova (2013),	1,243				X	X			BPD	11	24	Mixed (50%	good

US												male)	
XXVI – Anglin (2008), US	766							X	SPD (Cluster A)	9	20	Mixed (51% male)	low
XXVII – Mannuzza (1991), US	172	X							ASPD	7.3	18.5	Male	good
XXVIII – Mannuzza (1993), US	186	X							ASPD	9.3	18.6	Male	good
XIX – Mannuzza (1998), US	158	X							ASPD	7.3	24.3	Male	good
XXX – Gittelman (1985), US	200	X							ASPD	9.3	18.5	Male	good
XXXI – Klein (2012), US	271	X							ASPD	8.3	41	Male	good
XXXII – Hechtman (1984), CA	108	X							ASPD	6-12	20.5	Male	low

EXT/INT – Externalising/Internalising problems; ADHD – Attention Deficit Hyperactivity Disorder; CD – Conduct Disorder; ODD – Oppositional Defiant Disorder; Depr – Depressive symptoms; Anx – anxiety/fearfulness symptoms; (AS, B)PD – (Antisocial, Borderline) Personality Disorder; LCM – least control model; MCM – most control model

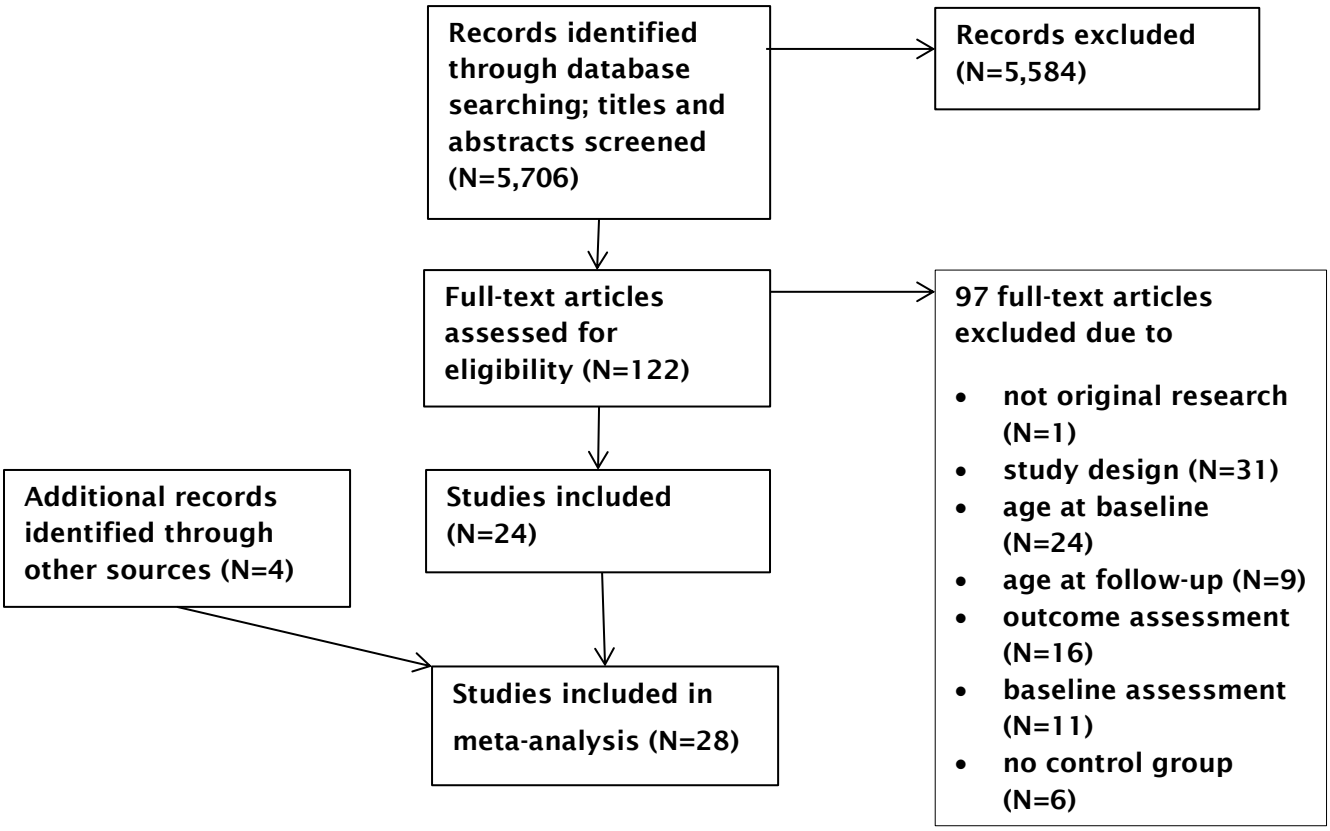


Figure 1: PRISMA flow chart of exclusion process of papers

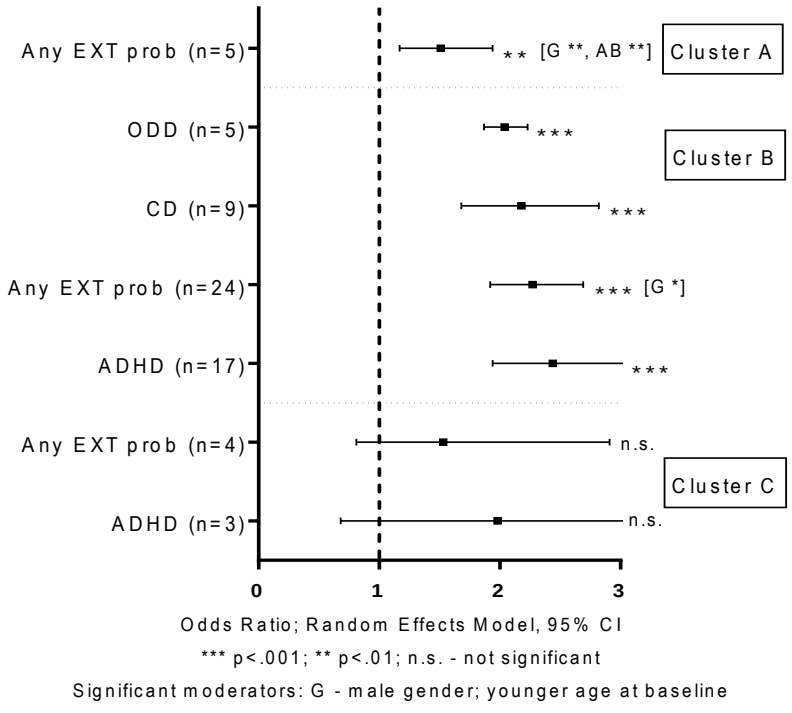
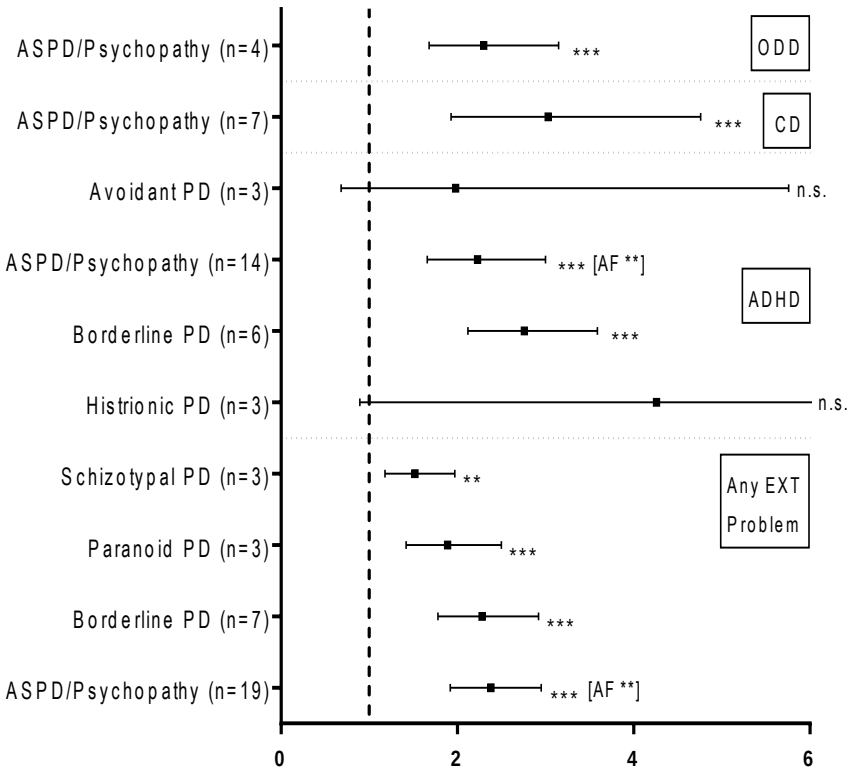


Figure 2: Childhood Externalising Problems and PD Clusters



Odds Ratio; Random Effects Model, 95% CI; *** p<.001; ** p<.01; n.s. - not significant

ODD - Oppositional Defiant Disorder; CD - Conduct Disorder; EXT - Externalising

Significant moderator: AF - Older age at follow-up

Figure 3: Childhood Externalising Problems and specific PDs

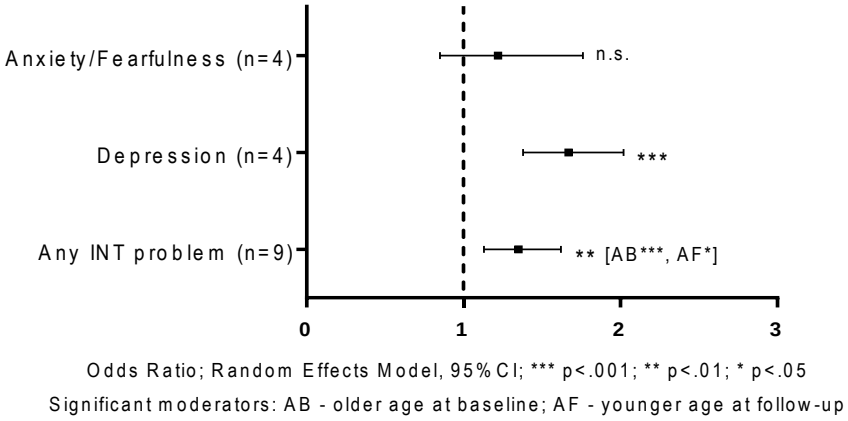


Figure 5: Childhood Internalising Problems and Cluster B

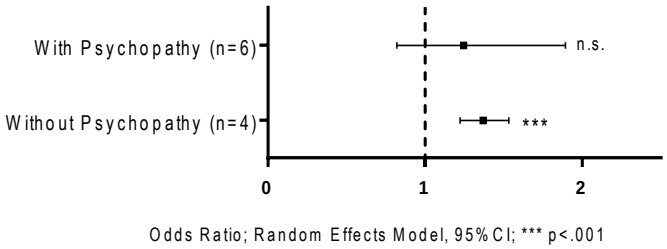


Figure 4: Childhood INT Problems and ASPD, with and without Psychopathy

2.3.3 Externalising problems and Cluster B PDs

Twenty-eight papers [I-V, VII-XIII, XV-XXIII, XXV, XXVII-XXXI] from 24 separate studies were included in the meta-analysis for childhood EXT problems and Cluster B PDs (N = 15,464). The results from both LCM (Figure 2) and MCM (OR=2.01, CI=1.70-2.39, p<.001) suggested children with EXT problems were over twice as likely to develop Cluster B PDs as children without EXT problems. The effect size dropped from 2.27 to 2.01 in MCM compared to LCM, but both models were highly significant (p<.001). Removal of individual studies did not change these results in either model. Heterogeneity among studies was high (LCM: Q = 280.33; p<.001; I^2=92%; MCM: Q = 294.70; p<.001; I^2=92%). Meta-regressions showed that gender was the only significant moderator for both LCM (b=0.55; p<.05) and MCM (b=0.61; p<.05) – the higher the proportion of males in the sample, the higher the effect size.

2.3.3.1 Specific externalising childhood problems (ADHD, CD, ODD) and Cluster B PDs

Twenty-one papers [I-V, VII-XI, XIII, XV, XIX, XX, XXII, XXVII-XXXII; N=10,771] from 17 separate studies were included in a meta-analysis about ADHD and Cluster B PDs (Figure 2). Nine studies [III, VII, X, XI, XII, XV, XVII, XXI, XXIII; N=7,935] looked at CD and Cluster B PDs (Figure 2) and five studies [III, IV, VII, XI, XV; N=2,818] looked at ODD and Cluster B PDs (Figure 2). ADHD (LCM: OR=2.44, CI=1.94-3.07, p<.001; MCM: OR=2.24, CI=1.78-2.82, p<.001), CD (LCM: OR=2.18, CI=1.68-2.82, p<.001; MCM: OR=1.78, CI=1.43-2.21, p<.001) and ODD (LCM: OR=2.04, CI=1.87-2.23, p<.001; MCM: OR=2.11, CI=1.70-2.61, p<.001) all independently increased the risk for Cluster B disorders. No significant moderator variables were found for any specific childhood EXT problems.

2.3.3.2 Childhood externalising problems and Antisocial Personality Disorder/Psychopathy

Twenty-four papers [III-V, VII-X, XII, XIII, XV-XXII, XXVII-XXXII; N=7,239] reporting data from 19 separate studies were included in the meta-analysis for childhood EXT problems and Antisocial PD (Figure 3). Results from both LCM (Figure 3, Appendix A.5), and MCM (OR=2.14; 95% CI =1.74-2.64, p<.001)

suggested that children with EXT problems are over twice as likely to develop Antisocial PD in adolescence/adulthood than children without EXT problems. The effect size dropped from 2.38 to 2.14 in MCM compared to LCM, but both models were highly significant (p<.001). In both models, removal of individual studies did not significantly change these results. Included studies were considerably heterogeneous (for LCM: Q = 75.10; p< .001; I^2=76%; MCM: Q = 83.06; p< .001; I^2=78%). Meta-regressions revealed two marginally significant moderators for LCM: age at follow-up (b=0.09; p<.10), where studies with older participants at follow-up reported higher effect sizes; and gender (b=0.86; p<.10) – the higher the proportion of males in the sample, the higher the effect size. For MCM, age at follow-up was significant (b=0.10; p<.05) and gender was marginally significant (b=0.78; p<.10).

2.3.3.3 Specific Childhood Externalising Problems (ADHD, CD, ODD) and Antisocial PD/psychopathy

ADHD. Eighteen papers [III-V, VII-X, XIII, XV, XIX, XX, XXII, XXVII-XXXII; N=7,235) from fourteen studies were included in the meta-analysis for childhood ADHD and Antisocial PD. Results from both LCM (Figure 3) and MCM (OR=2.22; 95% CI =1.65-3.00, p<.001) suggested that children with ADHD in childhood were over twice as likely to develop Antisocial PD/psychopathy in adolescence/adulthood. ORs in both models were almost identical, and in both models, removal of individual studies did not significantly change these results. High heterogeneity was found in both LCM (Q = 55.31; p< .001; I^2=77%) and MCM (Q = 55.88; p< .001; I^2=77%). Meta-regressions revealed that age at follow-up was a significant moderator in both LCM (b=0.18; p<.01) and MCM (b=0.18; p<.01), where studies with older participants at follow-up reported higher effect sizes. Quality of outcome assessment was marginally significant in both LCM (b=0.69; p<.10) and MCM (b=0.67; p<.10). In both LCM (b=1.19; p<.10) and MCM (b=1.25; p<.10), gender was a marginally significant moderator – the higher the proportion of males in the sample, the larger the effect size.

Conduct Disorder. Seven [III, VII, X, XII, XV, XVII, XXI; N=6,061] papers were included in a meta-analysis for childhood CD and Antisocial PD/psychopathy. Results from both LCM (Figure 3) and MCM (OR=2.40; 95%

CI =1.60-3.62, p<.001) suggested that childhood CD significantly increases the risk for Antisocial PD/ psychopathy. The effect size dropped from 3.03 to 2.40 in MCM compared to LCM, but both models were highly significant (p<.001). In both models, removal of individual studies did not significantly change these results. Studies included in both the LCM (Q = 36.77; p< .001; I^2=84%) and in the MCM were highly heterogeneous (Q = 33.23; p< .001; I^2=82%). No significant moderators were found.

Oppositional Defiant Disorder. Four [III, IV, VII, XV; N=1,585] papers were included in the meta-analysis for childhood ODD and Antisocial PD/ psychopathy. Results from both LCM and MCM (OR=2.22; 95% CI =1.40-3.50, p<.01) suggested that childhood ODD significantly increases the risk for Antisocial PD/psychopathy. The effect size dropped from 2.30 to 2.22 in MCM compared to LCM. In both models, removal of individual studies did not significantly change these results. Heterogeneity was low in LCM (Q = 3.26; p=0.354; I^2=8%) and moderate in MCM (Q = 5.17; p=0.160; I^2=42%). No significant moderators were found.

2.3.3.4 Childhood externalising problems and Borderline Personality Disorder

Seven studies [I, II, VIII, XI, XIII, XXII, XXV; N=5,226] were included in the meta-analysis for child EXT problems and Borderline PD. Results from both LCM (Figure 3) and MCM (OR = 1.90; 95% CI = 1.44-2.50; p < .001) suggested that childhood EXT problems significantly increase the risk for Borderline PD. The effect size dropped from 2.28 to 1.90 in MCM compared to LCM but both were highly significant (p<.001). In both models, removal of individual studies did not significantly change the results. Heterogeneity was high in both models (LCM: Q = 62.45; p< .001; I^2=90%; MCM: Q = 76.94; p< .001; I^2=92%). Meta-regressions revealed two marginally significant moderators for LCM: the higher the proportion of males in the sample (b=0.26; p<.10), and the higher the quality of PD assessments (b=0.09; p<.10) the stronger the association between EXT problems and Borderline PD. Two significant moderators were found for MCM: the lower the age both at baseline (b=-0.03; p<.05) and at follow-up (b=-0.01; p<.05), the stronger the association between EXT problems and Borderline PD.

2.3.3.4.1 Specific Childhood Externalising Problems (ADHD) and Borderline Personality Disorder

Six papers [I, II, VIII, XI, XIII, XXII; N=3,983] were included in the meta-analysis for ADHD and Borderline PD. The results from both LCM (Figure 3) and MCM (OR = 2.36; 95% CI = 1.77-3.14; p < .001) suggest that childhood ADHD is independently associated with Borderline PD. The effect size dropped from 2.76 to 2.36 in MCM compared to LCM, but both models were highly significant (p<.001). In both models, removal of individual studies did not significantly change these results. Both models were considerably heterogeneous (LCM: Q = 21.02; p< .001; I^2=76%; MCM: Q = 25.15; p< .001; I^2=80%). Meta-regressions revealed three highly significant moderators for LCM: the higher the proportion of males in the sample (b=0.17; p<.001), the higher the quality of PD assessments (b=0.08; p<.001), and the lower the age at baseline (b=-0.01; p<.001) the stronger the association between ADHD and Borderline PD. In addition, three significant moderators were found for MCM: the higher the proportion of males in the sample (b=0.15; p<.001), the higher the overall quality of the paper (b=0.18; p<.05), and the lower the age at both baseline (b=-0.02; p<.001) and follow-up (b=-0.01; p<.05), the stronger the association between ADHD and Borderline PD.

2.3.3.4.2 Externalising problems (ADHD) and Histrionic Personality Disorder

Three studies were included in the meta-analysis for childhood EXT problems and Histrionic PD [VII, XIII, XXII; N = 447], all of which were about childhood ADHD and Histrionic PD. Heterogeneity among studies was low (Q=1.98; p=0.371; I^2=0%). The two models, LCM and MCM, were identical. The results suggested a positive but only marginally significant association between childhood ADHD and Histrionic PD (OR=4.26, CI=0.89-20.23, p<.10; Figure 3). However, when study XXII was removed the resulting OR became statistically significant (OR=9.99; CI=0.89-20.23; p<.05).

2.3.4 Internalising problems and Cluster B PDs

Nine papers [I, III, IV, VI, X, XI, XV, XVIII, XXIII; N = 9,443) were included in the meta-analysis for childhood INT problems and Cluster B PDs. The results

from both LCM (Figure 5) and MCM (OR=1.35, CI=1.13-1.62, p<.01) suggested that childhood INT problems increase the risk for Cluster B PDs. Both models were significant (p<.01). ORs were identical in the two models; removal of individual studies did not change the results in either model. Heterogeneity was high (LCM: Q = 50.00; p<.001; I^2=84%; MCM: Q = 45.54; p<.001; I^2=82). Meta-regressions revealed two significant moderators: the higher the age at baseline (LCM: b=0.14; p<.001; MCM: b=0.15; p<.001) and the lower the age at follow-up (LCM: b=-0.01; p<.05; MCM: b=-0.05; p<.05), the stronger the association between INT problems and Cluster B PDs.

2.3.4.1 Specific Childhood Internalising Problems (Anxiety and Depression) and Cluster B PDs

Four studies [III, VI, XV, XXIII; N=1,975] were included in the meta-analysis for childhood anxiety and Cluster B PDs. The results from both LCM (Figure 5) and MCM (OR=1.10, CI=0.58-1.97, p=0.842) were non-significant, suggesting that childhood anxiety does not significantly increase the risk for Cluster B PDs. ORs decreased slightly in MCM. In both models, removal of study VI resulted in marginally significant ORs (LCM: OR=1.52, CI=1.00-2.32, p<.10; MCM: OR=1.50, CI=0.99-2.279; p<.10). Heterogeneity was high (LCM: Q =16.99; p<.01; I^2=82%; MCM: Q = 17.94; p<.001; I^2=83).

Meta-regressions revealed three significant and one marginally significant moderators for LCM: the higher the age at baseline (b=0.05; p<.001) and the lower the age at follow-up (b=-0.02; p<.05), the higher the quality of PD assessment (b=0.23; p<.01) and the higher the proportion of males in the sample (b=0.37; p<.10), the stronger the association between anxiety and Cluster B PDs. For MCM, only age at baseline and follow-up remained significant: the higher the age at baseline (b=0.04; p<.001) and the lower the age at follow-up (b=-0.01; p<.05) the stronger the association between anxiety and Cluster B PDs.

Four studies [X, XI, XV, XXIII; N=4,749] were included in the meta-analysis for childhood depression and Cluster B PDs. The results from both LCM (Figure 5) and MCM (OR=1.67, CI=1.38-2.03, p<.001) were highly significant suggesting that childhood depression increases the risk for Cluster B PDs. ORs were identical in the two models. In both models, removal of individual studies did not change the results. Heterogeneity was moderate (LCM: Q =

6.76; p<.10; I^2=56%; MCM: Q = 6.76; p<.10; I^2=56%). Meta-regressions revealed one marginally significant moderator: the higher the age at baseline (LCM and MCM: b=0.03; p<.10) the stronger the association between childhood depression and Cluster B PDs.

2.3.4.2 Childhood Internalising problems and Antisocial Personality Disorder/Psychopathy

Six papers [III, IV, VI, X, XV, XVIII; N=5,428] were included in the meta-analysis for childhood INT problems and Antisocial PD/psychopathy. Results from both LCM (Figure 4) and MCM (OR=1.20; 95% CI =0.79-1.85, p=0.395) suggested that childhood INT problems do not significantly increase the risk for Antisocial PD. The effect size dropped from 1.23 to 1.20 in MCM compared to LCM. However, removal of the two studies that were specifically about psychopathy rather than Antisocial PD [VI, XV] resulted in a significant association between childhood INT problems and Antisocial PD (for both models: OR=1.37; 95% CI =1.22-1.53, p<.001; N=4,932). This suggests that there may be differences in the predictive validity of childhood INT problems with regard to Antisocial PD and psychopathy. In both models, included studies were considerably heterogeneous (for LCM: Q = 35.88; p< .001; I^2=81%; for MCM: Q = 39.36; p< .001; I^2=87%). Meta-regressions revealed two significant and two marginally significant moderators for LCM: the higher the age at baseline (b=0.05; p<.001) and the lower the age at follow-up (b=-0.03; p<.001), and the higher the proportion of males in the sample (b=0.33; p<.10) and the higher the quality of PD assessments (b=0.17; p<.10), the stronger the association between childhood INT problems and Antisocial PD/psychopathy.

2.3.5 Externalising problems and Cluster A and C PDs

Cluster A Five studies [VIII, XXIII, XIV, XXVI, XXII; N = 1,818] were included in the meta-analysis for childhood EXT problems and Cluster A PDs (Figure 2). Heterogeneity among studies was high (Q = 11.52; p<.05; I^2=65%). LCM and MCM were identical. The results suggested that childhood EXT problems increase the risk for Cluster A PDs (OR=1.51, CI=1.17-1.94, p<.01). Removal of study XXVI resulted in a slightly decreased, yet significant, OR (p<.05). Meta-regressions revealed two significant moderators: the higher the age at

baseline (b=0.08; p<.01) and the higher the proportion of males in the sample (b=2.66; p<.01), the stronger the association between childhood EXT problems and Cluster A PDs.

Cluster C. Four studies [VIII, XIII, XXIII, XXIII; N =1,088] were included in the meta-analysis for childhood EXT problems and Cluster C PDs (Figure 2). LCM and MCM were identical. The results suggested that childhood EXT problems do not increase the risk for Cluster C PDs (OR= 1.53, CI=0.81-2.91). However, removal of study VIII lead to a significant result (OR=1.19, CI=1.08-1.32, p<.01). Heterogeneity among studies was moderate (Q = 5.68; p=0.128; I^2=47%). Meta-regressions revealed a marginally significant moderator: the higher the age at baseline (b=0.27; p<.10), the stronger the association between childhood EXT problems and Cluster C PDs.

2.3.5.1 Externalising problems (ADHD) and Avoidant Personality Disorder

Three studies [VIII, XIII, XXII; N = 447] were included in the meta-analysis for childhood EXT problems and Avoidant PD (Figure 3). All of these studies were about ADHD. Between study heterogeneity was low (Q test: Q = 2.60; p=0.273; I^2=23%). LCM and MCM were identical. The results were marginally significant (OR=2.83, CI=0.89-8.99; p<.10), and removal of study XIII resulted in a significant OR (OR=2.83, CI=0.89-8.99, p<.05), suggesting that childhood EXT problems (ADHD) may increase the risk for Avoidant PD.

2.3.5.2 Externalising problems and Paranoid Personality Disorder

Three studies [VIII, XIV, XXII; N = 411] were included in the meta-analysis for childhood EXT problems and Paranoid PD (Figure 3). Between study homogeneity was high (Q test: Q = 0.56; p=0.757; I^2=0%). LCM and MCM were identical. The results suggest that childhood EXT problems significantly increase the risk for Paranoid PD (OR=1.89; CI=1.42-2.50; p<.001; see Figure 12). However, further sensitivity analyses did not confirm this: when study XIV was removed, the resulting OR did not retain its statistical significance. This suggests that child EXT problems might increase the risk for Paranoid PD; however, single studies are highly influential and interfere with the statistical model.

2.3.5.3 Externalising problems and Schizotypal Personality Disorder

Three studies [VIII, XXVI, XXII; N = 1,003] were included in the meta-analysis for childhood EXT problems and Schizotypal PD (Figure 3). Between study homogeneity was high (Q test: $Q = 0.57$; $p=0.751$; $I^2=0\%$). LCM and MCM were identical. The results suggest that childhood EXT problems significantly increase the risk for Schizotypal PD (OR=1.52; CI=1.18-1.97; p<.01). However, further sensitivity analyses did not confirm this: when study XXVI was removed, the resulting OR did not retain its statistical significance. This suggests that child EXT problems might increase the risk for Schizotypal PD; however, single studies are highly influential and interfere with the statistical model.

2.3.6 Internalising problems and Cluster A and C PDs

Separate meta-analyses for internalising childhood problems and Cluster A and C PDs could not be carried out due to a lack of published studies in these areas.

Table 3: Summary of relationships between childhood predictor and young adult outcome variables

		EXT	*ADHD*	*CD*	*ODD*	INT	*ANX*	*DEPR*
Cluster B		***	***	***	***	**	a	***
	Antisocial PD/ Psychopathy	***	***	***	***	n.s. (*** w/o psychopathy)	n.s. (*** w/o psychopathy)	-
	Borderline PD	***	***	-	-	-	-	-
	Narcissistic PD	-	-	-	-	-	-	-
	Histrionic PD	a	a	-	-	-	-	-
Cluster A		*	-	-	-	-	-	-
	Paranoid PD	***	-	-	-	-	-	-
	Schizotypal PD	*	*	-	-	-	-	-
	Schizoid PD	-	-	-	-	-	-	-
Cluster C		n.s.	*	-	-	-	-	-
	Dependent PD	-	-	-	-	-	-	-
	Avoidant PD	*	*	-	-	-	-	-
	Obsessive-Compulsive PD	-	-	-	-	-	-	-

**** p<.001; ** p<.01; * p<.05; ª <.10; n.s. - not significant*

2.4 Discussion

The aim of this research was to systematically collate and meta-analyse the findings from prospective longitudinal studies that examined childhood risk factors for adolescent or adult PDs. The focus was on child precursors, specifically on child EXT and INT problems. The results supported the general consensus that PD symptom constellations identified in adulthood can be predicted on the basis of common childhood problems. However, results also revealed a profound lack of published research: with the exception of Borderline PD and Antisocial PD, searches produced mostly insufficient numbers of well-designed prospective longitudinal studies to produce reliable estimates of associations, which is striking, considering that over 30 years of published studies were searched. The findings are discussed below, first focusing on Cluster B, and particularly Borderline PD and Antisocial PD/psychopathy, followed by Clusters A and C.

2.4.1 Childhood Externalising Problems and Cluster B PDs

Strong and robust associations were found between childhood EXT problems and Cluster B PDs. These associations were consistent across specific PDs (Antisocial PD, Borderline PD and Histrionic PD) and specific child EXT problems (CD, ADHD, ODD). They remained significant when adding control variables into the model, and (with the exception of Histrionic PD) when removing individual studies from the model. These findings are in line with our predictions: Cluster B is the "dramatic-erratic" cluster with symptomatology on the externalising spectrum. DSM diagnostic criteria for Cluster B PDs share similarities with the diagnostic criteria for childhood EXT disorders, such as failure to conform to social norms with respect to lawful behaviours (Antisocial PD and CD, ODD), or impulsiveness (Antisocial PD, Borderline PD, ADHD). In fact, childhood CD is regarded as the 'childhood version' of Antisocial PD, and onset before age 15 is a diagnostic criterion for adult Antisocial PD in DSM (APA, 2013).

The findings were, however, rather unspecific both in relation to predictors and to outcome variables in that, all childhood EXT problems were predictive of all Cluster B PDs. This is in line with the view that

"externalising", as a broad, higher-order psychopathology factor, underpins the most commonly occurring EXT mental disorders and accounts for the covariance among childhood and adult EXT disorders (Krueger, 2002a; Krueger et al., 2001). Evidence suggests that there is a coherent genetic basis to EXT problems: numerous adoption, family and twin studies have demonstrated that EXT disorders share a common genetic liability (Kendler et al., 2003) and a highly heritable general vulnerability to all EXT disorders has been found to account for most of the familial resemblance (Hicks, Markon, Patrick, Krueger, & Newman, 2004). Further, evidence demonstrates moderate to strong continuities of EXT behaviours from early to middle childhood through adolescence and into adulthood (Fergusson, 1998). It is therefore not surprising that EXT problems detected in childhood show homotypic continuity over time and are expressed as adolescent/adult Cluster B PD in adolescence or adulthood.

Interestingly, CD, ODD and ADHD were all independently associated with Antisocial PD, yielding similar levels of risk for developing the disorder. CD is usually regarded as the childhood version of Antisocial PD and, according to DSM criteria, CD before the age of 15 needs to be confirmed in order to diagnose Antisocial PD (APA, 2013). Thus, one would have expected a unique, particularly strong association between CD and Antisocial PD. However, this was not the case, as both ODD and ADHD were also predictive of Antisocial PD.

ODD has often been conceptualised as a milder form of CD (Rey et al., 1988). Thus, the results could indicate that within the spectrum of conduct disorders, relatively mild behaviour problems (i.e. ODD) and more severe behaviour (i.e. CD) are both independently associated with Antisocial PD and show a similar level of risk for PD in adolescence/adulthood. Alternatively, the results could be in line with the view that ODD is an early stage in CD development (APA, 2000). In support of the latter are two studies included in this review that investigated the influence of both CD and ODD on Antisocial PD whilst controlling for the effect of the respective other disorder. In both cases, CD was predictive of Antisocial PD whilst controlling for ODD. In fact, ORs for the association between CD and Antisocial PD increased when controlling for the effects of ODD. Conversely, when controlling for CD, the association between ODD and Antisocial PD

became insignificant. These findings are in line with the theory that ODD only increases the risk for Antisocial PD if the child makes the developmental transition from ODD to CD during childhood (Lahey & Waldman, 2003; Loeber, Burke, & Lahey, 2002). Due to insufficient numbers of included papers, however, this hypothesis could not be formally tested.

Some have argued that childhood ADHD predicts Antisocial PD independent of CD (Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Mannuzza et al., 1993). Indeed, the results from this study support this argument as ADHD was independently predictive of Antisocial PD. However, all three studies that investigated the differential predictive effects of childhood ADHD and CD on Antisocial PD showed that CD predicted Antisocial PD when controlling for ADHD, but ADHD did not predict Antisocial PD when controlling for CD. Unfortunately, one of these papers [VII] did not provide exact data on nonsignificant relationships, reducing the number of papers suitable for pooling effect sizes to two, so differential effects of ADHD and CD on Antisocial PD could not be formally tested. More prospective longitudinal research is needed to clarify the associations between specific childhood EXT problems and Antisocial PD.

2.4.2 Childhood Internalising Problems and Cluster B PDs

Overall childhood INT problems were associated with Cluster B PDs, as were specific INT problems, i.e. depressive symptoms (strongly) and anxiety (marginally). This relationship may on the face of it seem surprising given that Cluster B symptomatology is on the externalising spectrum, so indicating heterotypic continuity of childhood INT problems. The findings are, however, in line with current theories and cross-sectional evidence regarding the relationship between Cluster B PDs and INT problems. Whilst Cluster B symptomatology is mostly on the externalising spectrum, it is very often co-morbid with INT problems. Community studies indicate that 30-40% of persons with Antisocial PD have lifetime major depression (Hamdi & Iacono, 2013) and 34–54% have a lifetime anxiety disorder (Goodwin & Hamilton, 2003; Lenzenweger, Lane, Loranger, & Kessler, 2007). These rates are even higher in individuals with Borderline PD diagnosis, where about 75% of individuals having a lifetime mood disorder (Grant et al., 2008; Zimmerman & Mattia, 1999). Significant associations between

childhood INT problems and adolescent/adult Cluster B PDs are in line with this, as was confirmed by this research.

The relationship between INT problems and Antisocial PD/psychopathy was not significant. However, removal of the two studies from the model that were specifically about psychopathy resulted in a highly significant association between INT problems and Antisocial PD. The initially non-significant association between Antisocial PD/psychopathy and INT problems appeared to be the result of included data about a negative relationship between fearfulness and psychopathy, which masked the otherwise significant relationship between child INT problems and Antisocial PD.

Antisocial PD and psychopathy have a lot of diagnostic overlap; they stem from the same underlying construct first described by Cleckley (Cleckley, 1941, 1976) and they were proposed to be combined in the revised conceptual model of PD in DSM-5. However, they differ in significant ways, with differences pertaining to the emphasis placed on personality traits. Psychopathy, on the one hand, is generally conceptualised through two broad factors: primary and secondary psychopathy (Karpman, 1941; Lykken, 1995; Mealey, 1995; Porter, 1996), which is supported by the two-dimensionality of widely used psychopathy assessment instruments such as the Psychopathy Checklist – Revised (PCL-R) (Hare, 1991, 2003). PCL-R Factor 1 describes psychopathic personality traits (e.g. lack of empathy, lack of remorse, glib charm) whereas Factor 2 captures behavioural indicators of antisocial deviance. Antisocial PD, on the other hand, is mostly conceptualised through the behavioural aspects of the disorder, similar to PCL-R Factor 2, with which it correlates highly (Hart & Hare, 1989), but it does not cover the personality trait aspects of PCL-R Factor 1. Thus, Antisocial PD and psychopathy show behavioural overlap, but Antisocial PD does not cover the personality aspects crucial for a diagnosis of psychopathy. Lykken (1995) explained these two psychopathy factors through underlying temperamental dispositions. He argued that primary psychopathy (Factor 1) is related to an innate fearless temperament, as a consequence of which it is associated with diminished sensitivity and responsiveness to threats and punishments. In contrast, he argued that secondary psychopathy (Factor 2/Antisocial PD) is related to abnormal

sensitivity to cues of rewards. These hypotheses have been supported by studies with incarcerated psychopaths compared to non-psychopaths (Newman, MacCoon, Vaughn, & Sadeh, 2005).

Psychopathy Factor 1 and Factor 2/Antisocial PD also show distinct correlates with EXT and INT symptomatology. Both psychopathy factors and Antisocial PD are clearly associated with EXT problems (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005; Hare, 1991; Patrick, Zempolich, & Levenston, 1997). This is in line with the findings of this research – childhood EXT problems were associated with Antisocial PD, with or without the inclusion of psychopathy in the model. Contrary relationships have been found between the two psychopathy factors and INT measures, however. In fact, fearlessness has been proposed to be the distinguishing factor between primary and secondary psychopathy (Lykken, 1995). PCL-R Factor 1 has been found to correlate positively with fearlessness and negatively with anxiety, whereas the opposite has been found for Factor 2/Antisocial PD among samples of both children (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999) and adults (Verona, Patrick, & Joiner, 2001).

The current results are in line with this: the significant relationships between both Antisocial PD and psychopathy with anxiety/fearfulness were in opposite directions, and initially cancelled each other out when included in the same model, resulting in an overall non-significant result. Removing studies about psychopathy from the model resulted in a highly significant association between Antisocial PD and INT problems. This finding suggests that not only are psychopathy and Antisocial PD differentiable in adulthood, but they are also likely to have distinct developmental profiles. INT problems appear to predict Antisocial PD, but not psychopathy, in line with the argument that fearless temperament distinguishes between primary psychopathy and secondary psychopathy/Antisocial PD (Lykken, 1995).

However, more longitudinal research is needed to clarify the developmental course of early EXT and INT problems and their prospective associations with Antisocial PD/psychopathy in adolescence/adulthood. Early identification and prevention is particularly important in this area, in light of evidence that 15-20% of the prison population have a diagnosis of psychopathy, and that psychopaths are more likely to reoffend, more

dangerous, more consistently violent and more driven by goal-directed violence (Porter, ten Brinke, & Wilson, 2009) than non-psychopaths. EXT childhood problems are predictive of both Antisocial PD and psychopathy, but the role of INT problems still needs to be clarified. The absence of INT problems, and a negative relationship with fearfulness, appears to be indicative of a higher risk for psychopathy. Regarding Antisocial PD, there is opposing evidence, with some showing that a combination of EXT and INT problems put children at a higher risk of developing Antisocial PD than EXT problems alone (Sourander et al., 2007) arguing for a generally higher level of psychopathology. Others show that INT problems, in fact, act as a protective factor and that EXT problems without INT problems lead to higher psychopathology. An alternative explanation is that, whilst child INT problems are on the face of it related to Antisocial PD, the effects can be explained by covariation with EXT problems and do not add to the prediction of Antisocial PD over and above the effects of EXT problems. Future research should address these issues.

2.4.3 Externalising childhood problems and Clusters A and C

The results for Clusters A and C were inconsistent and not robust. Childhood EXT problems were initially associated with Cluster A, but significance levels dropped when removing individual studies. In contrast, childhood EXT problems initially were not associated with Cluster C; however, when removing individual studies, the results became significant. The same pattern of inconsistency/non-robustness was found for specific Cluster A and C PDs (Paranoid PD, Avoidant PD, Schizotypal PD) – removal of individual studies resulted in significant changes of probability levels. These results may have been due to the very low number of included papers in these areas.

The findings highlight two points: Firstly, the amount of published research in this area is remarkably low. Over 30 years of studies were searched and only two to three papers were found for all Cluster A and C PDs and EXT problems. One of the major criticisms about the PD conceptualisation in DSM is that it lacks evidence and is not supported by empirical research. This review confirmed this for all PDs except Antisocial PD and Borderline PD: prospective longitudinal research is mostly lacking. Secondly, no clear

predictive pattern was found for Clusters A and C. This may have been due to the low number of included papers. Whilst EXT child problems showed strong homotypic continuity with Cluster B PDs, this was not found for Clusters A and C. One would expect mostly non-significant associations between childhood EXT problems and Clusters A and C – they are mostly associated with internalising symptomatology, and with core communalities and main characteristics described as: "a strong desire to control one's environment, restrained emotional expression, limited social interactions, problems with close relationships and cognitive and behavioural rigidity" (Lynch, Hempel, & Clark, 2012). Internalising disorders, such as major depressive disorder, are also more often comorbid with Clusters A and C compared with Cluster B PDs (Corruble, Ginestet, & Guelfi, 1996).

The inconsistency of these findings for Clusters A and C are in line with the critique of both the DSM cluster system, as well as with specific DSM PDs. Even the DSM acknowledges that the cluster system has "serious limitations" and has not been "consistently validated" (p.646). Most studies have rejected the cluster structure and reported evidence for four or five factors (Austin & Deary, 2000; Livesley, 1998; Mulder & Joyce, 1997; Sheets & Craighead, 2007). Further, specific PDs have been criticised for a number of reasons, including extreme heterogeneity among patients receiving the same diagnosis and poor coverage of personality psychopathology by the specific PDs. A large body of evidence supports the notion that personality pathology is much better captured in dimensional trait models. For instance, Markon et al. (2005) found a common hierarchical structure integrating both normal and maladaptive personality traits. Further, behaviour genetic studies (Kendler, Aggen, Czajkowski, & et al., 2008) found no evidence of distinct genetic factors that contributed to Clusters A, B, and C. Instead, the first of the three factors they found implied a general vulnerability for any PD (across all clusters). The other two factors had high loadings on just two PDs each, the first one on Antisocial PD and Borderline PD (both Cluster B), and the second one on Schizoid PD (Cluster A) and Avoidant PD (Cluster C). The majority of specific PDs only loads on the "general vulnerability" factor. The findings from Clusters A and C in this research are in line with the criticism of the DSM PD system, highlighting limitations with regard to validity.

2.4.4 Moderators and Covariates

Included studies varied a lot in terms of covariates considered in their models. In order to account for the impact of these differences, effect sizes were pooled in 'least control' and 'most control' models. This approach was adopted to provide a sense of the range of possible effects within each study. Surprisingly, for none of the separate meta-analyses carried out were there any significant differences between LC and MC models. Generally, adding covariates to the models only slightly lowered pooled effect sizes. However, none of the models significantly changed, which could argue for the robustness of the associations. However, this finding could also be due to the approach chosen to address the issue of heterogeneity in included studies. More specifically, for each predictor-outcome pair, the same studies were included, sometimes with identical data: for example, if a study only reported univariate data, this was included in both the LC and MC models. As a consequence, LC and MC models were often similar, in some cases even identical. However, it should be borne in mind that the aim of this approach was not to compare two entirely different models, but rather to provide a sense of the range of possible effect sizes within each model.

An overall trend indicated a moderate effect of gender on the association between EXT problems and Cluster B. Specifically, we found that the more males in the sample, the stronger the association between child EXT problems and Cluster B, particularly between childhood ADHD and Borderline PD. These findings are in line with evidence showing higher prevalence of EXT problems among males than females and with differential effects regarding the continuity of EXT problems, where significant associations were found among boys only (Rowe et al., 2010). Regarding INT problems, gender was marginally significant for anxiety and Cluster B and Antisocial PD/psychopathy, but these effects disappeared in MCM.

We also found moderating effects of age. The general trend for child INT problems was that, the older the children were at baseline and the younger the adolescents/adults were at follow-up, the stronger the association between INT problems and PD. This is in line with the notion that INT problems are more difficult to detect in younger children (Tandon, Cardeli,

& Luby, 2009), that is, assessments made later in childhood are likely to be more reliable than those made earlier on. It could also indicate that INT problems have less predictive validity in terms of PD development, at least when investigating heterotypic continuity (i.e. predictive validity of INT problems on EXT PDs). Due to insufficient numbers the effect of moderators on the relationship between INT child predictors and PDs could only be investigated in the domain of Cluster B.

For EXT childhood problems, we found diverging effects of age on the relationship with Antisocial PD and Borderline PD. Within the area of Antisocial PD, we found that the higher the age at follow-up, the stronger the association between EXT problems (especially ADHD) and Antisocial PD. For Borderline PD we found that the lower the age at baseline and the lower the age at follow-up the stronger the association between EXT problems (especially ADHD) and Borderline PD which might support findings that BPD symptoms decrease with age.

2.4.5 Limitations

The findings of this meta-analysis should be regarded in light of the following points:

1. A profound lack of studies investigating and Cluster A and C PDs did not enable us to draw any conclusions regarding the relationship between these PDs and INT problems. Further, inconsistencies between EXT problems and Clusters A and C could be due to the limited number of included papers, as well. Prospective longitudinal studies are needed to clarify these relationships.

2. Most of the meta-analyses conducted were subject to very high levels of heterogeneity between included studies. This can be explained by two main reasons: (i) A large variety of different assessment methods were used in included studies. It is therefore not entirely clear whether included studies are directly comparable. In fact, one of the major criticisms of the DSM categorical system has been poor convergent validity across PD assessments. Thus, heterogeneity among included studies using different assessment instruments is to be expected even among well-established PDs. (ii) PD categories have been criticised for high heterogeneity among

patients with the same disorder. For instance, even using well established, standardised, validated assessment instruments, such as the SCID II, there is a lot of variability in symptomatology among patients with the same disorder. For instance, it is possible that two individuals with a Borderline PD diagnosis only have one symptom in common. Thus, heterogeneity of samples even within the same group of PD is to be expected.

3. The choice of inclusion/exclusion criteria may have limited the findings. For instance, 32 papers were excluded due to the age cut-off, which may have resulted in inclusion/exclusion of papers that potentially might have shown different results. Further, only including studies that specifically assessed for PD in adolescence/adulthood may have excluded literature that implicitly assessed PD symptoms. However, this was mostly the case in the area of Antisocial PD, where some research was excluded that investigated certain behavioural aspects of Antisocial PD longitudinally (e.g. delinquency, criminality or violence) but did not specifically assess Antisocial PD. It is unlikely that exclusion of these studies biased the results as those studies that were excluded showed the same pattern as the studies that specifically assessed Antisocial PD and were included, i.e. homotypic continuity of EXT symptomatology.

4. Due to the limited number of studies, and given the overlap between different domains both in predictor and outcome variables, we could not specify precisely which factors were driving the observed effects. More prospective longitudinal research is needed to disentangle these relationships.

2.5 Chapter Summary

The findings from this review suggest that 1. Childhood EXT problems (ADHD, CD, ODD) are predictive of Cluster B PDs, specifically Antisocial PD/psychopathy and Borderline PD. Within the domain of EXT childhood predictors, CD appears to have a unique association with Antisocial PD; however, this relationship could not be formally tested due to insufficient number of included papers; 2. Childhood INT problems predict Antisocial PD when analysed without studies about psychopathy; 3. Results for Clusters A and C were mostly inconsistent and not robust, and it was not possible to

investigate INT childhood predictors for Cluster A and C due to the lack of studies in this area.

In short, signs and symptoms do appear in childhood that can predict personality pathology in adolescence/adulthood. However, the results were rather unspecific, and with the exception of Antisocial PD and Borderline PD, prospective longitudinal research is mostly lacking. More research is clearly needed to gain a better understanding of the developmental pathways leading to PD.

Chapter 3: Methodological Challenges

The research carried out for this thesis looked at early childhood externalising (EXT) and internalising (INT) predictors of adult personality disorders (PDs) in a prospective longitudinal study. Prospective longitudinal research is a strong research methodology and has many advantages over cross-sectional research. Clinically, longitudinal studies are immensely valuable because longitudinal data can provide information about individual change, whereas cross-sectional data cannot. Because individual change can be studied within individuals, longitudinal studies can be helpful in determining who might be most at risk of a negative development and, as such, most benefit from a particular intervention. However, longitudinal studies also have their challenges. This chapter will provide an overview about the methodological challenges encountered when conducting this research. In order to put these points into context, an outline of the methods applied in this research will first be given.

3.1 Outline of methods applied in this research

The research conducted for this PhD investigated early childhood predictors of PD using a prospective longitudinal design. Study one (Chapter 4) investigated childhood predictors of PD in the form of EXT problems and INT problems. Studies two (Chapter 5) and three (Chapter 6) addressed whether these associations were influenced by negative parenting behaviours or continuities of child psychopathology into adolescence/adulthood, respectively. This research was carried out as part of a subproject of, and in collaboration with, a larger scale research initiative, namely the Programme for Early Detection and Intervention for ADHD (PEDIA).

3.1.1 Participants

Baseline data was derived from an existing database of a population cohort of 4,199 preschool children and their families living in the Southampton / New Forest area who were assessed with a set of standardised instruments

at the time of their three year development check, between 1989 and 1997, by the family health visitor. The baseline sample consisted of three different cohorts: data for these different cohorts was collected in 1990-1992, 1994-1995 and 1998-1999, respectively. Specific assessments carried out varied for each cohort; however, all three cohorts were assessed for childhood overactivity, behavioural problems and temperament. At follow-up, a subgroup of these children (now aged 16-25) was assessed for PDs as well as for several other EXT and INT psychopathological variables. In addition, negative parenting behaviour was assessed. Details about how this subgroup of participants was selected will be given below. Both PD and negative parenting was assessed solely for the purposes of this PhD. The instruments assessing these constructs were added to the PEDIA assessment battery after ethical approval was obtained for this PhD research. EXT and INT psychopathological variables were already part of the assessments carried out for PEDIA, but used for this PhD as well. In addition, several psychometric assessments, as well as an interview addressing family background and other variables, were administered; however, these were only used for PEDIA and will not be described here.

3.1.2 Procedure

The majority of data for this study was collected jointly with Research Fellows of PEDIA. There was a small group of families that only took part in the PEDIA study without completing measures for the PhD; these families will not be considered here, only the procedure that involved families who took part in the PhD research will be described. Prior to the commencement of my PhD, I was a Research Fellow on the PEDIA project myself; therefore, I was involved in all parts of the data collection, even though it was initially not collected for the purposes of this PhD.

All families were approached and invited to participate in the study by letter. If no response was obtained after 2-3 weeks, a reminder letter was posted out, followed by a second reminder after another 2-3 weeks. Initially, all assessments were carried out face to face by two researchers who met with the families, usually in their own homes. Visits lasted between 30 – 180 minutes, on average around 60 minutes. Families received £40 for taking part. This phase of data collection was carried out prior to the

commencement of the PhD. After the questionnaires for the PhD were added to the assessment battery, they were offered the option of completing the measures for the present study by post, or online. If they agreed to take part by post, the questionnaires were posted out to them, together with a freepost return envelope. If they agreed to take part online, they were provided with a unique username and password to complete the questionnaires on iSurvey, which is a secure online software developed by the University of Southampton to complete online surveys. Questionnaire completion took up to one hour. Those participants, who had already taken part in PEDIA before the PhD measures were added to the assessment battery, were re-approached and paid an additional £10 for completing the extra measures. Recruitment and data collection by post and face-to-face was carried out in collaboration with PEDIA Research Fellows. Online recruitment and data collection was carried out by the author.

Current addresses and contact details of target families were traced by the author in collaboration with PEDIA Research Fellows, using public records, namely 192.com, which is an online public registry of electoral roll data; the rate of correctly traced families was 79% (i.e. addresses of 21% of potential participants could not be identified). A total number of N=423 suitable families were approached (details about how these families were selected are presented on pp. 84). Participants were selected randomly from a pool of suitable families.

164 of these families (39%) did not respond to the invitations sent out; 13 (3%) only partly completed the assessments or did not return questionnaires sent out by post, so the data could not be used for analyses; and ten families refused to take part (2%). A total of N=216 families completed all assessments and were included in analyses (consent rate: 51%).

3.1.3 Baseline assessments

The following baseline assessments were carried out for all three cohorts (see Table 4 for an overview). All of the scales/subscales were considered as the basis for selecting the follow-up sample and most of them were also used as childhood predictor variables (see Table 4 for an overview).

3.1.3.1 The Werry-Weiss-Peter Activity Scale (WWP) (Routh, 1978)

The WWP is a 27-item screening measure for children's hyperactivity levels. Psychometric properties have been reviewed by Barkley (1988), who reported discrimination between hyperactive and normally developing children to be good. Agreement between both parents has also been found to be good (r = 0.82) (Mash & Johnson, 1983). The WWP has been shown to have high levels of internal consistency, to correlate with other measures of hyperactivity and to identify children who have activity problems 5 years later (Sonuga-Barke, Stevenson, Thompson, & Viney, 1997). Items are rated by a caregiver and are scored from 0 – "no, or hardly ever"; to 2 – "yes, very often". The total score can range from 0 – 54 and gives an indication of the child's hyperactivity levels. This measure has been shown to identify the top 15 to 18% of the population using a score of 20 as a cut-off (Thompson et al., 1996). Cronbach's alpha in this sample was .93. For the full measure, please see Appendix A.6.

3.1.3.2 Behavioural problems – The Behaviour Checklist (BCL) (Richman, 1977)

The BCL is a 19-item screening questionnaire for parents and gives ratings of behaviour problems in a range of different domains. Items are rated by a caregiver and are scored from 0–2. The scale provides an aggregate Total Problems Score ranging from 0-38, as well as factor scores for Poor Social Adjustment, Poor Emotional Adjustment, Sleep Problems, Overactivity/Inattention, Eating Problems and Soiling, with a score range of 0-6 for each subscale (Sonuga-Barke, Thompson, Stevenson, & Viney, 1997). The validity and reliability of the BCL have been demonstrated through various methodologies, including observations of children, comparisons between clinical and nonclinical populations, and comparisons with other screening questionnaires (Boyle & Jones, 1985; Koot, Van den Oord, Verhulst, & Boomsma, 1997; McGuire & Richman, 1986; Sonuga-Barke, Stevenson, et al., 1997). Several researchers have used the instrument to detect preschoolers at risk for behaviour problems (Thompson et al., 1996). For the purposes of this study, only the subscales concerned with behavioural problems were considered. In this sample, Cronbach's alpha was .72 for the total score and ranged from .45 (Overactivity/Inattention

and Poor Emotional Adjustment) to .68 (Poor Social Adjustment) for subscales. These values are comparable to alpha values obtained in other preschool samples (Mathiesen & Sanson, 2000). Poor Social Adjustment was included to assess conduct problems, Poor Emotional Adjustment was included to assess emotional problems. Overactivity was used for sample selection only, not as a predictor due to overlap with the content of the WWP. For the full measure, please see Appendix A.8.

3.1.3.3 EAS Temperament Scale (Buss & Plomin, 1984)

The EAS is a 15-item temperament questionnaire used in children from 1-9 years on a 5-point Likert-type scale, ranging from 1 - not characteristic or typical of your child, to 5 - very characteristic or typical of your child. It consists of 3 subscales: Emotionality, Activity, and Shyness. The scale provides a total score for difficult temperament, ranging from 0-75, as well as scores for individual subscales, with scores ranging from 0-25 for each subscale. Only subscales Emotionality and Shyness were available for all three cohorts, so Activity was not considered. In this sample, only the subscale Shyness was used as a predictor variable; emotionality was not considered as a predictor variable due to content overlap with emotional problems (BCL). Cronbach's alpha was .82 for shyness, which is similar to alpha values that have been obtained in comparable samples (Mathiesen & Tambs, 1999; Mathiesen & Samson, 2000). Previous research has established a mean score of 3 or higher as a good indicator for high levels of shyness (Thompson et al, 1996). For the full measure, please see Appendix A.7.

Table 4: Baseline assessments used for sample selection and/or as longitudinal predictors of personality disorder

Measure	Assesses	Number of items	Total score range	Cronbach alpha	Used for sample selection	Used as predictor variable
Werry-Weiss-Peter Activity Scale (WWP)	Hyperactivity (EXT)	27	0-54	.93	✓	✓
Behaviour Checklist (BCL) subscales:						
Overactivity	Hyperactivity (EXT)	3	0-6	.45	✓	-
Poor Emotional Adjustment	Emotional Problems (INT)	3	0-6	.45	✓	✓
Poor Social Adjustment	Conduct Problems (INT)	5	0-10	.68	✓	✓
EAS Temperament Scale subscales:						
Shyness	Shyness (INT)	5	25	.82	✓	✓

EXT – externalising; INT – internalising

3.1.3.4 Socio-economic status

Socio-economic status (SES) at baseline was estimated using a measure of deprivation, namely the Carstairs score (Carstairs & Morris, 1991) which was calculated for all wards in the UK using 1991 census data about the characteristics of families living in different postcode regions. The four components of the score are the proportion of male unemployment, proportion of people living in overcrowded households, proportion of people in social classes IV and V, and proportion of people in households without access to a car. Each component of the score was standardised across Great Britain to have zero mean and unit variance and combined in a single continuous score. High scores reflect greater deprivation. Carstairs scores can also be classified into quintiles enabling comparisons to be made to the general UK population. Within the sample followed up for this PhD, 7.5% of participants scored within the fifth quintile (most deprived 20% of the UK population) and 14.2% scored within the first quintile (least deprived 20% of the UK population). The remaining 78.3% scored within in the middle 60% (quintiles 2, 3 or 4). Carstairs scores were estimated from postcodes of the original dataset.

3.1.4 Follow-up assessments

For an overview about all included follow-up measures, please see Table 5.

Table 5: Overview of follow-up assessments

Measure	Assesses	Number of items	Likert scale	Total score range	Cronbach alpha
Conners Behavior Rating Scale – Self-Report	ADHD	10	0-3	0-30	.89
	CD	14		0-42	.97
	ODD	8		0-24	.88
	MDD	15		0-45	.94
	GAD	13		0-39	.94
	SP	6		0-18	.87
PID-5	BPD	64	0-4	0-256	.96
	ASPD	66		0-264	.95
	NPD	14		0-56	.90
	OCPD	19		0-76	.92
	AVPD	33		0-132	.94
	SPD	57		0-228	.96
MOPS	Mother overcontrol	4	0-4	0-16	.81
	Mother indifference	6		0-24	.94
	Father overcontrol	4		0-16	.78
	Father indifference	6		0-24	.96

ADHD – Attention Deficit Hyperactivity Disorder; CD – Conduct Disorder; ODD – Oppositional Defiant Disorder; MDD – Major Depressive Disorder; GAD – Generalised Anxiety Disorder; SP – Social Phobia; B, AS, N, OC, AV, S (PD) – Borderline, Antisocial, Narcissistic, Obsessive-Compulsive, Avoidant, Schizotypal (Personality Disorder); PID-5 – Personality Inventory for DSM-5; MOPS – Measure of Parental Style

The following assessments were carried out at follow-up:

3.1.4.1 Personality Inventory for DSM-5 (Krueger et al., 2012)

PD was assessed using the Personality Inventory for DSM-5 (PID-5) (Krueger et al., 2012). The PID-5 was created specifically for assessing personality pathology in accordance with the new personality model proposed for the DSM-5 at the time of designing the method for this project (January 2012). The revisions were discarded before publication of the DSM-5; nevertheless, the PID-5 has become a widely used instrument, showing good reliability, specificity and sensitivity (Morey & Skodol, 2013). It is a 220-item self-report questionnaire with a four-point Likert-type response scale, ranging from 0 "very false or often false" to 3 "very true or often true". It has 25 primary scales that load onto 5 higher-order scales, namely Negative Affect, Detachment, Antagonism, Disinhibition, and Psychoticism which closely align with the Big Five dimensions of normal personality. Due to ethical considerations, three items of the depressivity subscale relating to suicidality ("I talk about suicide a lot"; "I know I'll commit suicide sooner or later", "The world would be better off if I were dead") were removed from the questionnaire for this study. This resulted in a total number of 117 items, taking approximately 20 minutes to complete. For the full measure, please see Appendix A.9.

Whilst the PID-5 is a variable-centred multi-dimensional measure, it can also be used to assess the specific DSM PDs (Morey & Skodol, 2013). Diagnostic decision rules based on thresholds of combinations of traits can also be employed for six of the specific DSM-IV PDs; they show good kappa coefficients of agreement between DSM-IV and PID-5 diagnoses (Morey & Skodol, 2013). That is, based on PID-5 scores of specific combinations of subscales, diagnostic decisions can be made for Borderline PD, Antisocial PD, Avoidant PD, Schizotypal PD, Narcissistic PD and Obsessive-Compulsive PD that closely align with diagnostic decisions made based on DSM-IV criteria. Dimensional scores for each of these PDs were created using the subscale score combinations suggested by Morey and Skodol (2013). BPD dimensional scores consisted of four subscale scores from Negative Affectivity (Emotional Lability, Anxiousness, Separation Insecurity,

Depressivity), two from Disinhibition (Impulsivity, Risk Taking) and one from Antagonism (Hostility). Avoidant PD consisted of four subscale scores from Detachment (Withdrawal, Intimacy Avoidance, Anhedonia) and one from Negative Affectivity (Anxiousness). Obsessive-Compulsive PD consisted of one subscale from Negative Affectivity (Perseveration) and one from Disinhibiton (inverse: Rigid Perfectionism). Antisocial PD consisted of four subscales from Antagonism (Manipulativeness, Deceitfulness, Callousness, Hostility) and three from Disinhibition (Irresponsibility, Impulsivity, Risk Taking). Narcissistic PD consisted of two subscales from Antagonism (Grandiosity, Attention-Seeking), and Schizotypal PD consisted of three subscales from Psychoticism (Eccentricity, Cognitive and Perceptual Dysregulation, Unusual Beliefs and Experiences), two from Detachment (Restricted Affectivity, Withdrawal) and one from Negative Affectivity (Suspiciousness). Alpha values were excellent: Borderline PD: alpha=.96, Avoidant PD: alpha=.94, Obsessive-Compulsive PD: alpha=.92, Antisocial PD: alpha=.94, Narcissistic PD: alpha=.90, Schizotypal PD: alpha=.96.

3.1.4.2 The Measure of Parental Style questionnaire (MOPS) (Parker et al., 1997)

Negative parenting dimensions overcontrol and lack of warmth were assessed through the subscales parental overcontrol and parental indifference of the Measure of Parental Style questionnaire (MOPS) (Parker et al., 1997). Assessments were made retrospectively by the young person, rating maternal and paternal parenting behaviours separately in the first 16 years of his/her life. That is, the scale consisted of four subscales: maternal overcontrol, maternal indifference, paternal overcontrol and paternal indifference. Subscales are scored on a 4-point Likert scale indicating the degree of agreement with the item statement. The subscale overcontrol consists of four items, the subscale indifference consists of 6 items. The indifference subscale measures the degree to which the parent was empathic and caring or cold and indifferent, while the overprotection subscale measures the extent to which the parent was intrusive and infantilising, or fostered independence in the child, in his/her first 16 years of life, as recalled by the child. In this study, the Cronbach coefficient alpha was 0.94 for mother indifference, 0.96 for father indifference, 0.81 for

mother overcontrol and 0.78 for father overcontrol. For the full measure, please see Appendix A.10.

3.1.4.3 Conners Behavioural Rating Scale – Self Report (CBRS-S) (Conners, 2008)

Adult EXT and INT psychopathology was assessed through the Conners Behavioural Rating Scale – Self Report (CBRS-S) (Conners, 2008) which was designed to provide a complete overview of child and adolescent concerns and behaviours. It consists of 179 items. Items are scored on a four point Likert scale as 0 (not at all true), 1 (just a little true), 2 (pretty much true), and 3 (very much true) with responses indicating the extent to which each symptom applies to the individual's behaviour over the past month. Conners CBRS has been found to have good psychometric properties including good validity, internal consistency, inter-rater reliability and test-retest reliability (Conners, 2008). Cronbach's alpha values range from .69 to .97, and 2- to 4-week test-retest reliability coefficients range from .56 to .96. Inter-rater reliability coefficients range from .50 to .89 (Conners, 2008). Support for the validity of the structure of the Conners CBRS was obtained using factor analytic techniques on derivation and confirmatory samples. Convergent and divergent validity were supported by examining the relationship between Conners CBRS scores and other related measures. Overall, scales that assess similar constructs tended to be moderately to strongly intercorrelated, while scales that did not assess similar constructs tended to have smaller correlations. Results from discriminative validity analyses indicated that the Conners CBRS scores accurately discriminate between relevant groups. Results from a series of multivariate analysis of covariance revealed that, for all scales, the means for the target clinical groups were significantly higher than the means for the general population and other clinical groups. In terms of the classification accuracy of the scores (as determined by a series of discriminant function analyses), the mean overall correct classification rate was 78% across all forms.

The CBRS-S is designed for use between the ages of 8-18yrs. The version used in this study was adapted slightly (with agreement of the publisher) to be used with older respondents as this sample included participants aged 16-25. More specifically, a number of items were modified to make them

developmentally relevant for the young adults in the study. This version omitted items relating to separation fears (e.g. 'I get scared if I am not with my family or other adults I know well.'), and one ADHD item inappropriate for older respondents ('I run around even when I am not supposed to'). Scoring was completed using the Conners CBRS Software programme. The double entry feature was used to verify accuracy of the data entry. The software generates raw scores, t-scores, percentiles and standard error scores for a range of subscales. In this study, subscales ADHD, Conduct Disorder and Oppositional Defiant Disorder were used to assess EXT disorders, and Major Depressive Episode, Generalised Anxiety Disorder, and Social Phobia were used to assess INT disorders. For the purpose of analysis, only raw scores were considered: t-scores convert the raw scores to a standardised score that reflects what is typical or atypical for that age and gender; however, as the scales are normed up to age 18 only, and the age ranged from 16-25 in this sample, raw scores were more appropriate. In this sample, alpha values ranged from .87 (Social Phobia) to .97 (Conduct Disorder); please see Table 5 for details. For the full measure, please see Appendix A.11.

3.2 Main challenges encountered

The following sections will provide an overview about the methodological challenges encountered when conducting this research. The key challenges were issues related to (1) participant attrition, (2) sampling, (3) exploiting an existing database, and (4) follow-up measures, including choice of instruments and shared method variance. Below, I will assess how each of these key challenges affected this research, outlining the literature associated with each, as well as describing how each issue was addressed.

3.2.1 Attrition

One of the main challenges associated with carrying out prospective longitudinal research is the high likelihood of attrition. Attrition refers to participants removing themselves from the research, prior to the end of the study. Attrition can be problematic because it can threaten the internal and

external validity of the study through a selection bias (Frees, 2004), by creating a significantly reduced sample size, producing non-representative groups, and causing a decrease in statistical power (Prinz et al., 2001). In addition, if attrition is non-random, i.e. systematically related to characteristics of the participants, any conclusions drawn from the study may be erroneous (Wolke et al., 2009), particularly if those characteristics are variables of interest in the study.

Two main reasons for attrition have been proposed (Capaldi & Patterson, 1987): (1) Losing contact with the participants; and (2) Participants' refusal to continue participation. In the current sample, attrition was expected to be high due to both of these aspects. These issues and how they were handled are discussed below.

3.2.1.1 Losing contact with participants

In longitudinal research, it is almost inevitable that a percentage of the sample will become difficult to locate (Cotter, Burke, Loeber, & Navratil, 2002), especially if the follow-up periods are extensive. However, it has been argued that most participants can be retrieved and that "if retention is ultimately poor in a longitudinal study, it is usually because little or no effort was made to do so" (Cotter et al., 2002; p. 488).

Locating participants was expected to be difficult in this study due to the substantial length of time that had passed between initial assessments at offspring age 3 and follow-up, ranging from 11 years for the youngest and 23 years for the oldest participants, increasing the likelihood that families would have relocated in the meantime. In addition, whilst at the time of the baseline assessments families had consented to be contacted again for any further research, these initial assessments were originally not carried out for the purposes of conducting a follow-up study. Thus, no efforts were made by the initial research team to prevent attrition, e.g. through examining potential issues or maintaining contact with the families and update their contact details if they relocated, which have been suggested to be vital steps in reducing attrition in longitudinal studies (Cotter et al., 2002; Stouthamer-Loeber, Van Kammen, & Loeber, 1992). Furthermore, most original contact details of families were missing, and participants had to be located using public records only.

3.2.1.2 Refusal to continue participation

Previous research has also shown that certain factors predict drop-out rates: in general, longer follow-up periods are associated with higher attrition (Schaffer, 1996). Further, studies have demonstrated that EXT problems and general psychopathology among children were associated with a higher risk of parents dropping out of studies (Cotter, Burke, Stouthamer-Loeber, & Loeber, 2005). In addition, certain socio-demographic variables, such as low educational level, being out of work, and not being married, are typically related to an increased risk of non-response and attrition (Badawi, Eaton, Myllyluoma, Weimer, & Gallo, 1999; Bjerkeset, Nordahl, Larsson, Dahl, & Linaker, 2008; Tambs et al., 2009). Based on these factors, drop-out rates were expected to be high. Selection criteria for this sample, i.e. levels of problem behaviour, have been linked with high levels of attrition. Furthermore, the families of interest often also had certain other characteristics that made a low response rate somewhat more likely than a randomly selected sample, i.e. socio-demographic variables, such as low educational level, and being a single mother.

In addition, the research team that collected data at baseline did not increase the chances of successful follow-up by making efforts to minimise the risk for dropping out. Retaining reluctant participants has been argued to be the most difficult task for project staff (Cotter et al., 2002). It has been suggested that, in order to retain participants in a study, careful examination of the reasons for attrition is required, followed by implementation of procedures to address these issues (Ullman & Newcomb, 1998). For instance, it has been proposed that a crucial task of the interviewer is to uncover potential issues participants may have with future participation (Cotter et al., 2002), and to maintain regular contact between assessments in order to retain participants in a study (Stouthamer-Loeber et al., 1992). However, the baseline assessments were initially not carried out for the purposes of conducting a follow-up study. Therefore, the initial research team did not maintain contact with the families, or attempted to prevent attrition by assessing individual barriers for taking part in the follow-up study. That is, whilst families did not have any objections to being contacted again in the future, they were not made aware that they would be contacted over 10 years later to take part in the follow-up. The majority of

those families that did consent to follow-up commented that they did not remember taking part in the first part of the study.

3.2.1.3 Strategies to increase participation rate

Several strategies were applied to increase the participation rate. Firstly, to increase the rate of correctly traced families, several approaches were used. As we were only able to use public records, several sources were consulted: initially, an online database was searched, namely 192.com, which is an online directory listing people and businesses, including addresses, current and historical electoral rolls, birth death and marriage registers. In addition, social network sites (e.g. Facebook) were searched where public profiles were available, and general internet searches (via Google) were performed. Secondly, several attempts were made to increase the number of responses received by families. They were initially contacted by post, with an information pack to participate in the study. They were also sent two reminder letters if they failed to respond to the first one. The contact letter was amended several times, according to comments from families who had taken part. In addition, participants were offered a variety of response methods (i.e. returning an expression of interest form, responding by email or text message). Participants were also offered several options to complete the assessments. Initially, they were asked to meet face to face with the researchers. At a later stage in the study, however, they were also provided with the option of completing the assessments online, via secure online software, or by post, to accommodate most preferences. Finally, the amount of money offered to families for taking part was increased. Initially they were offered £20 per family; this amount was increased to £40 per family.

3.2.1.4 Dealing with high attrition rates

High attrition rates in longitudinal research are not unusual - attrition rates from 30 to 70% are often reported (Badawi et al., 1999; Bjerkeset et al., 2008; Fischer, Dornelas, & Goethe, 2001; Goodman & Blum, 1996; Miller & Wright, 1995; Tambs et al., 2009). In general, higher attrition rates are expected after a long period before follow-up compared to short-term follow-up (Gustavson, Von Soest, Karevold, & Røysamb, 2012). Two main approaches are usually adopted to handle the issue of missing data (Mostafa & Wiggins,

2014). Firstly, weighting of cases to adjust the distributions of the responders so that the relative importance of each participant's characteristic is adjusted according to the importance of the characteristics of those who dropped out. Whilst weighting is an easy method to apply, it has a number of disadvantages. For instance, if certain variables are used to predict non-response (and thus are used to construct the weights) then the results of analyses using these variables as DVs and IVs will yield unbiased results. However, if other variables, which are not included in the process of constructing weights, affect the sample, then the sample will still be biased, because these characteristics were not considered. Secondly, random multiple imputation, (Little & Rubin, 2002; Rubin, 1987) can be applied. The two main advantages of multiple imputations are (1) Multiple imputations allow the treatment of both item and unit non-response. (2) Multiple imputations can be custom-made and are robust and generate valid inference. The downside is that the technique depends on the assumption that data is missing at random (MAR) as opposed to data missing not at random (MNAR) (Little & Rubin, 2002).

3.2.2 Sample selection

The second key challenge encountered when conducting this research was the issue of whether to approach the entire sample of N=4,199 for follow-up, or whether to select an appropriate subsample. Due to time and financial constraints, it was decided to test the relationships between baseline and outcome variables in a subgroup of this population. The challenge was to systematically select a subsample within which any predictive relationship between baseline and follow-up variables would be observable despite a reduced sample size. As argued by McClelland (1997), using nonoptimal sample designs can lead to either (1) increased costs to compensate for design inefficiencies or to (2) reduced statistical power for detecting the effects of interest. To ensure sufficient power, an enrichment strategy (described below), based on empirical examination of the baseline questionnaires, was applied.

Power is the probability of rejecting H0 when H1 is true; i.e. power represents the probability that effects have the chance of producing statistically significant results (Tabachnick & Fidell, 2013). Issues relating to

power need to be addressed before designing a study so that the chances of failure to produce a significant effect are decreased. Many of the choices in research design are made in order to increase power. A power level of .80 has been suggested as a minimum by Cohen (1988). There are several ways to enhance statistical power; two of the most common are (1) to increase the variance in the independent variables, and (2) to increase the sample size (McClelland, 1997).

In order to increase the range of scores in the independent variables (and therefore the variance), the subsample that was chosen for follow-up was not chosen at random but selected based on specific predetermined criteria. As put forth by McClelland (1997), the optimal design for a linear effect is to select participants at the most extreme levels of the independent variable, where one half are allocated to each extreme. Even designs with unequal proportions, McClelland argues, are reasonably efficient as long as all the observations are at extreme levels, unless the ratio of the number of observations at the two extreme levels exceeds 5.8:1.

Significance testing, and therefore statistical power, is related to sampling error, i.e. the error that arises as a result of taking a sample from a population rather than using the whole population (Lipsey & Hurley, 2009). Because sampling errors are smaller for large samples, they are less likely to obscure real effects and statistical power is greater. For any study, the sample has to be large enough so that it facilitates revelation of meaningful effects (Tabachnick & Fidell, 2013). Green (1991) suggested that, as a rule of thumb, $N \geq 50 + 8m$ (where m is the number of DVs) are required for testing multiple correlation, and $N \geq 104 + m$ for testing individual predictors. If both are tested, the larger sample size should be used. Alternatively, Harris (1985) proposed that, 1. For regression equations with five or fewer predictors, the number of participants should exceed the number of predictors by at least 50 (i.e., total number of participants equals the number of predictor variables plus 50). 2. When using six or more predictors, an absolute minimum of 10 participants per predictor variable is appropriate; however, a small effect size would be better detected with approximately 30 participants per variable. For instance, it has been demonstrated that for a single predictor that correlates with the DV at .30 in the population, 124 participants are needed to maintain 80% power

(Cohen & Cohen, 1975). Larger effect sizes are needed if the predictor variable is skewed, if the effect size expected is small, if there is substantial measurement error, or if stepwise regression is being used (Tabachnick & Fidell, 2013). Increased sample size is thus an effective way to boost statistical power and should be employed whenever feasible, but its costs and limited availability of participants may restrict the researcher's ability to use this approach.

3.2.2.1 Enrichment strategy

In order to maximise statistical power with a reduced sample size, an enrichment strategy was applied to select the follow-up sample following the suggestions by McClelland (1997). In order to increase the variability of baseline scores, the sample was not selected at random but chosen from the more extreme ends of the independent variables, i.e. based on childhood EXT and INT problem severity. The sample was split into four groups with different levels of INT and EXT problems at baseline, namely: (1) High levels of both EXT and INT childhood problems; (2) High levels of EXT problems with normal levels of INT problems; (3) High levels of INT problems with normal levels of EXT problems; and (4) Normal levels of both EXT and INT problems.

In order to create the four groups, the following procedural steps were undertaken:

3.2.2.1.1 Creation of subgroups

Prior to any analyses, any cases with whole subscales missing, or with more than 5% of data missing, were removed from the dataset. 413 cases were removed, resulting in a dataset of N=3,786 cases. All further analyses were carried out with this dataset.

The second step was to factor analyse the baseline dataset in order to create overall factor scores for EXT and INT childhood problems, and to determine cut-off scores with which the sample could be classified as "high" vs "normal" levels of EXT and INT problems. The most parsimonious factor solution was sought with high loadings on one factor only and no subscales loading on more than one factor. A Principal Component Analysis (PCA) was

carried out with a Varimax rotation on all included subscales of childhood assessment measures, i.e. shyness (EAS), emotionality (EAS), overactivity (WWP overall), overactivity (BCL), poor emotional adjustment (BCL), and poor social adjustment (BCL). An examination of the Kaiser-Meyer Olkin measure of sampling adequacy suggested that the sample was factorable (KMO=.740). The analysis yielded a 2 factor solution; however, EAS Emotionality loaded equally on both factors, so this subscale was removed, and another PCA was carried out. The results of an orthogonal rotation of the solution are shown in Table 6.

Table 6: Obliquely rotated component loadings for baseline subscales

	Component	
	EXT	INT
Hyperactivity (WWP)	.863	
Overactivity (BCL)	.831	
Conduct Problems (BCL)	.725	
Shyness (EAS)		.856
Emotional Problems (BCL)		.710
Eigenvalues	2.20	1.31
Percentage of total variance	44.021	26.164

Extraction Method: Principal Component Analysis. Rotation Method: Varimax with Kaiser Normalization. WWP= Werry Weiss Peter Activity Rating Scale; BCL=Behaviour Checklist; EAS= Emotionality, Activity, Sociability

Three subscales loaded onto Factor 1: (1) hyperactivity (WWP), (2) overactivity (BCL), and (3) conduct problems (BCL). Inspection of subscale content and individual items of subscales loading on Factor 1 showed that this Factor was clearly related to "externalising" childhood problems. Therefore, this factor was used to determine participants' baseline levels of externalising problems.

Two subscales loaded onto Factor 2: (1) shyness (EAS), and (2) emotional problems (BCL). Inspection of subscale content and individual items of subscales loading onto Factor 2 showed that this factor was clearly related

to "internalising" childhood problems. Whilst "emotionality" on the face of it does not automatically classify as "internalising", closer inspection of the subscale's individual items showed that they were concerned with "internalising" aspects of emotionality, i.e. clinginess, worries and fearfulness ("very clinging, can't be left with others", "has many different worries, broods over things", "very fearful, has lots of different fears"). Therefore, this factor was used to determine participants' baseline levels of internalising problems.

In order to determine children's severity of internalising and externalising problems, factor scores were created for the two factors using a least squares regression approach. The sample was then ranked according to individuals' factor scores on each factor. The top 30% on each factor were classified as showing "high" levels of externalising and internalising problems respectively. The remaining 70% were classified as showing "normal" levels of EXT/INT problems. Four groups were created accordingly (see Table 7): (1) High levels of EXT and INT problems; (2) High levels of EXT problems, normal levels of INT problems; (3) Normal levels of EXT problems and high levels of INT problems; and (4) Normal levels of both EXT and INT problems.

Table 7: "High" vs "no" EXT and INT childhood problems

		INT problems		
		High	No	*Total*
EXT problems	High	369	*767*	*1136*
	No	*767*	1883	*2650*
Total		*1136*	*2650*	*3786*

EXT – Externalising; INT – Internalising

Using the most conservative approach by Harris (1985) to determine the target sample size, including four childhood predictor variables required a minimum total number of 120 participants (N=30 for each predictor). However, because all of the predictor variables were skewed, some of the effects were expected to be small, and the sample was biased, with

observable effects likely to be an underestimation of real word effects, the target sample size was set higher, with n=50 participants for each group. Thus, the aim was to recruit a total sample of n=200 participants; if any of the groups were smaller, these cases would have to be filled through additional cases in the other groups. In order to reach the target sample size for each cell, groups of n=50 participants were selected randomly for each of the four groups of suitable families and invited to participate using the procedure described above. If after any given round of recruitment the target number for any cell was not reached, an additional sample of n=50 was randomly selected from suitable participants and invited to participate.

3.2.3 Exploiting an already existing dataset

The third key issue encountered when carrying out this research was that large parts of the data, namely all baseline data, were derived from an already existing dataset. Exploiting existing datasets has advantages, but it is also associated with challenges. As summarised by Yorke (2011), the main advantages are (1) the data was already collected, implying both financial and time benefits; and (2) analyses can focus on points that were not addressed by those who analysed the primary data, enabling a more complete analysis of the dataset. The main disadvantage, however, is that available data may not capture exactly what the researcher would have preferred to collect, inevitably leading to a compromise between the available dataset and the "ideal" dataset. These issues summarise the main challenges encountered in this study with regards to exploiting an existing dataset: Whilst this research would not have been possible as part of this PhD without the already existing database, this also caused some limitations. Specifically, the following implications arose from using data from an already existing dataset:

(1) The choice of childhood predictors was limited to those assessments that had been carried out at baseline. Whilst a wide ranging assessment was carried out at baseline for the three cohorts, only three scales were collected for all three cohorts, thus limiting available baseline predictors to these three scales. In addition, we had no influence on the choice of instruments that were used to assess these problems. (2) Another issue was related to accuracy of the dataset. The original dataset was already entered

by the research team at baseline and when making random accuracy checks it emerged that a lot of it was entered incorrectly. For the majority of participants, original paper versions of the data were available and could be re-entered if necessary, but these were not available for all participants. This meant that a large proportion of participants had to be excluded from follow-up because accuracy of baseline scores could not be verified. (3) The conditions under which the original data was collected are unknown, including the instructions that were given to participants about how to complete the questionnaires. There was some evidence that this may not have been optimal as some of the questionnaires were completed wrongly. In addition, data was collected by varying health visitors, and data was entered by various people in the team, and there was some evidence that they did not all adhere to the same instructions and/or scoring systems, resulting in a further proportion of cases having to be excluded.

3.2.4 Issues related to follow-up assessments

Several additional methodological challenges were encountered in relation to follow-up assessments, including (1) choice of instruments, (2) issues related to item overlap across measures, and (3) shared method variance

3.2.4.1 Choice of assessment instrument

One additional issue that arose when designing the study was the selection of follow-up measures, in particular the choice of assessment instrument for PD. PD has historically been notoriously difficult to diagnose, and there is no consensus amongst experts as to what is the best method. Most experts agree that the most widely used classification and diagnostic system, namely the diagnostic system by the DSM, is flawed and in need of revision (Blashfield & Reynolds, 2012; Tyrer et al., 2011; Widiger, 2012). The DSM conceptualises PDs using a polythetic-categorical approach, whereby a specified number of criteria have to be met in order to make a diagnosis. Alternative models of personality pathology have been discussed, including dimensional and hybrid models (Krueger, 2002b; McGlashan et al., 2005; Simonsen, 2010; Widiger & Clark, 2000; Widiger & Costa, 2002; Widiger & Simonsen, 2005), but there is no consensus as to what model would be most suitable. Dimensional models make classifications by locating

individuals among graded dimensions. In addition, the usefulness of variable-centred versus person-centred approaches has been debated. Variable centred approaches focus on personality traits, on the relationship between these traits in populations, and on understanding how dimensions of personality variation are organized empirically. Person-centred approaches, on the other hand, focus on differences between individuals when examining relationships between variables.

The controversy over what constitutes a personality disorder, and how it is best assessed, is reflected in the range of available PD assessment instruments. Most diagnostic tools assess slightly different aspects of personality pathology, in accordance with the different conceptualisations of PD. In addition, tools vary according to whether they conceptualise PD categorically, dimensionally, or whether they use a variable-centred or person-centred approach. As such, any researcher carrying out studies in the field of PD is faced with the challenge of choosing the appropriate assessment instrument.

Due to time/financial constraints, carrying out clinical interviews for all participants was not feasible, and a psychometric test had to be chosen. For the purpose of this research the following criteria were set out to choose an assessment instrument: 1. The measure should be as short as possible – most PD questionnaires are rather lengthy with completion times of 45 minutes and more. Because the assessment battery of these participants was already quite extensive, priority was given to shorter scales. 2. Reliability and validity; 3. Sensitivity and specificity; and 4. Normed for the appropriate age groups. Most importantly, however, the measure should closely align with the conceptualisation of PD in the DSM. This criterion was chosen as the most important one because it was deemed most relevant in terms of clinical and theoretical utility. Despite criticism and controversies, the DSM is the most widely used classification system, and the concepts introduced in the DSM are therefore most meaningful and of most practical relevance to professionals.

At the time when the choice of measures for this study was made (approx. January 2012), the DSM-5 task force had developed a new model for conceptualising and assessing PD. The model was a dimensional model

with five domains (Negative Affectivity, Detachment, Antagonism, Disinhibition and Psychoticism) that closely aligned with the dimensions of the FFM. Only a subset of the 10 DSM-IV PDs were suggested to be retained in DSM-5, namely Borderline PD, Narcissistic PD, Schizotypal PD, Avoidant PD and Obsessive-Compulsive PD, as a set of PD types. Antisocial PD was suggested to be combined with psychopathy to create an Antisocial PD/psychopathy type. In line with this new model, an assessment instrument operationalizing the new model was created - The Personality Inventory for DSM-5 (PID-5) (Krueger et al., 2012). The PID-5 fulfilled all other criteria specified above and, in addition, it appeared to be the assessment method of choice for DSM-5 and was therefore chosen as the main outcome assessment instrument for PD used in this research. However, due to heavy criticism of this new model, the plans for revising the classification system of PD were aborted just before publication of the DSM-5, and the DSM-IV conceptualisation of PD was transferred verbatim to DSM-5 whilst the revisions were moved to the Appendix of the DSM, to be further researched.

As such, the criteria of alignment with the DSM were not met. However, the PID-5 remains a widely used instrument that shows good reliability and validity. In addition, it can be used to assess six of the ten DSM PD categories and has been found to align closely with assessments made using DSM-IV classification systems.

3.2.4.2 Item overlap between follow-up measures

An additional issue in relation to follow-up measures was the problem of potential item overlap which is always a possible issue when conducting research based entirely on psychometric scales. Item overlap can occur in several ways (Burns, 2000). Firstly, items can be identical in different scales despite assessing different contents, and as such weaken the discriminant validity of the two scales because they share identical items. Secondly, item overlap can occur when an item on one rating scale represents several more specific items on a second scale. This type of item overlap also weakens the discriminant validity of the two scales because the item on one scale is a general example of the items on the other scale. Thirdly, item overlap may occur if the wording of an item on one rating scale is ambiguous enough to

allow the item to be similar to items from different constructs on a second rating scale. An additional type of overlap in this study concerned the overlap not just of scale items, but also of scale contents and concepts of psychopathologies assessed. For instance, both Borderline PD and Antisocial PD overlap substantially with ADHD: a core feature of both these PDs and ADHD is impulsivity (APA, 2013), so overlap between items on these scales is expected because they all assess aspects of impulsivity.

A test suggested to assess whether item overlap exists between scales has been proposed by Burns (2000). This methods proposes that if the items of one scale, e.g. Borderline PD, are distinct from the symptoms of another scale, e.g. ADHD, then each Borderline PD item should have a stronger corrected item-total correlation with its own dimension than with the ADHD dimension. For example, if a Borderline PD item had a corrected item-total correlation of .50 with the Borderline PD scale, and a correlation of .50 with the ADHD scale, then this item would have no discriminant validity (Burns, Keortge, Formea, & Sternberger, 1996; Burns, Walsh, Owen, & Snell, 1997). Thus, if for example the PD items failed this test, this would imply issues with discriminant validity and would suggest issues with item-overlap. However, in the current study, item overlap was expected for some associations (e.g. CD and Antisocial PD) because these concepts do have significant overlap both conceptually as well as in clinical symptomatology.

3.2.4.3 Choice of Raters – Shared Method Variance

An additional issue encountered when conducting this research was to decide who should provide the assessments at follow-up, and the related problem of shared method variance. Shared method variance is a potential threat to a study's validity because it is variance that is attributable to the measurement method rather than to the constructs the measures represent (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003) and, as such, could present an alternative explanation for an observed association. Based on the results of a meta-analysis, it has been estimated that approximately one quarter (26.3%) of the variance in a typical research measure may be due to systematic sources of measurement error like common method biases (Cote & Buckley, 1987), even though estimates vary considerably across fields and contexts.

Some of these common method biases may result from shared rater variance, i.e. from one respondent providing ratings of predictor and criterion (outcome) variables, and therefore producing an artifactual covariance. Several reasons for this covariance have been listed (Podsakoff et al., 2003); for instance (1) The "consistency motif", which is the tendency of respondents to try to maintain consistency in their responses to similar questions or to organise information in consistent ways in order to appear consistent and rational in their responses (Johns, 1994; Podsakoff & Organ, 1986; Schmitt, 1994). This tendency may produce relationships that would not necessarily exist at the same level in real-life settings (Podsakoff et al., 2003). (2) Social desirability, i.e. the tendency to present oneself in a favourable light, regardless of one's true feelings about an issue or topic. This may become problematic, not only because this may bias the answers of respondents but also because it may mask the true relationships between two or more variables (Ganster, Hennessey, & Luthans, 1983) by producing spurious relationships, serving as a suppressor or moderator variable that influences the nature of the relationships between the variables. (3) Acquiescence, is the tendency to agree with attitude statements regardless of content (Winkler, Kanouse, & Ware, 1982) which may be problematic due to an increase of correlations among items that are worded similarly despite being conceptually unrelated. One obvious way to remedy the issue of shared rater variance is to collect the measures from different sources. Despite the obvious advantages of this approach, it is not feasible to use in all cases and may require considerably more time, effort, and/or cost on the part of the researcher. Another potential remedy is to separate the measurement of the predictor and outcome variables, either temporally through separate assessment sessions, or methodologically by using different response formats, media or locations.

In the current research, main predictor and outcome variables were from different sources; childhood predictor ratings were made by parents, and adolescent/adult PD outcomes were assessed through self-ratings by the young person. Thus, the main longitudinal study assessing childhood predictors of adult PD (Chapter 4) was not affected by shared rater variance. However, the studies exploring whether parenting affected the association between childhood problems and PD (Chapter 5) or whether these

associations could be explained by continuation of childhood symptoms into adolescence/adulthood (Chapter 6) may have been affected by shared rater variance. Assessments of PDs and other psychopathologies, as well as assessments of negative parenting, were all made by the young person at the same time. The methodologically most effective remedy would have been to collect these assessments from other sources; however, due to financial and time constraints this would not have been feasible for the purpose of this PhD research. Furthermore, response rates would likely been lower if an additional person would have had to be recruited to provide these ratings. Similarly, separating the assessments temporally, i.e. collecting data in separate sessions would have increased the risk of participants dropping out. These issues would have posed more serious threats to the validity of the findings, so these possibilities were dropped. The most likely effect of the shared rater variance was expected to be an overestimation of associations of PD with co-occurring psychopathologies as well as with negative parenting variables which needs to be borne in mind for the interpretation of the findings in Chapters 5 and 6.

3.3 Chapter Summary

Longitudinal research has many advantages, but it is also subject to methodological challenges. This chapter outlined the main methodological issues encountered in the conductance of this research. Four main challenges were discussed: (1) participant attrition due to losing contact with participants or participants' refusals to take part. Several strategies were used to increase the number of correctly traced families, as well as to increase the number of responses received (sending reminders, several amendments to letters posted out, offering a variety of response methods, offering a variety of methods to complete assessments, increasing financial rewards for taking part). Two statistical techniques to deal with high attrition rates were discussed, namely weighting of cases and random multiple imputation. (2) Sample selection, including the decision about whether to follow-up the entire sample or whether to select a subsample. Due to financial and time constraints, a subgroup was chosen for follow-up based on sample characteristics, using an enrichment strategy to ensure sufficient power. (3) Issues related to exploiting an existing dataset for

longitudinal research were discussed, and (4) issues related to follow-up assessments were presented, including issues regarding the choice of available instruments, choosing who would provide ratings and the related issue of shared method variance. All these challenges, as well as strategies to overcome these, were discussed in depth.

Chapter 4: Childhood externalising and internalising problems as predictors of early adult personality pathology

Although there is a consensus that personality disorders (PDs) have their origins in childhood (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Bromley, et al., 2006; Johnson, First, et al., 2005; Kernberg et al., 2000; Mervielde et al., 2005; Shiner, 2007; Westen & Chang, 2000), relatively little is known about the developmental pathways leading to PD, and few prospective longitudinal studies have focused on the development of PD as a result of childhood problems (Shiner, 2009; Widiger & Trull, 2007). This chapter presents a prospective longitudinal study investigating externalising and internalising problems in early childhood as predictors of personality pathology in adulthood.

4.1 Introduction

PDs are characterised by pervasive and stable patterns of inner experience and behaviour that deviate markedly from the expectation of the individual's culture, and are associated with impairment, emotional distress and health care burden (APA, 2013). Approximately 10-15% of the adult population are affected by a PD (APA, 2000; Grant et al., 2008; Johnson, Smailes, et al., 2000; Mattia & Zimmerman, 2001) and PD is associated with significant financial costs to the healthcare system, social services and wider society (Rendu, Moran, Patel, Knapp, & Mann, 2002; Smith, Shah, Wright, & Lewis, 1995; Soeteman et al., 2008). The DSM conceptualises PD using a polythetic-categorical approach, whereby a specified number of criteria have to be met in order to make a diagnosis. A total of ten different PDs are listed, classified into three separate clusters. Cluster A PDs are described as "odd or eccentric PDs" and include Paranoid PD, Schizoid PD and Schizotypal PD. Cluster B PDs are described as "dramatic, emotional, or erratic PDs" and include Antisocial PD, Borderline PD, Histrionic PD and Narcissistic PD. Cluster C PDs are described as "anxious or fearful PDs" and include Avoidant PD, Dependent PD and Obsessive-Compulsive PD. Cluster

A and C PDs are generally associated with negative emotionality, anxiety or distress, i.e. with internalising symptomatology; Cluster B, on the other hand, includes problems with poor inhibitory control, inabilities to delay gratification, and impulsive/reckless behaviours linked to chaotic relationships and/or poor interpersonal functioning, i.e. symptomatology on the externalising dimension (Beauchaine et al., 2009).

The DSM categorical system has been widely criticised for a number of reasons (Blashfield & Reynolds, 2012; Tyrer et al., 2011; Widiger, 2012), and alternative models of personality pathology have been discussed. These are mostly dimensional models (Krueger, 2002b; McGlashan et al., 2005; Simonsen, 2010; Widiger & Clark, 2000; Widiger & Costa, 2002; Widiger & Simonsen, 2005) which conceptualise personality pathology as extreme and/or maladaptive variants on a continuum with normal personality traits. This was reflected in alterations that had been suggested for DSM-5: to apply a dimensional model with five domains (Negative Affectivity, Detachment, Antagonism, Disinhibition and Psychoticism). Only six of the ten DSM-IV PDs were suggested to be retained in DSM-5, namely Borderline PD (BPD), Narcissistic PD (NPD), Schizotypal PD (SPD), Avoidant PD (AVPD) and Obsessive-Compulsive PD (OCPD). Due to criticism and disagreements amongst PD experts (Livesley, 2010), the plans for revising the classification system of PD were aborted, and the DSM-IV conceptualisation of PD was transferred more or less verbatim to DSM-5.

4.1.1 Longitudinal studies about the developmental pathways to PD

Although rarely diagnosed prior to adulthood (Allertz & van Voorst, 2007; Chanen & McCutcheon, 2008), there is a consensus that PDs have their origins in childhood (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Bromley, et al., 2006; Johnson, First, et al., 2005; Kernberg et al., 2000; Mervielde et al., 2005; Shiner, 2007; Westen & Chang, 2000). However, relatively little is known about the developmental pathways leading to PD. Instrumental to understanding the developmental pathways to any disorder are prospective longitudinal studies, but few have focused on the development of PD as a result of common childhood problems (Shiner, 2009; Widiger & Trull, 2007). Because the DSM-IV classification of disorders in childhood and adolescence was restricted to Axis I

psychopathology, most of the childhood predictors for PD are common Axis I disorders such as externalising disorders, e.g. conduct disorder (CD), oppositional defiant disorder (ODD) (Dowson et al., 2001), and attention-deficit disorders (ADHD) (Young et al., 2003) and internalising disorders, e.g. mood disorders (Kasen et al., 2001), anxiety disorders (Bienvenu & Stein, 2003). Research indicates moderate to strong continuities in EXT and INT behaviours from early to middle childhood through adolescence and into adulthood (Ferdinand & Verhulst, 1995; Fergusson, 1998). These continuities have been found to be both homotypic (i.e. within the same 'class' of disorder) and heterotypic (i.e. in a different 'class' of disorder). For example, childhood ADHD symptoms have been found to predict later externalising disorders such as adult ADHD (Biederman et al., 2006; Mannuzza et al., 1993, 1998), CD (Loeber, Farringdon, Stouthamer-Loeber, & Van Kammen, 1998; Mannuzza, Klein, Abikoff, & Moulton, 2004; Moffit, Caspi, Dickson, Silva, & Stanton, 1996), ODD (Harvey, Youngwirth, Thakar, & Errazuriz, 2009; Pardini & Fite, 2010) and substance use disorders (Wilens & Morrison, 2011). Similarly, conduct problems have been found to show long-term continuity. In fact, ODD, CD and ASPD are often viewed hierarchically, reflecting age-dependent expressions of the same underlying disorder (see (Moffit et al., 2008)), where ODD is conceptualised as a developmental precursor to CD, and CD is conceptualised as a developmental precursor to ASPD (Lahey, Loeber, Quay, Frick, & Grimm, 1997; Loeber et al., 2002; Loeber, Green, Keenan, & Lahey, 1995; Robins, 1966, 1978). In addition, for both ADHD and CD, heterotypic continuity has also been demonstrated, with longitudinal studies consistently reporting associations between childhood ADHD and adult internalising disorders, such as anxiety and depressive disorders (Biederman et al., 2006; Pardini, Stepp, Hipwell, Stouthamer-Loeber, & Loeber, 2012; Rasmussen & Gillberg, 2000) and childhood CD and internalising disorder such as mood disorders (Loeber et al., 2002).

In the area of PD, prospective longitudinal studies have focused mostly on ASPD and BPD. Regarding externalising childhood problems, for instance, Burke & Stepp (2012) showed that ODD and ADHD predicted BPD in males. Similarly, Stepp et al. (2012) found that higher levels of both ADHD and ODD scores at age 8 uniquely predicted BPD symptoms in adolescent girls. In a large follow-up study of hyperactive boys, ADHD was highly

significantly related to ASPD in young, middle, and later adulthood (Klein et al., 2012; Mannuzza et al., 1993, 1998). Particularly strong links have been found between CD and ASPD which is perhaps not surprising considering that childhood CD is regarded as the 'childhood version' of ASPD, and onset before age 15 is a diagnostic criterion for adult ASPD in the DSM (APA, 2000). For instance, Copeland et al. (2009) showed that childhood CD significantly increased the risk for adult ASPD. Another study found robust linear associations between child CD symptoms and ASPD, but no associations for ADHD without CD (Lahey et al., 2005). However, Sourander et al. (2007) found that both children with conduct problems without hyperactivity, and children with hyperactivity problems (and no conduct problems) in childhood had an increased risk of developing ASPD. In sum, in the area of ASPD, the links with specific childhood EXT problems still need to be clarified, and in the area of BPD, it is yet unclear, whether any specific links between childhood EXT problems and BPD exist.

Regarding internalising childhood problems, in the area of ASPD mixed results have been found. For example, both Lahey et al. (2005) and Ramklint et al. (2003) found no significant relationship between childhood depressive symptoms and ASPD in adulthood. Similarly, neither Copeland et al. (2009) nor Diamantopoulou et al. (2010) found significant associations between childhood depression or anxiety and ASPD in young adults. Some evidence suggests that it might be INT problems in combination with EXT problems in childhood, rather than INT problems alone, that increase an individual's risk to develop ASPD. For instance, Sourander et al. (2007) compared groups of young adult males based on their assessment of childhood problems at age 8. They found that the group of children who had only INT problems were not at an increased risk to develop ASPD. However, those children who had a combination of high INT and EXT problems had the highest risk for developing ASPD in adulthood (OR=5.4), even more so than individuals who had high CD (OR=3.5) or high hyperactivity problems (OR=2.7). Ramklint et al. (2003) showed that depression in children and adolescents (mean age 14.1 years) in psychiatric care predicted BPD at mean age 30.5, also after adjusting for sex, age, and other childhood disorders. The results of a prospective longitudinal study showed that INT problems, assessed at age 5, were associated with higher BPD features at age 12 (Belsky et al., 2012).

However, in a study with males only, Burke & Stepp (2012) found no associations between childhood depression and anxiety symptoms and BPD in adulthood.

4.1.1.1 Methodological flaws of existing longitudinal studies

Existing longitudinal studies about childhood predictors of PD have several shortcomings. Firstly, studies often included a wide age range at baseline, not only including children but also early and late adolescents (see Chapter 2). Not only do symptoms of childhood disorders investigated at baseline (e.g. ADHD) vary and change with age, therefore confounding the interpretability of the results, but also the symptoms of the follow-up disorder (i.e. PD) will be affected by age. That is, some studies included participants in their sample who were already adolescents at baseline, an age at which PD symptoms will already have developed (Bernstein et al., 1996; Johnson et al., 1999), therefore making it difficult to infer whether the childhood disorder really can be seen as a predictor of PD.

Secondly, most studies investigating the effects of specific childhood disorders on PD fail to assess or account for the effects of other, comorbid, childhood disorders. As mentioned above, some evidence suggests, for instance, that childhood ADHD is independently predictive of adult ASPD (Gittelman et al., 1985; Mannuzza et al., 1993). However, it has also been argued that any higher occurrence of psychiatric disorders in adulthood among hyperactive children could be a consequence of their coexisting childhood conduct problems rather than, or in addition to, their severity of childhood hyperactivity. For instance, childhood conduct problems have been shown to be predictors of adolescent and adult antisocial behaviour, ASPD, and substance use disorders (Hinshaw & Anderson, 1996; Kratzer & Hodgins, 1997; Lynam, 1998). Early follow-up studies failed to determine the extent to which the psychiatric disorders found at adult follow-up were likely to be a function of severity of comorbid childhood conduct problems rather than of severity of childhood hyperactivity/ADHD. The few studies that have investigated the differential predictive effects of childhood ADHD and CD on ASPD showed that CD predicted ASPD when controlling for ADHD, but ADHD did not predict ASPD when controlling for CD (Copeland et al., 2009; Lahey et al., 2005; Sourander et al., 2005).

Thirdly, any additive or interaction effects of comorbid childhood problems on PD remain largely unexplored. An additive effect refers to the role of a variable in an estimated model: a variable that has an additive effect can merely be added to the other variables in a model to determine their additive effect on the independent variable, whereas a variable that has an interaction effect will have a different effect on the dependent variable, depending on the level of some third variable with which it interacts. Whilst in the area of PD, research about additive or interaction effects of childhood predictors is mostly lacking, in other areas evidence has shown that children with comorbid disorders were at a higher risk of a negative outcome than children with a single disorder (Colder, Mott, & Berman, 2002). For example, children with both ADHD and conduct problems were found to be more poorly adjusted compared to children with either disorder alone. Loeber, Brinthaupt, and Green (1990) found that children with both ADHD type problems and conduct problems were considerably more delinquent in adolescence than children with either type of problem alone. Similarly, Moffitt (1990) showed that boys with both ADD and delinquent behaviours had worse outcomes compared to boys with either disorder alone. Molina, Smith, and Pelham (1999) found that in adolescents with a CD diagnosis only, the risk for substance abuse was increased, whereas the risk in those with ADHD only was not increased. However, the joint presence of ADHD and CD was associated with particularly high rates of substance use, and they reported much higher use of multiple substances than did adolescents with only CD.

Evidence also suggests that often risk factors are not merely additive, but rather that psychopathology is caused by a complex interplay of multiple factors (McBurnett, 1992; Rothbart & Mauro, 1990). One study showed, for example, that positive and negative emotionality interacted to predict inhibition in children, where a combination of low positive and high negative emotionality was associated with highest inhibition levels (Park, Belsky, Putnam, & Crnic, 1997). Colder and Chassin (1997) and Colder and Stice (1998) found that impulsiveness moderated the effects of emotionality in adolescents: high levels of anger were associated with delinquency in impulsive but not unimpulsive adolescents. In the area of PD, one study has investigated the combined effects of CD and emotional problems on ASPD

(Sourander et al., 2005). The results demonstrated that children with both CD and emotional problems were 5.4 times more at risk for ASPD than children with only one of these disorders. The risk was 3.5 for CD, and 0.9 for emotional problems alone, indicating that the effects of combined disorders were much stronger than the effects of each disorder on its own, implying that the effects of comorbid disorders were not simply additive, but rather, the joint effects of comorbid problems were stronger than the effects of single disorders, arguing for interactive effects. Thus, evidence suggests that specific childhood problems increase the risk for PDs, and, in addition, additive and/or interactive effects may further increase the risk for a particularly problematic outcome. Indeed, several authors have even argued that children with co-occurring disorders such as ADHD and CD may represent different subgroups with poorer prognoses than children with either disorder alone (Hinshaw, 1987; Lilienfeld & Waldman, 1990; Lynam, 1996; Moffitt, 1990).

4.1.2 The current study – research aims and hypotheses

Taken together, to date few longitudinal studies have investigated childhood predictors of PD, and those that have are often based on flawed research designs. In addition, few studies have investigated the effects of childhood disorders on adult personality pathology while controlling for the effects of other childhood disorders. The current study aimed to fill this gap in the literature: Applying a prospective longitudinal design, early childhood predictors of PD, in the form of common EXT and INT problems, were investigated. Specifically, it was explored whether patterns of EXT problems (conduct problems and hyperactivity) and INT problems (emotional problems and shyness) in early childhood were predictive of adult personality pathology. The effects of these childhood problems were investigated both individually, as well as in interaction with each other.

The following research questions were addressed:

(1) Do common externalising and internalising childhood problems predict personality pathology in adulthood?

> *It was hypothesised that both externalising and internalising childhood problems would predict early adult personality pathology.*

Both homotypic as well as heterotypic continuities were expected: i.e. it was hypothesised that both externalising childhood problems (conduct problems [CP], hyperactivity [HYP]) and internalising childhood problems (shyness [SHY], emotional problems [EP]) would predict PDs on both the externalising spectrum (Borderline PD [BPD], Antisocial PD [ASPD], Narcissistic PD [NPD]) and on the internalising spectrum (Schizotypal PD [SPD], Avoidant PD [AVPD], Obsessive-Compulsive PD [OCPD]).

(2) Will combinations of childhood problems show additive and/or interactive effects in the prediction of personality pathology in early adulthood?

Both additive and interactive effects were expected for co-occurring childhood problems (hyperactivity, conduct problems, emotional problems, shyness) in the prediction of adult PDs (ASPD, BPD, NPD, OCPD, SPD. Based on previous research, especially strong additive/interactive effects were expected for HYP and CP in the prediction of ASPD and BPD. In addition, internalising problems (EP, SHY) were expected to add to / interact with externalising problems (CP, HYP) in the prediction of ASPD.

(3) Can unique patterns between childhood externalising and internalising problems and PDs be found, i.e. will specific childhood problems remain significantly related to adult PD when the effects of all other childhood problems are controlled for?

It was hypothesised that childhood problems would predict adult PDs when the effects of all other childhood problems were controlled for. Based on previous evidence, it was expected that childhood CP would uniquely predict adult ASPD and that both CP and HYP would predict BPD. In addition, SHY was expected to uniquely predict AVPD.

4.2 Methods

Please see Chapter 3 for a detailed description about the methods applied in this study and the assessment instruments used.

4.2.1 Participants

For details about the procedure for selecting participants for this study, please see Chapter 3. For an overview about sample characteristics, please see Table 7. Using the sample selection methods described in Chapter 3, a total number of N=216 participants took part in this study. However, these participants were not distributed equally across the four groups of high and normal levels of EXT and INT problems (see Table 8). Specifically, group 1 (High EXT, high INT) was underrepresented and group 4 (normal EXT, normal INT) was overrepresented.

Table 8: Sample characteristics – demographics and assessments at baseline (age 3) and follow-up

Total N	**216**
High EXT, High INT	**36 (17%)**
High EXT, Normal INT	**52 (24%)**
Normal EXT, High INT	**53 (25%)**
Normal EXT, Normal INT	**75 (35%)**
Male gender	**85 (39%)**
Mean age at follow-up (SD)	**20.29 (3.09)**
Mean SES score	**-.073**
Childhood emotional/behavioural problems	***Mean (SD)***
Hyperactivity (HYP)	**0.57 (0.39)**
Conduct problems (CP)	**0.49 (0.36)**
Emotional problems (EP)	**0.38 (0.37)**
Shyness (SHY)	**2.84 (0.95)**
Personality pathology – dimensional symptom scores	***Mean (SD)***
Borderline PD (BPD) symptoms	**6.91 (3.70)**
Antisocial PD (ASPD) symptoms	**5.14 (2.86)**
Narcissistic PD (NPD) symptoms	**0.57 (0.50)**
Obsessive-Compulsive PD (OCPD) symptoms	**1.04 (0.34)**
Schizotypal PD (SPD) symptoms	**4.31 (2.84)**
Avoidant PD (AVPD) symptoms	**3.10 (2.06)**

SD – Standard Deviation; SES – socio-economic status ; PD – personality disorder

4.2.2 Analyses

Four hierarchical regressions were carried out to investigate the effects of childhood problems on PD while controlling for the effects of all other childhood predictors. In the first step, covariates (see Table 9) were entered. In the second step, all child predictors that were significantly correlated

with the DVs were added simultaneously. Significance levels were Bonferroni-corrected for multiple comparisons and were set to $p<.01$ (two-tailed).

For any PD that showed more than one significant predictor, possible additive and interaction effects were tested, following the procedure suggested by Holmbeck (1997). Interaction terms were calculated by multiplying the predictor dimensions with each other (e.g. CP x HYP). Following the recommendations of Aiken and West (1991) and Cohen and Cohen (1983), a hierarchical order of entry of the predictor variables was used: in step 1, covariates and one childhood problem were entered (testing main effects); in step 2, an additional childhood problem was entered (testing additive effects); and in step 3, the interaction term of these childhood predictors was entered (testing interactive effects). This order of entry allowed assessment of additive effects and interaction effects over and above the effects of covariates, by considering not only beta values in the regression model, but also significance levels of the R^2 change statistics.

Age at follow-up, sex, and deprivation at baseline were controlled for in all multivariate models due to known effects on PD. Firstly, PD symptoms tend to change with age: Cluster B symptoms have been found to naturally reduce with age, whereas some data suggests that Cluster A and C symptoms may increase with age (Gunderson, 2011; Morse & Lynch, 2004; Zanarini, Frankenberg, Hennen, Reich, & Silk, 2006). Because participants' age range at follow-up was quite wide (17-26) in this study, this was controlled for in multivariate models. Further, evidence suggests gender differences in PD, in terms of prevalence, expression of symptoms, as well as in pathogenesis and comorbidity with other disorders (Grilo et al., 1996; Johnson et al., 2003) so the effects of gender were controlled for. Thirdly, low socioeconomic background has been found to be independently predictive of PD (Cohen, 2008) and to mediate the relationship between other predictors and PD (De Genna & Feske, 2013), so the effects of deprivation at baseline were statistically controlled for.

Table 9 presents an overview of the childhood predictors, covariates, and adult outcomes included in analyses.

Table 9: Predictors, Outcome Variables and Covariates

	Construct	Assessment Tool
Childhood (age 3) Predictors (IVs)	Hyperactivity	WWP Activity Scale
	Conduct Problems	BCL Social Maladjustment
	Emotional Problems	BCL Emotional Maladjustment
	Shyness	EAS Shyness
Outcomes (DVs) – Personality Pathology	Specific PDs: Borderline, Antisocial, Narcissistic, Obsessive-Compulsive, Schizotypal, Avoidant	PID-5
Covariates	Age at follow-up	
	Sex	
	Socio-economic status	Carstairs deprivation score

IV – Independent variable, DV – dependent variable, WWP – Werry-Weiss-Peter-Activity-Rating-Scale, BCL – Behaviour Checklist, EAS – Emotionality, Activity, Shyness Temperament Scale; SES – Socioeconomic Status; PD – personality disorder

4.3 Results

4.3.1 Preliminary analyses

Prior to all analyses, attrition rates and representativeness of the sample were examined. Overall attrition rate was 49%. Contrary to expectations, refusal rates were relatively low, with only 7% refusing to take part. The main issue was non-response (39%). However, because families were contacted by letter, there was no way of knowing whether the addresses that were used were in fact correct. That is, it is not clear whether the letters were sent to the wrong address, or whether families were not

interested in participating. The overall trace rate was 71% for this sample: 723 out of 1,020 cases were classified as "successfully traced" or "probably traced". However, trace rates differed across cohorts, due to the amount of information available. The trace rate was highest for cohort 1 (80%) where most information was available, including full names of both parents and siblings, birth dates and full addresses. The trace rate was worse for cohort 2 (70%) and cohort 3 (59%) where much less information was available. In cohort 3, for 1/3 of the sample, only names and post codes at the time of the baseline assessment were available; however, for 2/3 of the cases the only information available was the full name of the young person, and the name of the main caregiver which had to be deciphered from the signature on the original questionnaires. Not surprisingly, the trace rate was considerably lower in this group. No systematic differences existed between traced and untraced families on any of the baseline measures.

As a first step to deal with attrition, examination of attrition patterns was carried out to establish whether any systematic differences existed between participants and non-participants. The group of "non-participants" included all families that were considered "successfully traced" *and* invited to participate but who did not take part. That is, non-participants included families who refused to participate, those who did not respond to the study invitation, or those who dropped out before completing all relevant outcome measures. One young person whose family was contacted was deceased. Non-participants were compared to participants on all baseline variables of interest in order to determine whether a bias was likely due to systematic differences between those who were selected for participation and did not take part, and those who were selected for participation and agreed to take part. Untraced participants were not considered because it was already established that no differences existed between traced and untraced families. As shown in Table 10, significant differences were found between participants and non-participants on both externalising predictors (hyperactivity and conduct problems), where non-participants scored higher on both measures than participants. In addition, significantly more males dropped out than females. Thus, the sample was likely to be biased: the group of families that consented to take part were more "healthy" at baseline than those who did not take part, and the results were, thus, likely

to be an underestimation of real world effects due to lower variability of scores.

Table 10: Comparisons of predictors and covariates between participants and non-participants

	Participants	Non-Participants	Comparison
N	216	207	
Male gender: N (%)	85 (39%)	120 (58%)	χ^2=27.519; p<.001
Deprivation at baseline: M (SD)	-0.73 (2.088)	-0.64 (2.210)	F(1,776)=0.401, n.s.
	M (SD)	*M (SD)*	
Hyperactivity	0.59 (0.390)	0.80 (0.412)	F(1, 776)=21.671; p<.001
Shyness	2.84 (0.945)	2.80 (0.861)	F(1, 776)=0.595, n.s.
Emotional Problems	0.39 (0.365)	0.44 (0.403)	F(1, 776)=1.655, n.s.
Conduct Problems	0.49 (0.363)	0.64 (0.416)	F(1, 776)=15.284; p<.001

n.s. – not significant

As mentioned in Chapter 3, the most common methods to deal with attrition are weighting of cases and multiple imputations (Mostafa & Wiggins, 2014). Random multiple imputation, (Little & Rubin, 2002; Rubin, 1987) was not possible because the technique depends on the assumption that data is missing at random (MAR) as opposed to data missing not at random (MNAR) (Little & Rubin, 2002), and in the current sample, data was MNAR. Weighting of cases adjusts the distributions of responders so that the relative importance of each participant's characteristic is adjusted. Whilst efforts were made to have equal numbers of n=50 in all four groups of recruited participants (1. high EXT and INT, 2. normal EXT and INT, 3. high EXT, normal INT, 4. normal EXT, high INT), recruited participants did not distribute equally across the groups (see Table 7). Therefore, all analyses were carried out twice: 1. With re-weighted cases such that the weight of all four groups was balanced, so that more weight was given to underrepresented groups and less weight was given to overrepresented groups, and 2. Using the original unweighted data. The pattern of results

were identical in both cases, i.e. even though specific values slightly changed, the overall patterns of significant and non-significant predictors were similar, regardless of whether the analyses were carried out with the original data or with reweighted data. Therefore only results from analyses carried out with original, unweighted data is presented here.

Next, the presence of outliers and influential cases was assessed, using the studentized deleted residual, leverage, DFFits, and DFBetas statistics (Bollen & Jackman, 1990). Studentized residuals were determined by dividing residuals by their estimated standard errors; observations with absolute values >3 in were considered outliers. Leverage measured of how far any observation was from the other observations in terms of the levels of the independent variables. Observations with values larger than 2(k+1)/n were considered to be highly influential, where k was the number of predictors and n was the sample size. DFFits measured how much an observation had affected its fitted value from the regression model. Values larger than 2*sqrt((k+1)/n) in absolute value were considered highly influential. DFBetas measured how much an observation affected the estimate of a regression coefficient. Values larger than 2/sqrt(n) in absolute value were considered highly influential. Two cases were identified as influential outliers. Analyses were conducted, dropping these cases to assess the degree to which the findings were influenced by their presence. No changes occurred in the pattern of significant effects. Because of the consistency in the results, the outliers were retained in all subsequent analyses.

Next, checks on assumptions of multiple regressions were conducted. Z-tests were applied for normality tests using skewness and kurtosis. Z-scores were obtained by dividing the skew and kurtosis values by their standard errors. As suggested by Kim (2013), for this medium-sized sample (50 < n < 300), non-normality was assumed at absolute z-value over 3.29, which corresponds with an alpha level 0.05. Absolute values of kurtosis ranged from 1.103 (emotional problems) to 2.37 (hyperactivity) in predictor variables and from -2.797 (SPD) to 2.118 (ASPD) in outcome variables. None of the variables represented a deviation from a normal distribution. Absolute values of skewness ranged from 4.795 (hyperactivity) to 5.229 (conduct problems) in predictors, and from 3.108 (SPD) to 6.271 (ASPD) in outcome variables. All predictor variables were positively skewed. Whilst it

has traditionally been suggested to perform transformations on skewed data, this method for handling non-normality of data has also been criticised (e.g. Osborne, 2002). Further, as pointed out by Hayes (2013), simulation research shows that only the most severe violations of the normality assumption affect the validity of statistical interferences from a regression analysis unless the sample size is quite small (e.g. Duncan & Layard, 1973; Edgell & Noon, 1984; Havlicek & Peterson, 1997; Hayes, 1996). Therefore, all further analyses were performed using the original, un-transformed, dataset.

Multicollinearity among the predictors was assessed using the variance inflation factor (VIF) statistic. In this sample, the VIFs ranged from 1.25 to 1.73, all within acceptable ranges.

Table 11: Intercorrelations between study variables

		SHY	*EP*	*HYP*	*CP*	*BPD*	*ASPD*	*SPD*	*AVPD*	*OCPD*
Baseline	*Emotional problems*	.273***								
	Hyperactivity	-.210***	.235***							
	Conduct Problems	.022	.386***	.519***						
Follow-up	*Borderline PD*	-.030	.148*	.255***	.286***					
	Antisocial PD	-.005	.181**	.190**	.284***	.752***				
	Schizotypal PD	.098	.108	.166*	.182**	.739***	.657***			
	Avoidant PD	.055	.159*	.197**	.178**	.742***	.465***	.795***		
	Obsessive-Compulsive PD	-.013	.029	.065	.087	.363***	.457***	.643***	.608***	
	Narcissistic PD	-.047	.012	.049	.115	.534***	.753***	.453***	.209**	.261***

*Note: N=216; *p<.05; **p<.01; ***p<.001; SHY – shyness; EP – emotional problems; HYP – hyperactivity; CP – conduct problems; (B, AS, S, AV, OC)PD – (Borderline, Antisocial, Schizotypal, Avoidant and Obsessive-Compulsive) PD*

Emotional problems correlated with BPD, ASPD and AVPD, and both hyperactivity and conduct problems correlated with BPD, ASPD, SPD and AVPD. Shyness did not correlate with any of the PDs, and none of the childhood problems correlated with OCPD or NPD, so shyness, OCPD and NPD were not considered in further analyses.

4.3.2 Main, additive and interactive effects

Table 12 shows multiple regression results for childhood externalising and internalising problems on adult PDs. The following significant predictors were found: both conduct problems and hyperactivity were significantly predictive of BPD, whereas conduct problems was predictive of ASPD, and hyperactivity was predictive of AVPD. No significant predictors for SPD were found.

Table 12: Hierarchical Linear Regression Analyses of the Effects of Child EXT and INT problems as Predictors of Personality Disorders

	Borderline PD		*Antisocial PD*		*Avoidant PD*		*Schizotypal PD*	
	ΔR²	*β*	*ΔR²*	*β*	*ΔR²*	*β*	*ΔR²*	*β*
Step 1	.032		.053*		.036		.028	
Gender		.098		-.161*		.091		-.062
SES		-.062		-.058		-.129		-.096
Age at follow-up		-.167*		-.149		-.163*		-.138
Step 2	.252***		.165***		.158***		.137***	
Hyperactivity		.278**		.181*		.262**		.232*
Conduct Problems		.290***		.239**		.161		.190*
Emotional Problems		.092		.131		.122		.066

** p<.05; ** p<.01; *** p<.001 ΔR² - R² change; results presented in bold are significant after correcting for multiple testing (p<.01)*

Table 13 shows multiple regression results for additive and interactive effects of childhood conduct problems and hyperactivity on BPD. Because none of the other PDs had more than one significant predictor, BPD was the only PD which was tested for additive and interactive effects. The effects of

conduct problems and hyperactivity were additive in the prediction of BPD; their interaction was not significant.

Table 13: Hierarchical linear regression analyses of the additive and interactive effects of childhood externalising problems as predictors of BPD

	Borderline PD	
	ΔR²	*β*
Step 1	.229***	
Gender		.151
SES		-.125
Age at follow-up		-.281***
Conduct Problems		.460***
Step 2	.047**	
Conduct Problems		.315***
Hyperactivity		.283**
Step 3	.002	
Conduct Problems		.401**
Hyperactivity		.351**
Conduct Problems x Hyperactivity		-.142

*** p<.01; *** p<.001 ΔR² - R² change; PD – personality disorder; SES – socio-economic status*

4.4 Discussion

The current study applied a prospective longitudinal design to investigate early childhood predictors of PD. Specifically, it was explored whether patterns of common externalising problems (hyperactivity and conduct problems) and internalising problems (emotional problems and shyness) in early childhood were predictive of adult personality pathology. The findings showed that common childhood problems such as hyperactivity and conduct problems do indeed predict PD in early adulthood. Specifically, several unique relationships between childhood problems and PDs were detected: conduct problems and hyperactivity predicted BPD, conduct

problems predicted ASPD, and hyperactivity predicted AVPD. However, only one additive effect was found – the effects of hyperactivity and conduct problems were additive in the prediction of BPD – and no interactive effects were found in the prediction of any PD.

Finding such consistent and robust relationships is striking considering that these children were only three years of age at the time of their baseline assessments, and considering how much time had passed before they were followed up; for the oldest participants, this was a time span of over 20 years. In addition, obtaining these findings is remarkable considering the methodological challenges encountered throughout this research. For example, one of the two strongest predictors (conduct problems) was assessed using a scale which consisted of only 5 items, and yet it emerged as one of the most useful indicators for later psychopathology. Moreover, the sample was subject to high attrition and significantly biased – two of the main predictor scales were significantly higher in non-responders than responders. Obtaining such robust and consistent findings *despite* all these challenges is remarkable. The present findings suggest that PD can be predicted as early as preschool age and highlight the importance of longitudinal studies in adult psychopathology (in this case, PD). Such early identification could aid the development of early intervention. Longitudinal research is especially needed in the area of PD, a field where research into childhood predictors is mostly lacking (see Chapter 2).

The results of this research were in line with the consensus that PDs can be predicted on the basis of common childhood problems (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Bromley, et al., 2006; Johnson, First, et al., 2005; Kernberg et al., 2000; Mervielde et al., 2005; Shiner, 2007; Westen & Chang, 2000). Specifically, strong associations between EXT childhood problems and PD where found: conduct problems predicted ASPD, hyperactivity predicted AVPD and both hyperactivity and conduct problems predicted BPD. Previous research has demonstrated the effects of EXT problems on PD (Burke, 2012; Copeland et al., 2009; Klein et al., 2012; Mannuzza et al., 1993, 1998; Stepp, Burke, et al., 2012). However, one major methodological flaw of these studies was that when the effects of one disorder on a PD were tested, most studies did not control for the effects of co-occurring disorders in childhood. This is surprising, given that

especially EXT problems such as ADHD, ODD and CD very often co-occur. We controlled for the effects of other co-occurring child problems in our analyses, so the results were not due to overlap with other disorders. As expected, both homotypic and heterotypic continuities were found. Externalising childhood problems (conduct problems and hyperactivity) predicted PDs on the EXT spectrum (BPD and ASPD) as well as on the INT spectrum (AVPD). This is in line with previous research showing both homotypic (Biederman et al., 2006; Loeber et al., 1995; Mannuzza et al., 2004; Mannuzza et al., 1993, 1998; Moffit et al., 2008; Moffit et al., 1996) and heterotypic (Barkley, Fischer, Smallish, & Fletcher, 2004; Biederman et al., 2006; Loeber et al., 2002; Pardini & Fite, 2010; Rasmussen & Gillberg, 2000) continuities of childhood disorders into adulthood. The current results extend previous research by demonstrating these continuities in the area of personality pathology.

In our study, childhood INT problems were not predictive of adult PD: neither shyness nor emotional problems were found to be associated with any of the PDs assessed in this study. Shyness did not predict any of the PDs assessed, and, whilst initially effects of emotional problems on PD were detected, these effects disappeared when other childhood problems were controlled for, suggesting that the initially significant associations were due to overlap with other childhood problems. Previous research about the effects of childhood INT problems on PD has been mixed, with some studies showing links between INT disorders and PDs, and some studies finding no such associations (Belsky et al., 2012; Copeland et al., 2009; Diamantopoulou et al., 2010; Lahey et al., 2005; Ramklint et al., 2003). Some previous evidence also suggested that it might be INT problems in combination with EXT problems, rather than INT problems alone, that increase an individual's risk for PD (Sourander et al., 2007). Our findings were not in support of this argument – in our study, INT problems did not by themselves or in combination with EXT problems predict PD. However, this may have been related to the scales used to assess INT problems in this study. Whilst both scales used (shyness and emotional problems) were clearly on the internalising spectrum, they may not have been the best assessments of childhood INT problems. The most common and reliably assessed INT problems in childhood are depressive or anxiety symptoms,

and most previous research in the area that found significant associations between INT problems and PD assessed anxiety or depressive symptoms in children. However, because the first part of this longitudinal study (i.e. the baseline assessments) had already been completed, the choice of predictors to be included in this research was limited to those assessments that had been carried out previously. Thus, associations between childhood INT problems and PDs still need to be clarified, using more appropriate scales to assess INT problems in childhood.

When exploring whether these effects were additive or interactive in the prediction of PD, unexpectedly, we found only one additive effect, namely for conduct problems and hyperactivity in the prediction of BPD, and no interactive effects at all. These results were surprising given that research has more or less consistently shown that children with co-occurring disorders are at a higher risk of a negative outcome than children with a single disorder (Loeber et al., 1990; McBurnett, 1992; Moffitt, 1990; Molina et al., 1999; Park et al., 1997; Rothbart & Mauro, 1990; Sourander et al., 2005). It has been suggested that ADHD so commonly co-occurs with CD that the effects of ADHD may not add to the effects of CD in the prediction of a disorder (Lahey, Loeber, Burke, & Rathouz, 2002; Lynam, 1998; Moffit et al., 2008). However, this was unlikely to be the case in the current sample because we did find one additive effect. It does seem possible, however, that some effects did not emerge in this study because of a bias in the sample due to high attrition. Specifically, those who responded to the invitations and took part in the study were much "healthier" in terms of baseline variables (hyperactivity and conduct problems) than those who did not respond. This may have diminished the strength of associations between baseline and follow-up variables due to a decrease in power related to diminished variability in scores. Thus, it is possible that only the strongest associations between baseline and follow-up variables were detected in this research. In support of this is the fact that several almost-significant associations emerged: for instance, there was a marginally significant effect of hyperactivity on ASPD in the presence of conduct problems, but it did not reach significant levels. In addition, the effects of conduct problems and hyperactivity were almost significant in the presence of SPD. Future research needs to further clarify the association between

specific childhood problems and their additive or interactive effects on adult PDs.

Instead of additive and interactive effects, this study detected several unique risk patterns between childhood problems and specific PDs. Specifically, conduct problems predicted ASPD, hyperactivity predicted AVPD, and both conduct problems and hyperactivity predicted BPD. These findings will be discussed in detail below.

4.4.1 Conduct problems predict ASPD

This study confirmed previous findings of a robust association between conduct problems in childhood and subsequent ASPD in adulthood (Copeland et al., 2009; Lahey et al., 2005; Robins, 1966, 1978). This finding was not surprising, given that conduct disorder is usually regarded as the childhood version of ASPD, and onset before age 15 is a diagnostic criterion for adult ASPD in DSM (APA, 2013). In fact, conduct problems such as CD or ODD are often viewed hierarchically with ASPD, reflecting age-dependent expressions of the same underlying disorder (Moffit et al., 2008), where ODD is conceptualised as a developmental precursor to CD, and CD is conceptualised as a developmental precursor to ASPD (Lahey et al., 1997; Loeber et al., 2002; Loeber et al., 1995; Robins, 1966, 1978). Our results add further support to this view by showing that conduct problems are strongly predictive of adult ASPD even when assessed as early as age 3. In addition, our findings showed that the effects of conduct problems on ASPD were not due to overlap with other co-occurring childhood problems such as hyperactivity, as these were statistically controlled for.

Our findings also showed that hyperactivity was not a significant predictor of ASPD. Previous research about ADHD as a predictor of ASPD has been mixed. Some have argued that childhood ADHD predicts ASPD independent of CD (Gittelman et al., 1985; Mannuzza et al., 1993), and some research has indeed found highly significant links between ADHD and ASPD in young, middle, and later adulthood (Klein et al., 2012; Mannuzza et al., 1993, 1998). The results from our meta-analysis (Chapter 2) appeared to support this, by showing that CD, ODD and ADHD were all independently associated with ASPD, yielding similar levels of risk. However, the findings of our meta-

analysis also showed that all three studies that investigated the differential predictive effects of childhood ADHD and CD on ASPD showed that CD predicted ASPD when controlling for ADHD, but ADHD did not predict ASPD when controlling for CD. The results of the current longitudinal study confirmed this by indicating that conduct problems, but not hyperactivity, were predictive of ASPD when controlling for the respective other childhood problem. In addition, our findings were not in line with a large body of evidence showing that the negative effects of CD on outcomes are increased in the case of co-occurring ADHD, for instance in terms of antisocial behaviour (Loeber et al., 1990; Moffitt, 1990) or substance abuse (Molina et al., 1999): in our study, hyperactivity did not add to the prediction of ASPD over and above the effects of CD. The findings therefore add further support to the argument that the links between ADHD and ASPD may be due to overlap with CD, even when assessed as early as age 3.

One explanation for the finding that hyperactivity did not predict ASPD could be that symptoms of ADHD and conduct problems co-occur in most children (Lahey et al., 2002; Lynam, 1998; Moffit et al., 2008), and hyperactivity may not have added to the effects of conduct problems in the prediction of ASPD due to this overlap. However, the findings did show a trend towards effects of hyperactivity in the presence of conduct problems, but these were non-significant. This non-significance might be due to the fact that there was a difference in hyperactivity scores of responders and non-responders in this sample, so the results were expected to be an underestimation of "real world" effects. In addition, assessments of conduct problems and hyperactivity at age three, using parent-based assessments only, should not be regarded as the equivalent of clinical diagnoses of "CD" and "ADHD", respectively, which a) should and cannot be diagnosed at age three, and b) cannot be made on the basis of one questionnaire, without independent observer ratings. Bigger studies with larger samples, more power, and more reliable assessments of childhood disorders are needed to clarify the role of ADHD in the prediction of ASPD, over and above the effects of conduct problems.

4.4.2 Hyperactivity and AVPD

The results revealed a unique relationship between childhood hyperactivity and AVPD, which remained significant when controlling for the effects of the other childhood problems. This result was unexpected: even though heterotypic associations for ADHD have been demonstrated with longitudinal studies consistently reporting associations with adult internalising disorders (Biederman et al., 2006; Pardini & Fite, 2010; Rasmussen & Gillberg, 2000), homotypic continuities tend to show stronger effects than heterotypic continuities. Thus, AVPD, which is on the internalising spectrum, would have been expected to show stronger associations with internalising childhood problems. In particular, a significant association with childhood shyness would have been plausible: Persons with AVPD tend to show difficulties with social relationships, social anxiety and social withdrawal and present either with a generalised shyness, or with difficulties in sustaining relationships with people. As a consequence, persons with AVPD tend to habitually avoid social situations (Kantor, 2003). Therefore, based on the notion of homotypic continuities, an association between childhood shyness and AVPD was hypothesised.

Instead, a significant unique relationship with hyperactivity was found. Limited previous research has shown an association between childhood ADHD and AVPD: one study found that hyperactive children were significantly more likely to develop AVPD than controls (Miller et al., 2008). However, in this same study, the effects of ADHD were more pronounced for ASPD and BPD than for AVPD. In addition, their results showed that the effects were entirely driven by continuation of ADHD symptoms into adulthood: none of the participants with childhood ADHD who discontinued to have ADHD symptoms in adulthood, met criteria for an AVPD diagnosis in adulthood. That is, childhood ADHD as such did not predict AVPD. Instead, it was the continuation of ADHD that was associated with AVPD.

One speculative explanation for our finding could be the social problems that previous research has found to be associated with both AVPD and ADHD: Evidence has shown that ADHD is often comorbid with social phobia, which is also closely linked to AVPD. Social phobia (SP) is characterised by persistent fear and avoidance of social situations in which embarrassment

may occur; a somatic anxiety response upon exposure to the social situation; and, in adults, recognition that this fear is excessive or unreasonable (APA, 2013). The main characteristic of both SP and AVPD is a fear of negative evaluation, resulting in avoidance of social situations or feeling uncomfortable in social situations. Studies using community samples found that approximately 32.5% to 39.5% of individuals with AVPD had comorbid SP, whereas 18.3% to 36.4% of individuals with SP had comorbid AVPD (Cox, Pagura, Stein, & Sareen, 2009; Reichborn-Kjennerud et al., 2007).

SP is also very highly comorbid with ADHD: The National Comorbidity Survey Replication (Kessler et al., 2006) revealed that 29.3% of adults with ADHD had comorbid SP within the previous 12 months, the highest rate of any other anxiety disorder comorbid with ADHD. In children with ADHD, social problems are also very common (Cantwell, 1996; Friedman et al., 2003). According to Greene et al. (1996), many social skills deficits are related directly to the core symptoms of ADHD, such as behavioural disinhibition (e.g. interrupting conversations, intrusiveness, impatience) whereas other impairments, such as misinterpreting social cues, may reflect auxiliary deficits associated with underlying deficits in information processing (Crick & Dodge, 1994). In addition, social skills deficits that are associated with impaired functioning in school and in family and peer interactions have been documented in children and adolescents with ADHD (Biederman, Faraone, & Chen, 1993; Biederman et al., 1996; Greene et al., 1996; Hoy, Weiss, Minde, & Cohen, 1978). Research has also demonstrated that children with ADHD are aware of their lack of social skills and are adversely affected by the knowledge that they are unpopular (King & Young, 1982; Lahey, Shanghency, Strauss, & Frame, 1984). Research on social skills deficits in adults with ADHD is sparse; however, evidence suggests that ADHD symptoms continue to cause substantial problems in interpersonal relationships (Friedman et al., 2003; Hechtman, Weiss, & Perlman, 1984; Mannuzza, Gittelman, & Addalli, 1991; Weiss, Hechtman, Milroy, & Perlman, 1985). In addition, adults with ADHD have been found to rate themselves as less socially skilled at regulating their social behaviour than controls (Friedman et al., 2003).

Thus, the association between childhood hyperactivity and early adult AVPD may be linked to the social impairments in those with ADHD symptoms. Hyperactive children may be more prone to develop social skills deficits based on difficulties associated with the symptoms of ADHD. These difficulties, as well as their awareness of these difficulties, may lead to a further manifestation of these problems through social learning processes. These negative experiences may in turn lead to social anxieties and avoidance of these negative experiences, perhaps culminating in a diagnosis of SP or AVPD. However, these points are speculative and need to be further clarified.

4.4.3 Hyperactivity, conduct problems and Borderline PD

Both hyperactivity and conduct problems in childhood were independently linked to early adult BPD, with slightly stronger effects for conduct problems than for hyperactivity. Whilst longitudinal studies are limited, both these disorders have previously been linked with an increased risk for BPD; however, existing research has linked several EXT childhood problems to the development of BPD, and no specific associations had been detected, i.e. the effects of other childhood disorders were mostly uncontrolled. The results from our meta-analysis (Chapter 2) showed that ADHD strongly predicted BPD. However, in a large proportion of these, the effects of co-occurring childhood disorders were not controlled for.

One previous study examined the developmental links between childhood ADHD and ODD (assessed at ages 8 and 10), and BPD (assessed at age 14) in a large sample of girls (Stepp, Burke, et al., 2012). Using latent growth curve models, they found that an increase in ODD severity from age 8–10, but not age 10–13, predicted BPD symptoms at age 14. Conversely, for ADHD, increases in scores from age 10–13, but not 8–10, predicted Borderline symptoms at age 14. The authors argue that this suggests that for adolescent Borderline symptoms, difficulties with emotion regulation and relationships (as assessed by ODD) may precede problems with impulse control (as assessed by ADHD. This was not supported by the results of the present study - both impulse control and conduct problems were independently predictive of BPD, and both at a much younger age than in the above study by Stepp et al.

The behavioural and neurodevelopmental impairments in ADHD and BPD overlap substantially (Stepp, Burke, et al., 2012). For example, impulsivity, as well as poor self-regulation, executive function, and inhibitory control are key clinical features of both BPD and ADHD (Barkley, 1997; Daruna & Barnes, 1993; Dowson et al., 2004; Philipsen, 2006). Both disorders are characterised by impairment in executive functioning processes, such as working memory and attentional regulation (Barkley, 1997; Nigg, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Dysfunction in the prefrontal cortex is associated with both ADHD and BPD, implying overlapping neurological and behavioural mechanisms for these two disorders (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004; Spencer, Biederman, Wilens, & Faraone, 2002). In addition, in adulthood the two disorders are often comorbid (Biederman et al., 2007; Ferrer et al., 2010), and the two disorders have a lot of overlap in terms of personality traits (Koerting et al., 2012). This overlap in behavioural and neurodevelopmental impairments in ADHD and BPD may explain the putative developmental links between these two disorders.

In the area of conduct problems, very few longitudinal studies have been conducted; most long-term research on the effects of conduct problems has focused on ASPD rather than BPD. Nevertheless, some significant links have been demonstrated. For instance, one study showed that childhood ODD was associated with BPD symptoms in adolescents (Stepp, Burke, et al., 2012). Some evidence also shows that these effects hold in the presence of the respective other disorder: for instance, in a group of hyperactive children, it was found that childhood conduct problems predicted BPD when controlling for ADHD (Fischer, Barkley, Smallish, & Fletcher, 2002). Two other studies demonstrated that both childhood ADHD and childhood ODD were associated with BPD symptoms in adolescence (Stepp et al., 2012; Burke & Stepp, 2012). However, the sample was still very young at follow-up, so the results need to be replicated with older participants as in early adolescence the symptoms for BPD are generally higher than in later adolescence or adulthood. Nevertheless, limited previous evidence did show that both ADHD and conduct problems may be predictive of BPD in the presence of the respective other disorder, and our findings are in support of this evidence. In fact it was the only additive effect detected in our study. As mentioned above, due to a bias in the sample, the results are likely to be

an underestimation of real world effects due to a decrease in power. Thus, the finding that the effects of conduct problems and hyperactivity were additive, is likely to be a robust association. The effects are additive, but not interactive, indicating that there may be several pathways to BPD; one via hyperactivity and one via conduct problems.

4.4.4 Strengths and Limitations

This study addressed several methodological shortcomings of previous research. Firstly, whereas most previous longitudinal studies in this area used samples with a wide age range at baseline, the sample of the current study only included families whose children had first been assessed at age 3. In addition, most previous studies did not control for the effects of other co-occurring disorders when assessing the effects of childhood EXT/INT disorders on PD. Thirdly, in the area of PD, to our knowledge the additive and interactive effects of common childhood problems have not been tested. As such, this study filled some important gaps in the literature by addressing these issues specifically.

However, the findings of this study need to be interpreted in light of several limitations. Firstly, the current sample was likely to be subject to sample bias. On the one hand, this was related to the low response rate of approached families. It should be borne in mind, however, that the sampling strategy was not to follow up as many of the original families as possible (in which case the low response rate would pose a more serious threat), but the aim was to recruit families with specific, predetermined criteria until the target number for each group was met. On the other hand, systematic differences between responders and non-responders were found. Specifically, there were significant differences between responders and non-responders on both hyperactivity and conduct problem measures, with lower scores in responders on both scales. It is therefore very likely that the results are an underestimation of "real world effects". That is, the effects that were detected in this study are likely to be real effects; however, it is possible that some associations that showed only trends towards significance were not detected due to these systematic differences.

When analyses were carried out with re-weighted cases, where more weight was given to underrepresented cases and less weight was given to overrepresented cases, the pattern of results was the same. That is, even though specific values slightly changed, the overall patterns of significance and non-significance were similar, regardless of whether the analyses were carried out with the original data or with reweighted data. Nonetheless, it seems plausible to assume that the sample was biased in other aspects not included in the assessments of this study. Future research should focus on testing the effects of EXT and INT childhood problems on PD in larger, unbiased samples.

However, previous research has shown that sample biases may not necessarily decrease the validity of results. For instance, it has been found that differences in mean levels of variables between those who drop out and those who stay in a study do not necessarily imply that there are differences in *associations* between variables (Gustavson et al., 2012). In addition, evidence has suggested that systematic attrition of participants may not necessarily reduce the validity of prediction from longitudinal analysis (Wolke et al., 2009). Contrary to common assumptions, the presence of a substantial selection bias does not necessarily markedly attenuate the relationship between predictor and outcome variables. However, we cannot rule out the possibility that the sample was biased in other aspects not included in the assessments of this study, so this limitation due to sample bias should be borne in mind for the interpretation of the results.

Secondly, some issues regarding assessments made in this study should be mentioned. For instance, whilst the PID-5 (Krueger et al., 2012) is a valid and now widely used instrument for personality pathology, it was not designed to assess the specific DSM PDs. For the purpose of this study, specific subscale combinations were combined to produce dimensional scores that give estimates for six of the specific DSM PDs. Whilst the scale has been shown to map well onto the specific PDs (Morey & Skodol, 2013), it was not originally designed and standardised for this purpose. In addition, assessment of PD was based solely on self-report measures. Making a clinical diagnosis of PD requires an in-depth clinical interview carried out by a trained professional. Self-report assessments of PD should ideally be

corroborated through other-ratings (e.g. by the parent). Unfortunately neither of these options was available for the purposes of this study due to time and financial constraints. Therefore, the results need to be interpreted with caution and should not be mistaken for clinical diagnoses of PD. Future research in this area should ideally use clinically valid assessments of PD.

Similarly, some issues in relation to the scales that were used to assess childhood problems should be mentioned. (1) Some of the scales were not very reliable, with low alpha values and very few items for some of the subscales; (2) ratings were made by the parent only; ideally assessments made by trained professionals should be used, or ratings should be corroborated through other-ratings (e.g. by a preschool teacher); (3) scales used to assess INT problems were not ideal. Whilst both scales used (shyness and emotional problems) were clearly on the internalising spectrum, they may not have been the best INT predictors of PD. Usually, INT problems are assessed in the form of depressive or anxiety symptoms, and previous research in the area has focused on these INT aspects as predictors of PD. We did not detect any significant relationships between childhood INT problems and adult PD, which may have been related to the relative weakness of these INT childhood scales. Future research should address this issue by using more appropriate and reliable assessments of INT problems in childhood.

Fourthly, there was only one time of follow-up, with a long period in between assessments. Therefore, any inferences about developmental pathways to PD are very limited. In order to assess developmental pathways, ideally participants should be followed up at several time points.

4.4.5 Chapter Summary

The study presented in this chapter applied a prospective longitudinal design to investigate early childhood predictors of PD. Specifically, it was explored whether patterns of common externalising problems (hyperactivity and conduct problems) and internalising problems (emotional problems and shyness) in early childhood predicted adult PDs (ASPD, BPD, NPD, OCPD, SPD, AVPD). The findings showed that common childhood problems do indeed predict PD in early adulthood. Specifically, several unique

relationships between childhood problems and PDs were detected: conduct problems and hyperactivity predicted BPD, conduct problems predicted ASPD, and hyperactivity predicted AVPD. However, only one additive effect was found – the effects of hyperactivity and conduct problems were additive in the prediction of BPD – and no interactive effects were found in the prediction of any PD. The results are likely to be an underestimation of real world effects due to systematic differences between responders and non-responders, and the results need to be interpreted in light of several limitations. Future studies with larger, unbiased samples need to address these issues.

Chapter 5: Parental moderators of the relationship between child emotional and behavioural problems and PD

As demonstrated in Chapter 4, personality pathology can be predicted on the basis of early childhood problems, such as hyperactivity and conduct problems. Research has also shown that not only individual differences within the child, but also environmental risk factors have an influence on the development of personality disorders (PDs) in adulthood. Negative parenting has been suggested as a particularly strong risk factor for the development of PD. This chapter will explore the effects of paternal and maternal negative parenting on PD. In addition, we will examine whether any associations between childhood problems and PD identified in Chapter 4 were moderated by negative parenting.

5.1 Introduction

The risk associated with adverse parent factors on the child's development has several aspects. Firstly, children may have inherited a genetic predisposition to psychopathology from their parents: as discussed in Chapter 1, research has found that PD is partly heritable (Coolidge et al., 2001; Kendler et al., 2006; Reichborn-Kjennerud et al., 2007; Torgersen et al., 2008; Torgersen et al., 2000), as is the predisposition (temperamental vulnerability) to be affected by environmental stressors. That is, genetic predispositions to develop personality pathology may run in families. Parenting plays a fundamental role in a child's socialisation process, and adverse parenting practices have been strongly associated with children's behavioural and emotional problems (Frick, 1994; Loeber & Stouthamer-Loeber, 1986). In addition, behaviour geneticists have shown that associations between family environments and child traits are often genetically mediated. That is, genetic differences are associated with exposure to different environments, and many "environmental" variables have, in fact, been found to be moderately heritable (Kendler & Baker, 2007; Plomin & Craig, 2001). Heritable parental traits can influence the family

environment such that parents not only pass on genotypes to their children, but also a certain family environment that correlates with the genotype. For example, parents with poor self-regulation not only pass on a genotype for development of poor self-regulation in their children, but are also likely to provide a more chaotic home environment with fewer routines and little predictability.

The effects of negative parenting have been found to be long-term: for instance, seminal longitudinal research by Baumrind (Baumrind, 1967, 1971, 1989, 1991) showed that parenting behaviours, first assessed in preschool age, had long-term effects on their offspring's adjustment: the effects of adverse parenting persisted into middle and late childhood, as well as into adolescence. In addition, Barnes, Reifman, Farrell, and Dintcheff (2000) found that negative parenting behaviours in adolescence were associated with externalising problems in early adulthood.

5.1.1 Parental overcontrol and warmth

There are several prominent theoretical models in the extant research on parenting practices in relation to child behaviour (Power, 2013). Factor analytic studies obtained from psychometric assessment studies, and studies using independent observer ratings have identified two broad and universal dimensions of parenting *(*e.g., Grusec et al. (1997); Suchman et al. (2007); see Power (2013) for a review). These are parental control (overcontrol vs lack of control) and parental warmth (i.e. warmth/sensitivity vs lack of affection/indifference). Both these parenting dimensions have a strong influence on children's adjustment: for instance, research has revealed a link between low levels of parental warmth and externalising problems (Lee & Gotlib, 1991; Shaw et al., 1998). Low levels of warmth (e.g., lack of support or involvement) has been argued to interfere with a child's capacity to modulate and regulate arousal, and with a child's capability of considering the consequences of his/her actions (Chang et al., 2011; Eisenberg et al., 2005; McKee et al., 2008; Walton & Flouri, 2010). In addition, studies have reported significant associations between low levels of warmth and high levels of internalising problems in children (Garber et al., 1997; Hammen et al., 2004). It has been suggested that children learn to avoid the dysregulation that results from insensitive or unresponsive

parenting (i.e., parenting characterised by a lack of warmth) by withdrawing (Tronick & Gianino, 1986). This internalising response may become the child's preferred coping strategy which in turn has been suggested to place the child at risk for developing a number of symptoms related to internalising disorders (Field, 1995).

Both overly high and overly low levels of parental control have also been linked with child emotional and behavioural problems. For instance, Thompson et al. (Thompson, Hollis, & Richards, 2003) demonstrated that overcontrolling parental behaviour was associated with externalising child behaviour (conduct problems), and Alizadeh, Talib, Abdullah, and Mansor (2011) showed that maternal overcontrolling (authoritarian) parenting was associated with both externalising and internalising problems in children. Another study (Lewis-Morrarty et al., 2012) indicated that higher maternal overcontrol at seven years predicted higher social anxiety symptoms and lifetime rates of social anxiety disorder during adolescence. In addition, it was found that overcontrolling mothers had children with higher levels of internalising problems (Affrunti & Ginsburg, 2012). They argued that overcontrolling parental behaviour may communicate to youths that they do not have the skills to successfully handle challenges in their environment, thereby causing the child to worry about his/her abilities which in turn may increase avoidance and reduce the opportunities for youth to develop appropriate social or problem-solving skills. In sum, available research shows that negative parenting dimensions such as low warmth and high overcontrol are associated with both externalising and internalising childhood problems.

Even though there have been some studies about the importance of the father's role in their children's social and behavioural development (Harper & McLanahan, 2004; King, 1994; Lamb, 1997; Patterson & Dishion, 1988), the majority of studies on the influence of parenting practices on young children's development have focused on mothers. The general assumption is that maternal negative parenting affects the development of the child more strongly than paternal negative parenting (Enns et al., 2002; Kimbrel et al., 2007). However, even though fewer studies have investigated fathers' influence, research has revealed that negative parenting by both the mother and the father affect the child's outcome (Black et al., 1999; Kelley et al.,

1998; Lamb, 1997; Tamis-LeMonda et al., 2004). For instance, Sandler et al (2008) showed that (lack of) parental warmth of both mothers and fathers was significantly related to the children's externalising and internalising problems after the parents' divorce. Further, research has shown that parenting by the mother and by the father affect children differently. For instance, it has been demonstrated that paternal (lack of) warmth/supportiveness influenced the child much more strongly than maternal lack of warmth, whereas maternal overcontrol/intrusiveness had much stronger effects on the child than paternal overcontrol/intrusiveness (Cabrera et al., 2007).

5.1.2 Negative parenting and PD

The deficits that are evident among individuals with PDs have also been linked to adverse parenting behaviours (Keinänen et al., 2012). Although research, particularly longitudinal research, investigating this is sparse, there is a consensus among researchers and clinicians that parenting is a strong factor in the development of PD (Johnson, Cohen, et al., 2006; Johnson, Cohen, Kasen, et al., 2001; Keinänen et al., 2012). Those studies that do exist have indeed demonstrated that maladaptive parenting behaviours significantly increase the risk for PD in early adulthood independent of earlier childhood difficult behaviour and psychiatric disorder (Johnson, Cohen, et al., 2006; Johnson, Cohen, Kasen, et al., 2001). Specifically, it has been found that a combination of high parental overcontrol and low parental warmth, i.e. greater exposure to "affectionless control" (Parker et al., 1999; p. 363) increases the risk for PD. For instance, it has been shown that patients diagnosed with a PD reported lower levels of parental care, and higher levels of overprotection (Stravynski, Elie, & Franche, 1989). In addition, in a sample of child molesters, those who had a diagnosis of PD experienced more problematic relationships with their fathers compared to child molesters without a PD (Bogaerts, Vanheule, & Declercq, 2005). Specifically, they reported less warmth and more indifference from the father. No differences in maternal parenting behaviours between the two groups were found. Unfortunately, the authors do not state which PD was assessed. In addition, the results of this study are based on an extreme sample, so generalizability is questionable.

Most research directly investigating negative influences of parenting on PD has been conducted in the area of Borderline PD (BPD), and less directly in the area of Antisocial PD (ASPD) by investigating the effects of negative parenting behaviours on antisocial behaviour. Research has demonstrated that both lack of parental warmth and parental overcontrol are linked to the development of BPD and antisocial behaviour. For instance, BPD patients were found to report lower parental warmth and higher parental overcontrol (Byrne, Velamoor, Cernovsky, Cortese, & Losztyn, 1990; Paris & Frank, 1989; Torgersen & Alnaes, 1992; Zweig-Frank & Paris, 1991) than controls. Schuppert et al. (2012) investigated differences in retrospectively reported parenting styles in a group of referred adolescents with BPD features and healthy controls. The BPD group reported significantly less emotional warmth and more overprotection from their mothers than the control group, and these parental rearing styles strongly differentiated between controls and adolescents with BPD symptoms. Similarly, Trentacosta & Shaw (2008) prospectively investigated the effect of maladaptive parenting in early childhood on early adolescent antisocial behaviour and found that maternal hostile and controlling responses to toddler noncompliance predicted children's self-reported antisocial behaviour at 11 years. Another study showed that low parental affection/nurturing, assessed by self-report at age 16 years, was associated with ASPD at age 22 (Horwitz et al., 2001). One study looked at differences in maternal and paternal negative parenting and their effects on the development of BPD (Parker et al., 1999). This study showed that overall patients with high BPD features rated their parents as uncaring and overcontrolling. Specifically, paternal indifference and maternal overcontrol were found to be most distinctly associated with disordered functioning. In sum, both parental overcontrol and lack of parental warmth were associated with the development of both BPD and antisocial behaviour, and there may be differential effects of negative parenting on negative outcomes.

5.1.3 Interactional / transactional models of the development of personality pathology

Most theoretical models of the developmental pathways to PD are interactional or transactional models that are based on the notion that

individual vulnerabilities and environmental risk factors interact throughout life to influence a child's development, beginning as early as prenatally (Fruzzetti et al., 2005; Linehan, 1993). Usually, interactional models are diathesis-stress models which emphasise that pre-existing vulnerabilities lead to disorder when exposed to external stressors (Fruzzetti et al., 2005). They tend to focus on how the environment impacts individuals but are relatively silent about how individuals affect their environments (Huh, Tristan, Wade, & Stice, 2006). Thus, in the view of interactional models, negative parenting behaviours are stressors that interact with child characteristics (vulnerabilities) on the pathway to PD; i.e. they moderate the association between child emotional and behavioural problems and personality pathology. Interactional models take into account both the additive effects of parent and child characteristics, as well as their interactions (Sanson & Rothbart, 1995). As such, they propose that, although both negative parenting and difficult child behaviours are expected to directly predict children's development, for some children the effect of negative parenting will be exacerbated, whereas for others negative parenting will have less of an effect on the child (Lengua, Wolchik, Sandler, & West, 2000).

Specifically, research has shown that negative parenting has negative effects on children with higher levels of externalising problems. For instance, Lengua et al. (2000) demonstrated that negative parenting had a stronger effect on adjustment problems in children who were high in impulsivity, and a lesser effect on children who were high in positive emotionality. Other studies demonstrated that parental control aggravated children's externalising behaviour (Degnan, Calkins, Keane, & Hill-Soderlund, 2008); and that in undercontrolled children, externalising problems were enhanced in the presence of negative parental control (Van Leeuwen, De Fruyt, & Mervielde, 2004). However, research has also shown that adverse parenting behaviours had negative effects on children with internalising problems (Rubin & Burgess, 2002; Van Leeuwen et al., 2004). For instance, van Leeuwen et al. (2004) demonstrated that parent overcontrol had a stronger negative effect on children who were high in internalising problems, and van Brakel, Muris, Bögels, and Thomassen (2006) found that in high internalisers parent overcontrol contributed to the development of

anxiety problems. Similarly, another study showed that there was a significant interaction between high behavioural inhibition in childhood and maternal overcontrol, such that high inhibition predicted higher adolescent social anxiety symptoms in the presence of high maternal overcontrol, whereas high inhibition was not associated with adolescent social anxiety symptoms in children who experienced low maternal overcontrol (Lewis-Morrarty et al., 2012).

Whilst interactional models are popular models in explaining the interplay of vulnerabilities and environmental factors in the development of PD (Beauchaine et al., 2009; Fruzzetti et al., 2005), data supporting such models has been very limited. One exception is a study that examined the joint effects of early childhood adversity and temperament on the later development of BPD and Avoidant PD (AVPD) in a sample of 188 depressed outpatients (Joyce et al., 2003). The study showed that AVPD developed through a combination of high internalising temperament (shy, anxious), childhood and adolescent internalising disorders and negative parenting (parental neglect). BPD developed through a combination of childhood abuse and/or neglect, a combination of externalising and internalising temperament (high novelty seeking and high harm avoidance), and a combination of externalising and internalising psychopathology. However, this study was limited in several ways: firstly, the sample consisted of depressed outpatients only, so the generalizability of these results is unclear. Secondly, a big limitation of this study was its cross-sectional nature – both childhood problems and abuse and neglect experiences were rated retrospectively which may be distorted due to recall bias (Mannuzza et al., 2002; Maughan & Rutter, 1997). Thirdly, only extreme forms of negative parenting were assessed, i.e. abuse and neglect, whereas less extreme, but also more common forms of adverse parenting (e.g. overcontrol and/or lack of warmth) were not considered. In sum, theoretical models assume a strong association between parenting and the development of PD, but prospective longitudinal studies supporting these theories are sparse and/or flawed.

5.1.4 The current study – research aims and hypotheses

The aim of the current study was to address the complex interplay between childhood problems and negative parent behaviours in predicting adult personality pathology in a prospective longitudinal study. The aim was to start exploring the extent to which parenting influences the relationship between child externalising and internalising problems and PD. Specifically, testing interactional models of the development of PD, interaction (moderation) effects of negative parenting dimensions on significant associations between childhood problems and PDs established in Chapter 4 were investigated.

Specific research aims and hypotheses were:

1. To investigate whether negative parenting (overcontrol and lack of warmth) had an effect on the development of personality pathology, and whether these effects would add to the associations between childhood problems and personality pathology established in Chapter 4.

> ***Based on previous evidence it was hypothesised that (1) both parental overcontrol and lack of warmth would predict both externalising (ASPD/BPD) and internalising (AVPD) PDs, and (2) both parental overcontrol and lack of warmth would add to the effects of childhood problems in the prediction of personality pathology. These effects were expected for both maternal and paternal overcontrol and lack of warmth.***

2. To investigate interaction effects between negative parenting and childhood problems in the development of PD, specifically to examine the potential role of parenting as moderator on the association between child problems on personality pathology.

> ***In line with interactional models of the development of PD, it was hypothesised that negative parenting would moderate the association between pre-existing child behavioural problems and personality pathology. Specifically, it was predicted that (1) the higher the children's levels of conduct problems, the more they would be affected by parental lack of warmth and overcontrol and thus the higher their ASPD symptom severity in adulthood; (2) the higher the***

children's levels of conduct problems and/or hyperactivity, the more they would be affected by parental lack of warmth and overcontrol and thus the higher their BPD symptom severity in adulthood; (3) the higher the children's levels of hyperactivity, the more they would be affected by parental lack of warmth and overcontrol and thus the higher their AVPD symptom severity in adulthood; (4) the association between conduct problems and ASPD was expected to be weaker among those children who did not experience parental overcontrol or lack of warmth; (5) the association between hyperactivity and/or conduct problems and BPD was expected to be weaker among those who did not experience parental overcontrol and/or lack of warmth; and (6) the association between hyperactivity and AVPD was expected to be weaker among those who did not experience parental overcontrol and/or lack of warmth.

All these effects were expected for both maternal and paternal overcontrol and lack of warmth.

5.2 Methods

5.2.1 Participants

For details about the procedure of selecting participants for this study, please see Chapter 3. An overview about sample characteristics at baseline is presented in Chapter 3.

5.2.2 Procedure

Please see Chapter 3 for details about the procedure.

5.2.3 Measures

Please see Chapter 3 for details about baseline and follow-up measures. An overview about the assessments used for analyses in this chapter is presented in table 14.

Table 14: Predictors, Outcome Variables, Mediators/Moderators and Covariates

	Construct	Assessment Tool
Childhood Predictors (IVs) - Behaviour Problems (age 3)	Hyperactivity	WWP Activity Scale
	Conduct Problems	BCL Social Maladjustment
Adult Outcomes (DVs) – Personality Pathology	Specific PDs: Borderline, Antisocial, Avoidant	PID-5
Mediators / moderators (assessed retrospectively at follow-up)	Maternal overcontrol	MOPS; Overcontrol Subscale (Mother)
	Paternal overcontrol	MOPS: Overcontrol Subscale (Father)
	Maternal lack of warmth	MOPS; Indifference Subscale (Mother)
	Paternal lack of warmth	MOPS; Indifference Subscale (Father)
Covariates	Age at follow-up	
	Sex	
	Socio-economic status	Carstairs deprivation score

IV – Independent variable, DV – dependent variable, WWP – Werry-Weiss-Peter-Activity-Rating-Scale, BCL – Behaviour Checklist, EAS – Emotionality, Activity, Shyness Temperament Scale; SES – Socioeconomic Status; PD – personality disorder

5.2.4 Analyses

The effects of parenting on associations between childhood problems and PDs were only carried out for those relationships that were found to be significant in Chapter 4.

5.2.4.1 Main, additive and interaction effects

The procedure applied for assessing main, additive and interactive effects was identical to the methods applied in Chapter 4; please see Chapter 4 for details.

All statistical analysis was carried out with SPSS 21 for Windows. Age at follow-up, sex, and deprivation at baseline were controlled for in all multivariate models due to known effects on PD. Firstly, PD symptoms tend to change with age: Cluster B symptoms have been found to naturally reduce with age, whereas some data suggests that Cluster A and C symptoms may increase with age (Gunderson, 2011; Morse & Lynch, 2004; Zanarini et al., 2006). Because participants' age range at follow-up was quite wide (17-26) in this study, this was controlled for in multivariate models. Further, evidence suggests gender differences in PD, in terms of prevalence, expression of symptoms, as well as in pathogenesis and comorbidity with other disorders (Grilo et al., 1996; Johnson et al., 2003) so the effects of gender were controlled for. Thirdly, low socioeconomic background has been found to be independently predictive of PD (Cohen, 2008) and to mediate the relationship between other predictors and PD (De Genna & Feske, 2013), so the effects of deprivation at baseline were statistically controlled for. In all analyses, socio-economic status (SES), age at follow-up and gender were controlled for.

5.3 Results

5.3.1 Preliminary analyses

The presence of outliers and influential cases was assessed, and checks on assumptions of multiple regressions were conducted, using the methods described in Chapter 4. Two cases were identified as influential outliers. Analyses were conducted, dropping these cases to assess the degree to

which the findings were influenced by their presence. No changes occurred in the pattern of significant effects. Because of the consistency in the results, the outliers were retained in all subsequent analyses. Absolute values of kurtosis ranged from 1.103 (emotional problems) to 5.523 (paternal indifference) in predictor/moderator variables and from -2.797 (SPD) to 2.118 (ASPD) in outcome variables. Only paternal indifference represented a deviation from a normal distribution. Absolute values of skewness ranged from 1.892 (maternal indifference) to 7.335 (paternal indifference) in predictors/moderators, and from 3.108 (SPD) to 6.271 (ASPD) in outcome variables. All predictor and moderator variables except maternal overcontrol and indifference were positively skewed. All further analyses were performed using the original, un-transformed, dataset. Multicollinearity among the predictors was assessed using the variance inflation factor (VIF) statistic. In this sample, the VIFs ranged from 1.25 to 1.73, all within acceptable ranges.

As mentioned in Chapter 4, recruited participants did not distribute equally across the groups (see Table 8). Therefore, all analyses were carried out twice: 1. With re-weighted cases such that the weight of all four groups was balanced, so that more weight was given to underrepresented groups and less weight was given to overrepresented groups, and 2. Using the original unweighted data. The pattern of results was the same in both cases, i.e. even though specific values slightly changed, the overall patterns of significant and non-significant predictors were identical, regardless of whether the analyses were carried out with the original data or with reweighted data. Therefore only results from analyses carried out with original, unweighted data is presented here.

Mean values for mother/father indifference and overcontrol as rated by the child are presented in Table 15. Mean values for father indifference were significantly higher than mean values for mother indifference. The intercorrelations between conduct problems, hyperactivity, negative parenting and PD dimensions are presented in Table 16.

Table 15: Mean values for mother and father overcontrol and indifference

	Mother	Father	t (df); sig.
Mean Overcontrol (SD)	1.29 (.762)	1.21 (.720)	1.702; n.s.
Mean Indifference (SD)	0.65 (.570)	0.86 (.846)	-4.184; p<.001

n.s. – not significant ; SD – Standard Deviation

Table 16: Intercorrelations between study variables

	HYP age 3	*CP age 3*	*Mother indifference*	*Mother overcontrol*	*Father indifference*	*Father overcontrol*	*Borderline PD*	*Antisocial PD*	*Schizotypal PD*
Mother Indifference	.032	.032							
Mother overcontrol	.124	.118	.545***						
Father indifference	.148*	.114	.564***	.457***					
Father overcontrol	.048	.048	.516***	.620***	.544***				
Borderline PD	.255***	.286***	.229**	.332***	.352***	.333***			
Antisocial PD	.190**	.284***	.194**	.321***	.264***	.202**	.752***		
Schizotypal PD	.166*	.182**	.285***	.351***	.383***	.344***	.739***	.657***	
Avoidant PD	.197**	.178**	.271***	.326***	.319***	.325***	.742***	.465***	.795***

Note: N=216; *p<.05; **p<.01; ***p<.001 ; HYP – hyperactivity; CP – Conduct Problems; PD – Personality Disorder

5.3.2 Main, additive and moderation effects of negative parenting

Please see Table 17 for an overview about main effects of negative parenting on adults PDs. Father indifference predicted BPD and SPD, and mother overcontrol predicted ASPD.

Table 17: Hierarchical linear regression analyses of the effects of negative parenting on adult PDs

	Borderline PD		*Antisocial PD*		*Avoidant PD*	
	ΔR²	*β*	*ΔR²*	*β*	*ΔR²*	*β*
Step 1	.026		.055*		.032	
Gender		.098		-.161*		.091
SES		-.062		-.058		-.129
Age at follow-up		-.167*		-.149		-.163*
Step 2	.152***		.139***		.122***	
Mother indifference		-.079		-.018		.004
Father indifference		.253**		.190*		.218*
Mother overcontrol		.192*		.299**		.145
Father overcontrol		.081		-.053		.056

** p<.05; ** p<.01; *** p<.001 ΔR² - R² change; results presented in bold are significant after correcting for multiple testing (p<.01)*

In order to investigate whether any of the significant parent variables added to, or interacted with previously established associations between child problems and PDs (Chapter 4), three hierarchical multiple regression analyses were conducted.

Table 18: Hierarchical Linear Regression Analyses of the Effects of Child Problems as Predictors of Personality Disorders

	Borderline PD		*Antisocial PD*	
	ΔR²	*β*	*ΔR²*	*β*
Step 1	.032		.053*	
Gender		.098		-.161*
SES		-.062		-.058
Age at follow-up		-.167*		-.149
Step 2	.243***		.126***	
Hyperactivity		.293***		-
Conduct Problems		.305***		.367***
Step 3	.043**		.075***	
Hyperactivity		.236**		-
Conduct Problems		.287**		.305***
Mother overcontrol		-		.281***
Father indifference		.216**		-
Step 4			.012	
HYP x father indifference		-.183		-
CP x father indifference		-.003		-
CP x mother overcontrol				-.324

** p<.05; ** p<.01; *** p<.001 ΔR² - R² change; results presented in bold are significant after correcting for multiple testing (p<.02)*

The results showed that hyperactivity, conduct problems and father indifference were all predictive of BPD, and conduct problems and mother overcontrol were predictive of ASPD. No significant interactions were found.

5.4 Discussion

In the current chapter we explored the complex interplay between early child behavioural problems and negative parent behaviours in the prediction of adult personality pathology. Using a longitudinal design, this study aimed to investigate the effects of negative parenting on PD, as well as the moderation effects of negative parenting on the association between child problems and personality pathology. Key findings were: (1) Negative parenting does indeed increase the risk of developing PD in adulthood; father overcontrol predicted BPD, and mother overcontrol predicted ASPD. (2) All significant parenting predictors added to the associations between childhood problems and PDs established in Chapter 4. (3) Negative parenting did not moderate the associations between child problems and PDs. Findings will be discussed in detail below.

5.4.1 The effects of negative parenting on the development of PD in adulthood

Overall, the results of this study indicate that negative parenting increases the risk of developing PD in adulthood, in line with previous research. Father indifference predicted BPD, and mother overcontrol predicted ASPD. No effects were found for maternal indifference and father overcontrol. Previous longitudinal research has demonstrated that a combination of high parental overcontrol and low parental warmth increases the risk for PD (Bogaerts et al., 2005; Byrne et al., 1990; Paris & Frank, 1989; Schuppert et al., 2012; Stravynski et al., 1989; Torgersen & Alnaes, 1992; Zweig-Frank & Paris, 1991); our findings support the consensus among researchers and clinicians that parenting is a strong factor in the development of PD. However, our results were more specific than previous studies by showing differential effects of maternal and paternal negative parenting. Whereas strong links were found between father indifference, mother indifference and PD, no associations were found for maternal indifference or paternal overcontrol and PD. These differential effects of negative parenting variables by the mother and the father may be related to the different roles mothers and fathers play in the upbringing of their child/-ren. Evidence shows that mothers and fathers engage in rather different types of

interaction with their children (Lamb, 1981), consistently showing that fathers tend to specialise in play, whereas mothers specialise in caretaking/nurturance. Thus, perhaps those negative parenting variables that affect children's development most negatively, are most opposed to the roles the parents traditionally fulfil in Western cultures.

Whilst some evidence does imply that negative parenting by both the mother and the father affect the child's outcome (Black et al., 1999; Kelley et al., 1998; Lamb, 1997; Sandler et al., 2008; Tamis-LeMonda et al., 2004), the majority of previous studies have focused on mothers, and it has been argued that maternal negative parenting affects the development of the child more strongly than paternal negative parenting (Enns et al., 2002; Kimbrel, Mitchell, Hundt, Robertson, & Nelson-Gray, 2012). Our results are not in support of this argument – both maternal and paternal parenting influenced the development of PDs. Research has shown that fathers are overall much less involved with their children than mothers and spend significantly less time with their children than mothers do, regardless of employment status of the mother (Pleck & Masciadrelli, 2004). This was the case in this study as well: young people rated their fathers as significantly more indifferent as compared to their mothers.

This finding may also have been related to actual absences of fathers in this sample. In a lot of families who took part, mothers were divorced or separated from the child's biological father and often lived with a new partner at follow-up, or without a partner. Thus, high ratings of father indifference may reflect the fact that a lot of these fathers were in fact absent and therefore much less involved in their children's lives. This absence may also explain why father indifference had such strong effects on the development of PD: evidence has shown that father absence is highly detrimental to the development of the child due to several different reasons (Lamb, 1997). Firstly, absence of a co-parent can be harmful - following divorce, children consistently do better when they are able to maintain meaningful relationships with both parents unless the levels of interparental conflict remain unusually high (Kelly, 2000). Secondly, the economic difficulties often associated with single motherhood (Pearson & Thoennes, 1990) can significantly add to the overall stress levels of the mother and also lead to emotional stress, which in turn may influence

parenting qualities. Thirdly, children of divorce are often affected by the perceived, and frequently actual, abandonment by one of their parents (Kelly & Lamb, 2000; Thompson & Laible, 1999), and there are often detrimental effects of predivorce and postdivorce marital conflict (Cummings & O'Reilly, 1997; Kelly, 2000). Because most single-parent families are the product of divorce and because divorce is often preceded and accompanied by periods of overt and covert spousal hostility, parental conflict may play a major role in explaining the problems of fatherless children. However, we did not account for the effects of father absences in our analyses, so whether it is father indifference or actual father absence which affects the development of PD still needs to be clarified.

5.4.2 Does negative parenting add to or moderate the associations between childhood problems and PD?

All of the significant parenting effects added to the associations between childhood problems and PDs established in Chapter 4. That is, father indifference added to the effects of both conduct problems and hyperactivity in the prediction of BPD, and to the effects of hyperactivity in the prediction of AVPD; mother overcontrol added to the effects of conduct problems in the prediction of ASPD. Thus, despite significant overlap between both outcomes and childhood predictors both conceptually and in terms of assessments, somewhat surprisingly, distinct risk patterns for different PDs emerged. BPD was independently predicted by conduct problems, hyperactivity and father indifference, whereas ASPD was predicted by conduct problems and mother overcontrol. However, whilst all these effects of both childhood problems and negative parenting were highly significant in the prediction of PD, no interaction effects emerged.

The finding that these child problems and negative parenting variables were independently predictive of PDs and that their effects were not affected by the presence or absence of the respective other factor suggest that they may represent different pathways to PD, one via negative parenting and one directly via childhood problems. This implies that either factor alone (i.e. childhood behaviour problems or negative parenting) significantly and independently increases a child's risk of developing PD. This is in contrast to theoretical models about the pathways to PD, most of

which are interactional models based on the notion that individual vulnerabilities and environmental risk factors interact throughout life to influence a child's development (Fruzzetti et al., 2005; Linehan, 1993), emphasising that pre-existing vulnerabilities in combination with stressors lead to disorder (Fruzzetti et al., 2005). These models also propose that, although both negative parenting and difficult child behaviours are expected to directly predict children's development, for some children the effect of negative parenting will be exacerbated, whereas for others negative parenting will have less of an effect on the child (Lengua et al., 2000). Our findings do not support these models: rather, they suggest that individual vulnerabilities (in this case, conduct problems and hyperactivity) and stressors (maternal overcontrol and father indifference) both lead to PD, irrespective of the presence of the other factor. These effects are additive. In addition, these results further highlight the robustness and consistency of the findings described in Chapter 4 by demonstrating that the associations between child predictors and adult PDs were unaffected by negative parenting.

5.4.3 Clinical implications

Finding such strong long-term effects of early childhood behaviour problems has implications in terms of early intervention/prevention strategies. On the one hand these findings demonstrate how strikingly early in life the course for a negative outcome may already be set. On the other hand, findings such as these can be regarded as an opportunity: identifying childhood predictors of PD enables the development of treatment approaches aimed at early identification and prevention. Some specific PDs such as Borderline PD have been suggested to be "leading candidates" for developing such programmes (Chanen & McCutcheon, 2013). However, the more behaviour patterns are established the more difficult they become to treat (Burke et al., 2007; Linehan, 1993). Thus, identification of risk patterns very early in life can be regarded as an opportunity to implement treatment approaches directly for those who may be most at risk.

The most optimal prevention method for PD has been suggested to be 'indicated prevention' where individuals displaying risk markers of the disorder are targeted (Chanen & McCutcheon, 2013). As such, identification

of these early predictors in children is useful for two reasons: Firstly, early signs and symptoms in children could be identified and directly targeted, and secondly, the risk markers themselves could become target of interventions. Further, whilst standardised prevention/early intervention programmes have not yet been implemented specifically for PD, some early interventions for antisocial behaviour, such as the High/Scope Perry Preschool Program have already been shown to be (cost) effective in the US (Schweinhart & Weikart, 1998), and have been highly recommended in a study explicitly investigating the economic cost of severe antisocial behaviour in children (Romeo et al., 2006).

One of the most often used interventions is parent training, which is commonly regarded as one of the most effective interventions for early behaviour disorders (Brestan & Eyberg, 1998; Lundahl, Risser, & Lovejoy, 2006; McCart, Priester, Davies, & Azen, 2006). Most parenting programmes are based on social learning theory and attachment theory: they teach parents appropriate contingency management, modelling, and strategies to strengthen the attachment relationship between parent and child. Parenting programmes are usually based on the premise that parenting practices contribute to children's disruptive behaviours across childhood and aim to influence children's behaviours indirectly by teaching parents skills and modifying parents' assumptions about child development and child rearing. The effectiveness of parenting programmes has been investigated in several reviews: McCart et al (2006) compared the effects of parent training and cognitive behaviour therapy (CBT) for behaviour problems in a meta-analysis and found that both could be effective even though for both parent training and CBT the effect sizes were in the small to medium range. For parent training, there was a small effect for parent adjustment as well, suggesting that parents who participate in such programmes experience reductions in their own psychosocial stress. Another meta-analysis (Lundahl et al., 2006) suggests that parent training for disruptive behaviour problems produces moderate effect sizes immediately after treatment and small but significant effect sizes up to one year at follow-up.

Our findings also highlight the importance of father involvement in a child's life, and the detrimental effects non-involvement or absence of the father may have in terms of a child's healthy development. As such, the results

have strong implications in terms of early intervention/prevention strategies for PD, namely to make efforts to increase father involvement in children's upbringing. There has been a growing interest in the development of effective programmes and policies that support and promote positive father-child relationships (Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000), and there is some evidence that programmes increasing father involvement strengthen families and improve father-child interactions (Lundahl, Tollefson, Risser, & Lovejoy, 2007; Magill-Evans, Harrison, Rempel, & Slater, 2006). However, in terms of early intervention/prevention parenting programmes, reviews indicate that only 20% of such programmes include fathers (Coplin & Houts, 1991; O'Brien & Budd, 1982). This is not surprising considering that historically, parent training was synonymous with mother training, consistent with the past emphasis on mothers as the primary socialising agent (Coplin & Houts, 1991; Lamb, 1997).

Nevertheless, interventions that involve both mothers and fathers demonstrate improvements in child behaviour (Cowan, Cowan, Pruett, Pruett, & Wong, 2009; Lundahl et al., 2007), father engagement (Cowan et al., 2009), and parent perceptions (Lundahl et al., 2007). Such interventions may have better outcomes than interventions with only mothers or only fathers (Lundahl et al., 2007). Programmes that focus on active father-child involvement have been shown to enhance fathers' interactions with their children and increase fathers' positive perceptions of their children (Magill-Evans et al., 2006). These interventions may also increase children's cognitive development (Magill-Evans et al., 2006) and reduce problem behaviours (Lundahl et al., 2007).

Thus, early intervention/prevention approaches for PD should focus on involving fathers in the upbringing of their children. The results of this study strongly suggest that increasing father involvement may greatly decrease the risk for a negative outcome. Limited evidence shows that initiatives aimed at fathers should begin at birth, when many fathers are highly motivated to remain involved in their infants' lives (Lamb, 1997). Despite their early commitment, many fathers in particularly vulnerable families drift out of their children's lives over time; therefore it has been suggested that fatherhood programs should take a preventive approach by

providing services to new fathers well before they distance themselves from their children (Lamb, 1997).

5.4.4 Limitations

The current findings need to be interpreted in light of several limitations. Firstly, several issues in relation to the assessments of negative parenting should be mentioned. Assessments were made retrospectively. Retrospective assessments are of course subject to recall bias (Maughan et al., 1995; Maughan & Rutter, 1997; Robins et al., 1985). In addition, this study only focused on negative parenting. The current study implied that parenting had no effect on the association between childhood EXT problems and PDs: childhood HYP and CP predicted PDs regardless of negative parenting. However, positive parenting can serve as a strong protective factor: whilst not much research has been carried out in the area of PD specifically, evidence suggests that parental empathy, support and warmth helps children to cope effectively with many types of adversity (Cowen, 1994; Luthar & Zigler, 1991), and strong and supportive relationships with family members are associated with healthy interpersonal functioning (Werner & Smith, 1982). Thus, positive parenting may have attenuated the effects of child behaviour, which could not be tested in the current research. Assessing these effects would have provided a more complete picture of the associations between childhood problems, parenting and adult PD.

Secondly, as mentioned in previous chapters, this sample was subject to sample bias due to the high attrition rate and systematic differences between responders and non-responders on hyperactivity and conduct problems, with lower scores in responders on both scales. Therefore, the results of this study are likely to be an underestimation of real world effects, so the chance of making Type II errors was increased, i.e. the risk that weaker associations would not reach significant levels was increased due to the sample bias. Nevertheless, when analyses were carried out with re-weighted cases, where more weight was given to underrepresented cases and less weight was given to overrepresented cases, the pattern of results was the same in both cases. That is, even though specific values slightly changed, the overall pattern of significant and non-significant predictors were identical, regardless of whether the analyses were carried out with the

original data or with reweighted data. Nonetheless, it seems plausible to assume that the sample was biased in other aspects not included in the assessments of this study. Future research should focus on testing the effects of EXT and INT childhood problems on PD in larger, unbiased samples.

However, previous research has shown that sample biases may not necessarily decrease the validity of results. For instance, it has been found that differences in mean levels of variables between those who drop out and those who stay in a study do not necessarily imply that there are differences in *associations* between variables (Gustavson et al., 2012). In addition, evidence has suggested that systematic attrition of participants may not necessarily reduce the validity of prediction from longitudinal analysis (Wolke et al., 2009). Contrary to common assumptions, the presence of a substantial selection bias does not necessarily markedly attenuate the relationship between predictor and outcome variables. However, we cannot rule out the possibility that the sample was biased in other aspects not included in the assessments of this study, so this limitation due to sample bias should be borne in mind for the interpretation of the results.

An additional bias in this study may have been related to ratings of mother indifference. Mother indifference was found to be relatively unimportant in the prediction of PD in this study which may have been due to the very low variance of mother indifference scores in this sample. In most cases, it was mothers who responded to study invitations (rather than fathers). It seems probable that mothers who choose to respond to, and take part in, research studies about their children, are systematically different from parents who refuse to take part in such research projects. It seems likely that those mothers who do take part are those who are *not* rated as indifferent. Of course, this is just speculative and cannot be tested. However, perhaps significant effects of mother indifference were not detected due to the low variance in maternal indifference scores in this study.

Some issues regarding follow-up assessments made in this study should be mentioned. The main outcome measure for PD – the PID-5 (Krueger et al., 2012), a valid and now widely used instrument for personality pathology,

was not designed to assess the specific DSM PDs. For the purpose of this study, specific subscale combinations were combined to produce dimensional scores that give estimates for six of the specific DSM PDs. Whilst the scale has been shown to map well onto the specific PDs (Morey & Skodol, 2013), it was not originally designed and standardised for this purpose. In addition, assessment of PD was based solely on self-report measures. Making a clinical diagnosis of PD requires an in-depth clinical interview carried out by a trained professional. Self-report assessments of PD should ideally be corroborated through other-ratings (e.g. by the parent). Unfortunately neither of these options was available for the purposes of this study due to time and financial constraints. Therefore, the results need to be interpreted with caution and should not be regarded as an equivalent of clinical diagnoses of PD.

An additional limitation concerns the age of the child at which the ratings of behaviour problems were made. It may be impossible to disentangle the effects of individual child characteristics from negative parenting, even at such an early age. The first three years in life are highly important in the development of a child, and the parents of course play a vital role in these years. Therefore, the parents may already have had an influence on the development of those childhood problems at age 3 that the child presented with.

In addition, it would have been helpful to control for the effects of parental psychopathology, both at baseline and at follow-up, as mental health is closely linked to parenting. For instance, several studies have shown that children of mothers with a diagnosis of PD have an increased risk of emotional and behavioural problems, including BPD symptoms (for a review, see Stepp et al. (2012)). Another study showed stronger associations between PD symptoms and negative parenting styles in students who were raised by a parent with PD, as compared with students raised by a parent without PD (Cheng, Huang, Liu, & Liu, 2011).

An additional issue was shared rater variance for the assessments completed by the young person, which could pose a potential threat to the study's validity because it may have produced variance that is attributable to the measurement method rather than to the constructs the measures

represent (Podsakoff et al., 2003) and, as such, could present an alternative explanation for an observed association due to artifactual covariance. One obvious way to remedy this issue would have been to collect the measures from different sources; however this was not feasible in this study due to time and financial constraints. The most likely effect of the shared rater variance was expected to be an overestimation of associations of PD with negative parenting variables.

Finally, because we had only two time points in this study where data was collected, conclusions should be drawn with caution about the causal relationship between the variables. Ideally, parenting should have been assessed at baseline, and both predictor variables and parenting variables should have been assessed at several points in time to make any inferences about developmental pathways.

5.4.5 Chapter Summary

The results of the study presented in this chapter indicated that negative parenting by both mother and father increase the risk for later PD, but that it is different aspects of parenting in mothers and fathers that increase this risk. Negative parenting added to the effects of childhood hyperactivity and conduct problems in the prediction of PDs. However, no interactive effects were found, suggesting different pathways to PD. Distinct risk patterns for different PDs emerged. BPD was independently predicted by conduct problems, hyperactivity and father indifference, whereas ASPD was predicted by conduct problems and mother overcontrol. These findings need to be regarded in light of some study limitations. Future research should systematically test the effects of parenting in interactional models, using prospective parenting data collected at several time points in larger, unbiased samples.

Chapter 6: Do continuities in psychopathology dimensions explain the associations between childhood problems and adult PD?

Chapters 4 and 5 presented evidence that PDs can be predicted on the basis of childhood problems. Previous research has also shown, however, that these childhood problems predict later psychopathology other than PD: adult internalising (INT) disorders such as anxiety and depression, and externalising (EXT) disorders such as substance abuse, have also been found to be predicted by behaviour problems in childhood. These adult disorders, in turn, have been found to be highly co-occurring with, and predictive of, PDs. This poses the possibility that the links between childhood problems and adult PD could be explained by co-occurring psychopathologies. The aim of this chapter was to explore whether any associations between childhood problems and PD identified in Chapter 4 were mediated by continuities of childhood symptoms into adulthood, i.e. by adult co-occurring psychopathologies.

6.1 Homotypic and heterotypic continuities of childhood disorders

As described in previous chapters, childhood problems have been found to show both homotypic and heterotypic continuity, i.e. continuity within the same (homotypic) or different (heterotypic) 'class' of disorder. Longitudinal studies have found, for instance, that EXT problems such as ADHD and CD predict both EXT and INT adult disorders. For example, childhood ADHD symptoms have been found to show long-term continuity, from preschool age to later childhood (Harvey et al., 2009), as well as from childhood to adulthood, where individuals continued to fulfil the criteria for a diagnosis of adult ADHD; estimates ranged from 7% (Mannuzza et al., 1993, 1998) to 58% (Biederman et al., 2006). Childhood ADHD was also found to predict other EXT disorders, such as adult CD (Loeber et al., 1995; Mannuzza et al.,

2004; Moffit et al., 1996), adult ODD (Harvey et al., 2009; Pardini & Fite, 2010) and substance use disorders (Wilens & Morrison, 2011). Similarly, conduct problems such as childhood ODD and CD have been found to show long-term continuity. In fact, ODD, CD and ASPD are often viewed hierarchically, reflecting age-dependent expressions of the same underlying disorder (Moffit et al., 2008), where ODD is conceptualised as a developmental precursor to CD, and CD is conceptualised as a developmental precursor to ASPD. Typically, ODD symptoms appear first, and then, in a subgroup of children with ODD, CD symptoms develop (Lahey et al., 1997; Loeber et al., 1995). Evidence from a follow-back study of clinic-referred boys shows that 80% of boys with childhood CD had prior ODD (Lahey et al., 1997). Two prospective studies showed that about 60% (Lahey et al., 1997) and 40% respectively of children with ODD progressed to develop CD (Rowe, Maughan, Pickles, Costello, & Angold, 2002). In turn, follow-forward studies show that about one-third to one-half of children with CD grow up to have adult ASPD (Robins, 1966, 1978). In addition, children with conduct problems are often found to develop other EXT problems later in life, such as substance abuse disorders and serious violent behaviour (Loeber et al., 2002). Thus, there is strong evidence for homotypic continuity of both ADHD and conduct problems. However, for both ADHD and CD, heterotypic continuity has also been demonstrated. Prospective longitudinal studies consistently report associations between childhood ADHD and adult internalising disorders, such as anxiety and depressive disorders (Barkley et al., 2004; Biederman et al., 2006; Pardini & Fite, 2010; Rasmussen & Gillberg, 2000). Similarly, children with CD have been found to develop mood disorders later in life (Loeber et al., 2002). Thus, evidence shows both homotypic and heterotypic continuities childhood hyperactivity and conduct problems.

6.2 Co-occurring internalising and externalising psychopathologies of adult PDs

As demonstrated in Chapter 4, childhood problems such as hyperactivity and conduct problems predict adult PD. Previous evidence has also shown that these childhood disorders predict adult psychopathology other than PD, across both EXT and INT domains. These adult EXT and INT disorders, in

turn, have been found to very often co-occur with, and are predictive of, personality pathology (Coid & Ullrich, 2010; Goldstein, Grant, & Ruan, 2006; Howard, Finn, Jose, & Gallagher, 2012).

In many cases, individuals do not display a single discrete disorder but several (Kaplan, Crawford, Cantell, Kooistra, & Dewey, 2006), which researchers and clinicians commonly refer to "comorbidity" or "co-occurrence" of disorders. When multiple diagnoses are applied, the term comorbidity has been used to refer to the fact that diagnostic criteria for more than one disorder are met. The term 'comorbidity' is a relatively recent concept: Comorbidity has been defined 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). The term comorbidity first appeared in psychiatric literature in the early 1980s (Boyd et al., 1984; Lilienfeld, Waldman, & Israel, 1994) and refers to the presence of two or more diagnoses, exclusively psychiatric or psychiatric and medical (Maj, 2005). The concept of comorbidity has gained increasing prominence in the psychiatric and psychological literature since the publication of the DSM-III (Andrews, Slade, & Issakidis, 2002; APA, 1980; Maj, 2005). Krueger & Markon (2006a) proposed a hierarchically organised liability-spectrum model of comorbidity, in which comorbidity is understood as a function of underlying vulnerability for psychopathology. This model offers an empirically based organisational structure that transcends putative distinctions between psychological disorders, suggesting that specific disorders are diverse expressions of underlying vulnerabilities. It further argues that psychopathology is dimensional in nature and structured hierarchically rather than as discrete disorders. Krueger & Markon's (2006a) model structure consists of the two broad spectra of internalising and externalising. It is supported by behaviour-genetic studies, which indicate a high degree of genetic risk associated with the aetiology of the liability spectrum (Krueger et al., 2005).

In contrast to comorbidity, the term co-occurrence holds no implications for relatedness (Kaplan et al., 2006). If two disorders co-occur, they are simply happening together, and may not be causally related. Co-occurrence is a purely temporal concept, and may reflect either an underlying causality or completely unrelated aetiologies. In contrast to medical diseases, which are

well-defined clinical entities whose aetiologies are often known, psychological disorders are psychological syndromes that deviate from some standard of normality (Angold, 1988). The term comorbidity presumes that co-occurring disorders are simultaneous, independent disorders when it may be the case that an individual is suffering from a single underlying condition that displays features of two arbitrarily defined and differentiated disorders (Bradshaw, 2001; Kaplan et al., 2006). Thus, the comorbidity found among disorders could be due to how the different symptoms are lumped together or split apart by the various classification systems used for diagnosis (Angold, Costello, & Erkanli, 1999; Bradshaw, 2001; Kaplan et al., 2006). In the current study, we do not make assumptions about the aetiology, distinctness or relatedness of co-occurring disorders; thus, we will use the term "co-occurrence" rather than comorbidity.

For all of the specific DSM PDs, co-occurring disorders on both EXT and INT spectra have been found. That is, both Cluster A PDs (e.g. Schizotypal PD [SPD]) and Cluster C PDs (e.g. Obsessive-Compulsive PD [OCPD] and Avoidant PD [AVPD]) with symptomatology on the internalising spectrum, and Cluster B PDs (e.g. Antisocial PD [ASPD] or Borderline PD [BPD]) with symptomatology on the externalising spectrum, have been found to co-occur with both externalising and internalising psychopathology.

6.2.1 Psychopathologies co-occurring with Cluster B PDs

Several community studies have found strong evidence of co-occurring INT disorders with ASPD in adults, e.g. mood and anxiety disorders (Coid & Ullrich, 2010; Goldstein et al., 2006; Howard et al., 2012). Goodwin & Hamilton (Goodwin & Hamilton, 2003) found significant associations for anxiety disorders (Social Phobia [SP] and Post-Traumatic Stress Disorder [PTSD]) and ASPD, but they ruled out depression as a co-occurring disorder of ASPD, which they argued was an artefact of other co-occurring disorders such as substance abuse and anxiety disorders. ASPD has also been found to be co-occurring with other EXT disorders, such as adult ADHD (Cumyn, French, & Hechtman, 2009; Miller, Nigg, & Faraone, 2007). Research about co-occurrence of ASPD and conduct disorder (CD) is mostly lacking, presumably because usually studies of adults have implemented the exclusionary rule in which CD is only diagnosed if ASPD is absent (Moffit et

al., 2008). However, one study showed that, in incarcerated youth, ASPD frequently co-occurred with CD, particularly in males (Eppright, Kashani, Robison, & Reid, 1993).

Similarly, adults with BPD have been found to have strong comorbidities with other EXT and INT psychopathology. For instance, one study found that in a community sample, in those with BPD, the rates of current substance abuse and mood and anxiety disorders exceeded 50% (Grant et al., 2008). Another study found significant associations between BPD and Generalised Anxiety Disorder (GAD), phobic disorders, depression, substance abuse/dependence and Obsessive-Compulsive Disorder (OCD) (Coid & Ullrich, 2009). BPD has also been found to be co-occurring with ADHD (Cumyn et al., 2009; Miller et al., 2007). In incarcerated youth, BPD frequently co-occurred with CD, particularly in females (Eppright et al., 1993).

6.2.2 Cluster A and C PDs

Schizotypal PD (SPD) has been associated with both co-occurring INT and EXT psychopathology. For instance, one study reported significant associations between SPD and bipolar disorder, social and specific phobias, posttraumatic stress disorder (PTSD), and GAD (Pulay et al., 2009). Another study found SPD to be associated with ADHD (Miller et al., 2008). However, the Collaborative Longitudinal Personality Disorders Study (CLPS) (Shea et al., 2004) did not support the notion of a strong association between SPD and any particular Axis I disorder.

Both Avoidant PD (AVPD) and Obsessive-Compulsive PD (OCPD) have mostly been associated with co-occurring internalising disorders. Avoidant PD has been most frequently evaluated in relation to social phobia (SP) due the overlap in symptomatology, in order to address whether the two disorders are the same or are alternative forms of the same domain. Studies using community samples found that approximately 32.5% to 39.5% of individuals with AVPD had co-occurring SP, whereas 18.3% to 36.4% of individuals with SP had co-occurring AVPD (Cox et al., 2009; Reichborn-Kjennerud et al., 2007). Similarly, both AVPD and OCPD have been found to be significantly associated with OCD and anxiety disorders, and OCPD has been additionally linked with anxiety disorders and anorexia nervosa (Coles, Pinto, Mancebo,

Rasmussen, & Eisen, 2008; Shea et al., 2004; Wentz, Gillberg, Anckarsäter, Gillberg, & Rastam, 2009). However, both PDs have also been found to be co-occurring with EXT disorders, such as adult ADHD (Miller et al., 2008; Miller et al., 2007).

6.3 Are the links between childhood disorders and adult PDs mediated by specific co-occurring mental health problems?

In sum, evidence has shown that 1. Childhood EXT/INT disorders are predictive of PD (See Chapters 2 and 4); 2. Childhood EXT/INT disorders are predictive of adult EXT/INT psychopathology other than PD, and 3. Adult PDs often co-occur with other EXT/INT adult psychopathologies. This poses the question of whether any links that can be found between childhood disorders and PD can be explained by continuities in psychopathologies, and co-occurrence of adult disorders rather than between childhood disorders and PDs as such. In other words: are the associations between childhood disorders and adult PD mediated by co-occurring psychopathology, and do continuities in symptoms account for the effects between childhood problems and PD? Whilst this question has not yet been directly investigated, there is some (limited) evidence that does suggest this may indeed be the case.

For example, one study investigated the rate of adult ASPD in a sample of children with ADHD, compared to non-ADHD-matched peers; the rate of development of ASPD was significantly more frequent in children with ADHD (37%) compared to controls (3%) (Gittelman et al., 1985; Mannuzza, Klein, et al., 1991). However, the higher rate of ASPD was completely accounted for by those participants who had retained ADHD into adolescence. In those children who had retained ADHD symptoms, the prevalence of co-occurring ASPD was 48%, as opposed to 17% in remitters who were not statistically different from controls (8%). The findings suggest that, rather than childhood ADHD as such, it may be the continuity of ADHD symptoms beyond childhood that explain the higher rate of adult ASPD. Similarly, a study by Harty et al. (2009) showed that early childhood ADHD initially predicted several dimensions of anger, as well as verbal aggression

in adolescence. However, when co-occurring ADHD symptom severity in adolescence was controlled for, most of these initial effects disappeared. The authors argue that these results indicate that elevations of these EXT problems in adolescence are best explained by the persistence of ADHD symptoms rather than childhood problems as such.

Another study which investigated prospective associations between childhood ADHD and specific PDs, showed that childhood ADHD was associated with ASPD, BPD, NPD and AVPD (Miller et al., 2008). However, when subgroups were created based on whether participants' ADHD had remitted or persisted, they found no significant differences between participants whose symptoms had remitted and control participants in terms of PD symptoms. PD symptoms of participants whose ADHD symptoms persisted into adulthood, on the other hand, strongly differed from controls in NPD, BPD, ASPD and AVPD symptoms. Again, these findings suggest that the associations between childhood ADHD and adult PD might not be due to childhood ADHD symptoms as such, but rather that these associations can be explained by the continuity of childhood symptoms into adulthood.

6.4 The current study – aims and hypotheses

Taken together, evidence suggests that associations between childhood disorders and PD might be explained by continuity of childhood symptomatology. However, to our knowledge, this has not yet been directly tested. Therefore, the aim of this study was to investigate whether any associations between childhood problems and adult PDs could be explained by continuities of those underlying problem dimensions. Specifically, we explored whether homotypic and/or heterotypic continuity of childhood problems into adolescence/adulthood mediated the association between these childhood problems and adult personality pathology.

> ***Previous studies in research areas other than PD have shown both homotypic and heterotypic continuities of childhood disorders into adulthood. Therefore, it was hypothesised that both homotypic and heterotypic associations between childhood problems and adult psychopathologies would be found, i.e. that both EXT and INT***

childhood problems would lead to both EXT and INT psychopathologies in adolescence/adulthood. Similarly, both INT and EXT psychopathologies in adolescence/adulthood were expected to co-occur with PDs on both EXT/INT spectra.

It was further predicted that adult EXT and/or INT psychopathology would mediate the associations between childhood problems and personality pathology. For example, based on previous evidence, it was expected that ADHD in adolescence would mediate the association between childhood hyperactivity and adult BPD, ASPD and AVPD.

6.5 Methods

6.5.1 Participants

For details about the procedure of selecting participants for this study, please see Chapter 3. An overview about sample characteristics at baseline is presented in Chapter 3.

6.5.2 Procedure

Please see Chapter 3 for details about the procedure.

6.5.3 Measures

For details about measures used in this study, please see Chapter 3. An overview about the assessments considered for current analyses is presented in Table 19.

Table 19: Outcome Variables, Mediators/Moderators and Covariates

	Construct	Assessment Tool
Childhood Predictors (IVs) - Behaviour Problems (age 3)	**Hyperactivity**	**WWP Activity Scale**
	Conduct Problems	**BCL Social Maladjustment**
Adult Outcomes (DVs) – Personality Pathology	**Specific PDs: Borderline, Antisocial, Avoidant**	**PID-5**
Mediators / moderators (assessed retrospectively at follow-up)	**Conduct Disorder** **Attention Deficit Hyperactivity Disorder** **Oppositional Defiant Disorder** **Major Depressive Episode** **Generalised Anxiety Disorder** **Social Phobia**	**CBRS-SR**
Covariates	**Age at follow-up** **Sex**	
	Socio-economic status	**Carstairs deprivation score**

IV – Independent variable, DV – dependent variable, WWP – Werry-Weiss-Peter-Activity-Rating-Scale, BCL – Behaviour Checklist; SES – Socioeconomic Status; PD – personality disorder; CBRS-SR – Conners Behavior Rating Scale – Self-Report

6.5.4 Mediation Analyses

For all mediation analyses, D.A. Kenny's (Baron & Kenny, 1986; Judd & Kenny, 1981; Kenny, 2014) approach to mediation was used. According to Kenny (2014), several steps must be met to establish mediation: (1) the predictor must be significantly associated with the outcome variable, (2) the predictor must be significantly associated with the mediator, and (3) the mediator must be significantly associated with the outcome variable. To establish that the mediator completely mediates the association between predictor and outcome, the effect of the predictor on the outcome controlling for the mediator should be zero. If the effect of the predictor on the outcome is not zero when controlling for the mediator, but all other steps are met, partial mediation is indicated.

As suggested by Kenny (2014), prior to all multivariate tests, initial bivariate correlations were carried out. For those variables where significant correlations were established, several sets of multiple regressions were subsequently carried out. (1) Multiple regression analysis entering the predictor variable simultaneously with covariates, to establish associations between predictor and mediator; and (2) multiple regression analysis including predictor, mediator and outcome simultaneously. Associations between predictor and the outcome variables, controlling for covariates, were already established in Chapter 4, so these associations were not investigated again. Only significant associations were further tested.

The amount of mediation is called the indirect effect (Kenny, 2014). The significance of any indirect effects was tested using bootstrapping procedures (Bollen & Stine, 1990; Shrout & Bolger, 2002) using the PROCESS macro for SPSS developed by Hayes (2013). Bootstrapping is a non-parametric method based on resampling with replacement; 10,000 bootstrapped samples are recommended. From each of these samples the indirect effect is computed and a sampling distribution can be empirically generated. Because the mean of the bootstrapped distribution will not exactly equal the indirect effect a correction for bias is usually made. With the distribution, a confidence interval can be computed and it is checked to determine if the interval includes zero. If zero is not in the interval, then it

can be inferred that the indirect effect is significant. In this study, unstandardised indirect effects were computed for each of 10,000 bootstrapped samples, and the 95% confidence interval was computed by determining the indirect effects at the 2.5th and 97.5th percentiles, as suggested by Hayes (2013).

6.6 Results

6.6.1 Preliminary analyses

Preliminary analyses were conducted prior to the testing of hypotheses (please see Chapter 4 for details). Two cases were identified as influential outliers. Analyses were conducted, dropping these cases to assess the degree to which the findings were influenced by their presence. No changes occurred in the pattern of significant effects. Because of the consistency in the results, the outliers were retained in all subsequent analyses.

Absolute values of kurtosis ranged from 1.103 (emotional problems) to 35.98 (Conduct Disorder) in predictor/mediator variables and from -2.797 (SPD) to 2.118 (ASPD) in outcome variables. Only Conduct Disorder represented a deviation from a normal distribution. Absolute values of skewness ranged from 3.36 (Social Phobia) to 17.31 (Conduct Disorder) in predictors/mediators, and from 3.108 (SPD) to 6.271 (ASPD) in outcome variables. All predictor and mediator variables were positively skewed. Whilst it has traditionally been suggested to perform transformations on skewed data, this method for handling non-normality of data has also been criticised (Osborne, 2002). Further, simulation research shows that only the most severe violations of the normality assumption affect the validity of statistical interferences from a regression analysis unless the sample size is quite small (Duncan & Layard, 1973; Edgell & Noon, 1984; Havlicek & Peterson, 1977). Therefore, all further analyses were performed using the original, un-transformed, dataset. Finally, multicollinearity among the predictors was assessed using the variance inflation factor (VIF) statistic. In this sample, the VIFs ranged from 1.25 to 1.73, all within acceptable ranges.

As described in detail in Chapter 4, all analyses were carried out twice: 1. with re-weighted cases, and 2. using the original unweighted data. As the

pattern of results was the same in both cases, only results from analyses carried out with original, unweighted data is presented here.

Table 20 presents means, standard deviations and score ranges for adult EXT and INT psychopathologies in this sample. For possible total score ranges, see Table 5 in Chapter 3. As mentioned above, all mediator variables were positively skewed, which was reflected in low mean values on all scales, especially CD. Table 21 presents intercorrelations between study variables.

Table 20: Externalising and internalising psychopathologies at follow-up, as assessed by CBRS-SR subscale scores

Psychopathology dimension	Mean (SD)
Conduct Disorder	1.64 (2.515)
Attention Deficit Hyperactivity Disorder	6.63 (5.336)
Oppositional Defiant Disorder	5.37 (3.928)
Major Depressive Episode	9.65 (8.121)
Generalised Anxiety Disorder	10.37 (8.189)
Social Phobia	4.71 (3.573)

Note: Possible score ranges of all subscales are presented in Table 5

Table 21: Intercorrelations between study variables

	HYP-3	*CP-3*	*ADHD*	*CD*	*ODD*	*MDE*	*GAD*	*SP*	*BPD*	*ASPD*	*SPD*
ADHD	.148*	.086									
CD	.019	.114	.470***								
ODD	.121	.205**	.726***	.608***							
MDE	.064	.088	.594***	.463***	.614***						
GAD	.046	.058	.584***	.392***	.569***	.886***					
Social Phobia	.045	.030	.313***	.306***	.371***	.603***	.680***				
Borderline PD	.255***	.286***	.554***	.455***	.618***	.685***	.680***	.556***			
Antisocial PD	.190**	.284***	.565***	.573***	.676***	.432***	.402***	.242***	.752***		
Schizotypal PD	.166*	.182**	.471***	.486***	.509***	.569***	.591***	.527***	.739***	.657***	
Avoidant PD	.197**	.178**	.397***	.361***	.478***	.655***	.703***	.667***	.742***	.465***	.795***

*Note: N=216; *p<.05; **p<.01; ***p<.001; HYP-3 – Hyperactivity assessed at age 3; CP-3 – Conduct problems assessed at age 3; ADHD – Attention Deficit Hyperactivity Disorder; CD – Conduct Disorder; ODD – Oppositional Defiant Disorder; MDE – Major Depressive Episode; GAD – Generalised Anxiety Disorder; SP – Social Phobia*

At follow-up, all EXT/INT psychopathologies and PDs were positively correlated. Childhood EXT/INT problems correlated with only two EXT/INT adult psychopathologies: hyperactivity correlated with ADHD, and conduct problems correlated with ODD at follow-up, no other correlations with EXT/INT psychopathologies at follow-up were detected.

6.6.2 Mediation analyses – co-occurring EXT/INT psychopathologies at follow-up as mediators of the association between child emotional and behavioural problems and specific PDs

Mediation analyses were only considered for significant associations between childhood predictors and outcome variables that were established in Chapter 4. Mediation analyses were carried out for those variables where significant initial associations were established between predictor and mediator, and mediator and outcome variables. Based on these criteria, the following two mediational models were tested: (1) Hyperactivity → ADHD → BPD; (2) Hyperactivity → ADHD → AVPD; (3) Conduct Problems → ODD → BPD; (4) Conduct Problems → ODD → ASPD.

Due to the close relationship between ADHD, ODD and CD, in all models tested, the effects of the respective other two disorders were controlled for. All significance levels were adjusted for multiple comparisons and set to α=0.03.

6.6.2.1 Hyperactivity

Please see Figures 8 and 9 for path diagrams illustrating the associations between hyperactivity, ADHD and BPD and AVPD.

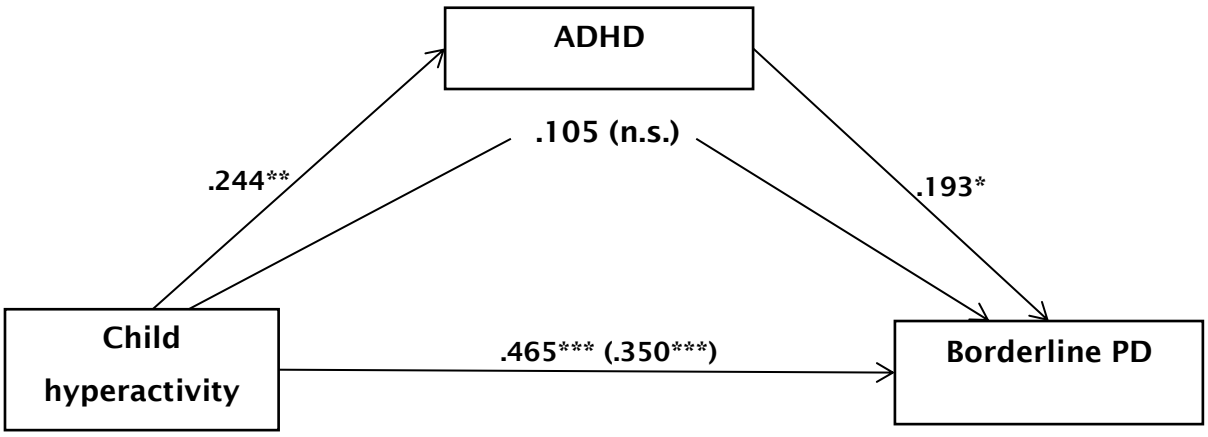


Figure 6: Path diagram illustrating associations between hyperactivity, ADHD and BPD

Figure 8 illustrates associations between hyperactivity, ADHD and Borderline PD. The coefficients in parentheses are standardised regression coefficients of the predictor when controlling for the mediator. As shown in Figure 8, child hyperactivity significantly predicted BPD both with and without controlling for the mediator. The amount of mediation was assessed by determining the indirect effect of the predictor on the outcome variable, via the mediator, using bootstrapping procedures. The bootstrapped unstandardized indirect effect of hyperactivity on BPD, mediated by ADHD was .105 (95% CI: -0.032 -0.503), so the indirect effect was not significant. This indicates that ADHD did not mediate the relationship between childhood hyperactivity and adult BPD.

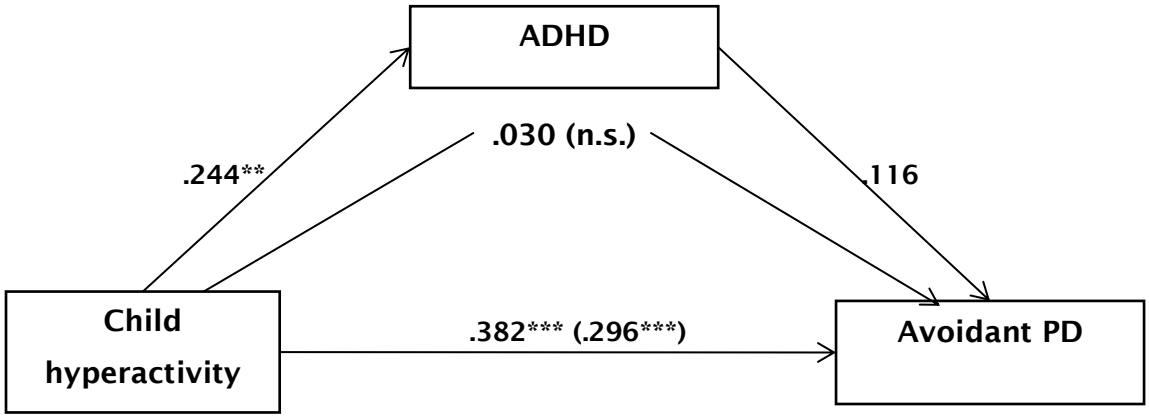


Figure 7: Path diagram illustrating associations between hyperactivity, ADHD, and Avoidant PD

Figure 9 illustrates associations between hyperactivity, ADHD and Avoidant PD. The coefficients in parentheses are standardised regression coefficients of the predictor when controlling for the mediator. As shown in Figure 9, child hyperactivity significantly predicted AVPD both with and without controlling for the mediator. The amount of mediation was assessed by determining the indirect effect of the predictor on the outcome variable, via the mediator, using bootstrapping procedures. The bootstrapped unstandardized indirect effect of hyperactivity on AVPD, mediated by ADHD, was .030 (95% CI: -0.028 - 0.205), so the indirect effect was not significant. Thus, ADHD did not mediate the relationship between childhood hyperactivity and adult AVPD.

6.6.2.2 Conduct Problems

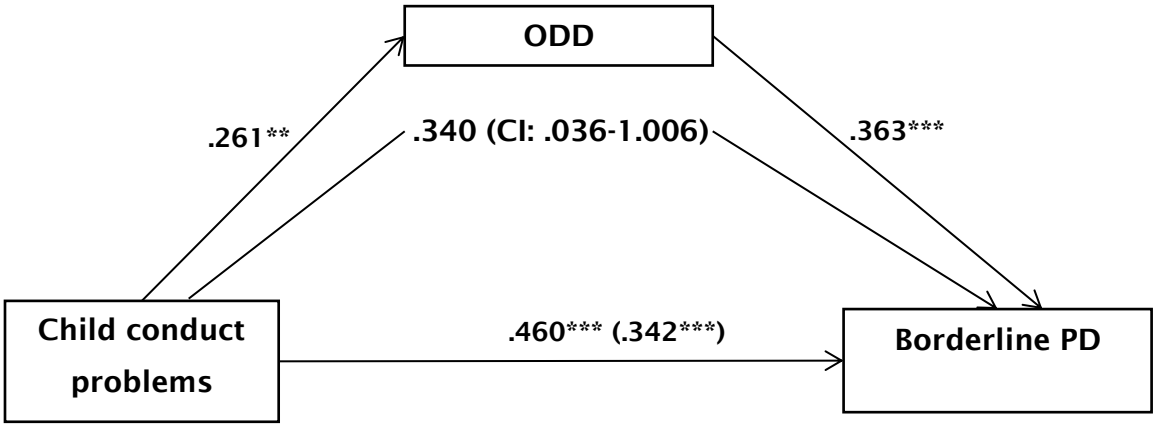


Figure 8: Path diagram illustrating associations between conduct problems, oppositional defiant disorder and BPD

Figure 10 illustrates associations between conduct problems, ODD and Borderline PD. The coefficients in parentheses are standardised regression coefficients of the predictor when controlling for the mediator. As shown in Figure 10, child conduct problems significantly predicted BPD both with and without controlling for the mediator. The amount of mediation was assessed by determining the indirect effect of the predictor on the outcome variable, via the mediator, using bootstrapping procedures. The bootstrapped unstandardized indirect effect of conduct problems on BPD, mediated by ODD, was 0.340 (95% CI: 0.036 – 1.006), so the indirect effect was statistically significant. This indicates that ODD partially mediated the relationship between childhood conduct problems and adult BPD symptoms.

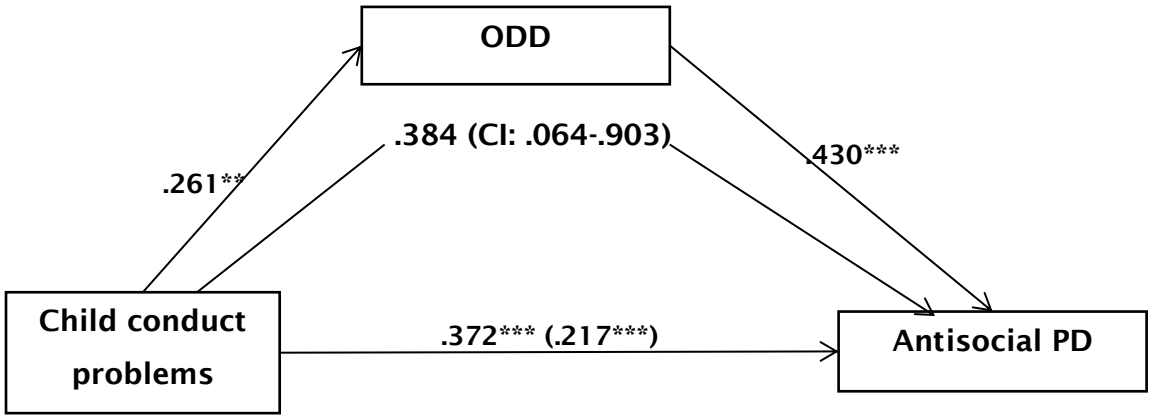


Figure 9: Path diagram illustrating associations between Conduct problems, Oppositional Defiant Disorder and Antisocial PD

Figure 11 illustrates associations between conduct problems, ODD and ASPD. The coefficients in parentheses are standardised regression coefficients of the predictor when controlling for the mediator. As shown in Figure 11, child conduct problems significantly predicted ASPD both with and without controlling for the mediator. The amount of mediation was assessed by determining the indirect effect of the predictor on the outcome variable, via the mediator, using bootstrapping procedures. The bootstrapped unstandardized indirect effect of conduct problems on ASPD, mediated by oppositional defiant disorder, was 0.384 (95% CI: 0.064 – 0.903), so the indirect effect was statistically significant. Thus, ODD partially mediated the relationship between conduct problems in childhood and ASPD symptoms in adulthood.

6.7 Discussion

The aim of this study was to investigate whether the associations between childhood EXT/INT problems and adult PDs could be explained by co-occurring adult psychopathology. Specifically, it was investigated whether homotypic and/or heterotypic continuity of childhood problems mediated the associations between childhood problems and adult PDs detected in Chapter 4.

Key findings were: (1) only homotypic continuities of childhood problems were found, that is, hyperactivity predicted adolescent/adult ADHD, and conduct problems predicted adolescent/adult ODD, but no other associations emerged. (2) In all models tested, childhood problems remained significant predictors of PDs while controlling for continuities of symptoms into adolescence/adulthood, i.e. childhood hyperactivity directly predicted BPD and AVPD, and conduct problems directly predicted ASPD even when controlling for the effects of ADHD or ODD in adolescence/adulthood; (3) none of the indirect effects via adolescent ADHD were significant, so the effects of childhood hyperactivity on PD could not be explained by the continuity of symptoms into adolescence/adulthood. (4) ODD partially mediated the association between conduct problems and BPD and ASPD. In other words, childhood conduct problems directly predicted BPD and ASPD, controlling for continuity of symptoms, but significant indirect effects of conduct problems on these PDs via ODD were also found, suggesting different pathways to PD. These findings further highlight the robustness and consistency of the associations found between childhood problems and adult PD, as none of the associations were affected by the continuation of symptoms into adulthood. The findings will be discussed in detail below.

6.7.1 Homotypic continuities of childhood problems into adolescence/adulthood

It was hypothesised that both homotypic and heterotypic associations between childhood problems and adult psychopathologies would be found, i.e. that both EXT and INT childhood problems would lead to both EXT and INT psychopathologies in adolescence/adulthood. Against expectations, only homotypic continuities were found. Specifically, conduct problems predicted later ODD, and hyperactivity predicted later ADHD. The results are in line with previous research demonstrating that ADHD shows long-term continuity into adolescence and adulthood (Biederman et al., 2006; Mannuzza et al., 1993, 1998). They are also in line with evidence about continuity of childhood conduct problems, with ODD, CD and ASPD being viewed hierarchically, reflecting age-dependent expressions of the same underlying disorder (Moffit et al., 2008), where typically ODD symptoms

appear first, and then, in a subgroup of children with ODD, CD symptoms develop (Lahey et al., 1997; Loeber et al., 1995).

Interestingly, in our sample, childhood conduct problems did not directly predict CD in adolescence/adulthood, but ODD predicted CD, and both ODD and CD predicted ASPD. This might indirectly support the hierarchical view of ODD, CD and ASPD, where CD only develops in a subgroup of those with ODD symptoms. However, this possibility could not be directly tested because ODD, CD and ASPD were all assessed at the same time, in adolescence/adulthood; therefore, causality could not be established. These findings of EXT continuities also partly support Krueger & Markon's model of "externalising" as a big vulnerability factor (Krueger et al., 2005). However, one would have also expected continuities with other EXT disorders in adolescence/adulthood. For example, one might have expected associations between childhood hyperactivity and not just ADHD, but also with ODD and CD, and between childhood conduct problems and ADHD and ODD, which was not the case.

In addition, no heterotypic associations were found, i.e. no links between childhood conduct problems or hyperactivity with later internalising psychopathology were detected. One might have expected heterotypic continuities of childhood hyperactivity and conduct problems, too – previous research has demonstrated associations between childhood ADHD and adult INT disorders (Barkley et al., 2004; Biederman et al., 2006; Pardini & Fite, 2010; Rasmussen & Gillberg, 2000), and childhood CD and INT disorders (Loeber et al., 2002). This finding may have been related to the bias detected in our sample – responders had significantly lower conduct problems and hyperactivity scores than non-responders at baseline, so it was expected that effects were likely to be underestimations of real-world effects. Thus, it is likely that only the most robust associations were detected in this study, and that less strong links between childhood and follow-up variables did not reach significant levels. Thus, further associations not detected in this study possibly exist between childhood and adult variables; these need to be clarified in future research, using larger, unbiased samples and a prospective research methodology.

Overall, it was striking that hardly any correlations between childhood problems and psychopathology in young adulthood were found. This was especially surprising given that robust and consistent associations between childhood problems and PDs were found, as demonstrated in Chapter 4. In fact, one would have expected stronger associations between childhood problems and (former) Axis I psychopathologies than between childhood problems and (former) Axis II psychopathologies (i.e. PDs). However, this was not the case in this sample – whilst strong associations were found with PDs, very few associations were detected with other psychopathologies.

Several explanations for this finding are possible: Firstly, childhood problems were rated by the parent/main caregiver of the child, whereas adult psychopathologies were self-ratings. It is likely that stronger associations would have been detected if parent ratings were used at follow up rather than self-ratings. Secondly, the findings may have been related to a lack of power due to the sample bias described above. That is, associations were likely to be underestimations of real world effects, and only the strongest associations were likely to be detected, whereas weaker associations may not have reached significant levels. In support of this argument is the finding that some associations were detected and that these associations were those showing direct homotypic continuity from childhood into adulthood (e.g. childhood hyperactivity and adult ADHD). It is possible that, due to a lack of power, weaker associations were not detected in this study. However, these two points would have also been relevant for PDs where several significant associations between childhood problems and adult psychopathology were found.

Thirdly, it is likely that the lack of significant associations was related to the assessment instrument used for adult psychopathology, i.e. the CBRS-SR. The CBRS-SR is a widely used instrument with good validity and reliability estimates. The scales were originally designed for 8-18 year-olds but a significant proportion of participants in this study were 19 years or older. Even though the scale was adapted for use with older participants and reliability values in this sample were good (see Chapter 3), the CBRS-SR was not normed for the age group of participants in this research. It is possible that, because of this, the scales did not properly capture the associated

disorders, which may be the reason for the lack of correlations between childhood predictors and adult EXT/INT disorders in this study. In support of this argument is the fact that all of the CBRS-SR subscales were highly skewed. On the one hand this may indicate that the scales were not appropriate for this older age group, and, in addition, highly skewed data may not be optimal for capturing correlations (Norris & Aroian, 2004).

6.7.2 Continuities of childhood hyperactivity or conduct problems do not mediate the associations with adult PDs

The hypothesis that adult EXT and/or INT psychopathology would mediate the associations between childhood problems and personality pathology was not supported by the results of this study. In all models tested, childhood problems remained significant predictors of PD while controlling for continuities of symptoms into adolescence/adulthood (ADHD and ODD), i.e. childhood hyperactivity and conduct problems directly predicted BPD, ASPD, SPD and AVPD, even when controlling for the effects of continuity of these symptoms into adolescence/adulthood. However, none of the indirect effects via adolescent ADHD were significant. In other words, the results of this study did not indicate that the effects of childhood hyperactivity on PD were due to homotypic continuity of these symptoms into adolescence/adulthood. This is in contrast to previous research showing that continuity of ADHD symptoms into adolescence explained the association of childhood ADHD with ASPD (Gittelman et al., 1985; Mannuzza, Gittelman, et al., 1991) and with ASPD, BPD, NPD and AVPD in adulthood (Miller et al., 2008). Instead, the results of this study indicate that childhood hyperactivity is directly related to BPD, ASPD, SPD and AVPD, irrespective of whether these symptoms continue into adolescence/adulthood or not. Alternatively, childhood hyperactivity might increase the risk for ASPD via some other variable not accounted for in the current study.

ODD, on the other hand, partially mediated the association between conduct problems and BPD, ASPD and AVPD. Regression coefficients of conduct problems were decreased (but still highly significant) when ODD was included, and ODD was also independently predictive of ASPD. In other words, childhood conduct problems directly predicted BPD, ASPD and AVPD, but significant indirect effects of conduct problems on these PDs, via ODD,

were also found. Thus, even though previous research has identified ODD as a predictor of PDs such as ASPD and BPD, in the current study, co-occurring ODD in adulthood only accounted for some but not all of the relationship between childhood conduct problems and ASPD, BPD and AVPD. This also suggests that these early conduct problems create a risk for later psychopathology (ODD) *as well as* PD, but these do not appear to be overlapping. This is especially surprising given the content overlap of ODD and BPD and ASPD, in particular.

The results were unlikely to be due to shared rater variance even though the ratings of EXT/INT psychopathologies and PDs potentially could have been affected because all these assessments were made by the young person at the same time. The most likely effect of the shared rater variance was expected to be an overestimation of associations of PD with co-occurring psychopathologies. However, this turned out not to be a very pronounced effect. ADHD was only very weakly associated with PDs in adulthood. ODD was strongly associated with BPD and ASPD, but this association did not mediate the association between childhood CP and PD. Thus, the results were unlikely to be due to shared rater variance.

The findings of this study add further support to our initial finding that childhood hyperactivity and conduct problems are very strong predictors of adult PD. In contrast to previous research showing that continuity of symptoms into adolescence explained the association of childhood disorders with PD (Gittelman et al., 1985; Mannuzza, Gittelman, et al., 1991) the associations we detected in Chapter 4 were not affected by continuities of childhood symptoms into adulthood, that is, these childhood problems predicted adult PD regardless of whether these symptoms continued into adulthood or not. Thus, our findings provide evidence for the consensus that PDs have their origins in childhood by showing that childhood problems are strongly and very robustly associated with adult PD.

6.7.3 Limitations

Several limitations need to be borne in mind when interpreting the results of the current study. Firstly, the current sample was likely to be subject to sample bias due to high attrition and comparisons of responders and non-

responders on all relevant baseline measures revealed systematic differences between the groups. In the current research, the systematic differences between responders and non-responders imply that the results in this research are likely to be an underestimation of "real world effects", i.e. the levels of problem severity and the size of associations between variables were likely to be lower than in more representative samples. Nevertheless, when analyses were carried out with re-weighted cases, where more weight was given to underrepresented cases and less weight was given to overrepresented cases, the pattern of results was the same. That is, even though specific values slightly changed, the overall pattern of significance and non-significance were similar, regardless of whether the analyses were carried out with the original data or with reweighted data. Nonetheless, it seems plausible to assume that the sample was biased in other aspects not included in the assessments of this study. Future research should focus on testing the effects of EXT and INT childhood problems on PD in larger, unbiased samples.

However, previous research has shown that sample biases may not necessarily decrease the validity of results. For instance, it has been found that differences in mean levels of variables between those who drop out and those who stay in a study do not necessarily imply that there are differences in *associations* between variables (Gustavson et al., 2012). In addition, evidence has suggested that systematic attrition of participants may not necessarily reduce the validity of prediction from longitudinal analysis (Wolke et al., 2009). Contrary to common assumptions, the presence of a substantial selection bias does not necessarily markedly attenuate the relationship between predictor and outcome variables. However, we cannot rule out the possibility that the sample was biased in other aspects not included in the assessments of this study, so this limitation due to sample bias should be borne in mind for the interpretation of the results.

Some issues regarding follow-up assessments made in this study should be mentioned. The main outcome measure for PD – the PID-5 (Krueger et al., 2012), a valid and now widely used instrument for personality pathology, was not designed to assess the specific DSM PDs. For the purpose of this study, specific subscale combinations were combined to produce

dimensional scores that give estimates for six of the specific DSM PDs. Whilst the scale has been shown to map well onto the specific PDs (Morey & Skodol, 2013), it was not originally designed and standardised for this purpose. In addition, assessment of PD was based solely on self-report measures. Making a clinical diagnosis of PD requires an in-depth clinical interview carried out by a trained professional. Self-report assessments of PD should ideally be corroborated through other-ratings (e.g. by the parent). Unfortunately neither of these options was available for the purposes of this study due to time and financial constraints. Therefore, the results need to be interpreted with caution and should not be regarded as an equivalent of clinical diagnoses of PD. In addition, the assessment instrument used for adult psychopathology (the CBRS-SR) was designed for use with adolescents, and a significant proportion of participants in this study were 19 years or older. The lack of continuity of childhood to adulthood psychopathology may have been related to the fact that it was not normed for the age group of participants in this research. It is possible that more significant associations would have been found if an age-appropriate instrument for use with adults would have been used. Future research should clarify this issue by assessing adult psychopathology as a mediator between childhood problems and adult PD longitudinally, using an age-appropriate assessment instrument.

In addition, the problem of potential item overlap needs to be borne in mind. In the current study, item overlap was expected due to substantial overlap between BPD and ODD. An additional issue related to follow-up assessments was that they were all made at the same point in time. Conceptually, psychopathology was regarded as occurring "before" PD, i.e. as having a causal influence on the development of PD. However, because they were assessed at the same time as PD, the temporal and relationship between these variables is not clear, and should be interpreted with caution.

6.7.4 Conclusion

The current study explored whether the associations between childhood problems and PD that were detected in Chapter 4 could be explained by continuities in symptomatologies into adulthood, i.e. whether associations

with PD were mediated by concurrent EXT/INT psychopathologies. Only homotypic continuities were detected - hyperactivity predicted adult ADHD, and conduct problems predicted adult ODD. ADHD did not mediate the associations between childhood hyperactivity and BPD, ASPD, SPD or AVPD, and ODD only partially mediated the associations between childhood conduct problems and PDs. The findings suggest that the associations between childhood hyperactivity/conduct problems and PD was unrelated to the continuities of symptoms into adulthood, i.e. these childhood problems predicted PD regardless of whether these problems continued into adulthood or not, suggesting separate pathways to PD. The findings were discussed in light of several methodological limitations.

Chapter 7: General Discussion

7.1 Introduction

Despite a consensus that personality disorders (PDs) have their origins in childhood (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Bromley, et al., 2006; Johnson, First, et al., 2005; Kernberg et al., 2000; Mervielde et al., 2005; Shiner, 2007; Westen & Chang, 2000) few prospective longitudinal studies have focused on the development of PD as a result of childhood problems (Shiner, 2009; Widiger & Trull, 2007). Theoretical models argue for an interaction between individual vulnerabilities and environmental risk factors such as negative parenting as the pathway to PD (Linehan, 1993). However, these models have hardly been empirically tested. Those studies that do exist indicate that PDs can indeed be predicted on the basis of childhood problems and that environmental stressors such as negative parenting do increase the risk for a PD in adulthood, but a lot of these studies are methodologically flawed. For example, studies often include a wide age range at baseline, fail to account for the effects of other, comorbid childhood disorders, and they do not assess for additive or interactive effects of childhood disorders.

The current research used a prospective longitudinal study design with the following research aims: (1) To investigate whether common externalising (EXT) and internalising (INT) childhood problems predict personality pathology in adulthood; (2) To assess whether any unique associations between specific childhood EXT/INT problems and specific PDs exist; (3) To explore whether any combinations of significant childhood predictors show additive and/or interactive effects in the prediction of PD; (4) To investigate whether negative parenting by both the mother and the father affect the development of personality pathology, and whether the effects differ for mothers and fathers; (5) To test whether the effects of negative parenting by the mother and/or father add to, moderate or mediate the effects of child problems in the prediction of PD; (6) To investigate whether any associations between childhood problems and PD can be explained by a continuation of childhood symptoms into adulthood (i.e. whether the

associations between childhood problems and adult PD are mediated by adult co-occurring psychopathology).

7.2 Summary of key research findings

The following key findings emerged: (1) The results in Chapter 4 showed that PDs can indeed be predicted on the basis of common childhood problems. EXT problems (hyperactivity, conduct problems) but not INT problems (emotional problems, shyness) predicted personality pathology in adulthood. These associations remained significant when the effects of negative parenting were added to the models (Chapter 5), and they were not explainable by the continuation of symptoms into adulthood (Chapter 6). These findings are striking considering that these children were only preschoolers at baseline, that the time span until follow-up was 15-20 years, and considering the many methodological challenges encountered throughout this research. Obtaining such robust and consistent findings *despite* all these challenges is remarkable and highlights not only the strength of these associations, but also the importance of longitudinal studies which are mostly lacking in the area of PD. (2) The results in Chapter 5 showed that negative parenting by both the mother and the father significantly predicted adult PD. Paternal indifference was the strongest predictor, which was associated with Borderline PD (BPD) and Schizotypal PD (SPD), while maternal overcontrol was associated with Antisocial PD (ASPD). These significant associations added to all effects of childhood problems identified as predictors of PD in Chapter 4. In contrast to the current consensus, however, negative parenting did not interact with child predictors, indicating that these negative parenting variables increased the risk for PD regardless of child characteristics, suggesting different pathways to PD. (3) The results in Chapter 6 showed that co-occurring psychopathology in adulthood did not account for the associations between childhood problems and adult PD. Thus, these associations were not due to the continuation of symptoms into adulthood. Overall, these results of this research show strong and robust associations between early childhood EXT problems and adult PD (Chapter 5), irrespective of exposure to negative parenting (Chapter 6) and adult psychopathology (Chapter 7) despite the many methodological challenges

encountered carrying out this research, highlighting the strength of these associations. In addition, contrary to current opinion, this research does not support interactive models of PD, where personality pathology develops through a process of interaction or transaction of the child with his/her environment (Chapter 6). Instead, the results suggest that PD may develop through different pathways, supporting the notion of equifinality in the development of PD.

7.3 Implication of findings

The results of this thesis raise a number of important issues. These issues will now be addressed by answering a number of key questions.

7.3.1 Do common externalising and internalising childhood problems predict personality pathology in adulthood?

Our results indicate that indeed PD in early adulthood can be predicted on the basis of common childhood problems (Chapter 4), even as early as age three. Large proportions of previous studies that investigated these issues were often based on retrospective data and/or contained methodological flaws, which the current research addressed. Whereas most previous longitudinal studies included a sample with a wide age range at baseline, all children in our sample were assessed for childhood problems around their third birthday, so the results were not confounded by variations of baseline symptoms due to a wide age range. In addition, almost all previous studies failed to account for the effects of other co-occurring childhood problems when investigating the predictive effects of childhood problems on PD. The results of this research addressed these methodological issues. The results strongly support the consensus that personality pathology may originate in childhood, and that PDs can be predicted on the basis of common childhood behavioural problems (Bleiberg, 2001; Cohen & Crawford, 2005; Geiger & Crick, 2001; Johnson, Bromley, et al., 2006; Johnson, First, et al., 2005; Kernberg et al., 2000; Mervielde et al., 2005; Shiner, 2007; Westen & Chang, 2000): strong and robust associations between childhood EXT problems and PDs were found, which remained strongly predictive of personality

pathology even when the effects of negative parenting (Chapter 5) and continuity of childhood symptoms (Chapter 6) were controlled for.

This research addressed many methodological flaws of previous studies; however, it was also affected by its own numerous methodological challenges (see section 7.5). For example, one of the two strongest predictors (conduct problems) was assessed using a scale which consisted of only 5 items, and yet it emerged as one of the most useful indicators for later psychopathology. In addition, the sample was significantly biased on two of the main predictor scales, and was subject to a significant amount of attrition. Obtaining such robust and consistent findings *despite* all these challenges is remarkable and highlights not only the strength of the associations under investigation, but once again shows how crucial the conductance of longitudinal studies is. This research shows that adult PD, as is the common consensus, can be predicted on the basis of childhood problems – as early as preschool age – but longitudinal research in this area is mostly lacking. Evidence suggests that, the earlier psychopathological problems can be detected, the more they may be subject to successful intervention. Longitudinal research is especially needed in the area of PD, a field where research into childhood predictors is mostly lacking (see Chapter 2). Future studies urgently need to address this.

There is a common consensus among clinicians and researchers in the field that theoretically grounds the development of PD in interactional/transactional models, assuming that PDs develop through an interaction/transaction of individual child characteristics (vulnerabilities) with environmental influences (stressors). Interactional models commonly assume that a negative outcome is much increased if a child with certain vulnerabilities is exposed to environmental stressors. The current findings are not in line with this; rather, they indicate that certain child characteristics – in this case, conduct problems and hyperactivity – are predictive of PD *regardless* of environmental influences, once again highlighting the robustness of these associations. It should be borne in mind, however, that even at age three children will already have been influenced by their parents' behaviours (or other environmental influences). The results, thus, do not imply that children who show certain characteristics early in life are "destined" for a negative outcome and

cannot be influenced by environmental factors. However, the results do indicate that if certain externalising behavioural problems such as hyperactivity and conduct problems are apparent in early childhood – as early as preschool age – these problems are strong indicators that the child may be at a very high risk of developing a personality disorder later in life. The clinical implications of this finding will be discussed below.

7.3.2 Are there any unique associations between specific childhood EXT/INT problems and specific PDs?

The results of our meta-analysis in Chapter 2 showed that the combined findings of all published longitudinal studies were rather unspecific both in relation to predictors and to outcome variables. That is, no unique associations between specific childhood problems and specific PDs were detected. For instance, all childhood EXT problems were predictive of all Cluster B PDs, in line with the view that "externalising", as a broad, higher-order psychopathology factor, underpins the most commonly occurring EXT mental disorders and accounts for the covariance among childhood and adult EXT disorders (Krueger, 2002a; Krueger et al., 2001). This finding was probably related to the methodological flaw in almost all studies that the effects of other, co-occurring childhood disorders were not controlled for. Establishing distinct associations between specific childhood problem patterns and specific adult PDs would be most valuable for the development of targeted intervention or prevention approaches.

The current research did indeed reveal three distinct patterns between childhood problems and PDs, i.e. specific patterns of childhood problems were predictive of specific PDs when controlling for the effects of all other childhood problems. (1) Childhood conduct problems predicted ASPD, confirming previous findings of a robust association between conduct problems in childhood and subsequent ASPD in adulthood (Copeland et al., 2009; Lahey et al., 2005; Robins, 1966, 1978) and showing that the effects of conduct problems on ASPD were not due to overlap with other co-occurring childhood problems. Hyperactivity was not predictive of ASPD when controlling for other childhood problems, adding support to the argument that associations between childhood ADHD and adult ASPD may be due to overlap with CD. (2) Unexpectedly, hyperactivity predicted AVPD. Homotypic

continuities tend to show stronger effects than heterotypic continuities; thus, AVPD, which is on the internalising spectrum, would have been expected to show stronger associations with internalising childhood problems. This finding was speculated to be linked to social problems, which are often prominent in both AVPD and ADHD (Kessler et al., 2006), and the association between childhood hyperactivity and early adult AVPD was hypothesised to be linked to the social impairments in those with ADHD symptoms. This theory was not supported by findings in Chapter 6, where results indicated that childhood hyperactivity was not linked to social phobia. However, social difficulties and anxieties may present in other, perhaps less extreme forms than social phobia which were not assessed as part of this PhD. In addition, these associations may be more subtle and may not have been detected in the current sample which was biased due to high attrition, and with the assessment instruments used, and should therefore be further clarified. (3) Both hyperactivity and conduct problems in childhood were independently linked to early adult BPD, with slightly stronger effects for conduct problems than for hyperactivity. Whilst existing research had linked several EXT childhood problems to the development of BPD, no specific associations had been detected. This finding may be related to the two core features of BPD, i.e. impulsiveness and emotional instability. Perhaps childhood hyperactivity and conduct problems are differentially associated with these two aspects of BPD – hyperactivity may have affected the impulsivity feature, whereas conduct problems may have affected the emotional instability aspect. Previous research has argued that the difficulties with emotion regulation and relationships may precede problems with impulse control (Stepp, Burke, et al., 2012). This was not supported by the results of this study - both impulse control and conduct problems were independently predictive of BPD at a very young age. However, determination of the different subtypes of BPD, or dimensional assessments of these two features was not possible with the instrument applied in this study. Therefore, these aspects are hypothetical and need to be clarified in future research.

In sum, distinct patterns between specific childhood problems and specific PDs were found. However, it needs to be borne in mind that our findings may have been influenced by methodological issues in this study, in

particular high attrition rates and systematic differences between responders and non-responders in our sample. Due to decreased power, only the strongest associations were detected in our study, whereas weaker links did not reach significance levels. Nevertheless, finding these associations despite the methodological shortcomings is remarkable, arguing for the robustness and strength of these associations and highlighting that further research in this area is crucial.

7.3.3 Do combinations of childhood problems show additive and/or interactive effects in the prediction of personality pathology?

In the area of PD, research about additive or interaction effects of childhood predictors is mostly lacking; however, in other areas evidence has shown that children with comorbid disorders, e.g. ADHD and CD, are at a higher risk of a negative outcome than children with a single disorder (Colder et al., 2002; Loeber et al., 1990; Moffitt, 1990; Molina et al., 1999). In the area of PD, the results of one study (Sourander et al., 2005) showed that the combined effects of different childhood problems on ASPD were not simply additive, but rather, the joint effects of comorbid problems were stronger than the effects of single disorders, arguing for interactive effects. Identifying additive and/or interactive effects are of high clinical importance: if the risk for a negative outcome does indeed amplify through a combination of different co-occurring childhood disorders, then detection of specific risk patterns (i.e. specific additive/interactive effects of the most common childhood problems) would enable identification of those children who are most at risk of a negative outcome and enable targeted treatment approaches.

However, unexpectedly, we found almost no childhood problems which showed additive or interaction effects in the prediction of personality pathology. Only one additive effect was detected (the effects of conduct problems and hyperactivity were additive in the prediction of BPD) and no interactive effects were found. These results were surprising given that research has more or less consistently shown that children with co-occurring disorders are at a higher risk of a negative outcome than children with a single disorder (Loeber et al., 1990; McBurnett, 1992; Moffitt, 1990; Molina et al., 1999; Park et al., 1997; Rothbart & Mauro, 1990; Sourander et

al., 2005). Indeed, several authors have even argued that children with co-occurring disorders such as ADHD and CD may represent different subgroups with distinct developmental trajectories and poorer prognoses than children with either disorder alone (Hinshaw, 1987; Lilienfeld & Waldman, 1990; Lynam, 1996; Moffitt, 1990). Specifically, ADHD has been argued to set the stage for later CD, laying a pathway toward greatest risk for adverse outcomes (Molina et al., 1999). This, too, was not the case in this research: as demonstrated in Chapter 6, childhood hyperactivity was only predictive of later ADHD, but not CD, thus showing purely homotypic continuity of symptoms into adulthood. In addition, neither ADHD nor ODD at follow-up mediated the associations between childhood problems and PDs.

Instead, our results suggest that specific childhood problems are uniquely predictive of specific PDs in adulthood. However, these findings could have been due to the sample bias described above: because non-responders had significantly higher conduct problems and hyperactivity scores at baseline, results were likely to be an underestimation of real world effects. Thus, most probably only the strongest associations were detected in this study, whereas less strong relationships may not have reached significant levels. The results did show trends towards additive and interaction effects, but these were not significant after controlling for multiple testing. Future research needs to clarify this with a larger unbiased sample.

7.3.4 Does negative parenting affect the development of personality pathology?

Overall, the results of this research support the consensus among researchers and clinicians that parenting is a strong factor in the development of PD (Fonagy & Luyten, 2009; Fruzzetti et al., 2005; Johnson, Cohen, et al., 2006; Johnson, Cohen, Kasen, et al., 2001; Linehan, 1993): negative parenting overall strongly increased the risk of developing PD in adulthood. However, our findings were not in line with the common assumption that maternal negative parenting affects the development of the child more strongly than paternal negative parenting (Enns et al., 2002; Kimbrel et al., 2012). Father indifference predicted BPD, and mother overcontrol predicted ASPD. Previous longitudinal research has

demonstrated that a combination of high parental overcontrol and low parental warmth increases the risk for PD (Bogaerts et al., 2005; Byrne et al., 1990; Paris & Frank, 1989; Schuppert et al., 2012; Stravynski et al., 1989; Torgersen & Alnaes, 1992; Zweig-Frank & Paris, 1991). However, our results were more specific than previous studies by showing differential effects of maternal and paternal negative parenting which may be related to the different roles mothers and fathers play in the upbringing of their child/-ren (Lamb, 1981). These findings highlight the importance of father involvement in a child's life, and the detrimental effects of perceived non-involvement or absence of the father may have in terms of a child's development. As such, the results have strong implications in terms of early intervention/prevention strategies for PD, namely to make efforts to increase a father's involvement in the child's upbringing. The results of this research strongly suggest that increasing father involvement may decrease the risk for a negative outcome.

7.3.5 Do the effects of negative parenting add to or moderate the effects of child problems in the prediction of personality pathology?

We found several additive, but no interactive effects of negative parenting in the prediction of PD. Most theoretical models about the pathways to PD are interactional models based on the notion that individual vulnerabilities and environmental risk factors interact throughout life to influence a child's development (Linehan, 1993), emphasising that pre-existing vulnerabilities, in combination with external stressors (Fruzzetti et al., 2005), lead to a negative outcome. These models also propose that, although both negative parenting and difficult child behaviours are expected to directly predict children's development, for some children the effect of negative parenting will be exacerbated, whereas for others negative parenting will have less of an effect on the child (Lengua et al., 2000). Our findings do not support these models; rather, they suggest different, independent pathways to PD, indicating that individual vulnerabilities (in this case, conduct problems and hyperactivity) and stressors (maternal overcontrol and father indifference) both increase the risk for PD, irrespective of the presence of the other factor. As such, the findings support the notion of equifinality in the

development of PD, i.e. the notion of diverse pathways to PD (Cicchetti & Crick, 2009).

7.3.6 Does continuity of childhood problems into adolescence/adulthood mediate the association between these childhood problems and adult personality pathology?

Previous research has suggested the possibility that the effects of childhood problems on adult outcomes might be explained by continuity of these childhood symptoms into adulthood (Gittelman et al., 1985; Mannuzza, Gittelman, et al., 1991). The results of this research were not in support of this: our findings did not indicate that the effects of childhood hyperactivity on BPD and AVPD were due to continuity of childhood symptoms into adolescence/adulthood. Instead, the results of this study showed that childhood hyperactivity was directly related to BPD and AVPD, irrespective of whether these symptoms continued into adolescence/adulthood or not. Alternatively, childhood hyperactivity might increase the risk for ASPD via some other variable not accounted for in the current study. Adult ODD, on the other hand, partially mediated the association between conduct problems and BPD and ASPD. In other words, childhood conduct problems directly predicted BPD and ASPD, but significant indirect effects of conduct problems on these PDs, via ODD, were also found. Thus, co-occurring ODD in adulthood only accounted for some but not all of the relationship between childhood conduct problems and ASPD/BPD. This also suggests that these early conduct problems create a risk for later psychopathology (ODD) *as well as* PD, but these do not appear to be overlapping. This is especially surprising given the content overlap of ODD and ASPD, in particular. These results were unlikely to be due to shared rater variance. These results add further weight to the robustness of the association between childhood problems and adult PD, which were unaffected by the effects of negative parenting and the effects of continuity of symptoms.

7.4 Clinical Implications

Finding such strong long-term effects of early childhood behaviour problems has implications in terms of early intervention/prevention strategies. On the one hand these findings demonstrate how strikingly early in life the course for a negative outcome may already be set. On the other hand, findings such as these can be regarded as an opportunity; identification of such strong early predictors in children is useful for two reasons: Firstly, early predictors enable identification of those children who may be most at risk of a negative outcome, and an implication about what form this negative outcome may take; and secondly, the risk markers themselves could become target of interventions. Distinct patterns can enhance insight into the risk for a particular outcome and may enable the development of intervention programmes that can be specifically tailored towards the particular needs of a family. One of the most common interventions is parent training (Brestan & Eyberg, 1998; Lundahl et al., 2006; McCart et al., 2006) which is usually based on social learning theory and attachment theory, teaching parents appropriate skills and strategies to strengthen the relationship between parent and child and to improve the child's behaviour. The effectiveness of parenting programmes has been investigated in several reviews, indicating that indeed they can improve children's behavioural problems (Linehan, 1993; McCart et al., 2006).

Our findings highlight that targeting both child symptoms and parenting behaviour may decrease a child's risk of a negative outcome in adulthood. Because the effects appear to be relatively independent of each other, targeting both child symptoms directly and indirectly through parent behaviour might be beneficial. Our findings also highlight the importance of father involvement in a child's life, and the detrimental effects non-involvement or absence of the father may have in terms of a child's healthy development. As such, the results have strong implications in terms of early intervention/prevention strategies for PD, namely to make efforts to increase father involvement in children's upbringing. There is some evidence that programmes increasing father involvement strengthen families and improve father-child interactions (Lundahl et al., 2007; Magill-Evans et al., 2006), but reviews indicate that only 20% of such programmes

include fathers (Coplin & Houts, 1991; O'Brien & Budd, 1982). Our findings strongly suggest that early intervention/prevention approaches for PD should focus on involving father in the upbringing of their children, and involve them in intervention strategies wherever possible.

7.5 Limitations

The findings of this research need to be interpreted in light of several limitations.

7.5.1 Sample bias

Several limitations need to be borne in mind when interpreting the results of the current study. Firstly, the current sample was likely to be subject to sample bias due to high attrition: around half of all approached families did not respond to study invitations. It should be borne in mind, however, that the sampling strategy was not to follow up as many of the original families as possible (in which case the low response rate would have posed a more serious threat to the representativeness of the sample), but the aim was to recruit families with specific, predetermined criteria until the target number for each group was met. Even though it may have taken longer, and more families needed to be approached to reach the target number of families, the groups as such were most likely not affected by this.

Secondly, comparisons of responders and non-responders on all relevant baseline measures revealed systematic differences between the groups on hyperactivity and conduct problems: non-responders had lower hyperactivity and conduct problem scores. As summarised in Chapter 3, sample biases can pose threats to validity of a study - in the current research, the systematic differences between responders and non-responders imply that the group of families that consented to take part were more “healthy” at baseline than those who did not take part. As such, the results based on this sample were likely to be an underestimation of real world effects; that is, effects may have been less pronounced due to lower variability of scores and decrease in power, i.e. the levels of problem severity and the size of associations between variables may have been lower than in more representative samples. Thus, the most likely threat to

validity in this research was the possibility of making Type II errors, that is, not detecting meaningful associations that only showed trends towards significance due to these systematic differences. The effects that were detected in this study, however, are likely to be real effects.

As mentioned in Chapter 3, multiple imputations was deemed an unsuitable method to deal with this bias because the data was not missing at random. When analyses were carried out with re-weighted cases, where more weight was given to underrepresented cases and less weight was given to overrepresented cases, the pattern of results was the same. That is, even though specific values slightly changed, the overall pattern of significance and non-significance was similar, regardless of whether the analyses were carried out with the original data or with reweighted data. Nonetheless, it seems plausible to assume that the sample was biased in other aspects not included in the assessments of this study. Future research should focus on testing the effects of EXT and INT childhood problems on PD in larger, unbiased samples.

On the other hand, previous research has shown that sample biases may not necessarily decrease the validity of results. For instance, it has been found that differences in mean levels of variables between those who drop out and those who stay in a study do not necessarily imply that there are differences in *associations* between variables (Gustavson et al., 2012). For example, Gustavson et al. (2012) showed that estimates of associations were quite robust, even when selective attrition was substantial. The results of their simulation study showed that regression estimates were only minimally affected by attrition rate, with similar estimates at both lower and higher attrition rates. Of course, the proportion of samples that rejected the false null hypothesis of a zero association between the two study variables was higher with stronger population associations. In addition, estimates of associations between variables seemed to be generalizable. In their study, baseline variables (sociability and educational level) predicted attrition; however, the associations between these variables and mental health were the same among those who later dropped out and those who remained in the study. Of the 15 correlations between variables examined at baseline, none were significantly different for participants and nonparticipants at short-term or long-term follow-up. The authors suggest

that even if those who stay and those who drop out of a study differ regarding mean levels of some variables, estimates of associations can be robust to such differences.

In addition, evidence has suggested that systematic attrition of participants may not necessarily reduce the validity of prediction from longitudinal analysis (Wolke et al., 2009). Contrary to common assumptions (Hernan, Hernandez-Diaz, & Robins, 2004; Rothman & Greenland, 1998), the presence of a substantial selection bias does not necessarily markedly attenuate the relationship between predictor and outcome variables. Wolke et al. (2009) found that a follow-up sample that was biased according to a range of relevant predictor variables, did not invalidate the prediction of disruptive behaviour disorders. That is, the same predictors for disruptive behaviour problems (e.g. gender, maternal psychopathology, maternal smoking during pregnancy, low education, financial difficulties) were found for those who were still participating in the longitudinal study as well as for those who had dropped out. Thus, although prevalence rates do have an impact on statistical power, differences in prevalence per se may not alter associations.

However, we cannot rule out the possibility that the sample was biased in other aspects not included in the assessments of this study. It is likely that other factors at baseline were systematically different between responders and non-responders that were not assessed. In addition, it is possible that attrition was related to follow-up variables. Systematic differences in follow-up variables pose a more serious threat to validity: it implies that attrition is dependent on variables with missing data because the researcher generally only has information on follow-up variables from those who stayed in the study, rendering it impossible to control for these biases (Gustavson et al., 2012).

7.5.2 Baseline assessments

As summarised in Chapter 3, several issues arose because the study was based on an already existing dataset that was collected 15-20 years earlier. For instance, the choice of childhood predictors was limited to those assessments that had been carried out at baseline. Whilst a wide ranging

assessment was carried out at baseline for the three cohorts, only three scales were collected for all three cohorts, thus limiting available baseline predictors to these three scales. Whilst two of the three most common childhood externalising problems were covered by baseline assessments, i.e. conduct problems and hyperactivity, the third most common externalising problem (oppositional defiant behaviour), was not assessed, which would have enabled a more complete assessment of childhood externalising problems as predictors of PD. In addition, we had no influence on the choice of instruments that were used to assess these problems. The subscale assessing conduct problems, for instance, only consisted of five items. The assessments of internalising childhood problems were limited to shyness and emotional problems. Whilst both these subscales were clearly on the internalising spectrum, reliability values for one of them (emotional problems) was poor (Cronbach's alpha = .45), and they may not have been the best indicators for internalising problems as compared to other assessments. Both in older and younger children, the most common indicators for internalising problems are anxiety disorders (e.g. generalised anxiety disorder) and mood disorders (e.g. depressive symptoms). However, as internalising problems were not the focus of interest in the original baseline studies, these were not assessed. In addition, ratings were made by the parent only; ideally assessments made by trained professionals should be used, or parent ratings should be corroborated through other-ratings (e.g. by a preschool teacher). We did not detect any significant relationships between childhood INT problems and adult PD, which may have been related to the relative weakness of these INT childhood assessments.

Another issue was related to accuracy of the dataset. The original dataset was already entered by the research team at baseline and when making random accuracy checks it emerged that a large proportion of it was entered incorrectly. For a large proportion of participants, original paper versions of the data were available and could be re-entered if necessary, but these were not available for all participants. This meant that a large proportion of participants had to be excluded from follow-up because accuracy of baseline scores could not be verified.

The conditions under which the original data was collected are unknown, including the instructions that were given to participants about how to complete the questionnaires. There was some evidence that this may not have been optimal as some of the questionnaires were completed wrongly. In addition, data was collected by varying health visitors, and data was entered by various people in the team, and there was some evidence that they did not all adhere to the same instructions and/or scoring systems, resulting in a further proportion of cases having to be excluded.

7.5.3 Follow-up assessments

Some issues regarding follow-up assessments made in this study should be mentioned. The main outcome measure for PD – the PID-5 (Krueger et al., 2012), a valid and now widely used instrument for personality pathology, was not designed to assess the specific DSM PDs. For the purpose of this study, specific subscale combinations were combined to produce dimensional scores that give estimates for six of the specific DSM PDs. Whilst the scale has been shown to map well onto the specific PDs (Morey & Skodol, 2013), it was not originally designed and standardised for this purpose. In addition, assessment of PD was based solely on self-report measures. Making a clinical diagnosis of PD requires an in-depth clinical interview carried out by a trained professional. Self-report assessments of PD should ideally be corroborated through other-ratings (e.g. by the parent). Unfortunately neither of these options was available for the purposes of this study due to time and financial constraints. Therefore, the results need to be interpreted with caution and should not be regarded as an equivalent of clinical diagnoses of PD. In addition, it is possible that the lack of significant associations between childhood problems and adult psychopathology was related to the assessment instrument used for adult psychopathology, i.e. the CBRS-SR. The CBRS-SR is a widely used instrument with good validity and reliability estimates. However, the appropriate age group for the CBRS-SR is 8-18 years, and a significant proportion of participants in this study were 19 years or older. It is possible that, even though the scale was adapted for use with older participants, and reliability values in this sample were good, the lack of continuity of childhood to adulthood psychopathology may have been related to the fact that it was

not normed for the age group of participants in this research. In support of this argument is the fact that all of the CBRS-SR subscales were highly skewed. On the one hand this may indicate that the scales were not appropriate for this older age group, and, in addition, highly skewed data may not be optimal for capturing correlations (Norris & Aroian, 2004). It is possible that more significant associations would have been found if an age-appropriate instrument for use with adults would have been used. Future research should clarify this issue by assessing adult psychopathology as a mediator between childhood problems and adult PD longitudinally, using an age-appropriate assessment instrument.

Moreover, several issues in relation to the assessments of negative parenting should be mentioned. Assessments were made retrospectively. Retrospective assessments are of course subject to recall bias (Mannuzza et al., 2002; Maughan & Rutter, 1997). In addition, this study only focused on negative parenting. The current study implied that parenting had no effect on the association between childhood EXT problems and PDs: childhood hyperactivity and conduct problems predicted PDs regardless of negative parenting. However, positive parenting can serve as a strong protective factor, and may have attenuated the effects of child behaviour, which could not be tested in the current research. Assessing these effects would have provided a more complete picture of the associations between childhood problems, parenting and adult PD.

An additional issue related to follow-up assessments was that they were all made at the same time. Conceptually, parenting and psychopathology were regarded as occurring "before" PD, i.e. as having a causal influence on the development of PD. However, because they were assessed at the same time as PD, the temporal relationship between these variables is not clear, and should be interpreted with caution. In order to test developmental pathways of a disorder, several follow-up points are required, ideally testing the same parameters in regular intervals over time. We only had one time of baseline assessment and one time of follow-up, with a long follow-up period in between, so inferences that can be made about the pathways to PD are relatively limited.

7.6 Future Directions

The prospective longitudinal research carried out for this thesis was subject to some methodological limitations. Despite these limitations, robust and consistent associations between childhood problems and adult PD were found, arguing for the strength of these associations, making a strong case for a need for research in this area. Future research should focus on overcoming some of these issues. Firstly, the focus should be on testing the effects of childhood problems on PD in larger, more representative samples. Preventative measures to minimise participant attrition should be taken, such as making contact with participants on a regular basis, especially if the follow-up period is long. Ideally, in order to assess the developmental pathways to PD more thoroughly, participants should be followed up at several time points. Several assessments are necessary to assess transactional models of child characteristics and parent variables, which was not possible with the two assessments available in this study.

Future research should also ensure that both baseline and follow-up assessments are reliable and valid. At baseline, age-appropriate reliable and valid assessment instruments should be used to assess all of the most common EXT and INT disorders. Ideally, ratings should be made by trained professionals, or be corroborated by two independent raters (e.g. parent and teacher). At follow-up, PD should ideally be assessed by clinical interview; alternatively, self-reports should be corroborated by other-reports. Adult psychopathology should be assessed using an age-appropriate instrument. Parent ratings should ideally be made at baseline rather than follow-up, so that ratings are not affected by recall bias. In particular, the predictive validity of childhood INT problems with regard to PD needs to be addressed in future studies by including a wider range and more reliable measures for INT problems.

7.7 Concluding Remark

The aim of this thesis was to investigate early childhood predictors of PD. Specifically, we investigated whether EXT (conduct problems and hyperactivity) or INT childhood problems (shyness and emotional problems)

assessed at age 3 were predictive of PD symptoms assessed in early adulthood. We further explored whether these associations were influenced by negative parenting, and whether these associations could be explained by a continuation of childhood symptoms into adulthood. Despite the young age at baseline, the long follow-up period and the many challenges encountered throughout this research, we found consistent and robust associations between childhood externalising problems and PD, arguing for the strength of these associations. These associations were not influenced by negative parenting, and they were not mediated by continuation of symptoms into adulthood. Negative parenting, especially paternal indifference, additionally increased the risk for a PD, but parenting did not interact with childhood problems in the prediction of PD. Specifically, the following risk patterns were found: (1) childhood hyperactivity, conduct problems and paternal indifference predicted Borderline PD; (2) childhood conduct problems and maternal overcontrol predicted Antisocial PD; and (3) childhood hyperactivity predicted Avoidant PD. These findings have several implications for early intervention and prevention strategies.

Appendices

A.1 Meta-Analysis Search Strategy

Search Strategy - searches run in December 2014

AB (conduct disorder or CD or conduct problems or adhd or attention deficit or hyperactivity or hyperactive or hyperkinetic or attention problems or ODD or Oppositional or defiant or disruptive disorder or disruptive problems or behavior problems or behaviour problems or behavior disorder or behavior problem or depression or depressive or anxiety or temperament or emotionality or shyness or emotional or externalising or externalizing or internalising or internalizing) AND AB (cluster a or paranoid or Schizoid or schizotype or schizotypal or Paranoid PD or Schizotypal PD or cluster b or Antisocial PD or Antisocial or Borderline PD or Borderline or Narcissistic PD or narcissistic or Histrionic PD or histrionic or psychopathy or psychopath or psychopathic or cluster c or Dependent PD or apd or Avoidant PD or ocpd or dependent or avoidant or obsessive compulsive personality or (personality disorder) or personality pathology) AND AB (longitudinal or predictor or outcome or prospective or risk factor or precursor)

Limiters - Date of Publication from: 19800101-; Scholarly (Peer Reviewed) Journals; Publication Year from: 1980-; Publication Type: Peer Reviewed Journal; English; Population Group: Human, Male, Female, Inpatient, Outpatient; Exclude Dissertations; English Language; Review Articles; Human; Year of Publication from: 1980-; Exclude Book Reviews; Exclude Non-Article Content; Population Group: Human, Male, Female, Inpatient, Outpatient; English Language; Research Article; Meta-Synthesis; Human; Language: English; Inpatients; Outpatients

Search modes - Boolean/Phrase

A.2 Quality Criteria

Representativeness of sample at baseline

2 – representative, population-based sample

1 – clinic-referred group or high risk group

0 – basis for sampling not clear

Sample size

1 – appropriate sample size

0 – inappropriate sample size

0 - Sample size not/insufficiently reported

Attrition – possibility of bias due to drop-outs/loss to follow up

2 – attrition rate <25% of sample lost at follow-up

1 - attrition rate > 25% lost but information about completers vs drop-outs given and no bias expected based on differences between completers/drop-outs on predictors/confounders

0 – attrition rate >25% and bias expected based on differences between completers/drop-outs on predictors/confounders

0 - attrition rate >25% and no information about completers/drop-outs given

0 - attrition rate (number of drop-outs) not given

Assessment of baseline variables

2 – structured clinical assessment made by clinician or trained researcher or additional independent evaluation used AND valid assessment instrument used (standardised instrument or Cronbach alpha of .7 or higher)

1 - assessment by 1 person (e.g. parent, teacher, self-report) AND valid assessment instrument used (standardised measure or Cronbach alpha .7 or higher)

0 - assessment made by only one person and instrument not valid (not standardised and Cronbach alpha lower than .7)

0 - insufficient information about assessment given

Assessment of outcome variable

2 – structured clinical assessment made by clinician or trained researcher or additional independent evaluation used) AND valid assessment instrument used (standardised or Cronbach alpha .7 or higher)

1 - assessment by 1 person (e.g., self-report) AND valid assessment instrument used (standardised measure or Cronbach alpha .7 or higher)

0 - assessment made by only one person and instrument not valid (not standardised and Cronbach alpha lower than .7)

0 - insufficient information about assessment given

A.3 Conversions

1. Calculation of effect sizes through cases vs non-cases:

	PD at outcome	No PD at outcome
Childhood problems	A	B
Controls	C	D

OR= $\frac{AD}{BC}$

2. Conversion to OR from r:

$$OR = \left[\frac{180^{\circ}}{\cos^{-1}(r)} - 1 \right]^2$$

3. Conversion from t to r:

$$r = \sqrt{\frac{t^2}{(t^2 + df)}}$$

4. Conversion from Fisher's z to r:

$$r = \sqrt{\frac{z^2}{(z^2 + df)}}$$

5. Conversion from χ^2 (df=1) to r:

$$r = \sqrt{\frac{\chi^2}{N}}$$

6. Conversion from χ^2 (df>1) to r:

$$r = \sqrt{\frac{\chi^2}{\chi^2 + N}}$$

4. Conversion from Z-score to r: $r = \sqrt{\frac{Z^2}{N}}$

A.4 Comparison of Least Control Model (LCM) and Most Control Model (MCM)

	Included papers	Covariates LCM	Covariates MCM
	Hechtman (1984)	-	-
	Weiss (1985)	-	-
	Fischer (2002)	-	-
	Mannuzza (combined)	-	-
	Fergusson (2005)	-	Withdrawal/ANX, ADHD, Sex, SES, ethnicity, adjustment problems, family background, child abuse, IQ
	Claude & Firestone (1995)	-	-
	Miller (2008)	-	-
	Burke (2007)	Sex	Sex
Cluster B – EXT	Carlson (2009)	-	Behavioural instability, relational disturbance, emotional instability, self-representation, parent-child disturbance
	Caspi (1996)	Sex	Sex
	Belsky (2012)	-	-
	Schaeffer (2003)	-	-
	McMahon (2010)	-	ADHD, ODD, CU traits
	Moffitt (2002)	-	-
	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ADHD, ODD, CD
	Stepp (2012)	-	-
	Lahey (2005)	-	ADHD, CD, SES, maternal ANTISOCIAL PD
	Diamantopoulou (2010)	-	-
	Sourander	-	Family background, school performance, emotional

	(2005)		problems, CD, DEPR, Psychosomatic problems
	Hellgren (1994)	-	-
	Forsman (2007)	-	-
	Crawford (2009)	Separation from parents, crying/demanding	Separation from parents, crying/demanding, abuse, inconsistent mothering, maternal satisfaction with child
	Bernstein (1996)	Age, sex	Age, sex
	Shi (2012)	Sex	Sex
Cluster B – ADHD	Hechtman (1984)	-	-
	Weiss (1985)	-	-
	Fischer (2002)	-	-
	Mannuzza (combined)	-	-
	Claude & Firestone (1995)	-	-
	Miller (2008)	-	-
	Carlson (2009)	-	Behavioural instability, relational disturbance, emotional instability, self-representation, parent-child disturbance
	Shi (2012)	Sex	Sex
	Belsky (2012)	-	-
	Stepp (2012)	-	-
	Burke (2007)	Sex	Sex
	Sourander (2005)	-	Family background, school performance, emotional problems, CD, DEPR, Psychosomatic problems
	Hellgren (1994)	-	-
	Forsman (2007)	-	-
	Diamantopoulou (2010)	-	-

	Lahey (2005)	-	CD, SES, maternal ANTISOCIAL PD
	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ODD, ADHD
Cluster B – CD	Burke (2007)	Sex	Sex
	Fergusson (2005)		
	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ODD, CD
	Lahey (2005)	-	ADHD, SES, maternal ANTISOCIAL PD
	McMahon (2010)	-	ADHD, ODD, CU traits
	Moffitt (2002)	-	-
	Sourander (2005)	-	Family background, school performance, emotional problems, CD, DEPR, Psychosomatic problems
	Stepp (2012)	-	-
	Bernstein (1996)	Age, sex	Age, sex
Cluster B – ODD	Burke (2007)	sex	Sex
	Diamantopoulou (2010)	-	-
	Lahey (2005)	-	CD, SES, maternal ANTISOCIAL PD
	Stepp (2012)	-	-
	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ODD, CD
Cluster B – INT	Burke (2007)	Sex	Sex
	Stepp (2012)	-	-
	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ODD, CD
	Belsky (2012)	-	-
	Sourander (2005)	-	Family background, school performance, emotional problems, CD, DEPR, Psychosomatic problems

	Diamantopoulou (2010)	-	-
	Bernstein (1996)	Age, sex	Age, sex
	Glenn (2007)	-	Physiological measures, sociability
	Caspi (1996)	Sex	Sex
Cluster B – ANX	Bernstein (1996)	Age, sex	Age, sex
	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ODD, CD
	Burke (2007)	sex	Sex
	Glenn (2007)		Physiological measures, sociability
Cluster B – DEPR	Bernstein (1996)	Age, sex	Age, sex
	Sourander (2005)	-	OAD, SAD, GAD, DEPR, ODD, CD
	Burke (2007)	sex	Sex
	Stepp (2012)	-	-
Cluster B – EMO	Carlson (2009)	-	Behavioural instability, relational disturbance, emotional instability, self-representation, parent-child disturbance
	Stepp (2012)	-	-
	Crawford (2009)	Separation from parents, crying/demanding	Separation from parents, crying/demanding, abuse, inconsistent mothering, maternal satisfaction with child
	Sourander (2005)	-	Family background, school performance, emotional problems, CD, DEPR, Psychosomatic problems
ANTISOCIAL PD - EXT	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ADHD, ODD, CD
	Diamantopoulou (2010)	-	-
	Forsman (2007)	-	-
	Lahey (2005)	-	ADHD, CD, SES, Maternal

			ANTISOCIAL PD
	Mannuzza (combined(	-	-
	Miller (2008)	-	-
	Shi (2012)	Sex	Sex
	Sourander (2005)	-	Family background, school performance, emotional problems, CD, DEPR, Psychosomatic problems
	Fergusson (2005)	-	Withdrawal/ANX, ADHD, Sex, SES, ethnicity, adjustment problems, family background, child abuse, IQ
	Fischer (2002)	-	-
	Burke (2007)	Sex	Sex
	Schaeffer (2003)	-	-
	Moffitt (2002)	-	-
	Caspi (1996)	Sex	Sex
	Weiss (1985)	-	-
	Claude (1995)	-	-
	Hechtman (1984)	-	-
	McMahon (2010)	-	ODD, ADHD, CU traits
	Hellgren (1994)	-	-
ANTISOCIAL PD – ADHD	Copeland (2009)	-	OAD, SAD, GAD, DEPR, CD, ODD
	Diamantopoulou (2010)	-	-
	Forsman (2007)	-	-
	Lahey (2005)	-	CD, SES, Maternal ANTISOCIAL PD
	Miller (2008)	-	-
	Sourander (2005)	-	Family background, school performance, emotional problems, CD, DEPR,

			Psychosomatic problems
	Fischer (2002)	-	-
	Weiss (1985)	-	-
	Claude (1995)	-	-
	Hellgren (1994)	-	-
	Hechtman (1984)	-	-
	Shi (2012)	Sex	Sex
	Burke (2007)	Sex	Sex
	Mannuzza (combined)	-	-
ANTISOCIAL PD – CD	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ADHD, ODD
	Lahey (2005)	-	ADHD, SES, Maternal ANTISOCIAL PD
	Sourander (2005)	-	Family background, school performance, emotional problems, DEPR, Psychosomatic problems
	Fergusson (2005)	-	Withdrawal/ANX, ADHD, Sex, SES, ethnicity, adjustment problems, family background, child abuse, IQ
	Burke (2007)	Sex	Sex
	Moffitt (2002)	-	-
	McMahon (2010)	-	ODD, ADHD, CU traits
ANTISOCIAL PD – ODD	Copeland (2009)	-	OAD, SAD, GAD, DEPR, ADHD, CD
	Diamantopoulou (2010)	-	-
	Lahey (2005)	-	CD, SES, Maternal ANTISOCIAL PD
	Burke (2007)	Sex	Sex
ANTISOCIAL PD – INT	Copeland (2009)	-	ADHD, CD, ODD
	Diamantopoulou	-	-

	(2010)		
	Glenn (2007)	-	Sociability, physiological measures
	Sourander (2005)	-	Family background, school performance, emotional problems, CD
	Burke (2007)	Sex	Sex
	Caspi (1996)	Sex	Sex
ANTISOCIAL PD – ANX	Copeland (2009)	-	ADHD, CD, ODD
	Glenn (2007)	-	Sociability, physiological measures
	Burke (2007)	Sex	Sex
BORDERLINE PD – EXT	Belsky (2012)	-	-
	Carlson (2009)	-	Attentional disturbance, behavioural instability, relational disturbance, emotional instability, self-representation, parent-child disturbance
	Miller (2008)	-	-
	Stepp (2012)	-	-
	Fischer (2002)	-	-
	Crawford (2009)	Separation from parents, crying/demanding	Separation from parents, crying/demanding, abuse, inconsistent mothering, maternal satisfaction with child
	Hellgren (1994)	-	-
BORDERLINE PD – ADHD	Belsky (2012)	-	-
	Carlson (2009)	-	Behavioural instability, relational disturbance, emotional instability, self-representation, parent-child disturbance
	Miller (2008)	-	-
	Stepp (2012)	-	-
	Fischer (2002)	-	-

	Hellgren (1994)	-	-
BORDERLINE PD – EMO	Carlson (2009)	-	Attentional disturbance, behavioural instability, relational disturbance, self-representation, parent-child disturbance
	Stepp (2012)	-	-
	Crawford (2009)	Separation from parents, angry temperament	*MCM not carried out*
HISTRIONIC PD – EXT/ADHD	Miller (2008)	-	-
	Fischer (2002)	-	-
	Hellgren (1994)	-	-
Cluster A – EXT	Bernstein (1996)	Age, sex	Age, sex
	Miller (2008)	-	-
	Natsuaki (2009)	-	-
	Hellgren (1994)	-	-
PARANOID PD – EXT	Miller (2008)	-	-
	Natsuaki (2009)	-	-
	Hellgren (1994)	-	-
SCHIZOTYPAL PD – EXT/ADHD	Miller (2008)	-	-
	Hellgren (1994)	-	-
	Anglin (2008)	Anxious temperament, early separation, maternal affection	Anxious temperament, early separation, maternal affection
Cluster C – EXT	Bernstein (1996)	Age, sex	Age, sex
	Miller (2008)	-	-
	Hellgren (1994)	-	-
	Fischer (2002)	-	-
Cluster C – ADHD	Miller (2008)	-	-
	Hellgren (1994)	-	-
	Fischer (2002)	-	-

AVOIDANT PD – EXT/ADHD	Miller (2008)	-	-
	Hellgren (1994)	-	-
	Fischer (2002)	-	-

A.5 Forest Plots

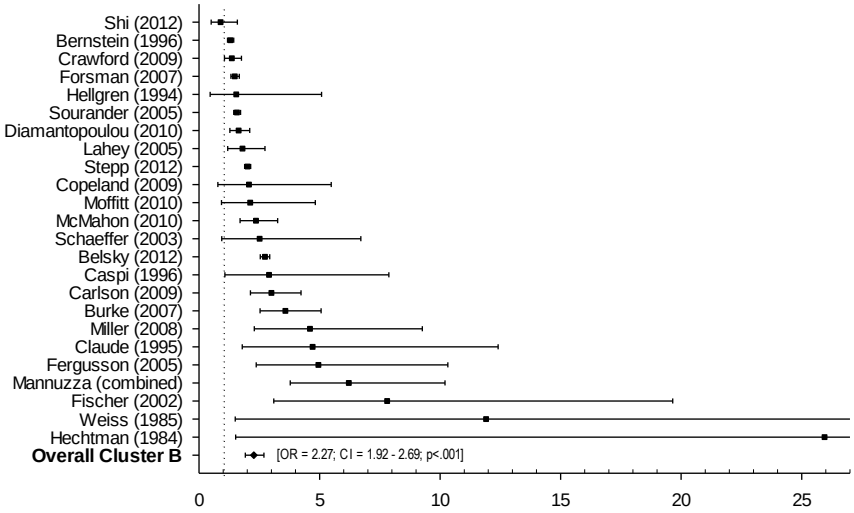


Figure 2: Externalising Childhood Problems and Cluster B
Random Effects Model, 95% CI; OR = Odds Ratio; CI = Confidence Interval

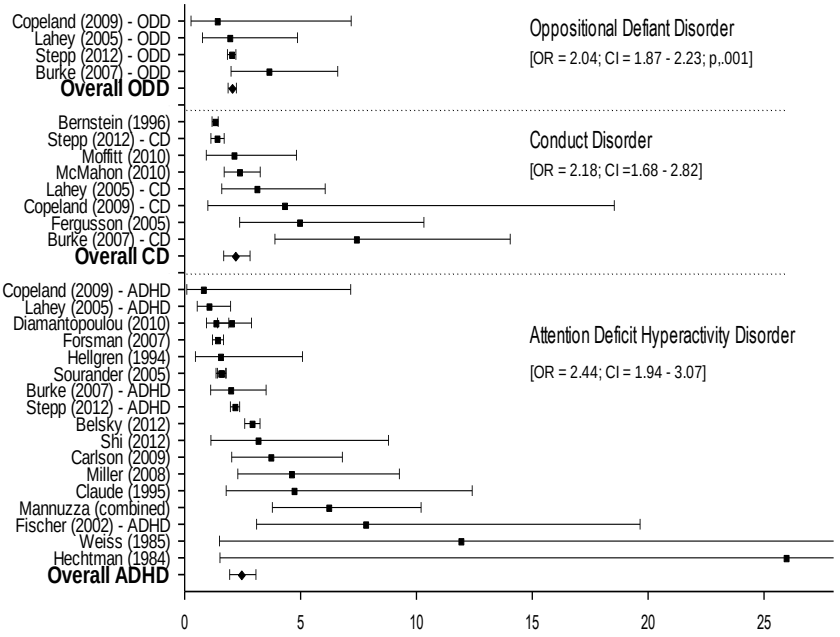


Figure 3: Childhood ODD, CD, ADHD and Cluster B
Random Effects Model, 95% CI; OR - Odds Ratio; CI - Confidence Interval

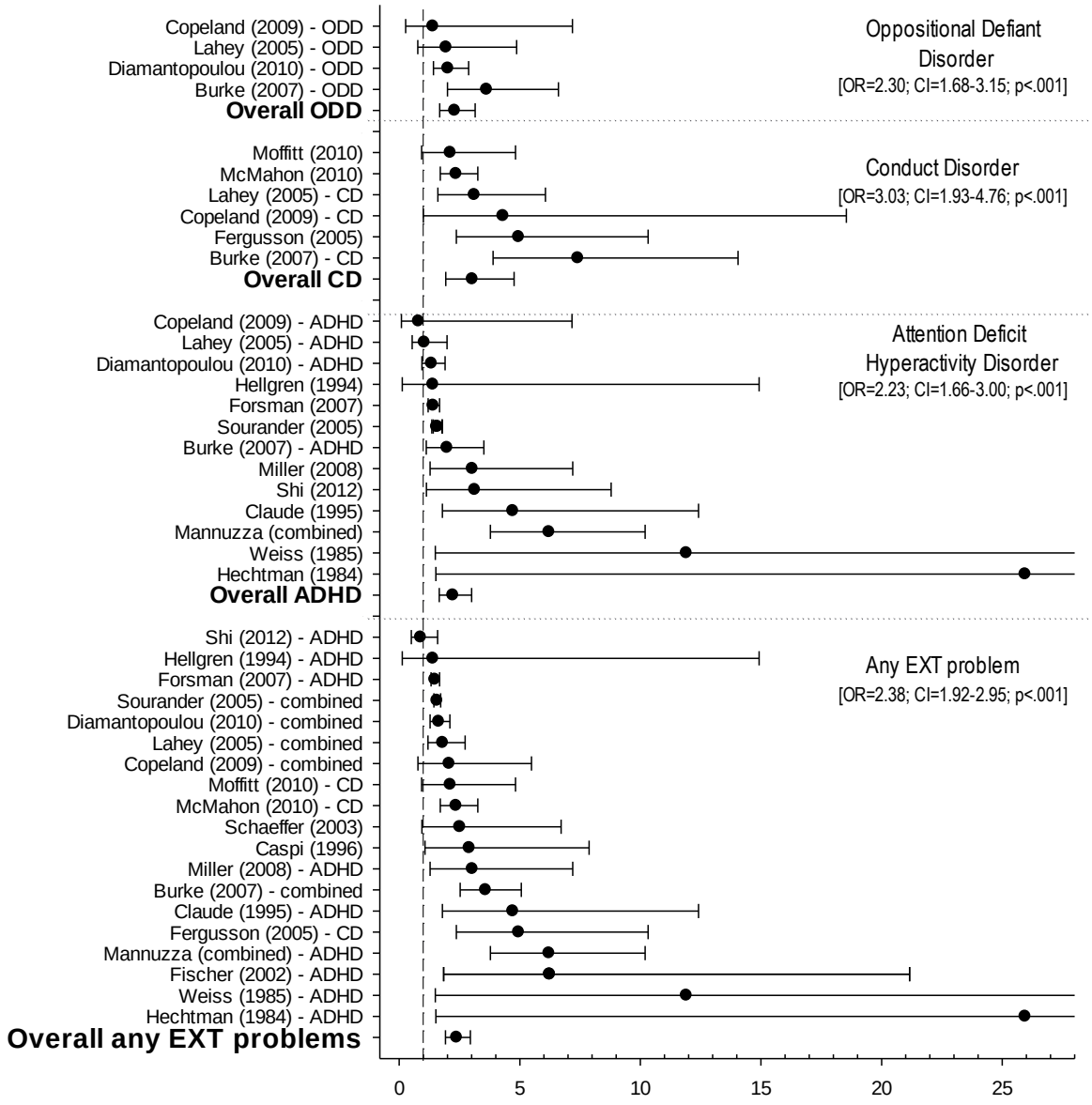


Figure 4: Childhood EXT Problems and Antisocial PD/Psychopathy

Random Effects Model, 95% CI; OR - Odds Ratio; CI - Confidence Interval

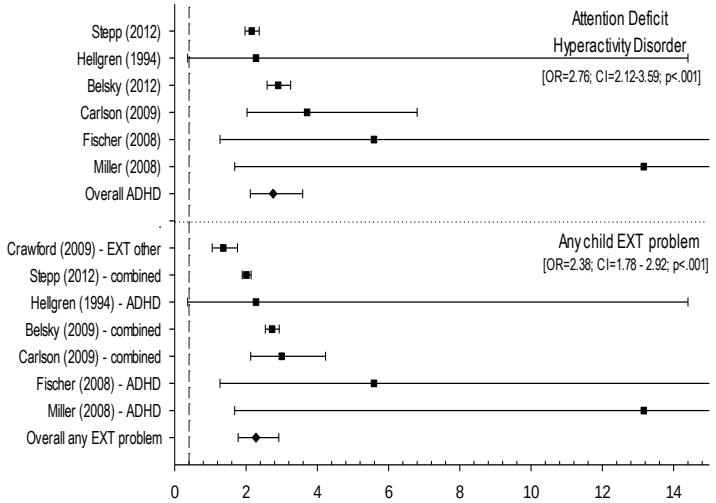


Figure 5: Childhood Externalising Problems and Borderline PD
Random Effects Model, 95% CI; OR - Odds Ratio; CI - Confidence Interval

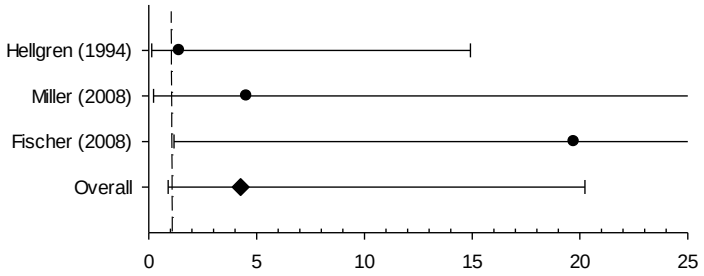


Figure 6: Childhood Externalising Problems and Histrionic PD
OR = 4.26; CI = 0.13 - 14.92; p<.10
Random Effects Model, 95% CI; OR - Odds Ratio; CI - Confidence Interval

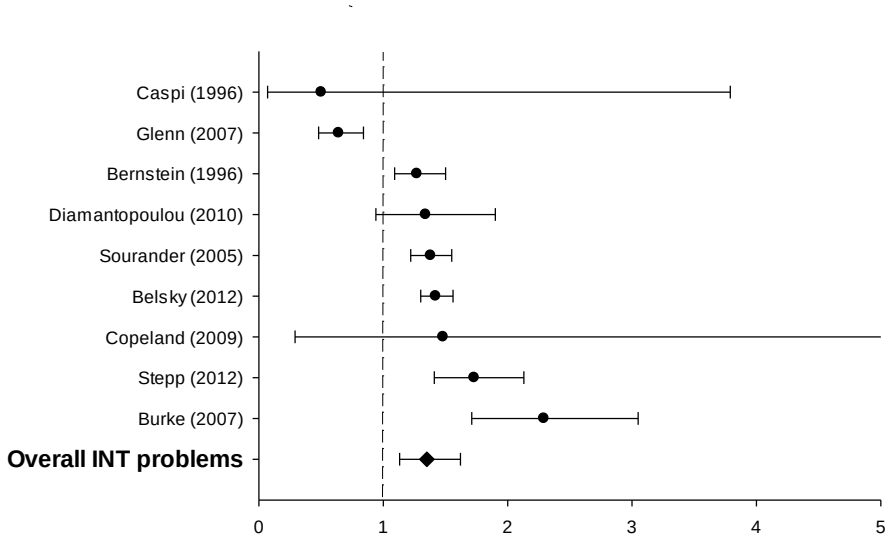


Figure 7: Childhood Internalising Problems and Cluster B PDs
OR = 1.35; CI = 1.13 - 1.62; p < .01
Random Effects Model; OR - Odds Ratio; CI - Confidence Interval

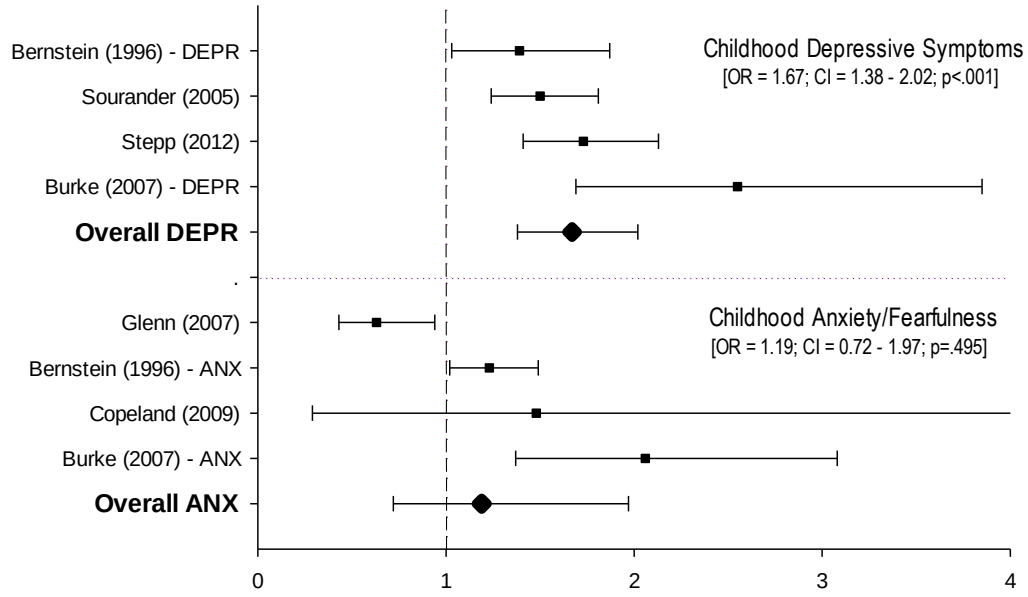


Figure 8: Childhood Anxiety/Depression and Cluster B
Random Effects Model, 95% CI; OR - Odds Ratio; CI - Confidence Interval

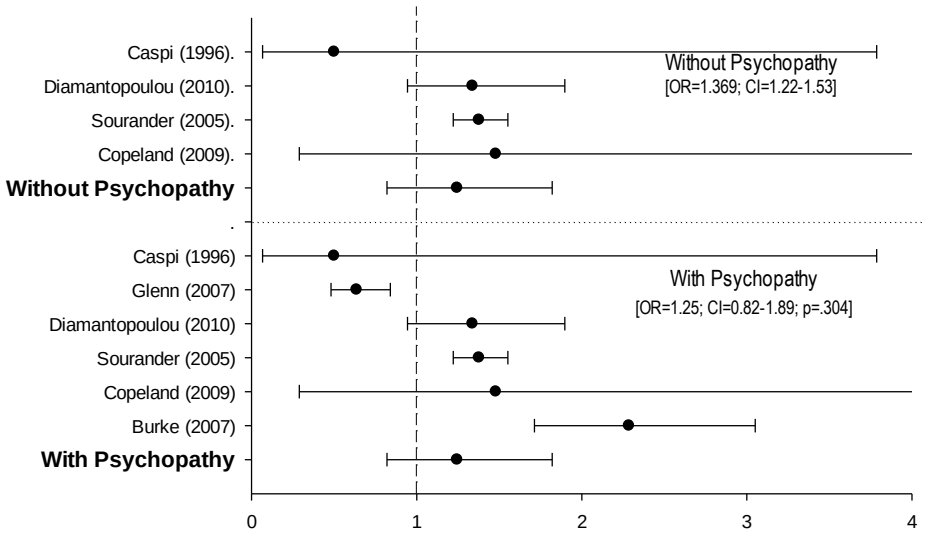


Figure 9: Childhood Internalising Problems and ASPD, with and without Psychopathy
Random Effects Model; 95% CI; OR - Odds Ratio; CI - Confidence Interval

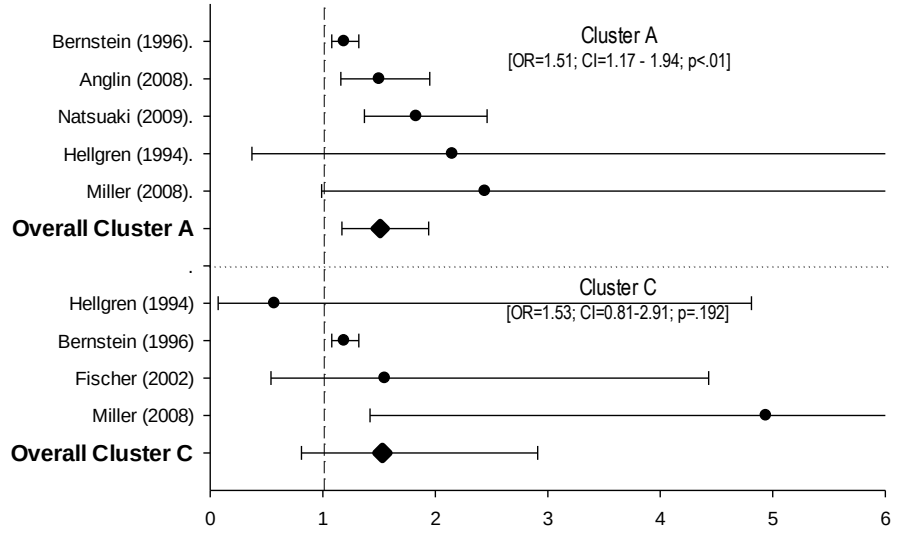


Figure 11: Childhood Externalising Problems and Clusters A and C
Random Effects Model, 95% Confidence Interval (CI); OR - Odds Ratio

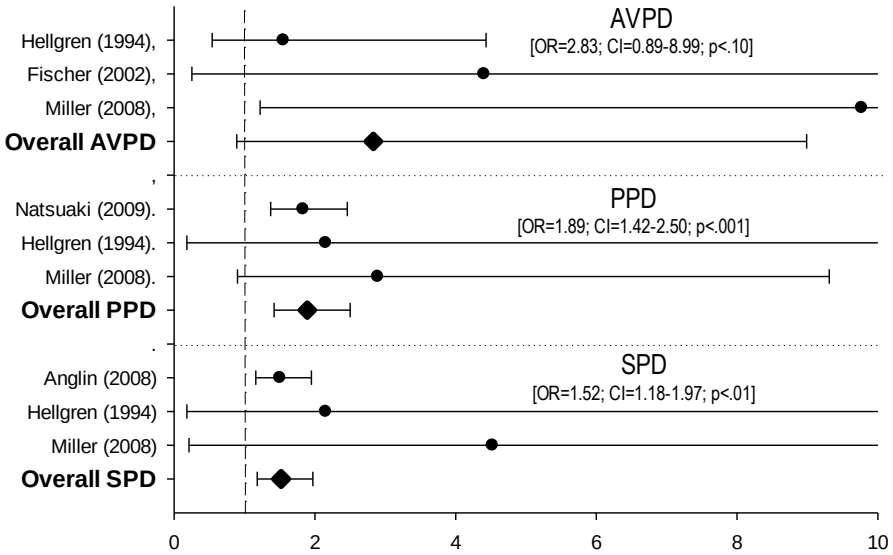


Figure 12: Externalising Childhood Problems and SPD, PPD, AVPD

Random Effect Models, 95% Confidence Interval (CI); OR - Odds Ratio

A.6 Werry-Weiss-Peter Activity Rating Scale

		No or Hardly Ever	Yes, Fairly Often	Yes, Very Often
1	During meals is the child up and down at the table?	☐	☐	☐
2	During meals, does the child interrupt without regard to what others are trying to say?	☐	☐	☐
3	During meals, does the child wriggle?	☐	☐	☐
4	During meals, does the child fiddle with things?	☐	☐	☐
5	During meals, does the child talk too much?	☐	☐	☐
6	When watching television, does the child get up and down during the programme?	☐	☐	☐
7	When watching television, does the child wriggle?	☐	☐	☐
8	When watching television, does the child play with objects or his/her own body?	☐	☐	☐
9	When watching television, does the child talk too much?	☐	☐	☐
10	When watching television, does the child play which interrupts others ability to watch the programme?	☐	☐	☐
11	When drawing, colouring, writing or doing homework, does the child get up and down?	☐	☐	☐
12	When drawing, colouring, writing or doing homework, does the child wriggle?	☐	☐	☐

13	When drawing, colouring, writing or doing homework, does the child play with objects or his/her own body?	☐	☐	☐
14	When drawing, colouring, writing or doing homework, does the child talk too much?	☐	☐	☐
15	When drawing, colouring, writing or doing homework, does the child require adult supervision or attendance?	☐	☐	☐
16	Is the child unable to play quietly?	☐	☐	☐
17	When at play, does the child keep going from one toy to another?	☐	☐	☐
18	When at play, does the child seek attention of an adult?	☐	☐	☐
19	When at play, does the child talk too much?	☐	☐	☐
20	When at play, does the child disrupt the play of other children?	☐	☐	☐
21	Does the child have difficulty settling down for sleep?	☐	☐	☐
22	Does the child get too little sleep?	☐	☐	☐
23	Is the child restless during sleep?	☐	☐	☐
24	Is the child restless during travel?	☐	☐	☐
25	Is the child restless during shopping (including touching everything)?	☐	☐	☐
26	Is the child restless during church, at the cinema or watching a school play for example?	☐	☐	☐
27	Is the child restless while visiting friends or relatives?	☐	☐	☐

A.7 EAS Temperament Questionnaire

Please circle the rating on each of the items for your child.

	NOT TYPICAL				VERY TYPICAL
1. Child tends to be shy	1	2	3	4	5
2. Child makes friends easily	1	2	3	4	5
3. Child is very sociable	1	2	3	4	5
4. Child takes a long time to warm up to strangers	1	2	3	4	5
5. Child is very friendly with strangers	1	2	3	4	5
6. Child cries easily	1	2	3	4	5
7. Child tends to be somewhat emotional	1	2	3	4	5
8. Child often fusses and cries	1	2	3	4	5
9. Child gets upset easily	1	2	3	4	5
10. Child reacts intensely when upset	1	2	3	4	5

A.8 Behaviour Checklist

Not active enough / not markedly active	0
very active	1
too active, won't sit still for meals or at other times for more than 5 minutes	2
Concentrates on play indoors for 15 minutes or more	0
concentration 5-15 minutes or very variable	1
hardly ever concentrates for more than 5 minutes on play indoors	2
Not clinging, can easily be left with people s/he knows	0
Gets upset if away from mother but gets over it	1
Very clinging, can't be left with others	2
Independent, doesn't ask for a lot of attention	0
sometimes asks for a lot of attention, sometimes follows mother around all day	1
demands too much attention, follows mother around all day	2
Easy to manage and control	0
sometimes difficult to manage and control	1
frequently very difficult to manage and control	2
Doesn't have temper tantrums	0
sometimes has temper tantrums (lasting a few minutes)	1
has frequent or long temper tantrums	2
Usually happy except for brief periods, when tired for instance	0
sometimes miserable or irritable	1
frequently miserable or irritable	2
Not a worrier	0
sometimes worried for short periods	1
has many different worries, broods over things (e.g. accidents, illnesses, monsters)	2
Few or no fears	0
has some fears	1
very fearful, has lots of different fears	2
gets on well with other children	0
some difficulties playing with other children	1
finds it very difficult to play with other children	2

A.9 Personality Inventory for DSM-5 (PID-5)

0 – very false or often false

1 - Sometimes or somewhat false

2 – sometimes or somewhat true

3 – very true or often true

1	I don't get as much pleasure out of things as others seem to.	0	1	2	3
2	Plenty of people are out to get me.	0	1	2	3
3	People would describe me as reckless.	0	1	2	3
4	I feel like I act totally on impulse.	0	1	2	3
5	I often have ideas that are too unusual to explain to anyone.	0	1	2	3
6	I lose track of conversations because other things catch my attention.	0	1	2	3
7	I avoid risky situations.	0	1	2	3
8	When it comes to my emotions, people tell me I'm a "cold fish".	0	1	2	3
9	I change what I do depending on what others want.	0	1	2	3
10	I prefer not to get too close to people.	0	1	2	3
11	I often get into physical fights.	0	1	2	3
12	I dread being without someone to love me.	0	1	2	3
13	Being rude and unfriendly is just a part of who I am.	0	1	2	3
14	I do things to make sure people notice me.	0	1	2	3
15	I usually do what others think I should do.	0	1	2	3
16	I usually do things on impulse without thinking about what might happen as a result.	0	1	2	3
17	Even though I know better, I can't stop making rash decisions.	0	1	2	3
18	My emotions sometimes change for no good reason.	0	1	2	3
19	I really don't care if I make other people suffer.	0	1	2	3
20	I keep to myself.	0	1	2	3
21	I often say things that others find odd or strange.	0	1	2	3
22	I always do things on the spur of the moment.	0	1	2	3
23	Nothing seems to interest me very much.	0	1	2	3

24	Other people seem to think my behaviour is weird.	0	1	2	3
25	People have told me that I think about things in a really strange way.	0	1	2	3
26	I almost never enjoy life.	0	1	2	3
27	I often feel like nothing I do really matters.	0	1	2	3
28	I snap at people when they do little things that irritate me.	0	1	2	3
29	I can't concentrate on anything.	0	1	2	3
30	I'm an energetic person.	0	1	2	3
31	Others see me as irresponsible.	0	1	2	3
32	I can be mean when I need to be.	0	1	2	3
33	My thoughts often go off in odd or unusual directions.	0	1	2	3
34	I've been told that I spend too much time making sure things are exactly in place.	0	1	2	3
35	I avoid risky sports and activities.	0	1	2	3
36	I can have trouble telling the difference between dreams and waking life.	0	1	2	3
37	Sometimes I get this weird feeling that parts of my body feel like they're dead or not really me.	0	1	2	3
38	I am easily angered.	0	1	2	3
39	I have no limits when it comes to doing dangerous things.	0	1	2	3
40	To be honest, I'm just more important than other people.	0	1	2	3
41	I make up stories about things that happened that are totally untrue.	0	1	2	3
42	People often talk about me doing things I don't remember at all.	0	1	2	3
43	I do things so that people just have to admire me.	0	1	2	3
44	It's weird, but sometimes ordinary objects seem to be a different shape than usual.	0	1	2	3
45	I don't have very long-lasting emotional reactions to things.	0	1	2	3
46	It is hard for me to stop an activity, even when it’s time to do so.	0	1	2	3

47	I'm not good at planning ahead.	0	1	2	3
48	I do a lot of things that others consider risky.	0	1	2	3
49	People tell me that I focus too much on minor details.	0	1	2	3
50	I worry a lot about being alone.	0	1	2	3
51	I've missed out on things because I was busy trying to get something I was doing exactly right.	0	1	2	3
52	My thoughts often don’t make sense to others.	0	1	2	3
53	I often make up things about myself to help me get what I want.	0	1	2	3
54	It doesn't really bother me to see other people get hurt.	0	1	2	3
55	People often look at me as if I'd said something really weird.	0	1	2	3
56	People don't realize that I'm flattering them to get something.	0	1	2	3
57	I’d rather be in a bad relationship than be alone.	0	1	2	3
58	I usually think before I act.	0	1	2	3
59	I often see vivid dream-like images when I’m falling asleep or waking up.	0	1	2	3
60	I keep approaching things the same way, even when it isn’t working.	0	1	2	3
61	I'm very dissatisfied with myself.	0	1	2	3
62	I have much stronger emotional reactions than almost everyone else.	0	1	2	3
63	I do what other people tell me to do.	0	1	2	3
64	I can't stand being left alone, even for a few hours.	0	1	2	3
65	I have outstanding qualities that few others possess.	0	1	2	3
66	The future looks really hopeless to me.	0	1	2	3
67	I like to take risks.	0	1	2	3
68	I can't achieve goals because other things capture my attention.	0	1	2	3
69	When I want to do something, I don't let the possibility that it might be risky stop me.	0	1	2	3
70	Others seem to think I'm quite odd or unusual.	0	1	2	3
71	My thoughts are strange and unpredictable.	0	1	2	3

72	I don't care about other people's feelings.	0	1	2	3
73	You need to step on some toes to get what you want in life.	0	1	2	3
74	I love getting the attention of other people.	0	1	2	3
75	I go out of my way to avoid any kind of group activity.	0	1	2	3
76	I can be sneaky if it means getting what I want.	0	1	2	3
77	Sometimes when I look at a familiar object, it's somehow like I'm seeing it for the first time.	0	1	2	3
78	It is hard for me to shift from one activity to another.	0	1	2	3
79	I worry a lot about terrible things that might happen.	0	1	2	3
80	I have trouble changing how I'm doing something even if what I'm doing isn't going well.	0	1	2	3
82	I keep my distance from people.	0	1	2	3
83	I often can't control what I think about.	0	1	2	3
84	I don't get emotional.	0	1	2	3
85	I resent being told what to do, even by people in charge.	0	1	2	3
86	I'm so ashamed by how I've let people down in lots of little ways.	0	1	2	3
87	I avoid anything that might be even a little bit dangerous.	0	1	2	3
88	I have trouble pursuing specific goals even for short periods of time.	0	1	2	3
89	I prefer to keep romance out of my life.	0	1	2	3
90	I would never harm another person.	0	1	2	3
91	I don't show emotions strongly.	0	1	2	3
92	I have a very short temper.	0	1	2	3
93	I often worry that something bad will happen due to mistakes I made in the past.	0	1	2	3
94	I have some unusual abilities, like sometimes knowing exactly what someone is thinking.	0	1	2	3
95	I get very nervous when I think about the future.	0	1	2	3
96	I rarely worry about things.	0	1	2	3
97	I enjoy being in love.	0	1	2	3

98	I prefer to play it safe rather than take unnecessary chances.	0	1	2	3
99	I sometimes have heard things that others couldn’t hear.	0	1	2	3
100	I get fixated on certain things and can’t stop.	0	1	2	3
101	People tell me it's difficult to know what I'm feeling.	0	1	2	3
102	I am a highly emotional person.	0	1	2	3
103	Others would take advantage of me if they could.	0	1	2	3
104	I often feel like a failure.	0	1	2	3
105	If something I do isn't absolutely perfect, it's simply not acceptable.	0	1	2	3
106	I often have unusual experiences, such as sensing the presence of someone who isn't actually there.	0	1	2	3
107	I'm good at making people do what I want them to do.	0	1	2	3
108	I break off relationships if they start to get close.	0	1	2	3
109	I’m always worrying about something.	0	1	2	3
110	I worry about almost everything.	0	1	2	3
111	I like standing out in a crowd.	0	1	2	3
112	I don't mind a little risk now and then.	0	1	2	3
113	My behaviour is often bold and grabs peoples' attention.	0	1	2	3
114	I'm better than almost everyone else.	0	1	2	3
115	People complain about my need to have everything all arranged.	0	1	2	3
116	I always make sure I get back at people who wrong me.	0	1	2	3
117	I'm always on my guard for someone trying to trick or harm me.	0	1	2	3
118	I have trouble keeping my mind focused on what needs to be done.	0	1	2	3
120	I'm just not very interested in having sexual relationships.	0	1	2	3
121	I get stuck on things a lot.	0	1	2	3
122	I get emotional easily, often for very little reason.	0	1	2	3
123	Even though it drives other people crazy, I insist on	0	1	2	3

	absolute perfection in everything I do.				
124	I almost never feel happy about my day-to-day activities.	0	1	2	3
125	Sweet-talking others helps me get what I want.	0	1	2	3
126	Sometimes you need to exaggerate to get ahead.	0	1	2	3
127	I fear being alone in life more than anything else.	0	1	2	3
128	I get stuck on one way of doing things, even when it's clear it won't work.	0	1	2	3
129	I'm often pretty careless with my own and others' things.	0	1	2	3
130	I am a very anxious person.	0	1	2	3
131	People are basically trustworthy.	0	1	2	3
132	I am easily distracted.	0	1	2	3
133	It seems like I'm always getting a “raw deal” from others.	0	1	2	3
134	I don't hesitate to cheat if it gets me ahead.	0	1	2	3
135	I check things several times to make sure they are perfect.	0	1	2	3
136	I don’t like spending time with others.	0	1	2	3
137	I feel compelled to go on with things even when it makes little sense to do so.	0	1	2	3
138	I never know where my emotions will go from moment to moment.	0	1	2	3
139	I have seen things that weren’t really there.	0	1	2	3
140	It is important to me that things are done in a certain way.	0	1	2	3
141	I always expect the worst to happen.	0	1	2	3
142	I try to tell the truth even when it's hard.	0	1	2	3
143	I believe that some people can move things with their minds.	0	1	2	3
144	I can't focus on things for very long.	0	1	2	3
145	I steer clear of romantic relationships.	0	1	2	3
146	I'm not interested in making friends.	0	1	2	3
147	I say as little as possible when dealing with people.	0	1	2	3

148	I'm useless as a person.	0	1	2	3
149	I'll do just about anything to keep someone from abandoning me.	0	1	2	3
150	Sometimes I can influence other people just by sending my thoughts to them.	0	1	2	3
151	Life looks pretty bleak to me.	0	1	2	3
152	I think about things in odd ways that don't make sense to most people.	0	1	2	3
153	I don’t care if my actions hurt others.	0	1	2	3
154	Sometimes I feel "controlled" by thoughts that belong to someone else.	0	1	2	3
155	I really live life to the fullest.	0	1	2	3
156	I make promises that I don't really intend to keep.	0	1	2	3
157	Nothing seems to make me feel good.	0	1	2	3
158	I get irritated easily by all sorts of things.	0	1	2	3
159	I do what I want regardless of how unsafe it might be.	0	1	2	3
160	I often forget to pay my bills.	0	1	2	3
161	I don’t like to get too close to people.	0	1	2	3
162	I'm good at conning people.	0	1	2	3
163	Everything seems pointless to me.	0	1	2	3
164	I never take risks.	0	1	2	3
165	I get emotional over every little thing.	0	1	2	3
166	It's no big deal if I hurt other peoples' feelings.	0	1	2	3
167	I never show emotions to others.	0	1	2	3
168	I often feel just miserable.	0	1	2	3
169	I have no worth as a person.	0	1	2	3
170	I am usually pretty hostile.	0	1	2	3
171	I've skipped town to avoid responsibilities.	0	1	2	3
172	I've been told more than once that I have a number of odd quirks or habits.	0	1	2	3
173	I like being a person who gets noticed.	0	1	2	3
174	I'm always fearful or on edge about bad things that might happen.	0	1	2	3

175	I never want to be alone.	0	1	2	3
176	I keep trying to make things perfect, even when I've gotten them as good as they're likely to get.	0	1	2	3
177	I rarely feel that people I know are trying to take advantage of me.	0	1	2	3
179	I've achieved far more than almost anyone I know.	0	1	2	3
180	I can certainly turn on the charm if I need to get my way.	0	1	2	3
181	My emotions are unpredictable.	0	1	2	3
182	I don't deal with people unless I have to.	0	1	2	3
183	I don’t care about other peoples’ problems.	0	1	2	3
184	I don't react much to things that seem to make others emotional.	0	1	2	3
185	I have several habits that others find eccentric or strange.	0	1	2	3
186	I avoid social events.	0	1	2	3
187	I deserve special treatment.	0	1	2	3
188	It makes me really angry when people insult me in even a minor way.	0	1	2	3
189	I rarely get enthusiastic about anything.	0	1	2	3
190	I suspect that even my so-called “friends” betray me a lot.	0	1	2	3
191	I crave attention.	0	1	2	3
192	Sometimes I think someone else is removing thoughts from my head.	0	1	2	3
193	I have periods in which I feel disconnected from the world or from myself.	0	1	2	3
194	I often see unusual connections between things that most people miss.	0	1	2	3
195	I don't think about getting hurt when I'm doing things that might be dangerous.	0	1	2	3
196	I simply won't put up with things being out of their proper places.	0	1	2	3
197	I often have to deal with people who are less important than me.	0	1	2	3
198	I sometimes hit people to remind them who's in charge	0	1	2	3

199	I get pulled off-task by even minor distractions.	0	1	2	3
200	I enjoy making people in control look stupid.	0	1	2	3
201	I just skip appointments or meetings if I'm not in the mood.	0	1	2	3
202	I try to do what others want me to do.	0	1	2	3
203	I prefer being alone to having a close romantic partner.	0	1	2	3
204	I am very impulsive.	0	1	2	3
205	I often have thoughts that make sense to me but that other people say are strange.	0	1	2	3
206	I use people to get what I want.	0	1	2	3
207	I don't see the point in feeling guilty about things I've done that have hurt other people.	0	1	2	3
208	Most of the time I don't see the point in being friendly.	0	1	2	3
209	I've had some really weird experiences that are very difficult to explain.	0	1	2	3
210	I follow through on commitments.	0	1	2	3
211	I like to draw attention to myself.	0	1	2	3
212	I feel guilty much of the time.	0	1	2	3
213	I often "zone out" and then suddenly come to and realize that a lot of time has passed.	0	1	2	3
214	Lying comes easily to me.	0	1	2	3
215	I hate to take chances.	0	1	2	3
216	I'm nasty and short to anybody who deserves it.	0	1	2	3
217	Things around me often feel unreal, or more real than usual.	0	1	2	3
218	I'll stretch the truth if it's to my advantage.	0	1	2	3
219	It is easy for me to take advantage of others.	0	1	2	3
220	I have a strict way of doing things	0	1	2	3

A.10 The Measure of Parental Style

During your first 16 years how 'true' are the following statements about your parents' behaviour towards you. Rate each statement either as:

0 - not true at all
1 - slightly true
2 - moderately true
3 - extremely true

For each statement, please CIRCLE the appropriate number for both your mother and your father.

	Mother					Father			
1. Overprotective of me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
3. Over controlling of me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
4. Sought to make me feel guilty	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
6. Critical of me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
5. Ignored me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
8. Uncaring of me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
10. Rejecting of me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
11. Left me on my own a lot	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
12. Would forget about me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**
13. Was uninterested in me	**0**	**1**	**2**	**3**		**0**	**1**	**2**	**3**

A.11 Conners Behavior Rating Scale – Self Report (CBRS-S)

In the past month, this was….. 0 = Not true at all (Never, Seldom) 1 = Just a little true (Occasionally) 2 = Pretty much true (Often, Quite a bit) 3 = Very much true (Very often, very frequently)					
1.	I wake up during the night and have trouble falling back to sleep.	0	1	2	3
2.	I worry about things that are different from what other young people my age worry about.	0	1	2	3
3.	I feel nervous or jumpy.	0	1	2	3
4.	People say I am violent.	0	1	2	3
5.	I worry more than other young people about being embarrassed.	0	1	2	3
6.	I bully or threaten other people.	0	1	2	3
7.	I get into trouble.	0	1	2	3
8.	My appetite or weight has changed a lot.	0	1	2	3
9.	I have trouble keeping my mind on what people are saying to me.	0	1	2	3
10.	I have trouble sleeping because I am worrying about stuff.	0	1	2	3
11.	I am lonely.	0	1	2	3
12.	I can't make up my mind about things anymore.	0	1	2	3
13.	My muscles get tense when I am worried about something.	0	1	2	3
15.	I have to stay home from college/university or work because of aches and pains.	0	1	2	3
16.	The future seems hopeless to me.	0	1	2	3
17.	I interrupt other people.	0	1	2	3
18.	I am behind in my academic work or tasks at my job.	0	1	2	3
19.	It is easy for me to make mistakes when reading.	0	1	2	3
20.	When I get mad at someone, I get even with them.	0	1	2	3
21.	I make sounds that are hard to control (like clearing my throat or sniffing).	0	1	2	3

22.	Upsetting thoughts or pictures get stuck in my mind and I try to make them go away.	0	1	2	3
23.	I get along with people once I am comfortable with them.	0	1	2	3
25.	I blurt out the answer before the question is finished.	0	1	2	3
26.	I feel very slowed down in my movements.	0	1	2	3
27.	My thoughts come so fast it is hard to keep up with them.	0	1	2	3
28.	I don't like doing things that make me think hard.	0	1	2	3
29.	I feel like I am driven by a motor.	0	1	2	3
30.	I am perfect in every way.	0	1	2	3
31.	I create upsetting thoughts or pictures that get stuck in my mind.	0	1	2	3
32.	I have trouble keeping myself organised.	0	1	2	3
33.	I do what my parents or other adults ask me to do.	0	1	2	3
34.	I like getting gifts.	0	1	2	3
35.	I get worn out with worrying.	0	1	2	3
36.	I know where to get a gun or another serious weapon when I need one.	0	1	2	3
37.	I make mistakes by accident.	0	1	2	3
38.	I have trouble controlling my worries.	0	1	2	3
39.	In maths, word problems are hard for me.	0	1	2	3
40.	I like making threats against other people.	0	1	2	3
41.	People don't show me the respect I deserve.	0	1	2	3
42.	Spelling is hard for me.	0	1	2	3
43.	I steal important things when no one is watching.	0	1	2	3
44.	I worry that other people might laugh at me or make fun of me.	0	1	2	3
45.	I have thoughts or rituals that are unusual.	0	1	2	3
46.	I get panicky when I have to do things in front of other people (like answer questions or give a talk).	0	1	2	3
47.	I get headaches.	0	1	2	3
48.	I destroy stuff that belongs to other people.	0	1	2	3
50.	I eat too much.	0	1	2	3

51.	It is hard for me to sit still.	0	1	2	3
53.	I act like an angel.	0	1	2	3
54.	It's hard to stop myself from doing certain things over and over again (like counting, checking locks or other things, or washing my hands).	0	1	2	3
55.	An awful thing happened to me where I thought I was going to die or get badly hurt.	0	1	2	3
56.	I carry a weapon (like a bat, brick, broken glass, knife, or gun).	0	1	2	3
57.	It is hard for me to think of ideas for stories or papers.	0	1	2	3
58.	I lose my temper.	0	1	2	3
59.	I'm so afraid of some things (like animals, bugs, blood, doctors, water, storms, heights, or places) that it stops me from doing things that I want to do.	0	1	2	3
60.	I steal from other people (by mugging, purse snatching, or armed robbery).	0	1	2	3
62.	I like to set things on fire.	0	1	2	3
63.	I feel like I can't stop talking.	0	1	2	3
64.	I run away from home.	0	1	2	3
65.	I get distracted by things that are going on around me.	0	1	2	3
66.	I have trouble with carrying and borrowing in maths.	0	1	2	3
67.	I skip classes or work.	0	1	2	3
68.	I take drugs that I'm not supposed to.	0	1	2	3
69.	It is hard for me to understand what I read.	0	1	2	3
70.	I have trouble falling asleep.	0	1	2	3
71.	I like to be on the go rather than being in one place.	0	1	2	3
72.	I feel like things are not going well in my life and that I can't do anything about it.	0	1	2	3
73.	I get bullied or picked on.	0	1	2	3
74.	I avoid or get really stressed out about doing things in front of other people.	0	1	2	3
75.	I do dangerous things.	0	1	2	3
76.	I talk too much.	0	1	2	3
77.	When I get mad, I break, throw, or destroy things.	0	1	2	3

78.	I worry about lots of things.	0	1	2	3
79.	I have made plans to hurt others.	0	1	2	3
80.	I tell the truth; I do not even tell “little white lies”.	0	1	2	3
81.	It is hard for me to pay attention to details.	0	1	2	3
82.	I have trouble doing leisure activities quietly.	0	1	2	3
83.	I act okay on the outside, but inside I am unsure of myself.	0	1	2	3
84.	I avoid or get really stressed out by talking to unfamiliar people.	0	1	2	3
85.	I start fights with other people.	0	1	2	3
86.	I am restless.	0	1	2	3
87.	I break into houses, buildings, or cars.	0	1	2	3
88.	I blame others for things I do wrong.	0	1	2	3
89.	I become unusually happy or irritable for a week or longer.	0	1	2	3
90.	I like trying new things.	0	1	2	3
91.	I make mistakes.	0	1	2	3
92.	It is fun to make people look foolish.	0	1	2	3
93.	I don’t feel like doing things that I used to enjoy.	0	1	2	3
94.	Upsetting thoughts or pictures get stuck in my mind and it’s hard to make them go away.	0	1	2	3
95.	I have muscle twitches that are hard to control (like blinking a lot or jerking my head).	0	1	2	3
96.	I tell lies to get out of doing things or to get stuff.	0	1	2	3
97.	I feel like nobody cares about me.	0	1	2	3
98.	I eat things that are not food (like wallpaper, dirt, or garbage).	0	1	2	3
99.	I have trouble waiting for my turn.	0	1	2	3
100.	People like being around me.	0	1	2	3
101.	I have trouble keeping my mind on what I am doing.	0	1	2	3
102.	Reading is hard for me.	0	1	2	3
103.	I have trouble finishing things.	0	1	2	3

104.	I get stomach aches.	0	1	2	3
105.	I smoke cigarettes or chew tobacco.	0	1	2	3
106.	I lose my place when I am reading.	0	1	2	3
107.	I am happy and cheerful.	0	1	2	3
108.	I sleep much less than I used to but I don't feel tired.	0	1	2	3
109.	I suddenly get dizzy, shaky, or sweaty when I am worried.	0	1	2	3
110.	I get out of my seat when I am not supposed to.	0	1	2	3
111.	I mix up my maths signs (like +, -, x, ÷).	0	1	2	3
112.	I am mean to animals.	0	1	2	3
113.	I have trouble keeping my mind on things.	0	1	2	3
115.	I feel sad, gloomy or irritable for many days at a time.	0	1	2	3
116.	I lose stuff that I need.	0	1	2	3
117.	I argue with adults or authority figures.	0	1	2	3
118.	I feel more guilty than I should.	0	1	2	3
119.	Doing things over and over again helps me feel less worried.	0	1	2	3
120.	I don't care if I hurt other people, as long as I get what I want.	0	1	2	3
121.	I feel really tired during the day.	0	1	2	3
122.	I worry about what is going to happen.	0	1	2	3
123.	I'm good at some things.	0	1	2	3
124.	I pull my hair from my scalp, eyelashes, or other places (so much that you can see bald patches).	0	1	2	3
125.	I sleep too much.	0	1	2	3
126.	I get distracted by things that are not important.	0	1	2	3
128.	I have trouble stopping myself from worrying.	0	1	2	3
129.	I have trouble following instructions.	0	1	2	3
130.	When I get mad at someone, I start a fight.	0	1	2	3
131.	I struggle to complete hard tasks.	0	1	2	3
132.	I enjoy myself when I do my favourite activities.	0	1	2	3
133.	I am happy, even when I'm waiting in a long line.	0	1	2	3

134.	I try to annoy other people.	0	1	2	3
135.	I feel worthless.	0	1	2	3
136.	I feel better protected when I am part of a street gang.	0	1	2	3
137.	I feel tired, like I don't have enough energy.	0	1	2	3
138.	I suddenly feel sick or get stomach aches when I'm worried.	0	1	2	3
139.	Something awful has happened and I thought someone was going to get hurt or die.	0	1	2	3
141.	I use stuff around the house, at college/university, or at work to get high (like glue or paint).	0	1	2	3
142.	When I feel nervous, things irritate me.	0	1	2	3
143.	People make me angry.	0	1	2	3
144.	I do things to hurt people.	0	1	2	3
146.	I think about hurting myself.	0	1	2	3
147.	I am no longer able to keep my mind on one thing.	0	1	2	3
148.	I am easily annoyed by others.	0	1	2	3
149.	I suddenly have many more plans and activities than I used to.	0	1	2	3
150.	When I'm worried, I suddenly have trouble breathing, or my heart pounds really fast.	0	1	2	3
152.	I'd rather be by myself when I am supposed to be with other people.	0	1	2	3
153.	I like it when people say good things about me.	0	1	2	3
154.	I forget stuff.	0	1	2	3
155.	I like gossiping and spreading rumours.	0	1	2	3
156.	I don't feel well-rested, even after I sleep all night.	0	1	2	3
157.	People make me so mad that I lose control.	0	1	2	3
158.	I wake up too early (and not just because of the alarm clock or because other people wake me up).	0	1	2	3
159.	I don't eat enough.	0	1	2	3
160.	When I do a good job or when I am interested in something, I like to tell other people about it.	0	1	2	3
161.	I worry about what others think of me.	0	1	2	3

162.	I go out at night even when I'm supposed to be at home.	0	1	2	3
163.	Even when I know the right answer, it is hard for me to write it down.	0	1	2	3
164.	I have lots of fears.	0	1	2	3
165.	I call people mean names.	0	1	2	3
166.	I do things that feel good, no matter what bad things might happen afterwards.	0	1	2	3
167.	I am discouraged.	0	1	2	3
168.	I drink beer, wine, or other alcoholic beverages (e.g., spirits or alcopops).	0	1	2	3
169.	I am a slow reader.	0	1	2	3
170.	I use a weapon (like a bat, brick, broken glass, knife, or gun) to scare or hurt people.	0	1	2	3
171.	I feel really good, like I'm better than everyone else and I can do anything.	0	1	2	3
172.	I get even with people.	0	1	2	3
173.	Maths is hard for me.	0	1	2	3
174.	I worry about little things.	0	1	2	3

Think about your answers so far and then answer the next three items.

175.	I have problems that make college/university or work really hard for me.	0	1	2	3
176.	I have problems that make friendships really hard for me.	0	1	2	3
177.	I have problems that make things really hard for me at home.	0	1	2	3

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