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The Relationship between Child ADHD and Maternal Expressed Emotion: A longitudinal Analysis of Child and Family Effects

by

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ABSTRACT

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High parental expressed emotion (EE) is often associated with ADHD in childhood. However, the direction of causation in the relationship is not well understood: is it the behaviour of the child with ADHD (i.e., child effects) or shared characteristics of the parent or family more generally that are independent of a specific child (i.e., family effects) that predict parental EE? Furthermore, does parental EE predict child problems over time? In this thesis, child and family effects on maternal EE and child problems and the specific child and family characteristics that explain these effects were examined using cross-sectional and longitudinal multilevel models of sibling pair data in families of children with ADHD sampled from a longitudinal study. The results revealed a complex picture with both child and family effects implicated in predicting both maternal EE and child behaviour. Studies 1 and 2 (which cross-sectionally used Time 1 [T1; n = 72 families] and Time 2 [T2] data [n = 48 families] respectively) and the longitudinal analysis of Study 3 (n = 45 families) demonstrated that, except for warmth, child effects were stronger in predicting maternal EE. Child effects seemed to be driven by oppositional/conduct problems (OPP/CP) and emotional problems, rather than ADHD per se. Mothers' depressive symptoms and overall family levels of child OPP/CP largely predicted family effects on maternal EE. Study 4 (n = 45 families), the second longitudinal analysis, found similar T1 child and family effects on T2 child problems. Increase in negative maternal EE from T1 to T2 significantly predicted T2 child OPP/CP. T1 family effects on T2 child problems were predominantly predicted by T1 maternal ADHD symptoms and average family (i.e., sibling pair) levels of EE. The results suggested a potential causal role of both child (especially OPP/CP) and family effects (especially average family levels of child OPP/CP) in predicting maternal EE. In addition, high EE may be a risk factor for child OPP/CP over time and maternal ADHD for both behavioural and emotional child problems. This may have important clinical implications for interventions with families of children with ADHD.

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Academic Thesis: Declaration of Authorship

I, Kim Cartwright, 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.

The Relationship between Child ADHD and Maternal Expressed Emotion: A Longitudinal Analysis of Child and Family Effects

I confirm that:

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Definitions and Abbreviations

ADHD	Attention-Deficit/Hyperactivity Disorder
APA	American Psychiatric Association
CFI	Camberwell Family Interview
CD	Conduct Disorder
CBCL	Child Behaviour Checklist
CBRS	Comprehensive Behaviour Rating Scale
CPRS-R: L	Conners Parent Rating Scale – Revised, Long Version
CTRS-R: L	Conners Teacher Rating Scale – Revised, Long Version
CC	Critical comments
CRIT	Criticism
CSS-SR	Current Symptoms Scale – Self-Report Form
DBD	Disruptive behaviour disorders
DEP	Depressive symptoms
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders
EDP	Emotional dysregulation problems
EP	Emotional problems
EOI	Emotional over-involvement
EE	Expressed emotion
FMSS	Five Minute Speech Sample
GHQ-12	General Health Questionnaire – 12-item Version
HADS	Hospital Anxiety and Depression Scale
IMAGE	International Multicentre ADHD Genetics project
IS	Initial statement
MDD	Major Depressive Disorder

MLM	Multilevel modelling
OCD	Obsessive-compulsive disorder
ODD	Oppositional Defiant Disorder
OPP/CP	Oppositional/conduct problems
PACS	Parental Account of Childhood Symptoms
PC	Positive comments
REL	Relationship
Sz	Schizophrenia
SDQ	Strengths and Difficulties Questionnaire
SES	Socioeconomic status
T1	Time 1
T2	Time 2
WAR	Warmth

Chapter One

ADHD and its Developmental Course

1.1 Overview of Chapter One

Chapter 1 presents a review of the literature pertaining to attention-deficit/hyperactivity disorder (ADHD) in childhood and its developmental course. Initially, the disorder is defined and its core symptoms are set out followed by descriptions of its prevalence, diagnostic criteria and subsets. This is followed by an overview of comorbid psychiatric conditions and functional impairment associated with the condition in childhood. Patterns of homotypic and heterotypic continuity are then discussed with particular focus on findings from controlled prospective follow-up studies which have tracked the progress of children with ADHD into adolescence and adulthood. Finally, a brief introduction to the literature reviewed in Chapter 2 on the relationship between parental expressed emotion (EE) and child behaviour, with a specific focus on its relationship with ADHD, is provided.

1.2 Introduction to ADHD

ADHD is a clinically heterogeneous and highly prevalent neurodevelopmental disorder with a complex aetiology that manifests across the lifespan and is characterised by developmentally inappropriate, enduring and impairing inattention, hyperactive and impulsive behaviours (Barkley, 1998). The substantial economic, emotional and social burden the disorder imposes on the individual, their families and society alike makes it a considerable public health problem and has led to it being the most intensely researched mental health condition. The inattention dimension of ADHD is expressed as poor sustained attention (e.g., problems maintaining attention on/listening to what people are saying, following instructions, and sticking to and finishing tasks and activities, particularly those that require greater mental effort) and focused attention (e.g., distractibility, trouble paying close attention to detail, and often making accidental mistakes), difficulty keeping organised, often losing things and forgetfulness in daily activities. The hyperactive component is manifested as developmentally inappropriate levels of motor activity such as fidgeting, problems remaining seated, restlessness, running or climbing that is unfitting to the situation, often being "on the go" and as excessive levels of vocal activity such as

talking too much and having trouble playing or doing things quietly. The impulsive element presents as responses that lack sufficient planning or control such as not being able to wait one's turn, interrupting or intruding on others in conversation or activities and blurting out answers before questions have been asked.

1.3 Epidemiology of ADHD

Epidemiological data on the global and European incidence and prevalence of ADHD is somewhat limited and prevalence estimates of the disorder have been reported in numerous literature reviews to vary considerably between different countries across the world from as low as 1% to as high as almost 20% among school-aged children (Faraone, Sergeant, Gillberg, & Biederman, 2003). Reasons for this discrepancy remain poorly understood and are discussed further on in this section. Most recently in the broadest systematic review of this subject to date and consistent with the majority of previous reviews on this topic (Faraone et al., 2003; Scahill & Schwab-Stone, 2000; Szatmari, 1992), the worldwide pooled prevalence was estimated at 5.29% (Polanczky, Silva de Lima, Horta, Biederman, & Rohde, 2007), making ADHD the most common childhood disorder. It is now well documented that ADHD manifests early in life and is present across the lifespan with prevalence rates of 2-6% in preschool children (Schmidt & Petermann, 2009), 6-13% in adolescents (Skounti, Philalithis, & Galanakis, 2007) and 2-5% in adults (Kooij, Bejerot, Blackwell, et al., 2010).

In general, ADHD is more prevalent in males than females with gender incidence ratios found to range from 2:1 to 9:1 (DSM-IV-TR; American Psychiatric Association, 2000). This is dependent on the subset of ADHD and setting. The ratio is higher in studies of clinic-referred children than in community-based epidemiological studies. For example, the ratio of boys to girls with ADHD in community studies has been found to be 4:1(Cantwell, 1996). Referral bias related to symptoms of disruptive behaviour may account for differences between sexes since boys have more hyperactive/impulsive symptoms and more conduct and oppositional symptoms than girls (Swanson, Sergeant, Taylor, Sonuga-Barke, Jensen, & Cantwell, 1998). It may also be due to girls being under-identified or because girls' symptoms are different to those in boys. Biederman et al. (2002) documented inattentive type ADHD is more common in girls than boys, the risk for learning disability and problems at school is less in girls as is comorbid depression. In adults, similar prevalence is found in both genders.

Returning briefly to the reasons for different prevalence rates of ADHD, several assumptions have been made. First, the high variability in estimates have been attributed to geographical and demographic factors (Rappley, 2005). In the past, ADHD has often been

perceived as an American disorder partly because the US has dominated research in this field, but also due to the higher prevalence rates found in North America compared to other nations (Anderson, 1996; Timimi & Taylor, 2004). Experts consequently debated at length the possibility that ADHD might be the result of cultural factors. However, evidence from several studies has confirmed a lack of difference in prevalence rates in ADHD between American and non-American populations (Faraone et al., 2003; Taylor, 1986; Fergusson, Horwood, & Lynskey, 1993; Taylor & Sandberg, 1984).

Second, an alternative school of thought has been that the variability of estimates is most likely due to differences in how cases are defined. Furthermore, variability of actual prevalence across geographical sites should not exist when case definitions used in different studies are the same. Whereas at present, for example, some studies use the World Health Organisation (WHO, 1992) ICD-10 (especially in Europe), others use the American Psychiatric Association (APA, 2004) Diagnostic and Statistical Manual for Mental Health Disorders Fourth Edition (DSM-IV) definition of ADHD. Furthermore, numerous revisions have been made to the definitions by APA and WHO over the years. Studies also vary in the degree to how vigorous they are in applying the specifics of the diagnostic criteria with some investigators not adhering to the requirement for symptoms to be present in at least two settings or for the presence of symptom caused functional impairment.

Third and similarly, methodological differences across studies such as method of ascertainment, diagnostic systems and associated criteria (e.g., situational versus pervasive, degree of impairment), informants (i.e., parents and/or teachers), the population studied (i.e., community vs. school samples) have been identified as contributing to differences in rates. In a systematic review and metaregression analysis, Polanczky et al. (2007) compared 171,756 subjects from regions on all continents of the world to determine factors implicated in the variability of estimates. The large variability of ADHD prevalence rates worldwide was explained primarily by methodological differences rather than geographic location: there was significant variability in rates between North America and both Africa and the Middle East, but not between North America and Europe. Similar rates between countries were found especially when the same diagnostic criterion and methodological procedures were followed. The authors noted that although culture does play a role in the aetiology of ADHD, the findings do not support the view that ADHD is a culturally-based construct to the North American culture.

1.4 Diagnostic Criteria and Subsets of ADHD

There has been considerable change to the terminology and definitions ascribed to ADHD with each successive modification to the APA's DSM and further modifications might

occur with the forthcoming publication of the DSM-5. However, the currently used version of the manual, the DSM-IV, allows ADHD to be classified into three subtypes/domains. These are:

(1) ADHD combined type (ADHD-C) which requires a minimum of six out of the nine aforesaid inattention symptoms and at least six of the above mentioned symptoms of hyperactivity/impulsivity

(2) ADHD predominantly inattentive (ADHD-I) which requires only the criteria for inattention to be met (i.e. six or more inattention symptoms)

(3) ADHD predominantly hyperactive-impulsive type (ADHD-HI) which requires only the criteria for hyperactive-impulsive to be met.

Diagnostic confirmation also requires symptom onset before the age of 7, presence of symptoms in two or more settings and sufficient evidence of impairment in social or academic functioning. The age-of-onset criterion has, however, received harsh criticism with some authors suggesting it is too strict and in need of revision (Faraone, Biederman, & Mick, 2006). Its validity has been thrown into question as a result of mounting empirical data providing evidence of late-onset ADHD (i.e., onset of symptoms after aged 7) as a valid diagnosis. For example, in one study no association was found between age at onset and severity of symptoms, types of adjustment difficulties or the persistence of the disorder in adolescents with onset before 13 and those with onset after 13 (Schaughency, McGee, Nada Raja, Feehan, & Silva, 1994). Furthermore, other studies have not found any differences regarding impairment between children who meet all the criteria for full ADHD and children with onset after age 7 (Rohde et al., 2000; Willoughby, Curran, Costello, & Angold, 2000). Some authors have also raised the issue that the DSM-IV does not explain cognitive (executive functioning) deficits associated with ADHD (Barkley 1997a).

1.5 Problems Associated with ADHD in Childhood

1.5.1 Psychiatric Comorbidity

Psychiatric comorbidity is a well-recognised key clinical feature observed in children with ADHD. It is much more common to find a child with comorbid ADHD than with ADHD alone (Biederman, Newcorn, & Sprich, 1991; Hazell, 1997, 2010; Kadesjo & Gillberg, 2001). Similar to previous findings of prevalence estimates of comorbidity (Jensen et al., 2001), recently, in the US, a study found that two thirds of school-aged children with a diagnosis of ADHD, as reported by their parents, were more likely to also have each of ten

other mental health and neurodevelopmental disorders (Larson, Russ, Kahn, & Halfon, 2011). The most commonly co-occurring disorders reported include disruptive/antisocial behaviour disorders, specifically oppositional defiant disorder (ODD) and conduct disorder (CD), reported to occur in 30% to 50% and 50% of children with ADHD in community and clinical samples respectively and internalising disorders such as mood and anxiety disorders (August, Realmuto, MacDonald, Nugent, & Crosby, 1996). Developmental disorders such as speech and language delays and learning disabilities are prevalent in children with ADHD too. The results of some studies have suggested that coexisting conditions are more frequent in children with the predominantly hyperactive-impulsive and combined subtypes (Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996).

It is widely documented that children with comorbid ADHD who also have other comorbid conditions have more negative outcomes compared with children who have ADHD alone. Children with ADHD and conduct problems often display a more diverse set of aggressive behaviours (Waschbusch, 2002), experience greater levels of peer rejection and academic underachievement (Hinshaw, 1992), are at greatest risk of chronic criminal offending (Lynam, 1996) and are at increased risk of substance use (Molina & Pelham, 2003; August, Winters, Realmuto, Fahnhorst, & Botzet, 2006). Although a less severe condition than CD, research has shown that ODD accounted for more numerous and intense family conflicts, negative parent-child interactions and higher ratings of maternal psychological distress among children with ADHD compared to controls (Barkley, Fischer, Edelbrock, & Smallish, 1991).

1.5.2 Functional Impairment

Over and above the heightened risk for comorbid psychiatric disorders children with ADHD are also more susceptible to problems in a range of functional domains. Most notably in preadolescents are impairments in educational and school functioning and social functioning. First, in relation to academic functioning, there is substantial evidence that very young and older children with ADHD are often educationally disadvantaged and experience a number of behavioural and social problems at preschool or school. DuPaul, McGoey, Eckert, and Van Brakle (2001) demonstrated that even prior to formal school entry preschool children scored lower on tests of pre-academic skills than children without the disorder. In addition, pre-schoolers with ADHD showed more noncompliant and inappropriate behaviour, especially during task situations. Numerous studies across different countries have shown poor academic performance including lower scores on academic standardised tests including reading and arithmetic achievement tests in children with ADHD compared to controls and in children who manifest symptoms of ADHD, but

who have not had a formal diagnosis of ADHD (Biederman et al., 1996; Merrell & Tymms, 2001; Fergusson, Lynskey, & Horwood, 1997). Children with ADHD are often also more likely to be expelled, suspended, repeat a year and use special educational services compared to controls (Farone et al., 1993; LeFever, Villers, Morrow, & Vaughn III, 2002).

More specifically, several studies using observational measures have shown that inattention symptoms often lead to off-task behaviour in the classroom including failure to listen to classroom or task instructions, forgetting to complete and turn in, losing or failing to finish assignments, frequent distraction from tasks and difficulty returning to activities once interrupted and more errors on tasks over time (Mash & Barkley, 2003; Shelton et al., 1998; Zentall, 1993). These studies have also found that compared to controls hyperactivity symptoms cause children with ADHD difficulty staying seated in the classroom, excessive fidgeting, touching objects more frequently, playing noisily, increased discipline, negative teacher attributions and lower levels of task completion. Furthermore, impulsivity symptoms often result in poor planning when studying for tests and completing long-term projects, failure to read directions or ask for help because this requires waiting and as a consequence often making errors because of not waiting long enough to consider the information.

Evidence suggests that in clinical samples, children with ADHD have an intelligence quotient (IQ) score nine points lower than children with ADHD (Frazier, Demaree, & Youngstrom, 2004) and a correlation of -.02 to -0.4 between dimensional measures of ADHD symptoms and IQ has been reported in general population samples (Fergusson et al., 1993; Goodman et al., 1995; Rapport et al., 1999). However, studies have shown that the effects of maladaptive behaviour tend to outweigh the effects of low IQ on later social and psychological outcomes. For example, data collected in New Zealand during the course of a 25-year longitudinal study on a birth cohort during middle childhood indicated that parent and teacher reported conduct problems at ages 7-9 years predicted a range of adverse psychosocial outcomes at age 21-25 years even after controlling for confounding factors including IQ (Fergusson, Horwood, & Ridder, 2005). However, conduct problems did not predict educational and occupational outcomes when early attentional problems and childhood IQ were controlled. The results of this more recent study, for the most part, contradicted earlier findings from the National Longitudinal Study of Youth a decade earlier which revealed strong associations between IQ and educational achievement, crime, welfare dependence, family functioning and similar outcomes (Herrnstein and Murray, 1994).

Second, with respect to social functioning, peer relationships and interactions with family members often suffer in children with ADHD. In preschool settings, children with ADHD have been shown to be less socially skilled, displaying more negative social behaviour than controls (DuPaul et al., 2001). Blachman and Hinshaw (2002) found that over a 5-week period, at each assessment period school-aged girls with ADHD had fewer friends, were more likely to have no friends and those that were able to make friends had more problems maintaining friendships and there were higher levels of conflict and relational aggression in their friendships. High rates of rejection found among children with ADHD, in particular boys, have been attributed to their intrusive, disruptive behaviours and limited understanding of how their behaviour impacts on others (Hinshaw & Melnick, 1995; Pelham & Bender, 1982). Indeed, Pelham & Bender (1982) reported that peer rejection often occurs nearly instantly upon social contact and even children with ADHD who do not display antagonistic behaviour tend to be rebuffed due to their ardent and insensitive behaviours. Children with ADHD also often facilitate negative social interactions among their peers (Whalen & Henker, 1985).

The difficult behaviour of children with ADHD often makes family relationships challenging. Parents of children with ADHD often find the symptoms of ADHD difficult to cope with (Woodward, Taylor, Downey, 1998) and as a consequence often experience heightened levels of stress (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; Baker & Heller, 1996; DuPaul et al., 2001; Mash & Johnston, 1982). Moreover, parental stress has been shown to affect the ability of parents of children with ADHD to adopt positive parenting practices to foster co-operative behaviour from their child (Dix, 1991), making parents more prone to use dysfunctional parenting strategies (Kendziora & O'Leary, 1993). Indeed, parents of children with ADHD often have more directive and negative, less socially engaged (Campbell, Breaux, Ewing, Szumowski, & Pierce, 1986; DuPaul et al., 2001) and have harsher and more demanding interactions with their children (Buhrmester, Comparo, Christensen, Gonzalez, & Hinshaw, 1992).

1.6 Developmental Course: ADHD in Adolescence and Adulthood

Contrary to the commonly-held belief that ADHD was a condition exclusive to childhood, it is now widely accepted that ADHD, particularly inattention symptoms, persists into adulthood (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Biederman, Mick, & Faraone, 2000; Swanson et al., 1998). On one hand, retrospective studies have provided evidence of the continuity of ADHD in adulthood; however, these studies are limited due to their reliance on retrospective diagnoses which are often unreliable and whose validity cannot be assessed (e.g., by comparing adult self-reports of childhood memories to those

obtained from a knowledgeable informant such as the individual's parent). On the other hand, controlled prospective follow-up studies of children into adolescence and adulthood have played a pivotal role in providing evidence for the validity of the ADHD diagnosis in adults. Importantly, they have also promoted the diagnosis and treatment of previously undiagnosed and untreated patients. Indeed, the initial many doubts and unwillingness to accept the notion of continuity of ADHD into adulthood led to adults with ADHD being largely undiagnosed and treated. First, this may have resulted at least in part because the clinicians who treat children and adolescents (i.e., paediatricians and child psychiatrists) are different to those who treat adults (i.e., general or family practitioners and general psychiatrists) and because of the lack of or limited teaching or training in adult ADHD provided to medical students until recently. Second, given the increase in comorbidity with age, ADHD often goes unnoticed and instead diagnoses and treatment for comorbid conditions are made in adults with ADHD. Several controlled, prospective studies have tracked the long-term progress of large samples of clinic-referred children into adolescence and/or adulthood and in doing so retained 50% or more of their original samples and had matched control groups to examine the persistence of the disorder. These are described first and then followed by a discussion of adolescent and adult outcomes in ADHD based on the findings of these studies.

1.6.1 Controlled Prospective Follow-up Studies

The first and earliest of these studies was the Montreal follow-up study which first reassessed 91 hyperactive children in early adolescence five years after initial assessment at the age of 6 to 12 years (Minde et al., 1971; Minde, Weiss, & Mendelson, 1972; Weiss, Minde, Werry, Douglas, & Nemeth, 1971). Seventy five hyperactive males of the original 103 and 44 matched controls of this cohort were assessed again at 10 years in late adolescence/early adulthood (aged 17 to 24 years) (Hechtman, Weiss, Perlman, Hopkins, & Wener, 1979; Weiss, Hechtman, Periman, Hopkins, & Wener, 1979). At the last wave of data collection 15 years later, 63 hyperactive adults and 41 matched controls of the cohort were ascertained for assessment (Weiss, Hechtman, Milroy, & Perlman, 1985).

In New York, another group of researchers recruited 207 boys (aged 6 to 12 years) diagnosed with DSM-II ADHD at a child psychiatric research clinic following referral by teachers because of behaviour problems between 1970 and 1977, divided them into two cohorts of 103 and 104 hyperactive boys and compared them in adolescence and adulthood with roughly equal numbers of controls matched on age and gender. At nine-year follow-up, 98% of the first cohort were reassessed when they were 16 to 23 years old (Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Mannuzza, Klein, Bonagura, Konig, & Shenker,

1988; Mannuzza, Klein, Konig, & Giampino, 1989) and 91 were reassessed in adulthood at a mean age of 26 years 16 years later (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993). From the second cohort, ninety four males were seen in adolescence (Mannuzza et al., 1991) and 85 at a mean age of 24.1 years in adulthood (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998).

A Swedish study by Rasmussen & Gillberg (2000) was the first community-based follow-up study of 62 males and 39 females aged 22 years who at the age of 6 were identified from the general population as having ADHD with developmental coordination disorder (DCD), ADHD alone or DCD only.

In 1987 to 1988 the Milwaukee study by Barkley, Fischer, Edelbrock, and Smallish (1990), followed-up at 8 years in adolescence 78% (123 of 158) and 81% (66 of 81) of an original sample of hyperactive and control children respectively initially recruited between 1979 and 1980. One hundred and forty seven hyperactive children and 73 controls were reassessed in 1992 to 1996 in young adulthood at a mean age of 20 to 21 years (Fischer, Barkley, Smallish, & Fletcher, 2002; Barkley, Fischer, Smallish, & Fletcher, 2004).

In Los Angeles, Satterfield, Hoppe, and Schell (1982) followed up into adolescence 110 boys who were diagnosed with attention-deficit disorder (ADD) between the ages of 6 and 12 years and 89 controls and subsequently reassessed this cohort in young adulthood when 19 to 25 years old and again at 32 to 42 years old 30 years after baseline assessment in 1970 to 1973.

Lastly, in the Iowa study, as part of an original family-genetic study of ADHD, 140 children with ADHD and 120 controls were originally assessed between 6 and 7 years old and then subsequently 4 years later in early adolescence (Biederman et al., 1996) and 10 years later in young adulthood (Biederman et al., 2006, 2008).

1.6.2 Homotypic versus Heterotypic Continuity

The co-occurrence of other disorders with ADHD makes it a particularly complex disorder to understand in terms of its developmental course. Subgroups of children with ADHD might be defined on the basis of the disorder's comorbidity with other disorders and these subgroups may have differing risk factors, clinical course and pharmacological response (Biederman, Newcorn, & Sprich, 1991). The continuity of the disorder may be homotypic, that is, a form of psychopathology which is consistently displayed over time, but the specific symptoms may have somewhat different manifestations over time. The persistence of the condition may also be heterotypic. In other words, one form of psychopathology (i.e., in this case ADHD) may contribute to the emergence of a different form of psychopathology. With this in mind, the continuity of ADHD into adolescence and

adulthood is discussed below in relation to the findings of the aforementioned controlled, prospective follow-up studies. These studies have addressed outcome in adolescence and adulthood in the domains of psychiatric status, educational and occupational functioning, social functioning, criminality and self-esteem.

1.6.3 Psychiatric Status in Adolescents and Adults with ADHD

In both cohorts of the New York study combined, at young adult follow-up, more than twice as many individuals with ADHD as controls had an on-going psychiatric disorder (50% vs. 19%). Three disorders significantly discriminated between groups: attention deficit disorder with or without hyperactivity 37% vs. 3%), antisocial personality/conduct disorder (APD, 29% vs. 8%) and non-alcohol substance use disorder (13% versus 2%), which in nearly all cases involved marijuana. Psychiatric status at follow-up in adolescence and adulthood is described in more detail below.

ADHD (Homotypic Continuity)

In relation to the persistence of ADHD symptoms, controlled, prospective follow-up studies have consistently demonstrated that a significant proportion of children with ADHD continue to meet the diagnostic criteria for the disorder in adolescence and adulthood. The Montreal study reported that more than half the hyperactive adult males followed up after 10 years had at least one impairing symptom, almost half manifested restlessness (fidgeting or changing sitting position frequently) during the interview as rated by psychiatrists and half had not outgrown all aspects of the disorder (Weiss et al., 1979). At 15-year follow-up, at a mean age of 25, one third of ADHD subjects compared to 2% of controls reported at least one symptom as moderately or severely disabling at adult follow-up (Weiss et al., 1985).

In the first New York cohort, full ADHD persisted in 31% of probands versus 3% of controls at 10 years follow-up (Gittelman et al., 1985). At a mean age of 26, the proportion of probands with full ADHD fell to 11% and in controls the percentage remained the same (Mannuzza et al., 1993). The Swedish study yielded similar results; severe hyperactivity was present in 15% of the index groups and 2% of comparison group. Severe inattention was present in 44% of the index groups and 7% of the comparison group. Continuation of both hyperactivity-impulsivity and inattention was seen in 9% of the index groups and no individuals in the comparison group. Half of the index cases and 9% of the individuals in the comparison group had marked symptoms at 22 years. These findings support the notion that whilst hyperactivity symptoms tend to decrease in adolescence and adulthood, inattention symptoms such as sustained attention, concentration and organisational

problems are likely to remain prominent and even become more apparent. This was also shown in the Milwaukee study (Fischer, Barkley, Edelbrock, & Smallish, 1990).

The results of the second cohort of the New York study showed the lowest rates of ADHD compared to the other prospective follow-up studies with 4% of adults previously diagnosed in childhood with ADHD still meeting the criteria for full ADHD at follow-up and none reporting clinically impairing symptoms in the absence of the full disorder (Mannuzza et al., 1998). In contrast, Barkley et al. (1990) found much higher rates of continuity with 80% of their sample at 8 years follow-up in adolescence still diagnosed with ADHD. The Milwaukee study reported widely discrepant findings. When self-reports were used, only 3% of ADHD subjects met full criteria for ADHD. This rate rose to 27% when diagnoses were based on parent reports. Differences found between these studies may be attributable to differences in attrition rates (e.g., attrition was 40% versus 15% in the Montreal and New York studies respectively), whether or not interviewers were blind to group membership (e.g., in the Montreal and Milwaukee studies, interviewers were not blind to group membership) and ascertainment procedures such as the age of individuals at follow-up.

Antisocial Personality Disorder (Heterotypic Continuity)

Prospective longitudinal studies have reliably found antisocial personality disorder (APD) to be the most prevalent disorder in grown-up children with ADHD with rates in adulthood across studies averaging around 26% (versus 2-7% in controls). In Weiss and colleagues' (1985) cohort, APD was the only DSM-III diagnosis that was significantly more prevalent at follow-up in 21 to 23 year olds with ADHD compared to controls (23% versus 2.5%) and the only other disorder that distinguished the two groups. Although APD was the most common diagnosis in the group with ADHD in the New York study, the lowest rates of the disorder were found in this study. In both cohorts combined 15% of individuals with ADHD versus 2% controls had APD at follow-up. The authors commented that differences between theirs' and others' findings might be due to the levels of CD in the initial childhood groups. Their cohort may have been relatively pure with respect to ADHD and the absence of CD because children were excluded at entry to the study if the primary reason for school referral involved aggressive or other antisocial behaviours. The other follow-up studies report relatively high rates of CD in their initial samples. They also made two conclusions. First, based on the results of the New York study, childhood ADHD with or without CD is at significantly increased risk for APD in adulthood. Second, based on rates of APD in other studies, childhood CD may increase the risk for APD among children with ADHD.

Substance Use and Substance Abuse Disorders (Heterotypic Continuity)

Findings with regard to adolescent and adult substance use in children with ADHD present a more complex picture. In the 10-year Montreal follow-up study, grown-up children with ADHD in their late teens had tried more nonmedical drugs (mainly hashish) than controls; however, group differences were not statistically significant in the year preceding reassessment (Weiss et al., 1979). In the same cohort, at 15-years follow-up, no differences between groups were found with respect to alcohol abuse. Similarly, at four-year follow-up in mid-adolescence, no differences in the rates of alcohol or drug abuse dependence were found between subjects with ADHD and controls in the Iowa study (Biederman et al., 1996). The Milwaukee study found higher rates of self-reported cigarette smoking in teenagers with ADHD than controls at 8-year follow-up as well as higher rates of marijuana use, but only in adolescents with ADHD and comorbid CD (Barkley et al., 1990). However, there were no significant group differences in parent or self-reported alcohol or drug use (other than marijuana use). Although in the Swedish study 24% of the index group versus 4% of the control group had abused alcohol, differences between groups in substance abuse disorders did not reach statistical significance (Rasmussen & Gillberg, 2000).

In contrast, the results of the New York study showed that in the first cohort more than half of the young adults with ADHD versus 3% of controls in the first wave of follow-up had an on-going substance use disorder (Gittelman et al., 1985) and in the second wave they were five times more likely than controls to have an on-going drug abuse disorder with marijuana and cocaine the most frequently used substances in both groups (Mannuzza et al., 1993). Similar results were found in the second wave of follow-up with the second cohort (Mannuzza et al., 1998). Indeed in the latter follow-up non-alcohol substance use disorder was one of the most common diagnoses found in the cohort and high rates of comorbid substance use disorders were found in the ADHD group with antisocial personality disorder (60%) compared to 13% without antisocial personality disorder.

Mood and Anxiety Disorders (Heterotypic Continuity)

The New York study found that in both the ADHD and control groups, affective and anxiety disorders were rare at follow-up in late adolescence (Gittelman et al., 1985). Similarly in their other cohort, at adult follow-up rates of mood and anxiety disorders did not differ significantly between groups (Mannuzza et al., 1998). Interestingly rates of mood disorders were nearly identical, anxiety disorders were more common in the control group and there were was no specific mood or anxiety disorder that distinguished the two groups.

Although Rasmussen and Gillberg (2000) found that major depression was very common in all groups, only three individuals had current depression all of whom were in the ADHD and DCD group. In the Montreal study, at the 15-year follow-up subjects in the ADHD group made significantly more suicide attempts and one subject successfully committed suicide (Weiss et al., 1985). In contrast to the New York Study, in the Iowa study, lifetime risk for major depression was elevated in adults with ADHD compared to controls at 10-year follow-up (Biederman et al., 2006), results that were consistent with the Milwaukee study (Fischer et al., 2002). A similar pattern was also found for anxiety disorders.

1.6.4 Functional Impairment in Adolescents and Adults with ADHD

Educational and Occupational Functioning

The aforementioned academic difficulties reported in short-term follow-up studies of children with ADHD in early to middle adolescence continue into young adulthood. These studies fairly consistently have shown that, compared to normal controls, children with ADHD exhibit impaired academic functioning (obtain lower test scores, more often repeat grades, etc.) and perform more poorly on cognitive tasks. In young adulthood, compared to their peers, ADHD subjects complete less formal schooling, achieve lower grades, fail more courses, perform worse on standardised achievement tests and are more likely to attend special schools. For instance, the Montreal study found that at follow-up in late teenhood/early adulthood, subjects with ADHD had completed less education and were less likely to finish high school than controls (Weiss et al., 1979). Furthermore, only about 15% of individuals with ADHD (versus half of comparisons) completed a bachelor's degree or higher and 3% (versus 15-16% of controls) were enrolled in, or had completed a graduate degree by their mid-twenties.

Similarly, in the New York study, young adults with ADHD had completed 2.5 years less schooling than controls, one quarter versus 2% had dropped out of school by 11th grade and 12% versus nearly half of controls had completed a bachelor's degree or higher (Mannuzza et al., 1993). Both the Montreal and New York studies found that ADHD subjects had significantly lower occupational ranks than controls. Differences were primarily attributed to a greater proportion of comparisons occupying higher-level positions than ADHD subjects. But these two studies did not find any significant differences between groups in rates of employment. Although 90% of adults in both groups were employed at follow-up or full-time students at mean age of 25 years, subjects with ADHD had significantly lower occupational rankings, significantly fewer held professional positions (e.g., lawyer, account). In fact, whereas a large proportion of adults diagnosed in

childhood with ADHD were owners of small businesses, the largest proportion of controls were accountants and stockbrokers.

Social Functioning and Self-Esteem

The previously mentioned poor social functioning characteristic of children with ADHD often continues into adolescence and adulthood. In early to middle adolescence children with ADHD are more often characterised by low self-esteem and poor social functioning (e.g., described by their mothers as having no steady friends). In their late teens to early adulthood, children with ADHD have fewer friends. In the Montreal study, grown-up children with ADHD scored significantly worse than controls at follow-up in adolescence, early adulthood and adulthood based on the same self-esteem and social skills tests administered at all three time points (Weiss et al., 1978; Weiss et al., 1979; Weiss et al., 1985). In the first reassessment they continued to have impulsive approaches to cognitive tasks (Hopkins, Perlman, Hechtman, & Weiss, 1979). In the Milwaukee study, at follow-up in mid to late adolescence, individuals with ADHD were rated by their mothers as having a greater number and more intense family conflicts than controls, although adolescents' self-reports of these conflicts did not differ between groups. More negative and controlling behaviours and less positive and facilitating behaviours during mother-child interactions were observed in the group with ADHD compared to the control group (Barkley et al., 1991).

Criminality

In general, prospective follow-up studies have demonstrated that individuals with ADHD fare poorly compared to controls on indices of criminality. At the second wave of reassessment in late adolescence and young adulthood, the Montreal study showed that grown-up children with ADHD had had more court appearances than controls, but this difference was not statistically significant in the year preceding reassessment. The New York and Los Angeles studies both used an objective index of the severity and pervasiveness of antisocial behaviour; they obtained official arrest measures (which are less susceptible to the usual sources of unreliability which sometimes characterise interview data –e.g., forgetting, minimising, denying, selective recall etc.). In the New York study, compared to controls, more individuals in the ADHD group had been arrested, convicted and imprisoned than controls in young adulthood. In this cohort, continuing ADHD alone at follow-up was not related to arrest history. Instead, the increased risk for criminality was almost completely accounted for by the presence of antisocial/conduct disorder (Mannuzza et al., 1989). In fact, when subjects without an antisocial disorder were

compared, ADHD and control groups did not differ significantly in arrest rates (28% vs. 16%). These results suggest that ADHD per se may not be a risk factor for criminality as it tends to be those who develop antisocial disorders that are more likely to engage in criminal acts. In the second cohort of the New York study, five subjects with ADHD were imprisoned at follow-up versus no controls. In the longest follow-up study to date, the Los Angeles study found that although the majority of subjects in the ADHD group did not become adult criminals, the high rate of adult arrests (44%), convictions (29%) and incarcerations (26%) indicated that a substantial subgroup became serious adult offenders (Satterfield et al., 2007).

1.7 Summary of Chapter One

The literature reviewed in this chapter demonstrates that ADHD is a highly prevalent and comorbid disorder that persists into adulthood and is associated with a range of functional impairments. In particular, long-term controlled prospective follow-up studies have documented that symptoms of the disorder, comorbid psychiatric problems and associated academic, social and emotional impairment continue into adolescence and adulthood and that other problems (e.g., substance misuse, criminality) also often emerge in children with ADHD as they grow up. Furthermore, these studies have laid the groundwork for the validity of the diagnosis of ADHD in adulthood. Within the developmental psychopathology framework, there is great emphasis on identifying risk factors that may influence negative trajectories or indeed protective factors that may foster positive long-term outcomes in children. This is particularly important for optimising preventive and treatment strategies for children with ADHD and their families. The next chapter provides a review of the literature pertaining to expressed emotion (EE), an aspect of the emotional atmosphere of the family environment, which has been identified as a potentially important environmental risk factor that contributes to the course and outcome of ADHD.

Chapter Two

Child ADHD and Parental Expressed Emotion: Child and Family Effects

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2.1 Overview of Chapter Two

Chapter 2 presents a review of the literature in the area of parental expressed emotion (EE) in paediatric and teen populations with specific emphasis on children and adolescents with ADHD. Initially, gene-environment interplay, specifically gene-environment correlations (rGEs) and gene-environment interactions (GxE) in explaining behaviour and child psychopathology is discussed, in addition to the role of family processes in child psychopathology, to provide a backdrop to the importance of research in this area. Following this, the EE concept is introduced and defined, its origins described and an overview of the methodology used to measure the construct is provided. Empirical evidence pertaining to the relationship between parental EE and child behaviour, including ADHD, and other child characteristics and parent-related/family characteristics is then discussed. Following this, four theoretical models are proposed to explain the ways that parental EE and ADHD might be related to each other. Finally, the primary aim of the first two studies of the thesis is set out in addition to the aim of the next chapter.

2.2. Gene-Environment Interplay

In the developmental psychopathology literature, a general consensus has been reached that behaviour and psychopathology reflect the interplay of nature and nurture. This stemmed from quantitative and molecular genetics. In the past, quantitative behavioural genetics, usually based on twin and adoptee studies, focussed on examining the separable and independent genetic and environmental influences on individual differences in psychological traits and mental disorders. This was made possible by quantifying the proportion of total population variance accounted for by genetic factors and was pivotal in demonstrating that nearly all behaviour and psychiatric disorders are genetically influenced

to a significant extent. In contrast, molecular genetics, which involves ascertaining individuals' DNA sequences in conjunction with linkage or association strategies, is concerned with the identification of susceptibility genes for traits or disorders. Until recently findings have been contradictory and inconclusive, largely due to difficulties in identifying genes of very small effect, the multiplicity of genes involved and genetic heterogeneity. However, there is now some evidence for four susceptibility genes implicated in schizophrenia, two in ADHD and the strongest evidence for three in Alzheimer's disease.

In the case of ADHD, twin and adoption studies have estimated heritability of ADHD to be in the range of 60% to 90% (Bergen, Gardner, & Kendler, 2007; Faraone et al., 2005; Ficks & Waldman, 2009; Waldman & Gizer, 2006; Waldman & Rhee, 2002). Most genetics studies have focussed their attention on associations between ADHD and candidate genes implicated in neurotransmitter production, functioning and degradation in the dopaminergic, serotonergic and noradrenergic systems, with the strongest evidence supporting links between ADHD and dopaminergic system genes (e.g., a polymorphism of the DRD4 gene) (Faraone & Khan, 2006; Gizer, Ficks, & Waldman, 2009; Waldman & Gizer, 2006). However, inconsistent findings across studies and small sample sizes stress the need for further studies to confirm these associations, in particular their magnitude and those sourced in serotonergic and noradrenergic systems.

However, simply measuring or quantifying heritability and not taking into account genetic-environmental interdependence does not shed light on the causal processes and mechanisms involved. Gene-environment interdependence should be considered. There are four main kinds of gene-environment interplay. Two of these include gene-environment correlations (rGEs) and gene x environment interactions (G x E). rGEs are concerned with the association between genetic factors and environmental influences and are usually passive, evocative and active in nature (Plomin, DeFries, & Loehlin, 1977). Passive rGE, which must be examined via twin studies of parents (e.g., Neiderhiser et al., 2004, cited in Rutter, 2007), has been described as "the association between a characteristic of the parent and a characteristic of the child that may result from an underlying shared genetic characteristic that simultaneously influences both the trait in the parent and the trait in the child, even if the parent and child traits have quite different behavioural manifestations" (Jaffee & Price, 2007, cited in Harold, Elam, Lewis, Rice, & Thapar, 2012). Evocative rGE suggests that "genetically influenced child characteristics (e.g., externalising problems) evoke patterned responses such as negativity from a parent" (Ge et al., 1996, cited in Harold et al., 2012) and active rGE refers to "the effects of the child's genes on those child behaviours that help the child's selection of environment" (Rutter, 2007), and both are

examined using children of twins studies (D'Onofrio et al., 2003; Silberg & Eaves, 2004, cited in Rutter, 2007) or adoptee designs (e.g., Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998, cited in Rutter, 2007).

Stimulated by Caspi and colleagues' seminal work that demonstrated a stronger effect of maltreatment early in life on later antisocial behaviour in males with the genotype conferring low MAOA activity compared to males with the genotype conferring high MAOA activity (Caspi et al., 2002; Caspi, Sugden, & Molfitt, 2003), research examining gene-environment interactions in ADHD has found strongest support for a role of two polymorphisms of dopaminergic genes (DRD4 exon 3 VNTR and DAT1 3'UTR VBTR – account for 50% gene-environment interactions examined)with significant interactions between these genes and environmental variables including indices of perinatal adversity (e.g., prenatal smoking and alcohol exposure). However, findings have been mixed. Gene-environment interactions involving EE and polymorphisms of the serotonin and dopamine transporters have also been found suggesting maternal expressions of warmth and hostility may act together with genetic factors in altering severity of ADHD (Sonuga-Barke et al., 2009).

It has been estimated that environmental influences on ADHD, represented predominantly by those that are nonshared (i.e., child-specific), explain 10% to 40% of its variance (Waldman & Rhee, 2002) and include biological and psychosocial factors. Biological aspects of the environment shown to be related to the disorder include elevated levels of toxins (e.g., lead, manganese and polychlorinated biphenyls), and pregnancy and delivery complications, such as maternal age at child's birth, duration of labour, poor maternal health, eclampsia, foetal distress, low birth weight and prenatal exposure to smoking and alcohol (Banerjee, Middleton, & Faraone, 2007). However, for some of these risk factors (e.g., maternal alcohol use and smoking via exerting negative influence on infant birth weight) the evidence is inconsistent. Parental and family factors, such as parent-child interaction, parenting practices and attitudes, marital distress and exposure to maternal psychopathology, have long been considered psychosocially salient environmental influences on ADHD and its developmental course with a wealth of studies demonstrating their importance and that rather than acting in isolation they often exert their influence in a cumulative and interactive manner.

Identifying both genetic and environmental influences that contribute to the course and outcome of ADHD remains a fundamental area of investigation within the developmental psychopathology framework and has important theoretical and therapeutic implications. Further research findings may help explain the risk and protective pathways that lead to adverse outcomes and impairment in this population. They may also provide

an evidence base to inform the development of effective risk reduction strategies in the long-term management of ADHD. Although investigation of gene-environment interactions in ADHD is beyond the scope of this thesis, this thesis aims to improve understanding of the role of the family environment indexed by parental expressed emotion (EE) in the course and outcome of ADHD.

2.3 Family Processes and Child Developmental Outcomes

It is widely accepted that a multitude of biopsychosocial factors that operate in tandem are implicated in child psychopathology. However, most theoretical models, including social learning, cognitive-behavioural, family systems and bio-behavioural models, place considerable emphasis on family relationships as essential to explaining the development of child outcomes. For example, drawing on social learning theory, Patterson's coercion model which postulates that parent-child interactions contribute directly to antisocial behaviour in children (Patterson, 1982), is supported by strong evidence demonstrating family functioning during a child's primary school years predicts adolescent antisocial behaviour (Dishion, French, & Patterson, 1995). Cognitive behaviour theories assume that distorted cognitions and misattributions of intent, instrumental in causing depression (Mash, 1988), are embedded in early socialisation experiences, in particular to negative interactions between family members (Meichenbaum, 1977). Inherent to family systems theory is the notion that families work as a system in establishing and maintaining maladaptive behaviour and psychopathology results from disturbed parent-child boundaries and relationships within this system (Wagner & Reiss, 1995). Support for these models comes from a wealth of studies demonstrating a role of family processes in the development and maintenance of child psychopathology. EE is a child-specific construct of family functioning with several methodological advantages (discussed in the next section) over other measures of the family environment that has received increasing attention due to the wealth of evidence demonstrating its link with child developmental outcomes.

2.4 Introduction to Expressed Emotion

Expressed emotion is conceptualised as a measure of the emotional atmosphere of the home and is considered an important marker of familial relationships. It is defined as the extent to which a family member expresses critical/hostile and emotionally over-involved attitudes and comments toward another family member. It is assumed that EE, which is measured by rating the way parents or relatives talk about their child or relative during a semi-structured interview, is representative of how they interact with their child on a daily

basis (Chambless, Bryan, Aiken, Steketee, & Hooley, 1999). The concept first emerged in investigations of family influences on the course of schizophrenia. These studies reported an increased likelihood of relapse in adult patients with schizophrenia who returned to high EE families (Brown, Birley, & Wing, 1972; Brown, Monck, Carstairs, & Wing, 1962). These findings have since then been consistently echoed in schizophrenic populations and across a wide spectrum of other mental health disorders in adults, including mood, anxiety, substance use, personality and eating disorders (Butzlaff & Hooley, 1998; Hooley, 1985, 2007) as well as medical conditions including Alzheimer's disease, asthma, diabetes, and Parkinson's disease (Wearden, Tarrier, Barrowclough, Zastowny, & Rahill, 2000).

Interest in the EE construct in child populations stemmed from early investigations which did not employ standard EE measures per se, but examined associations between individual facets that characterise EE, such as warmth and hostility, and child behaviour. For instance, Rutter et al. (1975) and Quinton and Rutter (1985) demonstrated that absent or low levels of warmth and hostility were related to child behavioural problems. Similar findings were reported by Richman, Stevenson, and Graham (1982) in non-referred three-year-old children. Studies that have used the EE measure in community and clinical child and adolescent samples have reliably documented links between high parental EE and child psychiatric disorders including ADHD (Christiansen, Oades, Psychogiou, Hauffa, Sonuga-Barke, 2010; Daley, Sonuga-Barke, & Thompson, 2003; Marshall, Longwell, Goldstein, & Swanson, 1990; Peris & Hinshaw, 2003; Peris & Baker, 2000), disruptive behaviour disorders (DBD) (Hibbs et al., 1991; Stubbe, Zahner, Goldstein, & Leckman, 1993; Vostanis & Nicholls, 1995; Vostanis, Nicholls, & Harrington, 1994), mood disorders (Asarnow, Goldstein, Tompson, & Guthrie, 1993; Asarnow, Tompson, Woo, & Cantwell, 2001; McCleary & Sanford, 2002; Silk et al., 2009; Vostanis et al., 1994) and anxiety disorders (Hibbs et al., 1991). Indeed, Schwartz, Dorer, Beardslee, Lavori, and Keller (1990) found a three-fold risk of receiving a DSM-III diagnosis of substance abuse, conduct disorder or depressive disorder in children of high EE mothers.

In addition to being a good discriminator between clinical and non-clinical populations, research has also revealed EE to be a powerful prognostic tool. Asarnow and colleagues (1993) reported that in boys and girls with depressive disorders none who returned to high EE homes compared to 53% who returned to low EE homes recovered one-year post-discharge from hospital. Associations have also been found between parental EE and child externalising and internalising measured broadly and dimensionally using questionnaires (Baker, Heller, & Henker, 2000; Hale III et al., 2011; Psychogiou, Daley, Thompson, & Sonuga-Barke, 2007). Furthermore, one study found maternal high EE was associated with higher Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983)

internalising and externalising scores in typically developing school-aged children (Vostanis & Nicholls, 1992). This well-recognised relationship between parental EE and behavioural and emotional problems has been found in both boys and girls and in preschool (Baker et al., 2000; Daley et al., 2003) and school-aged children (Peris & Baker, 2003; Psychogiou et al., 2007) and adolescents (Hale III et al., 2011; McCleary & Sanford, 2002; Stubbe et al., 1993), although less so in clinical populations of adolescents.

The EE measure has been of particular interest for a number of reasons. This is because this construct affords a number of methodological advantages over other measures of family functioning. Some of these apply generally to child development research while others apply specifically in the context of ADHD. The first is due to its reliability in predicting both child and adult psychopathology.

Second, EE is strongly related to observational data of actual behaviours manifested during parent-child interaction. Marshall and colleagues (1990) found that high FMSS-EE was significantly associated with more verbal coercion processes and negative affective style during a direct interaction task in families of school-aged boys with ADHD. An earlier study found similar results with parents who expressed a higher level of criticism during the interview expressing more mild and harsh criticism in the direct interactions with their children. Most recently, in a clinic-referred sample of children aged seven to 17 years parents the CRIT dimension of FMSS-EE predicted actual behaviour in interactions with parents rated high in CRIT rating higher on antagonism, negativity, disgust, harshness, less responsiveness (McCarty et al., 2004).

Third, EE allows child-specific expression to be estimated. As noted in the next chapter behavioural genetics studies have identified nonshared (child-specific) environmental experiences as an important influence on child psychopathology with some authors arguing their importance is greater than shared aspects of the environment. However, behavioural genetics studies have largely not measured child-specific experiences. Indeed, many measures of psychosocial adversity (e.g., marital conflict) are shared in nature and affect all children in the same family. Child-specific constructs allow measurement of experiences that can account for behavioural differences between children reared in the same family as comprehensively as possible. This prevents the potential interpretation that shared family effects might be attributable to child-specific effects that had not been estimated. In the case of the present thesis, EE can be used to assess whether different children are more or less likely to be 'subject' to different experiences in the home as a function of their ADHD. EE also affords individual-specific expression regarding the parent who expressed the particular emotion. Third, it does not focus on close-ended questions as other measures do but it takes into account the manner in which the parent

talks about the child. Fourth, the rating of EE considers what the person actually says about the child but also it considers vocal elements such as the tone of voice. Fifth, EE puts emphasis on emotions about the individual child rather than specific emotions associated with the ADHD symptomatology.

2.5 Origins of Expressed Emotion

High EE in relatives has been found to be associated with significantly increased relapse rates in adults with schizophrenia (Sz) (Vaughn, Snyder, Jones, Freeman, & Falloon, 1984), unipolar depression (Hooley, 1986; Hooley & Teasdale, 1989; Vaughn & Leff, 1976a) and bipolar disorder (Miklowitz, Nuechterlein, Snyder, & Mintz, 1988). These associations were independent of initial severity of symptoms, although several studies failed to find an independent association (e.g., McCreadie & Philips, 1988). High EE also linked in a prospective study with subsequent onset of Sz in adolescents diagnosed with Sz-spectrum disorder (Goldstein, 1985). The concept of EE originally evolved from the findings of a succession of epidemiological studies conducted in the late 1950s and early 1960s in the Camberwell area of south east London by George Brown and his colleagues (Brown, Carstairs, & Topping, 1958; Brown, 1959; Brown et al., 1962). These studies sought to explore environmental factors that might affect the adjustment of and prognosis for adults with schizophrenia when they returned home from hospital into the community. In the first of these studies, interviews were conducted with long-stay adult males with schizophrenia and their families at one-year follow-up post-discharge from hospital (Brown et al., 1958; Brown, 1959). It was found that upon their return home, clinical deterioration was associated with the patient's living arrangements, with higher rates of re-admission to hospital among patients who lived with close family members, such as a spouse or parent, compared to those who lived with siblings or more distant kin or alone in lodgings. Increased risk of symptomatic relapse (i.e., a clinically significant return of symptoms based on an independent assessment with a structured clinical interview) was also determined by unavoidable enduring contact with close family members (e.g., when neither patient nor parent or spouse was going out to work).

The notion that attributes of the family environment might be important in the course of Sz initiated a second study, this time including a sample of short-stay male patients with schizophrenia male aged 20-49 years (n = 128) (Brown et al. 1962). In this study, Brown at al. worked to overcome some of the shortcomings of the previous study (e.g., reliance on one interview at follow-up and lack of direct measures of the family environment). Clinical interviews with the patient alone a few days before discharge from hospital and on re-admission or one year post-discharge (if the patient was not re-admitted) usually with both

the patient and the relative were conducted to assess the patient's mental state and behaviour. Background information about the family and the patient's past behaviour and how the family felt about the patient's return was collated through an interview with the relatives a few days after discharge. In an interview 2 weeks post-discharge from hospital, patients and their key relatives (usually a mother or wife) were observed and ratings of the key relative's emotions and hostility expressed and dominant or directive behaviour toward the patient and ratings of the patient's emotions and hostility expressed toward the key relative were made. Based on these ratings, patients were divided into "high emotional involvement" and "low emotional involvement" groups. Brown et al.'s first hypothesis was confirmed with patients returning to homes where at the time of discharge "high emotional involvement" was shown by their relatives being significantly more vulnerable to clinical deterioration than patients who returned to homes where relatives showed "low emotional involvement". Furthermore, this relationship remained regardless of whether patients returned following hospitalisation to close or more distant relatives and when psychiatric status at the time of discharge was controlled for.

This research team working with Michael Rutter subsequently carried out a series of methodological studies over the next 3 years to tackle the question of how to reliably measure aspects within the family system that might be pertinent in the course of Sz. This work led to the development of a standardised family interview known today as the Camberwell Family Interview (CFI; Vaughn & Leff, 1976a) and to clarification of the term 'emotional involvement' which was renamed 'expressed emotion'. The CFI will be discussed in the next section.

2.6 Measuring Expressed Emotion

2.6.1 Camberwell Family Interview (Original Version)

The primary method used to measure emotions expressed by relatives in adult psychiatric populations was traditionally an extended version of today's widely recognised Camberwell Family Interview (Brown & Rutter, 1966; Rutter & Brown, 1966). This original instrument was an audio-recorded, semi-structured interview with the relatives of the individual with the mental health condition which lasted for two to four hours and aimed to solicit detailed information on family life as well as the attitudes and feelings of the relative toward the individual. In particular, the interview enquired into the onset and development of the patient's current psychiatric episode and the effects it endured on a wide range of activities and events in the home (e.g., occurrence of arguments, involvement in household chores and patient-relative contact) during the 3 months prior to the patient's hospitalisation.

Observations of the relative's behaviour were made throughout the interview and attention was paid in particular to the feelings the relative expressed in general about their family members and more specifically about the patient and his recent behaviour. Ratings were made on observed emotions including warmth, hostility and emotional over-involvement and on the number of positive and critical comments made by the relative about the patient in the interview. In the ratings, reliance was especially placed on vocal aspects of speech relatives made when talking about the patient. Brown and Rutter (1966) noted that family interviews used previously to the original CFI that attempted to capture the emotions expressed during the interview situation, placed emphasis on facial expressions and excluded consideration of speech. Furthermore, few of the studies that had used vocal aspects of speech in their ratings had published data on inter-rater reliability, and of those that had, inter-rater correlations were more often than not low except in Brown et al.'s (1962) study. Criticism was also made about the tendency of previous work of emotional expression to measure this aspect of the family system using what they termed 'artificial' emotions as ratings were based on feelings expressed at the request of the experimenter. Ratings of EE in the original CFI therefore also included observations of spontaneously expressed emotion during the interview.

Brown and Rutter (1966) had set out first to develop an improved family interview to address the methodological problems previous techniques measuring family variables were plagued with and criticised for. This research entailed interviewing husband and wife, one of whom was a psychiatric patient, individually and together. Their second aim, to assess the reliability and validity of the measure, was achieved by recruiting a further 30 families with whom interviews were conducted with the patient alone, spouse alone and both together with two different investigators present at each interview. The patient and spouse interviews, although administered slightly differently from each other, formed the main family interview for which high levels of inter-rater reliability ($r = .80 - .85$) were achieved. Despite their successful effort to develop a measure that could reliably rate the expression of emotions, some other questions remained unanswered. First and foremost, the concept 'emotional involvement' was considered somewhat vague and further clarification was required in understanding what the components of 'emotional involvement' were. Second, little was known about the cause-effect relationship between emotions expressed by relatives and patient deterioration. Third, the role of other factors (e.g., gender, marital status, patient-relative contact, patient's attitudes and medication) that might influence the course of schizophrenia was unclear and had been paid little attention.

With these issues in mind, with the aim to replicate and extend the findings of the previous study (Brown et al., 1962), Brown et al. (1972) conducted a prospective 9-month

follow-up study of 101 in- and out-patients identified from case records as having recently begun a new period of treatment and who might be suffering from schizophrenia. It was in this study that the term 'emotional involvement' was specified more precisely and replaced with 'expressed emotion' and hence the EE construct was developed. On admission to hospital, information of past behaviour of the patient (e.g., work impairment, social withdrawal, delinquency and aggressiveness) was obtained. A psychiatric interview administered at the time of hospitalisation to confirm clinical caseness for Sz and the CFI administered to the key relative whilst the patient was in hospital were later repeated 9-months post-discharge or at any readmission. The interview was slightly refined. Ratings of the number of critical comments made about another person in the home, hostility, dissatisfaction, warmth and emotional-overinvolvement were made using the CFI, and to a lesser extent, a joint interview with patient and relative 2 weeks after discharge that was similar to that administered in Brown et al.'s (1962) previous study. An overall index of EE was used to categorise families into high expressed emotion and low expressed emotion groups through the exploration of several methods, with the following indices being used to finally assign half of the families to the high expressed emotion group: ≥ 7 critical comments (≥ 2 comments in the joint interview), marked overinvolvement and hostility. The previously included component 'warmth' was dropped as deemed a complex variable. In line with the findings of earlier work (Brown et al., 1962), high expressed emotion among relatives at the time of hospitalisation predicted symptomatic relapse in the 9 months post-discharge.

2.6.2 Camberwell Family Interview (Abbreviated Version)

Even though proven to be a methodologically sound family research instrument to measure the quality of emotional relationships between patients and relatives, from a practical perspective, the length of the original CFI limited its utility even in research settings and made it a laborious task for both interviewer and respondent alike. Hence, Vaughn and Leff (1976a) sought to modify and shorten the interview to no more than 2 hours, but at the same time maintain its reliability and validity. Development of the modified version of the CFI was facilitated by the finding in the 1972 study that the number of critical comments made by the relative when talking about the patient and his condition was the single most salient measure in contributing to the overall index of EE and in predicting symptomatic relapse. Fifteen of the original tape-recorded interviews, with equal representation of high, medium and low EE interviews were listened to and individual time graphs representing the points at which critical remarks were made during the interview were plotted. The area of questioning when criticism occurred was also noted. It was found first that the majority

(67%) of critical remarks were made in the first hour when the areas of enquiry were psychiatric history, quarrelling and irritability and clinical symptoms during the preceding 3 months, second that the total amount of critical comments and length of interview were not significantly correlated and third that the occurrence of criticism differed according to the area of inquiry, with fewer critical remarks being made in the last sections (except for Household Tasks/Money Matters and in the case of the relatives being parents, Relationships) such as the Marital Relationship section and the particularly lengthy Kinship section. This was partly due to criticism spontaneously manifesting earlier on in the interview. Support for shortening the interview was justified on the basis of these results. The abbreviated interview schedule, which included fewer sections, was reordered according to which sections elicited the most critical comments, but the form and content of the questions remained the same.

The abbreviated version of the original CFI, referred to today simply as the CFI, is generally accepted as the 'gold standard' measure of EE (Vaughn & Leff, 1976). It has been used extensively in studies of adult psychiatric populations (e.g., Hooley & Licht, 1997) and to some extent in paediatric samples (e.g., Vostanis & Nicholls, 1995; Vostanis et al., 1994). Lasting 1-2 hours, the CFI is a standardised semi-structured interview conducted with the patient's key relation (typically a spouse or parents – interviewed separately) in the absence of the patient that is audio-recorded and later coded. During the interview, the relative is asked questions about the onset and course of the patient's current psychiatric episode, irritability and quarrelling in the household, symptoms noticeable in the three-month period prior to admission to hospital as well as the patient's involvement in household activities, daily routines of the patient and family members and how the relative generally gets along with the patient. The audiotape of the interview is scored on five scales (the same scales as the original CFI as mentioned above), based on what the relative says about the patient and their tone of voice used whilst talking about the patient, to yield an overall index which reflects the relative's attitudes and feelings toward the patient. The first dimension, criticism is derived from the total number of critical comments the relative makes about the patient. Remarks are rated as critical if the content demonstrates disapproval or dislike of the patient's behaviour or actions or when negative voice tone is used to describe specific situations involving the patient. Hostility typifies more global criticism and general disapproval or rejection of the patient rather than criticism of specific actions or behaviours. Emotional overinvolvement, characterised by extreme over-protective behaviours and remarks displayed by the relative toward the patient, is scored on a scale from 0-5 on the basis of the relative describing intrusive behaviour toward the patient and displays of extreme emotional stress during the interview. Positive remarks are

assessed according to the total number of positive remarks, defined by praise, approval and appreciation of the individual, made about the individual by the relative. Lastly, warmth is exemplified by the relative's tone of voice used during the interview when discussing the patient and is rated on a scale of 0-5.

Criticism, hostility and EOI are the three principal components considered in the overall dichotomised (high vs. low) EE index derived from the CFI. The criteria for defining relatives as high-EE is: six or more critical remarks, evidence of hostility or a score of three or more on the EOI scale. Warmth and positive comments are excluded in the overall rating of EE. Reasoning for the sole use of the three dimensions was based on several findings from Brown et al.'s (1972) seminal study. First, the authors identified these scales as the best predictors of relapse in individuals with schizophrenia. Second, low warmth was correlated with high rates of criticism and high warmth was associated with high levels of EOI. This component therefore perplexed the authors and its role in families has since tended to be neglected, with a few exceptions (e.g., Lopez et al. 2004). Criticism has proven to be the most important component of EE as shown initially in Brown et al.'s study and consistently in succeeding studies. The established overlap of criticism with hostility (Hooley & Licht, 1997; Chambless, Bryan, Aiken, Steketee, & Hooley, 1999) has led to a substantial proportion of EE research (Hooley, 1986, 1998; Vaughn et al., 1984) reducing the components considered in the overall EE rating to two – criticism and emotional overinvolvement, with relatives being classified as high-EE if they score high on emotional overinvolvement or on both. In Brown et al.'s study, a cut-off of seven critical remarks was statistically proven to best distinguish between adult with schizophrenia who relapse and those who do not. In the development of the CFI, this was further reduced to six critical comments. A lower cut-off score of 2-3 critical remarks has proven more appropriate for patients with unipolar depression. Good concurrent and predictive validity has been found for the CFI (Butzlaff & Hooley, 1998). In addition, it has acceptable inter-rater reliability even though the task of coding the interview is quite arduous (e.g., Hooley, 1986; Mueser et al., 1993).

Even though the CFI is deemed the benchmark research tool used to assess EE, the length of the interview (i.e., over one and a half hours) the additional two hours it takes to code and the extensive training required to administer and score the interview has limited its popularity particularly in research involving children and their families and large-scale studies. It does continue to be well-used in adult populations.

Having developed the CFI, in a replication (i.e., identical design and execution) and extension (i.e., use of the abbreviated main family interview) of Brown et al.'s (1972) previous work, Vaughn & Leff (1976) broadened their sample to two clinically different

groups of individuals with psychiatric disorders; those with Sz (n = 37) and those with neurotic depression (n = 30), however, the analyses were mostly concerned with the group of individuals with schizophrenia. In accordance with the earlier study, symptomatic relapse nine months post-discharge was best predicted by returning to a high EE home. In contrast to Brown et al.'s (1972) speculation that living with parents but without other household members explained the differences in deterioration between married and unmarried couples, parents who lived alone with the patient were just as likely to show high EE and differences in the amount of parent and spouse expressed emotion were not significant.

Some researchers have assessed parental EE toward children with behavioural and emotional problems using codes derived from the CFI, but with information (upon which the codes were applied) collected using the Parental Account of Childhood Symptoms (PACS; Taylor, Schachar, Thorley, & Wieselberg, 1986a), a semi-structured psychiatric interview. This method was followed by Sonuga-Barke et al. (2008) who found evidence for the role of warmth and criticism moderating the effects of genes on ADHD severity and comorbid CD.

2.6.3 Five Minute Speech Sample

The Five Minute Speech Sample (FMSS; Magaña et al., 1986) was developed as an alternative to Vaughn and Leff's (1976) abbreviated version of the CFI with the aim to further reduce interviewing time in mind and on the premise that placing a time limit on the interview and making it less structured would elicit critical and overinvolved responses under the time pressure. It has invariably been applied in studies of EE in families of children and adolescents (e.g., Asarnow et al., 1993; Hibbs et al., 1991; Marshall et al., 1990; Psychogiou et al., 2007; Peris & Hinshaw, 2003; Stubbe et al., 1993) and to a lesser extent in adult populations.

In administration of this newer instrument, respondents are asked to talk for five uninterrupted minutes about their thoughts and feelings about the relation, and about how the two of them have been getting along, using a standard prompt. The monologue is audio-recorded and transcribed for later coding which takes approximately 20 minutes to complete compared to 2-3 hours required to code the CFI. Extensive training in interview methodology and coding procedures is still required for this measure.

Coding the speech sample is in part parallel to the CFI in that a final index of EE that is dichotomised into high or low EE is attained. In addition, the criteria used to rate the core components, Criticism (CRIT) and emotional overinvolvement (EOI), that contribute to the overall FMSS-EE score, were derived from the CFI. In contrast to the CFI, there is

no hostility or warmth rating on the FMSS. Ratings on five scales (Criticism, Hostility, EOI, Warmth and Positive Remarks) are made using the CFI compared to nine scales on the FMSS. These are: initial statement (positive, neutral or negative), critical comments (frequency count), positive remarks (frequency count), quality of relationship (positive, neutral or negative), emotional display during the speech sample (presence/absence), statements of attitude (frequency count), evidence of self-sacrificing, overprotective behaviour or lack of objectivity (present or absent), excessive detail about the past (present or absent) and evidence of dissatisfaction with the child (present or absent). Eight of these subcomponents contribute towards CRIT and EOI scores. Ratings of CRIT qualify as high if the respondent's initial statement or quality of relationship is scored as negative, or if the respondent has made one or more critical remarks. The presence of emotional display or extreme levels of self-sacrificing, overprotective behaviour or lack of objectivity merits a high EOI rating. High EOI scores may also be given if two of the following are evident: presence of excessive detail about the past, an extreme statement of positive attitude or excessive praise (five or more positive remarks). Final EE is rated as high if either CRIT or EOI or both are high, and low if no criteria are met for either component. Borderline high CRIT is indicated if no criteria are met, but the presence of dissatisfaction is evident and a borderline high EOI rating is given if the relative makes an extreme statement of positive attitude, five or more positive comments and if evidence of excessive detail about the past is present.

In evaluating the concurrent validity of the FMSS, Magaña et al. (1986) found that 15 of 23 respondents defined as high EE and 15 of 17 rated as low EE on the CFI were also rated as high EE and low EE respectively on the FMSS, findings that were almost identically echoed in a Spanish-speaking sample (Magaña et al.). Validation in a German sample demonstrated 80% sensitivity (proportion of accurately identified high-EE relatives) and 71% specificity (percentage of low-EE respondents correctly identified) (Leeb et al., 1991). In all three samples, significant overall agreement between the FMSS and CFI ratings was found. Respondents classified as high EE on the FMSS for the most part were always rated as high EE on the CFI. On the surface this seems to suggest adequate concurrent validity. However, Magaña et al. reported that a considerable proportion of high-EE cases were misclassified as 20% of the respondents who were rated as being low-EE on the FMSS were defined as high-EE on the CFI. The same pattern was found by Leeb et al. (1991) with 52% of high-EE relatives being misclassified. These results suggest therefore that the FMSS has a tendency to under-identify high-EE cases. One explanation for this discrepancy may be that one of the rules in the FMSS coding manual is that upon hesitation between rating a respondent high, one must be conservative. When cases

identified at the borderline of low EE have been included as high EE sensitivity increased from 66.7% to 100% (Shimodera et al., 2002).

Calam and Peters (2006) attempted to establish comparability of the CFI and FMSS in a sample of children (n = 75) aged 3-10 years diagnosed with ODD or CD. Female primary carers were administered the FMSS first and then a modified version of the CFI which was made age-appropriate (e.g., questions about daily routine were changed to ask children about dressing, mealtimes, after-school activities, etc.) from which ratings were compared to parent-rated common behavioural problems after intervention at 12 months follow-up. Fifty seven mothers were classified as high EE using the CFI and 65 on the basis of the FMSS approach, results that do not converge with previous studies that found either adequate concordance levels (Magaña et al., 1986) or that high FMSS-EE was under-identified (Malla, Kazarian, Barnes, & Cole, 1991; Van Humbeeck, Van Audenhove, De Hert, Pieters, & Storms, 2002). Modifications made to the CFI for the study were approved by the first author of the original abbreviated CFI, criterion raters trained psychologists to reliability in coding the CFI and inter-rater reliability of FMSS-EE ratings was assessed using independent coders, one of whom was blind to all information about the mothers and the study hypotheses. However, levels of EE were exceptionally high in the sample compared to other published studies including samples of younger children. In addition, the CFI was administered immediately after the FMSS. By and large, the CFI is considered the most comprehensive assessment of EE and the most appropriate measure for use in adult populations. However, the CFI remains limited in terms of its practicality. In summary, the benefits of the FMSS include its brief duration, the ease of data collection in small and large-scale studies and studies involving paediatric populations in which use of the CFI is simply not practical, its valid use in children and adolescents and the relatively minimal training requirements for the interviewer. For these reasons, this measure remains the most commonly used in child populations.

2.6.4 Other Measures of EE

Researchers have searched for alternative and briefer methods to assess EE which unlike the CFI and FMSS do not require formal training (extensive training for CFI coding). In a review of EE assessment measures nine other measures of EE were identified (Van Humbeeck et al., 2002). Three of these, all questionnaires, are the only measures to have been validated against the CFI as ascertained in another review (Hooley & Parker, 2006). These are: the Level of Expressed Emotion questionnaire (LEE; Cole & Kazarian, 1988) which comprises 60 items, the Family Attitude Scale (FAS; Kavanagh et al., 1997) which consists of 30 items and the Perceived Criticism Scale (PC; Hooley & Teasdale, 1989)

which contains 1 item. Studies of adults have demonstrated predictive validity of these measures in a range of psychiatric disorders (Hooley & Parker, 2006; Van Humbeeck et al., 2002). To date, only the LEE, which is the first developed self-report measure using true-false items, has been used in child populations. Recently, a research group in the Netherlands used the 38-item version of the LEE (Gerlsma & Hale III, 1997; Gerlsma, van der Lubbe, & van Nieuwenhuizen, 1992) in a series of studies to examine the relationship between maternal EE and adolescent internalizing and externalizing behaviour (Hale III, Raaijmakers, Gerlsma, & Meeus, 2007; Hale III et al., 2011; Hale III, Raaijmakers, van Hoof, & Meeus, 2011). In the first of these studies, the four-factor structure (Lack of Emotional Support, Intrusiveness, Irritation, and Criticism) applied to adolescents in the same way it had previously been shown to apply to adults and that the four scales related to adolescent internalizing symptoms. In the other two studies, the four components as well as an additional scale (Constructive Criticism) were longitudinally associated with adolescent internalizing and externalizing symptoms. However, the 38-item version has not been tested against the CFI or FMSS and rather than providing an objective measure of EE, this questionnaire measures perceived EE which in one of the studies was that of parents and in the other two studies was adolescents' perceptions their parents' EE.

Docherty, Serper, and Harvey (1990) developed another self-report measure, the Questionnaire Assessment of Expressed Emotion, which comprises 99 items that measure criticism/hostility and EOI with good internal consistency found for both subscales. In a comparison of this measure with the CFI with 25 relatives of individuals with a diagnosis of schizophrenia, ratings were similar for 88% and 68% of individuals on the criticism/hostility and EOI scales respectively in addition to a 76% congruence rate between the two measures for distinctions between high and low EE.

Other self-report measures of EE include the Expressed Emotion Adjective Checklist (EEAC; Friedman & Goldstein, 1993), which uses ten positive (e.g., easy to get along with, loving) and ten negative (e.g., angry, hostile) adjectives to measure criticism and EOI displayed by family members toward the individual with the mental health condition and vice versa, and has also been shown to have high internal consistency, and the Family Emotional Involvement and Criticism Scale (FEICS; Shields, Franks, Harp, McDaniel, & Campbell, 1992), which comprises 23 items that assess perceived criticism and EOI and has demonstrated adequate reliability for both subscales. Whilst a review of the former questionnaire found this measure to have good concurrent validity with the CFI and the FMSS (Van Humbeeck et al., 2002), the authors of the latter questionnaire did not establish its concurrent validity or predictive power, limiting conclusions to be made as to whether it can be used as a suitable alternative to the CFI. That the EEAC has also been used in a

psychoeducation intervention study as a measure EE change over time demonstrates its utility as a treatment outcome.

2.7 Associations with Child Behaviour and Other Child Characteristics: Empirical Evidence

Research has shown that both child characteristics and family characteristics are related to parental EE. With respect to child characteristics, the focal point has been on child behaviour and the literature pertaining to the association between parental EE and child behaviour is growing. Next, empirical findings demonstrating this relationship is reviewed including studies which have specifically examined a link between parental EE and ADHD.

2.7.1 Child Behaviour

In one of the earliest studies of EE and childhood disorder, Schachar, Taylor, Wieselberg, Thorley, and Rutter (1987) adopted a double-blind crossover design to assess the effects of methylphenidate on family function and relationships indexed by maternal warmth and criticism, contact with each parent, positive and negative encounters with siblings and parental coping efficiency and consistency in 38 boys aged 6 to 10 years who had been referred to a child and adolescent psychiatric clinic because of antisocial, disruptive or overactive behaviour. With respect to maternal EE, the results revealed that among the families of the 18 children whose behaviour significantly improved, the affective tone of the parent-child relationship improved, maternal warmth increased and maternal criticism decreased. However, the study was limited for several reasons. First, a measure of EE (i.e., the FMSS or CFI) as such was not used. Second, the changes observed were only short-term and predominantly relied on parent ratings of child behaviour to assess response to treatment. Still, because ratings were based on descriptions of child behaviour they may have been less susceptible to bias. In addition, both parents and clinicians were blind to the drug condition when ratings were made and the same results were found when the data were reanalysed with teacher ratings.

Marshall et al. (1990) aimed to examine whether parental and children's EE, measured with the FMSS and a modified three-minute version of the FMSS respectively, and their interactional behaviour was associated with the presence or absence of concurrent aggressive symptomatology in families of children with ADHD. In their sample of boys aged 6 to 11 years attending an intensive summer programme for children with ADHD who met DSM-III-R criteria for a diagnosis of ADHD only or ADHD plus ODD or CD,

they found that maternal, paternal and child FMSS-EE were highly correlated and that neither differed significantly between children with a diagnosis of ADHD and those with comorbid ODD/CD. Parental EE did not predict degree of child aggressiveness. The same pattern held when between-group comparisons of parents' prosocial behaviour (e.g., acceptance, agreement, positive self-statements etc.) and coercive processes coded from observations during family interaction tasks (role-play and discussions between parents and children) were examined.

However, there were several limitations. First, although children's interactional behaviour in the direct observation was highly correlated with their aggressiveness, direct observations of negative parental behaviour were not related to child aggressiveness. Furthermore, whilst child-to-parent-EE (i.e., children's affective attitudes toward their parents) and parent-to-child-EE mimicked each other, children's observed interactional behaviour was not associated with their EE status toward their parents. In addition, child EE mirrored paternal EE less. The authors attributed these findings to the limited number of high EE fathers making it more difficult to rigorously test these data. With regard to lack of correspondence between parent and child behaviour, the authors speculated that it might be that the direct interaction task measured concurrent parent-child behaviour but not the subsequent long-term impact of this type of negative interaction on the child, and therefore the negative impact of high parental EE and interactional behaviours are more likely to be observable at a later time. It might also be argued that the family interaction task might not be representative of typical everyday behaviour. Nevertheless, good inter-rater reliability for scoring the FMSS and the three-minute version was found (Kappa = 0.76, $p < .001$) and both EE tasks were counter-balanced. Overall, the study highlighted that regardless of whether separate maternal and paternal or parental unit classifications of EE were made (e.g., parental unit was classified as high if at least one parent in dual parent families and the sole parent in single-parent families expressed high EE toward the child), parent and child EE did not differ significantly between the ADHD only and ADHD-ODD/CD group.

In a subsample of 96 male and 32 female children and adolescents (aged 8-17 years) participating in an ongoing longitudinal study of biological factors in childhood aggression (Kruesi, Lenane, Hibbs, Major, & Rapoport, 1990a; Kruesi et al., 1990b), Hibbs et al. (1991) investigated whether maternal and paternal FMSS-EE differed between families of children with disruptive behaviour disorders (including ADHD, CD & ODD) and OCD, as assessed with the Diagnostic Interview for Children and Adolescents (DICA; Herjanic & Campbell, 1977; Welner, Reich, Herjanic, Jung, & Amado, 1987), and a control group matched for age and gender with the clinical groups. They found a significantly larger

proportion of families of the two groups with a diagnosis of a psychiatric disorder were rated high in family EE (88% DBD group, 82% OCD group) compared to controls (42%), with high ratings allocated if one or both parents had a high EE rating. However, the FMSS was administered after the psychiatric diagnostic interviews which may have led parents to express more negative views. Further, the generalizability of the findings were limited due to the young age of the population, higher socioeconomic status (SES) of the families, difference in referral methods (e.g., self-referred, school referrals, physician/therapist referrals, newspaper adverts) and screening procedures of the control group (e.g., see Kruesi et al. 1990a).

Given that most previous studies of EE were limited to clinical samples of homogeneously diagnosed adult and adolescent patients in on-going treatment, in the US, Stubbe et al. (1993) attempted to examine the usefulness of the FMSS as a research tool in a representative community sample and the relationship between parental EE and childhood psychopathology in 108 6 to 11 year olds who scored above 63 on the parent or teacher forms of the CBCL and their primary caregivers (mothers, foster mothers, grandmothers). Overall EE was associated with an increased rate of a broad spectrum of child DSM-III-R (APA, 1987) psychiatric disorders assessed by clinicians via the DISC-R (Shaffer et al., 1993). Seventy two percent of the sample did not meet criteria for any diagnosable condition, 14.8% had a diagnosis of DBD (ADHD, ODD, CD) and 9.2% were diagnosed with an anxiety or depressive disorder (overanxious disorder, separation anxiety, phobias, panic disorder, OCD, major depression, dysthymia) and 3.5% fulfilled diagnostic criteria in both broad categories. Over half (56.1%) the children from high EE families reported one or more diagnosable conditions compared with only 18.9% from low EE families. However, the inner city sample in addition to the high proportion of employed and middle and upper class mothers, there being fewer Hispanic families and more children in the older range meant there were several biases and that the generalizability of the findings were limited to other populations. A comparison of families with and without FMSS data (i.e., those who were included in the analyses and those who weren't) demonstrated that a greater percentage of upper class families completed the FMSS, those who did not complete the FMSS had lower scores on the Family Environment Scale (Moos & Moos, 1986) and there was a non-significant positive trend in this group toward having younger children. Still, the study demonstrated parental EE was significantly higher in clinical groups versus control families.

Following on from findings that implicated the prognostic power of EE in predicting outcome in adults with depression, Asarnow et al. (1993) aimed to investigate whether the same was confirmed in a sample of 26 7 to14 year old children (17 boys), predominantly

Caucasian, admitted to hospital for between nine and 334 days (M = 92 days) with DSM-III diagnosed depressive disorders (major depression [MDD] and/or dysthymia disorder [DD]), assessed with the K-SADS-E. The FMSS was administered to each parental figure (e.g., mother and boyfriend living in the home) living with the child prior to hospitalisation (almost half were single parent homes), although data was only complete for mothers due to a new parent moving into the home, the child moving to the home of a previously uninvolved or absent parent and data not being available for the father. Controlling for socio-demographic variables, higher rates of persistent mood disorders and/or relapse were reported one year post discharge among children returning to homes with high levels of FMSS-EE in comparison to those returning to low EE homes, with all but two of the families of children with continuing mood disorder rated as high EE and 53% of children recovered in the low EE group. The effect of EE persisted despite a significant association found between higher chronicity (whether the child became acutely ill within a year of hospitalisation or had shown signs of illness for more than one year) and recovery and a marginal trend for comorbidity with disruptive behaviour disorder (DBD - ADHD, CD, ODD) associated with continued mood disorder. The results, however, cannot be generalised to outpatient and non-referred children with depressive disorders. Still, diagnosis of severe MDD or DD was rigorously assessed and even in the presence of potential mediating variables, the relationship between EE and outcome remained demonstrating children with depressive disorders returning to high EE homes are less likely to recover during the year post-hospitalisation than children returning to low EE homes.

Vostanis et al. (1994) examined whether maternal EE, measured with the CFI, differed between two referred diagnostic groups and a non-referred control group matched for age and gender. Three maternal EE scales distinguished the three groups of 6 to 11 year olds: warmth, critical comments and positive comments. Maternal warmth was significantly lower in the CD group compared to the ED group and was significantly lower in the ED group compared to the control group. Criticism was significantly higher in the CD group compared to the ED group and control group, but did not differ significantly between the latter two groups. Mothers made significantly fewer positive comments toward their children in the clinical groups compared to the control group, but there were no significant differences between the two clinical groups. The differences in criticism and warmth between the clinical groups remained when further analyses were conducted first when children with mixed CD and ED were excluded from the CD group and second when the ED group was divided into two subgroups based on the number of conduct symptoms.

Vostanis et al.'s (1994) cross-sectional findings were later extended using a longitudinal design. Vostanis & Nicholls (1995) tested whether CFI-assessed maternal and paternal EE predicted changes in child behaviour over a period of nine months in 6 to 11 year old children referred to a child psychiatric clinic with ICD-9 (WHO, 1978) diagnoses of conduct disorder (CD) (included those with anxiety and depressive symptoms) (n = 28) or emotional disorder (ED) (included those who presented with oppositional and aggressive behaviour, but not those diagnosed with ADHD) (n = 29) and for which they were receiving routine treatment. Results indicated that maternal CBCL and EE scores changed during the nine month follow-up with overall outcome remaining poor, but significant improvements found in CBCL scores for children in both groups, although unlike the CD group (broad range of externalising scores – broad range of problems) the ED group had moved significantly from being in the clinical range to the nonclinical range. However, no research instrument was used to assess for diagnosis, clinical diagnoses were compared with maternal ratings on the CBCL. Although the study aimed to ascertain changes in maternal EE simultaneously with child symptoms, type of treatment which was a potentially confounding variable that could have affected outcome, was not controlled for in the analyses, as was the case in similar studies (e.g., Asarnow et al. 1993) that also did not include a measure of impact of specific treatment types.

A Canadian study examined FMSS-EE levels in 20 clinic-referred and 19 non-referred two-parent families of children and adolescents (7-16 year olds) recruited from community mental health centres and through local media announcements respectively (Kershner, Cohen, & Coyne, 1996). Based on families being rated as high if one or both parents were rated high and borderline-high EE families being grouped with high EE families, 19 of 20 (6 high CRIT, 6 high EOI, 7 high CRIT and EOI) clinical families were rated high in EE in comparison to 10 of 19 (2 high CRIT, 7 high EOI, 1 high CRIT and EOI) nonclinical families with this difference reaching statistical significance. Further, this finding that EE predicted clinical-nonclinical group membership persisted controlling for mothers' psychological distress and parents' perceptions of overall family functioning indexed by the General Functioning Scale of Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983), which also made a significant contribution. The authors questioned the utility of using overall EE to predict outcome in the broader clinical population of families due to the 19:1 ratio of high EE to low EE in clinical families. They argued that, instead, it may be more important to focus on the individual components, a point that will be returned to later in this chapter.

Peris and Baker's (2000) longitudinal study investigated the extent to which maternal FMSS-EE and child difficulties, as indicated by parent and teacher ratings on the CBCL,

were simultaneously and subsequently related with each other over time from preschool to third grade in a high risk community sample of 45 boys and 46 girls who upon entry to the study were exhibiting externalising and internalising behaviour problems. At first grade (mean age 6.8 years), externalising symptoms, but not internalising symptoms were associated with maternal EE status; 72.7% of children of high EE mothers were in the high externalising group (CBCL T-score 64 or greater), 28.6% were in the moderate group (CBCL T-score 60-63) and 18.5% were in the low externalising group (CBCL T-score under 60). At third grade, 69 children were assessed for childhood psychiatric diagnoses using the Diagnostic Interview Schedule for Children (DISC; Costello, Edelbrock, Dulcan, Kalas, & Klaric, 1984), the results revealed that 35 met criteria for one or more DSM-IV disorders, 19 for internalising (mood, dysthymia, GAD, simple phobia, social phobia), 15 for ODD and 20 for ADHD. Maternal EE was not associated with internalising diagnoses or ODD, but a strong relationship with ADHD was found as 64.3% of children of mothers rated high in EE at preschool met the criteria for a diagnosis of ADHD and 20% of low EE mothers had ADHD. High CRIT, but not EOI was significantly associated with ADHD, controlling for preschool child behaviour and maternal stress indexed by the Family Impact Questionnaire (FIQ; Donenberg & Baker, 1993) which measures parent's perceptions of the impact of a child on the family relative to the impact most children the child's age have on families. Further, ADHD symptom scores were significantly higher for children of high EE mothers at preschool, again for CRIT, not EOI. EE accounted for 7.7% variance, maternal stress 17.7 and preschool behaviour problems 16%; the same pattern was found when the CRIT score was used. However, these findings may not be generalizable due to the majority of children coming from well-educated and middle to upper class socioeconomic backgrounds. These results do, nevertheless support Marshall et al.'s (1990) speculation that negative family environment may independently be linked to the developmental course of ADHD and that it may serve as an effective prognostic tool for clinicians.

Asarnow et al. (2001) compared levels of FMSS-EE among parents of two clinical groups (inpatients and outpatients) and one community-based control group of six to eighteen year olds (at the time of diagnosis), predominantly male and Caucasian, recruited to a project in the US on family studies of childhood psychiatric disorders. The first group comprised 83 with a current diagnosis of depressive disorder (defined as "continuing illness at time of diagnostic interview or offset within two months of diagnostic interview) of which 59 had major depressive disorder (MDD), 24 had dysthymic disorder (DD) and 20 had both. The second group was formed of 73 children and adolescents with a current diagnosis of ADHD or ADD without hyperactivity and the third control group included 73

children and adolescents with no lifetime diagnoses of MDD, DD, ADHD or ADD. It was found that overall EE and CRIT were significantly higher among the parents of children in the depressed group compared to parents of children with ADHD and the community controls. Interestingly, family and paternal EE rates were marginally higher toward children with ADHD compared to the control group, but this difference did not reach statistical significance. This is the first study to demonstrate that rather than scoring significantly higher in EE than parents of control children, parents of children with ADHD scored similarly to parents of community controls. The findings conflict with other studies demonstrating FMSS-EE and CFI-EE is related to non-depressive forms of childhood psychiatric disorder, including ADHD (although marginally higher rates of EE were observed among families of children with ADHD compared to controls). However, the study was limited by administration of the FMSS up to two months after administration of the diagnostic interview and potentially inadequate power to identify differences as a function of age. Levels of EE were higher in fathers of older children in the depressed group and overall sample demonstrating that developmental factors are important as previous studies have documented high-EE in adults toward patients with schizophrenia, but not toward adolescents with schizophrenia (Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994). The sample was also characterised by substantial comorbidity; in the depressed group comorbid anxiety disorder, ODD and ADHD was common; in the ADHD group almost half had learning disorders and some ODD and anxiety disorders. Further, the authors did not report on parental psychiatric status, a variable shown to be important in understanding characteristics that contribute to development of high EE (Hibbs et al., 1991) and also another issue that will be returned to later in this chapter.

Data collected from a subsample of 57 inpatient and outpatient adolescents with a mean age of 15.1 years recruited to a Canadian longitudinal study on the clinical course of CD and MDD (Sanford et al., 1995) examined whether high EE was predictive of one-year persistence of adolescent MDD (McCleary & Sanford, 2002). At baseline, thirty four (59.6%) of parents (47 mothers; one step-mother) were rated low in EE and 23 (40.1%) as high EE and children of high EE parents had significantly more MDD symptoms, as indicated by the DISC-R, than children of low EE parents. At one-year follow-up, MDD had persisted in 43.1% of the 51 families retained in the study, but contrary to Asarnow et al.'s (1993) findings of an association between parental EE and MDD in children, neither overall family EE nor EE subtypes predicted continuity of MDD. The incongruent results of these two studies may reflect differences in classification of EE; although both studies rated family EE based on the highest score of both parents, in 71% of the families in the

McCleary and Sanford study there was only one parent, possibly leading to misclassification of some high EE families as low.

In a community study, Daley et al. (2003) examined the reliability and validity of a modified version of the FMSS for young children in mothers of 80 pre-school aged boys and girls diagnosed with ADHD using a clinical interview and 20 without ADHD who scored below the clinical cut-off point on the Werry-Weiss-Peters Activity Rating Scale (WWPARS; Routh, 1978). They found a marked increase in levels of maternal EE in mothers of children with ADHD compared to mothers of children without ADHD. Maternal EE was also associated with greater problems in the social domain, lower parenting self-esteem and higher levels of negativity in relation to the impact of the child on the family (Daley et al., 2003).

Studies of the relationship between ADHD and EE have predominantly centred on samples of Caucasian children. Kwon et al. (2006) found that hyperactivity levels as measured using the Conner's Parent Rating Scale (CPRS; Conners, 1996) of 190 African-American children at early school age were significantly higher in children of high maternal EE. Decreased perceptions of cognitive competence and increased anxiety were also associated with high levels of maternal EE. The results provided early evidence of high parental FMSS-EE toward children with ADHD in families from other ethnic backgrounds.

Girls have traditionally been under-researched in the field of ADHD often due to the lower prevalence of the disorder in this population. Peris and Hinshaw (2003) assessed parental FMSS-EE in an ethnically and socioeconomically diverse sample of 131 six to 12 year old girls who met DSM-IV criteria for ADHD combined type or inattentive type assessed with the Diagnostic Interview Schedule for Children 4th edition (NIMH DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) and were attending a five week summer enrichment programme. They found that high parental EE was associated with both ADHD and ODD/CD and that the association with ADHD withstood control of comorbid ODD/CD.

Another study of 36 parent-adolescent dyads found an association between parental FMSS-EE CRIT and engagement during the past year in each type of self-injurious thoughts and behaviours (SITB) in 12 to 17 year olds and that this relationship was moderated by adolescent self-criticism assessed with the Self-Rating Scale (SRS; Hooley et al., 2002) (Wedig & Nock, 2007). High levels of CRIT in conjunction with high self-criticism resulted in increased SITB. Specifically, similar to distributions of FMSS-EE found in previous clinical and community studies (Asarnow et al., 2001; Hirshfield, Biederman, Brody, Faroane, & Rosenbaum, 1997; McCleary & Sanford, 2002; Peris &

Baker, 2000), 25% of parents, predominantly biological mothers (n = 28), were rated high EE and 75% low EE.

In their community study, Psychogiou et al. (2007) examined associations between both child and parent psychopathology and mothers' EE toward their school-aged sons (mean age of 8). They found a significant and positive association between ADHD symptoms measured using the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) and maternal FMSS-EE CRIT in 100 boys, predominantly white British and of mixed socioeconomic status, selected randomly from schools. However, this association no longer remained significant when conduct and emotional problems were entered in the regression model. But the relationship between maternal EE CRIT and conduct problems and emotional problems persisted after maternal ADHD, measured with mothers' self-reports on the Current Symptoms Scale (CSS-SR; Barkley & Murphy, 1998), low mood indexed by self-report ratings on the General Health Questionnaire – 12-item Version (GHQ-12; Goldberg, 2003; Goldberg & Williams, 1988) and aggression were entered. The same pattern was found when maternal symptoms of psychopathology were entered in the first step of the stepwise regression and child symptoms in the second step.

Two studies which conducted analyses using data from the same ongoing large-scale multicenter study on the molecular genetics of ADHD have examined associations between parental EE and genes in children with ADHD. The first of these found some evidence for the role of EE in moderating the effects of genes on ADHD severity and comorbid CD; however, these G X E effects did not reach genome wide significance (Sonuga-Barke et al., 2008). The second study confirmed an association between parental EE and the presence of conduct problems in children with ADHD and that this association was significantly moderated by genetic factors, effects that were independent of whether a parent or teacher had rated the child's behaviour and site (Sonuga-Barke et al., 2009).

In a sample of 62 clinic-referred German children with ADHD, their parents and 61 healthy controls and their parents, Christiansen et al. (2010) found significant differences between the ADHD and non-ADHD groups in ratings of parental EE with mothers and fathers of children with ADHD manifesting higher EE than those of control parents. These significant differences did not remain when oppositional problems were entered as a covariate. Interestingly, high warmth scores, but not high criticism scores were associated with oppositional problems. The link between parental EE and oppositional problems was mediated by stress-related cortisol.

Finally, in one of the most recent studies of association between parental EE and child behaviour, Hale III et al. (2011) assessed perceived maternal EE using a shortened parent version of the Level of Expressed Emotion questionnaire (LEE; Cole & Kazarian,

1988), completed by mothers, and obtained adolescent self-ratings of internalising and externalising symptoms on the Reynolds Adolescent Depression Scale (RADS; Reynolds, 2000) and Child Behavior Checklist Youth Self-Report (YSR; Verhulst, van der Ende, & Koot, 1997) respectively in 497 Dutch adolescents, of which 57% were male, from the general community who were prospectively studied annually for three years . Structural equation analyses indicated that internalising and externalising symptoms predominantly elicit maternal EE, and not the other way round. The bidirectional effect between mothers' EE irritation and adolescents' behaviour was the only exception to this. These findings contradict Peris and Baker's (2000) longitudinal study that demonstrated maternal EE at preschool predicted diagnosis of ADHD in third grade. This discrepancy may be a reflection of the different methodologies used (i.e., FMSS vs. questionnaire and EE measured at two time points vs. three time points). Hale III et al.'s study is limited by sole reliance on self-reports of adolescent symptoms and EE and the use of a questionnaire which, although shown to have good psychometric properties, has not been compared to the CFI or FMSS. Further, whilst a need for clinically useful and accessible CFI alternatives has been identified, issues have been raised with questionnaire-based measures of EE (Hooley & Parker, 2006). Although community samples help evade the problem of referral bias, the findings also need to be replicated in a clinical population with examination of longitudinal associations between actual diagnoses, assessed via structured clinical interviews, and maternal EE.

The studies thus far reviewed have demonstrated there is increasing empirical evidence for strong associations between parental EE and a wide range of child behaviour including ADHD in both clinical and community samples. The majority of studies conducted to date have been cross-sectional in design limiting causal inferences in the relationship between parental EE and child behaviour to be made and highlight the need for prospective longitudinal designs to be employed. Further, these studies highlight the complexity of the relationship. Specifically, the high levels of comorbidity in childhood psychiatric disorders has made it particularly difficult to tease out whether high parental EE is linked to specific disorders or whether it is a correlate of child psychopathology generally. This is discussed next.

Clinical Specificity of EE

It is widely accepted that high rates of psychiatric comorbidity exist among paediatric populations (Angold, Costello, & Erkanli, 1999; Caron & Rutter, 1991). In fact, seldom do childhood disorders occur in isolation. This pattern of comorbidity complicates the relationship between parental EE and child psychopathology especially given that parental

EE is associated with a range of mental health conditions in children and adolescents. As a consequence, it remains poorly understood whether EE predicts certain types of child disorder or whether it is a non-specific correlate or risk factor for multiple conditions (i.e. whether it is more globally associated with child psychopathology). Discrepant findings relating to this issue have contributed to lack of clarity on the clinical specificity of parental EE. Next, the literature relating to the clinical specificity of parental EE in the broader context of child psychopathology will be presented. Second, given the high rates of comorbid ODD and CD in ADHD and research that has demonstrated ODD and CD are related to parent-child interaction, particular emphasis will be placed on studies that have specifically examined the relationship between parental EE, ADHD and comorbid ODD and CD. Given that aggressive status is associated with poorer outcome for children with ADHD (Hinshaw, 1987; Loeber, Lahey, & Thomas, 1991; Waschbusch, 2002) clarification of the discrepancy as to whether it is ADHD or comorbid antisocial and oppositional behaviour that drives the relationship between EE and ADHD is essential to understanding risk factors associated with maintenance or exacerbation of negative course.

On one hand, the results of some studies seem to suggest that parental EE is a non-specific correlate of childhood psychopathology. In a three-group study, Hibbs et al. (1991) found that levels of family FMSS-EE (with high family EE rated when one or both parents had a high EE rating) were higher in the two diagnostic groups (DBD and OCD) than in the control group; however, no significant differences were found in the rates of high parental EE between the two clinical groups in a sample of 128 male and female children and adolescents. In accordance with these findings, Stubbe et al. (1993) found that although maternal FMSS-EE was significantly higher in 6 to 11 year old boys and girls with an emotional disorder (anxiety or depressive disorder) or DBD (ADHD, ODD, or CD) as compared to children in the control group, no significant differences were found in rates of high maternal EE between the two diagnostic groups. Further support comes from Schwartz et al.'s (1990) previously mentioned study in which high overall EE was linked to an increased likelihood of receiving a DSM-III-R diagnosis of ADHD, ODD, or any disruptive behaviour disorder, separation anxiety, or a global rating of one or more diagnosis. In African American children, maternal EE was related to both hyperactivity and anxiety scores (Kwon et al., 2006). Hibbs et al. (1991) showed that high rates of maternal, paternal and family EE were more prevalent in two clinical groups than in normal controls, but did not differ between two diagnostic groups (OCD and DBD).

On the other hand, there is evidence to suggest EE may be a risk factor or correlate for specific disorders. Asarnow et al. (2001) demonstrated significantly elevated rates of family ratings of FMSS-EE CRIT (generated using both mothers' and fathers' EE scores)

among children and adolescents with depression compared to controls, but no significant differences in parental EE toward non-depressed children and adolescents with ADHD compared to controls (parental EE among children with ADHD marginally higher). These findings suggest maternal criticism may show some specificity as a risk factor or correlate of depression in two independent samples that vary in age (children and adolescents) and severity of impairment (inpatient and outpatient). The authors hypothesised that because ADHD tends to have an early onset with symptoms evident early in life, parents may be less likely to be critical as they may view their child's difficulties as characteristics of the child. In contrast, with disorders, such as depression, which are episodic and characterised by distinct behavioural changes and for which the symptoms may be perceived by the parent as under the patient's control (Hooley, 1987), parents may be more likely to respond with emotional reactions such as high critical EE in their efforts to cope with the behavioural changes. That criticism, but not overall EE, discriminated between the depressed and non-depressed ADHD groups highlights that in the relationship between parental EE and child psychopathology there is also an issue of the specificity of the individual components of EE. This is a point that will be returned to later in this chapter. The findings of this study also highlight a lack of specificity of parental EE to ADHD. Most of the few studies of children with ADHD have not specifically examined the impact of comorbid depression in their samples (Hibbs et al., 1991; Stubbe et al., 1993), therefore the question of whether elevations in parent FMSS-EE are associated specifically with ADHD or specifically with depression or indeed are related broadly to multiple child psychiatric outcomes remains to be resolved. However, some studies have shown no association between EE and internalising behaviour (e.g., Nelson, Hammen, Brennan, & Ullman, 2003; Baker et al., 2000).

Further evidence, albeit much less robust, for specificity in the relationship of parental EE to depression comes from Psychogiou et al.'s (2007) study which aimed to examine which aspects of child behaviour, measured dimensionally using the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), predict maternal EE. The authors reported a significant relationship between ADHD and maternal EE, but that this did not remain when conduct and emotional problems were entered in the regression model. However, it is difficult to draw conclusions about the specificity of parental EE to mood, first, because conduct problems were also associated with maternal EE, and second because the measure of emotional problems taps more broadly into both mood and anxiety symptoms. In contrast to these findings, Peris and Baker (2000) found that parental EE measured at preschool was predictive of diagnosis of ADHD four years later, but was not predictive of any other condition diagnosed, including ODD.

Asarnow et al. (1993) found that the significant association between parental EE and outcome one-year post hospital discharge among 26 seven-to-fourteen-year old boys and girls with depressive disorders (major depressive disorder [MDD], dysthymic disorder [DD], and double depression [MDD/DD]) remained after controlling for DBD, including ADHD, CD or ODD. However, a marginal trend for comorbidity was found with DBD associated with persistent mood disorder. Although the sample used was small, it was composed of rigorously diagnosed psychiatrically hospitalised children and association between FMSS-EE and child outcome was robust and persisted in presence of potential mediating variables (e.g. – treatment type, depressive disorder type, SES, single versus parent household, child age and gender, comorbid DBD diagnosis or anxiety diagnosis). This study provides some evidence for the specificity of parental EE as a correlate for depression but is limited due to its design (i.e., controlling for comorbid conditions in one group of children with depressive disorders rather than between-group comparisons of children with a diagnosis of depression, one or more comparison psychiatric groups and controls). Another study examining whether FMSS-EE is a specific risk factor for depression in children and adolescents, parents of children/adolescents with depressive disorders had higher levels of EE compared to parents of children with schizophrenia-spectrum disorders or typically developing controls (Asarnow et al., 1994). However, in this study, within the depressed group high levels of maternal EE were more prevalent in mothers of children with depression and comorbid disruptive behaviour disorder compared to mothers of children with pure depression or depression with comorbid anxiety disorders. Similarly, Hirshfield et al. (1997) reported that mood disorders in conjunction with behaviour disorders was associated with borderline and elevated EE CRIT, but because 70% of the sample had comorbid mood and behaviour disorders, it was not possible to examine the separate effects for mood disorders as opposed to behaviour disorders.

Other patterns of clinical specificity with parental EE have been shown in adolescents experiencing self-injurious thoughts and behaviours. Wedig & Nock (2007) found that in their sample of adolescents the association between self-injurious thoughts and behaviours was not accounted for by the presence or comorbidity of mental disorders, therefore, providing evidence of a direct and specific relationship between EE and self-injurious thoughts and behaviours.

Studies that have aimed to examine the role of comorbid ODD and CD in the relationship between ADHD and parental EE have also yielded mixed results, with some offering evidence that ADHD is associated with high parental EE independent of comorbid ODD/CD and others suggesting the opposite. In one of the first investigations of this kind, the authors predicted that high parental EE would be associated with the presence of

ODD/CD in ADHD children (Marshall et al. 1990). Their hypothesis was based on previous research which indicated that concurrent aggressive symptomatology and negative parent-child relationship were highly correlated in children with ADHD and that these two factors either independently or together may be associated with poor prognosis in children with ADHD (Paternite & Loney, 1980; Patterson, 1962, 1975, 1979). In a sample of 29 boys aged 6 to 11 years who met DSM-III-R criteria for ADHD only or ADHD plus ODD or CD and were participating in an intensive summer programme for ADHD children, they found that, contrary to their hypothesis, FMSS rated parental EE toward the child and child's EE toward the parent were highly correlated and that both parental and child EE did not differ significantly between the ADHD only and the ADHD-ODD/CD group regardless of whether separate maternal and paternal or parental unit classifications of EE were made (e.g., parental unit was classified as high EE if at least one parent in dual parent families and the sole parent in single-parent families expressed high EE toward the child). The same pattern held when between-group comparisons of parental interactional behaviour, rated from observations using a family interaction task, were examined. In accordance with these findings, more recently, in their study of preadolescent girls diagnosed with ADHD also taking part in a summer five-week enrichment programme, Peris & Hinshaw (2003) found that high maternal EE was associated with both ADHD and comorbid ODD/CD, but that the link between ADHD and maternal EE withstood control of ODD/CD; the association between ODD/CD and maternal EE did not withstand control of ADHD. Further support for specificity of EE to ADHD comes from Peris & Baker's (2000) finding that high parental EE ratings at preschool predicted diagnosis of ADHD in children at third grade, but not any other condition.

In contrast to these findings, Taylor et al. (1986) found that in a sample of boys aged 6 to 10 years who had been referred to psychiatric clinics because of antisocial or disruptive behaviour, conduct problems, but not ADHD symptoms, were associated with less maternal warmth and more criticism. However, maternal EE in this study was measured with a general semi-structured interview of family functioning and not measured with the FMSS or CFI which are specific standardised measures of EE. Similarly, a community-based study of school-aged boys reported that the significant association between FMSS rated maternal EE and questionnaire measured ADHD symptoms did not remain significant when conduct and emotional problems were entered in the regression model (Psychogiou et al., 2007). This study suggests that conduct problems may play a more important role than ADHD in predicting high maternal EE; however, because the study was not conducted in a clinical sample of children with a diagnosis of ADHD it is difficult to make assumptions about the role of comorbid conduct problems in the relationship

between maternal EE and ADHD. These results as aforementioned perhaps explain about the specificity of parental EE more generally. More robust evidence to support the notion that comorbid ODD/CD plays an important role in the relationship between parental EE and ADHD comes from more recent studies.

Sonuga-Barke et al. (2009) reported that, in a large sample of male children and adolescents, parent and teacher rated comorbid conduct problems in children diagnosed with DSM-IV ADHD Combined Type were significantly predicted by mothers' and fathers' CFI rated negative EE with effects generalising across national and cultural settings and that sensitivity to the effects of parental EE on conduct problems was moderated by variants of the DAT1 and 5HTT genes. Congruent with these findings, a study of children with and without ADHD found higher ratings of parental EE among parents of children with ADHD as compared to parents of healthy controls, but that these significant effects were removed when children's parent and teacher rated oppositional problems were entered in the model as a covariate. Specifically, in the ADHD group, high warmth predicted low levels of oppositional problems (Christiansen et al., 2010).

Is EE a Unitary Construct?

In connection with the issue of which aspects of childhood behaviour problems are specifically associated with parental EE is the question of whether EE is a unitary/integrated construct or whether its individual components display different patterns of association with child and family factors. Daley and colleagues (2003) found that the individual components of a modified version of the FMSS were only moderately correlated suggesting that while the components correspond to the broad EE construct, at the same time, they may individually afford discrete information. Researchers have generally approached the exploration of EE in two ways. First, associations between overall EE and child and family factors have been examined whereby the EE construct has been dichotomised into high EE and low EE (e.g., Asarnow et al., 1993; Marshall et al., 1990; Hibbs et al., 1991; Kwon et al., 2006; McCleary & Sanford, 2002). Second, relations between overall EE and its individual EE components and child and family characteristics have been investigated. For example, the FMSS which, as previously noted, is the most commonly used measure of EE in child populations, codes for two different components: criticism (CRIT) and emotional over-involvement (EOI). The theory is that these two components of EE measure distinct parental attitudes and thus should be associated with different behaviours. Research that has dismantled the overarching EE construct into its subtypes has revealed a number of patterns with regard to the relationship between the individual components and child psychopathology.

First, on one hand, there is evidence to suggest that high parental FMSS-EE emerges predominantly from the CRIT component and that this component is more frequently associated with child behaviour than EOI often to the extent that EOI is not related at all to psychiatric problems in children. EOI scale demonstrates poor prognostic power when considered independently. Clinical studies have reported that mothers' and fathers' CRIT, but not EOI, is associated with depression in children and adolescents (Asarnow et al., 2001; Silk et al., 2009). Similarly, in community samples, maternal CRIT, but not EOI, predicted externalising symptomatology concurrently (Baker et al., 2000) and diagnosis of ADHD longitudinally in young children (externalising behaviour) (Peris & Baker, 2000), emotional symptoms in school-aged boys (Psychogiou et al., 2007) and self-injurious thoughts and behaviours in adolescents (Wedig & Nock, 2007). For example, Peris and Baker (2000) showed that 64% of parents of children in the high externalising symptom group rated high in CRIT, 19% rated high in the borderline group and 7% of the low externalising symptom group, but EOI did not relate to group status and CRIT and EOI were not correlated. In the same sample, preschool EE ratings were driven by CRIT; EOI rarely scored and was unrelated to child behaviour (Baker, Heller, & Henker, 1998). Consistent with these studies, Peris & Hinshaw (2003) demonstrated that although both CRIT and EOI were associated with ADHD and ODD/CD the relationship was stronger for CRIT. Specifically, whereas mothers high in CRIT were twenty times more likely than mothers low in CRIT to have a daughter with ADHD, mothers high in EOI were about four times more likely than mothers low in EOI to have a daughter with ADHD. Similar results have been found in samples of preschool children whereby EOI rarely coded (Daley et al., 2003). Indeed high EE is rarely derived from EOI alone; rather it emerges from critical ratings that on occasion are concurrent with EOI ratings (Hibbs et al., 1991; Miklowitz, et al., 1984). Psychogiou et al. (2007) reported only one significant correlation among EOI components (positive remarks and statements of attitude), but almost all CRIT components significantly related to each other.

Correspondingly, when the CFI was employed to measure EE rather than the FMSS, Vostanis and colleagues (1994) found that parental EOI did not differ between two clinical and one non-referred control group and maternal EE CRIT was higher in the DBD group compared to the ED group, although no differences were found in CRIT between the ED and control groups. WAR, which is not rated in the FMSS, was also important as it differentiated the three groups with levels of parental WAR highest in the CD group, followed by the ED group and then the control group. In a 9-month follow-up study of the same sample, baseline low levels of maternal warmth predicted the presence of CD as rated by the clinician at the last clinical contact (Vostanis & Nicholls, 1995). These studies

provide evidence to support the predictive value of WAR as well as CRIT in child populations and may reflect CD and ED are related to different maternal styles. Low EOI might be due to use of the CFI which was developed for adult patients.

Some researchers have suggested that EOI may be more related to internalising symptoms than externalising symptoms. Specifically, EOI may show some specificity as a correlate of anxiety in children. The first investigation that systematically examined the role of the separate components of FMSS-EE demonstrated that whilst high maternal CRIT was associated with diagnosis of a DBD (ADHD, ODD, or CD) high EOI was related to diagnosis of an anxiety disorder in 6 to 11 year olds, controlling for potentially confounding demographic variables (Stubbe et al., 1993) (high EOI in mothers children with anxiety - 52% high EOI vs, 45% high CC of high EE ratings). However, this finding converges with only one other study. A pilot study which examined the links between behavioural inhibition and child psychopathology and FMSS-EE reported associations between EOI and child separation anxiety disorder in an at-risk, psychiatric control group of young children (aged 4-10 years) of mothers with and without panic disorder (Hirshfield, Biederman, Brody, Faraone, & Rosenbaum, 1997). However, out of the two samples included in the study, the association between maternal EOI and child separation anxiety disorder was only found in the at-risk sample. This may have been accounted for by the younger age or male gender in the at-risk sample or due to ascertainment differences (i.e., in at-risk sample many mothers had had clinically significant anxiety or mood disorders and many of the children carried a genetic vulnerability for anxiety disorder whereas as in the other sample which was epidemiologically derived fewer parents had clinically significant disorders). The authors speculated that EOI may influence the development of separation anxiety disorder only through children with a high vulnerability to anxiety disorders (e.g., children of agoraphobics) and not in others. If child separation anxiety disorder elicits overprotection, it may do so chiefly in mothers who are themselves anxious. However, the results of this study presented a mixed pattern as child behavioural inhibition was associated with high or borderline maternal criticism, independent of other measures of child psychopathology.

Inconsistent with these findings, Psychogiou et al. (2007) found that conduct problems, not emotional symptoms were associated with EOI. Further analyses that were conducted based on evidence suggesting that EOI might reflect more than two dimensions (Wamboldt, O'Connor, Wamboldt, Gavin, & Klinnert, 2000) revealed that the correlation between EOI and conduct problems was accounted for by positive remarks and statements of attitudes about the future. Children in this sample with conduct problems therefore received less positive remarks from their mothers. Nevertheless, in contrast to Hirshfield et

al. (1997) and Hibbs et al. (1991) who interviewed mothers and used DSM-III and DSM-III-R criteria respectively to diagnose anxiety disorders, Psychogiou et al. (2007) employed a broad measure of emotional problems based on a composite score for anxiety and depressive symptoms derived from a 5-point rating scale. This less comprehensive measure of anxiety may explain the divergent findings. Baker and colleagues (2000) did not find an association between EOI and internalising symptoms, but this may have been precluded by the low incidence of EOI. Furthermore, in children and adolescents with depression, high EOI was less common among subjects with comorbid anxiety disorder than those without comorbid anxiety disorder (McCleary & Sanford, 2002), but this difference did not reach statistical significance. Sixty five percent of parents were rated high in CRIT compared with only 21.7% in EOI and only 13% both CRIT and EOI. This contradicts previous studies demonstrating specificity of anxiety disorders to EOI. Adult studies also report that criticism is more associated with high EE than EOI (Miklowitz et al. 1984). By and large, more often than not, high EE is related to a high number of critical comments, more so than EOI, high EOI ratings are normally accompanied by high levels of CRIT and EOI and CRIT are often not significantly related (e.g., Hooley & Licht, 1997).

That most studies have not found EOI to be related to child behaviour or even to code brings into question the utility of EOI ratings with regard to children. Indeed, an investigation of the correspondence between CRIT, EOI and observations of parent-child interactions in clinic-referred children and adolescents revealed that none of the observed behaviours correlated with parental EOI (McCarty, Lau, Valen, & Weisz, 2004). The authors concluded that either the dimension EOI lacks validity with children and adolescents or that the behaviours that correspond to EOI are difficult to observe. The criteria for rating EOI in the FMSS originated from the CFI which was developed for use in adults. It may be that the decision rules used to distinguish over-involvement and self-sacrificing may need to be refined when applying EOI to children. However, it may simply be the case that high EOI is nore developmentally appropriate for children, especially young children, and children are not as impacted by the overprotective instincts of their parents to the same extent as adults and therefore for adults may be more intrusive, problematic and have more of a negative effect on adolescents and adults. Adults are aware that the behaviour that their parents exhibit is developmentally inappropriate. However, as mentioned above, adult studies also find that criticism is a more powerful predictor of psychopathology than EOI.

2.7.2 Other Child Characteristics

Although the link between parental EE and child behaviour is well established, less is known about the determinants of EE. Adult studies of the construct have implicated that some of the variance in EE is explained by characteristics of the patients themselves, for example, longer duration of illness, poorer social functioning, higher chronicity of delusional thinking have been found to correlate with high EE (Barrowclough & Tarrier, 1990; Glynn et al., 1990; MacMillan, Gold, Crow, Johnson, & Johnstone, 1986; Stirling et al., 1991). Such associations appear to be isolated and not well-replicated results (Hooley, Rosen, & Richters, 1995).

There is limited evidence of child factors other than diagnosis or symptoms associated with parental EE in child populations. This may, in part, be due to a paucity of examination of child characteristics other than demographic variables and a proportion of studies that have been conducted in samples of boys (e.g., Marshall et al., 1990; Psychogiou et al., 2007) or girls (e.g., Peris & Hinshaw, 2003) only.

Child Age, Gender and IQ

By and large, most community and clinical studies have documented a limited role of gender and age in predicting parental EE. Hibbs et al. (1991) found that family EE, as well as maternal and paternal EE when examined separately were not significantly different toward boys or girls even though groups of children with DBD, OCD or controls which were matched for age and gender were made up predominantly of boys. Nor was EE related to age of child; high EE was spread evenly across age and gender. Likewise, in another study of children with DBD or ED, no significant associations were found between levels of maternal EE and child gender or age (Stubbe et al., 1993). Males were over-represented with a 2:1 ratio of males to females in Vostanis et al.'s (1994) study, but the effects of gender were not examined. The relationship between maternal EE and recovery in depressed children one-year post-hospitalisation remained when controlling for child age and gender (Asarnow et al. 1993). Asarnow et al. (2001) reported children were younger in the depressed group and marginally in the control group and a higher prevalence of paternal high CRIT in the depressed group and overall sample among older children (> 13 years of age). The subtypes of EE do not seem to differ according to sex or age either in depressed adolescents (McCleary et al., 2002). Edwards (2006) did not find any gender differences in the association between EE, as indexed by adolescents' ratings of their perceptions of their mothers' EE on an adjective checklist, and psychopathology in a clinical sample of 61 adolescents placed in residential care. Similarly, parental EE did not correlate with child gender or age in a sample of African-American children and their

mothers. There are very few exceptions to this consistent pattern. Cook, Strachan, Goldstein, & Miklowitz (1989) identified higher levels of EE in mother-son dyads than in mother-daughter dyads (73.1% versus 31.1% high/borderline) in a sample of adolescents with psychiatric problems. Contradictory to these findings, in a study of the relationship between maternal EE and child behavioural inhibition, child female gender was associated with maternal criticism and this effect remained when child age, behavioural inhibition and maternal anxiety were controlled (Hirshfield et al., 1997), but when the number of mood and behaviour disorders were controlled for the association fell to trend significance. EE scores did not differ according to child's age in Peris & Hinshaw's (2003) sample of children with ADHD and ODD/CD.

Few studies have examined the relationship between parental EE and IQ. Peris & Hinshaw found lower Verbal IQ scores among children of high EE parents. Other studies that have not used the construct of EE per se, but instead broader parent-child relationship quality indices have also found associations with IQ. For example, Croft, O'Connor, Keaveney, Groothues, and Rutter (2001) longitudinally examined the direction of effects underlying the association between adoptive parents' interaction quality with their children at age 4 and 6 years and their children's cognitive ability in a sample of 110 Romanian adoptees placed into UK homes before 24 months of age and 52 British adoptees who were placed into adoptive families before the age of 6 months. They found that cognitive/developmental delay mediated the association between parent-child relationship quality and duration of deprivation, that the most positive change in parent-child relationship quality was observed among children who exhibited cognitive catch-up between assessments and that the direction of effects seemed to be child to parent as the authors hypothesised.

Severity of Condition

Severity of illness in two diagnostic groups (DBD, OCD) assessed with the Child's Global Assessment Scale and NIMH global scales was not significantly associated with parental EE in Hibbs et al.'s (1991) study, but caution must be taken in interpreting these findings due to referral biases inherent in clinical samples.

2.8 Associations with Parent-Related/Family Characteristics: Empirical Evidence

In pinpointing determinants of EE, researchers have also looked at parent and family characteristics.

2.8.1 Family Socio-demographic Characteristics

On the whole, studies have found few significant associations between family demographic characteristics and parental EE. In relation to socioeconomic status (SES), Baker et al. (2000) reported that maternal EE scores were unrelated to SES; however, the large number of families of high SES may have precluded finding this relationship. Nevertheless, this finding is consistent with other studies which have also not found a relationship between SES and parental EE (Hibbs, Hamburger, Kruesi, & Lenane, 1993; Hirshfield et al., 1997; Stubbe et al., 1993). In one study, although children with CD were more likely to come from a manual occupation socioeconomic background and to have a younger mother compared to children with ED, differences between these groups on the EE scales were not accounted for by these factors as maternal CRIT had an independent effect in predicting CD status (Vostanis et al., 1994).

Interestingly, Stubbe and colleagues (1993) found that in their inner city community sample of children with conduct and emotional disorders, maternal CC was significantly higher in Roman Catholic families compared to non-Catholic families and there was a trend toward higher rates of overall EE. In addition, ratings of Hispanic mothers' EOI were significantly higher compared to other ethnicities. Aside from these two demographic variables, birth order, teenage mother, single parent status, maternal education and maternal employment were not found to be associated with overall EE, CC or EOI. Consistent with these findings, other studies of depressed children and adolescents have found neither overall EE nor the subtypes CRIT or EOI differed according to single parent status (Asarnow et al., 1993; McCleary & Sanford, 2002). Hibbs et al. (1993) found a higher proportion of married couples in the OCD and control groups compared to the DBD group and that parents of children with OCD had a higher level of education than the control group, but neither parental level of education or marital status were related to parental EE. In a more recent study, Boger, Tompson, Briggs-Gowan, Pavlis and Carter (2008) demonstrated that mothers' FMSS-EE was not associated with ethnicity, parent relationship status, maternal education, maternal work status or poverty status in a sample of young at-risk children. Together, these studies suggest that parental EE is not a proxy for broader demographic risk. In another study, neither EE nor subtypes differed according to whether adolescents with MDD or CD lived with family (McCleary & Sanford, 2002)

2.8.2 Parental Psychopathology

The high prevalence of psychological problems in the parents of children with ADHD (and other child disorders) may further complicate the relationship between parental EE and ADHD and indeed other childhood psychiatric disorders. The parents of children with

ADHD often have adult ADHD (Biederman et al., 2002; Faraone et al., 2005), depression, anxiety (Minde et al., 2003; Murphy et al., 1996; Young et al., 2003) and aggression (Cadoret & Stewart, 1991; Dowson & Blackwell, 2010). At a general level, most studies reveal an elevation in psychological problems among parents of children with ADHD across a range of child ages and in both clinic-referred and community samples (e.g., Befera & Barkley, 1985; Murphy & Barkley, 1996; Scahill et al., 1999; Shelton et al., 1998). Biederman et al. (1992) found elevated rates of mood, conduct, substance use and anxiety disorders as well as ADHD among first degree relatives of children with ADHD compared to controls. The extent to which these problems are specific to ADHD or vary across ADHD subtypes and comorbidity with conduct problems is not revealed in these studies. Parents with psychological illness may be especially vulnerable to the provoking effects of child ADHD on EE.

Asarnow and colleagues (1993) suggested the need for further research to clarify the relationship between parental EE and psychiatric status. Schwartz et al. (1990) found that maternal criticism was associated with the presence of depressive illness and that the increased risk of psychiatric diagnosis in children of high EE mothers' was not independent of mothers' affective disorder. Similarly, in another study, the parents of children in two diagnostic groups (DBD and OCD) had more psychiatric disorders than the parents of controls and maternal and paternal high FMSS-SEE was associated with parental psychiatric illness in all three groups combined (Hibbs et al., 1991). However, when the diagnostic groups were examined separately there was no association. Whilst for fathers no specific diagnostic category was predictive of high EE, mothers with a mood disorder were five times more likely to be of high EE status. Indeed, paternal psychiatric status was the only predictor of high levels of EE in fathers. For mothers, child characteristics were important too. Hirshfield and colleagues (1997a, b) reported that high levels of CRIT were associated with increased behavioural inhibition in children of anxious mothers. These studies suggest that parental psychiatric status plays an important role in the relationship between parental EE and child mental illness. Recently, maternal EE toward children aged 8 to 12 years at risk for depression was associated with maternal depression in a child's life time and current and history of maternal depression and maternal EE were both independently associated with children's externalising symptoms (Tompson et al., 2010). Together, these studies indicate a role of parental psychiatric illness in determining high levels of EE toward children with behavioural and emotional problems.

In contrast, although over half (59.6%) the parents of adolescents with depression reported current or history of depression, parent depression history was not related to EE status (8/23 – no history parents were high EE and 15/35 – current or no history parents

rated high EE). Differences in sensitivity of measures of parent depression may account for the differences in results between this and previous studies of children and adolescents with depression. The association between FMSS maternal EE and ADHD diagnostic status in girls aged 6 to 11 years was also independent of maternal depressive symptoms measured using the Beck Depression Inventory (BDI; Beck, Steer, & Carbin, 1988) (Peris & Hinshaw, 2003). Psychogiou and colleagues (2007) found few associations between maternal psychopathology and EE. Whereas maternal aggressive symptoms and low mood were not significantly associated with maternal EE, mothers' ADHD symptoms were significantly related to maternal CRIT; however, when child characteristics were entered in the regression model, this effect was lost. Unexpectedly, mothers with low mood expressed fewer critical comments. The authors speculated that this may have been due to mothers of low mood being less talkative or because the sample was drawn from the community. The findings of an earlier community study also did not reveal associations between FMSS measures of overall EE and CC and maternal depression, neuroticism or introspectiveness (Stubbe et al., 1993). When considered alongside parental EE and overall family functioning, Kershner et al. (1996) found mothers' mental health did not predict clinical versus nonclinical group membership.

2.8.3 Other Measures of the Family Environment

It is still not well known whether parental EE toward children represents a unique dimension, thus has independent explanatory power beyond other measures of the family environment, or whether it reflects other measures of parenting behaviours. Some authors have argued that from a clinical perspective it is important to have better understanding of this issue. This is because if other dimensions of the family environment (that are often measured with questionnaires that are much quicker to complete compared to the coding of EE) are found to have as much predictive power, then it may be of greater utility for service providers to use such alternative measures to seek to understand families of young children with behavioural and emotional problems.

Studies that have investigated the relationship between parental EE and family/parental stress and adjustment/coping have produced mixed results. Baker et al. (1998, 2000) reported that mothers' CRIT and two expanded FMSS codes (positive affect and worry) were moderately related to scores on the Parenting Daily Hassles Scales (Crnic & Greenberg, 1990) and the Family Impact Questionnaire (FIQ; Donenberg & Baker, 1993), which enquires about the target child's impact on the family, and that these measures differentiated three child groups (high behaviour problem>moderate behaviour problem>comparison) as well as or better than the EE scores. In addition, although high

maternal CRIT predicted child externalising problems at preschool, this association did not remain significant when parent al stress and adjustment measures were entered in the regression model. Indeed, FIQ negative impact was the only predictor that entered. The same pattern was found when associations between maternal EE at preschool and child problems 2 years later at 1st grade were explored. These findings seemed to suggest that parental EE may not be a unique dimension of the family environment, at least not beyond mothers' own reports of child-related stress. The authors speculated that FMSS codes are behavioural measures and may more directly reflect how mother's experiences of child-related stress translate into attitudes and expression that can influence the child.

In contrast to these findings, in 91 young children selected from the same longitudinal study as the latter study, that preschool maternal FMSS ratings at preschool predicted diagnosis of ADHD at 3rd grade was independent of FIQ scores. Similarly, although actual levels of maternal stress, measured using the Parental Distress subscale of the Parenting Stress Index (PSI; Abidin, 1997), were not reported, Peris & Hinshaw (2003) found that when PSI scores were entered as a covariate in the regression analyses, the link between ADHD in preadolescent girls and maternal EE remained.

Two studies, one using the FMSS and the other the CFI, compared parental EE ratings and scales of the Family Environment Scale (FES; Moos & Moos, 1981) provide further evidence of a limited relationship between parental EE and other measures of the family environment. Whereas EE is rated by an independent assessor, FEE scales reflect parents' perceptions of their family environment. Stubbe and colleagues (1993) who examined the conflict and expressiveness subscales of the FES found that only the conflict scale of the FES was associated with overall EE and EOI. This scale was not associated with CC. Vostanis and colleagues (1994) paired some of the scales of the FES and components of the CFI (criticism [EE] – conflict [FES], warmth [EE] – cohesion [FES], warmth [EE] – expressiveness [FES], & EOI [EE] – control [FES]). Similar to the latter study, they found that none of these pairs were significantly correlated, with the exception of a significant association between ratings of maternal CRIT and conflict within the group of children with conduct disorders. Moreover, in contrast to the EE ratings, none of the FES scales distinguished between the two diagnostic groups of children with conduct disorders and emotional disorders. In a 9-month follow-up of the same sample, mothers' ratings of family conflict at initial assessment were significantly associated with dropping out of treatment in the CD group and in the ED group, initial ratings of maternal cohesion predicted absence of psychiatric disorder at the last clinical contact.

Hibbs and colleagues (1993) reported an association between high levels of FMSS-EE in mothers (but not fathers) and the achievement orientation and conflict subscales of

the FES in two diagnostic groups and a control group combined and in the OCD group, but only in relation to achievement orientation. However, it was difficult to determine whether familial conflict was due to psychiatric disorders or vice versa and whether high EE was a consequence of parental psychopathology or conflict given that in the regression analysis 33% of the variance in high EE was accounted for by maternal psychopathology and 40% by conflict. These findings seem to suggest that the observational measure of EE is more strongly associated with ratings of child behaviour than maternal ratings of their family environment given that few FES scales were related to EE ratings. However, in the latter study it is difficult to draw conclusions because the link between child behaviour and parental EE was not examined.

With respect to marital adjustment, EOI, but not CRIT was related to disagreements with partner and quality of parental relationship in Stubbe et al.'s (1993) study, but not CRIT. Although in this study, EOI was related to anxiety disorder, given that few studies have found an association between EOI and psychiatric disorder, it may well be that EOI is related more to other measures of the family environment rather than EE. For mothers, but not fathers, low EE was related to marital adjustment as measured using the Dyadic Adjustment Scale (DAS; Spanier, 1976), but high scores on the DAS were also associated with absence of psychiatric illness in all three groups combined (DBD>OCD>controls) and in the OCD and control group, but not the DBD group, when examined separately in Hibbs et al.'s (1991) study. Psychogiou (2004) did not find any relationship between child ADHD symptoms on empathy and personal distress.

2.9 A Theoretical Framework of the Parent EE-ADHD Link

There is continuing debate and uncertainty in the literature about the direction of effects in the relationship between EE and ADHD. The cross-sectional nature of most studies that have examined how EE might relate to ADHD has precluded cause-effect relationships to be tested. As demonstrated in the literature reviewed above, both child and family characteristics are associated with parental EE. A key question that requires further attention is whether parental EE is driven by the behaviour of the child (i.e., child effects) or by general parent-related or family characteristics that are independent of a specific child (i.e., family effects). Furthermore, in any child effects found, determining which child characteristics explain these effects is complicated by high levels of comorbidity in this clinical population with studies thus far providing mixed evidence regarding the clinical specificity of parental EE. Although parental EE has been found to be related to family characteristics, little is known which family characteristics drive family effects on parental EE toward children with ADHD. It is also apparent that different child and family

influences may display different patterns of association with the individual components of EE as demonstrated by previous research. It also remains to be understood whether parental EE drives the development of child problems over time. These issues can only be addressed by considering child and family effects simultaneously. This is the approach the current thesis has adopted. In order to help understand and disentangle the relationship between parental EE and ADHD and the role of both child and parent/family influences on EE in ADHD families, first, four theoretical models are proposed. These models show the different ways that EE might be related to ADHD and are based on the literature investigating the relationship between family environment variables and ADHD. These models are represented graphically and a description of each model is presented followed by empirical evidence.

2.9.1 Parent Effects Model

Traditional views in the child socialisation literature prior to the late 1960s inferred that children are passive recipients of environmental influences and that parent-child interaction is unidirectional in a parent-to-child direction with only parents having effects on the behaviour of the child and not the other way around. In line with this view, it is speculated in the first model - the parent effects model - that high levels of parental EE contribute to and play a causal role in the onset and clinical course of ADHD symptomatology (see Figure 2.1). It is assumed that parent characteristics or circumstances alone are responsible for the high levels of EE and that the child's behaviour does not influence the parent. Limited evidence exists to support this model with a few exceptions. In a longitudinal study, Peris and Baker (2000) showed that maternal EE at preschool predicted diagnosis of ADHD four years later at third grade. In the broader context of child psychopathology, high parental EE has been found to predict the lack of recovery in children with depressive disorders (Asarnow et al., 1993). In contrast, however, Hastings et al. (2006) did not find a longitudinal relationship between maternal EE and child and adolescent externalising symptoms. Moreover, Lifford, Harold, and Thapar (2009) reported that a causal hypothesis of family relations (hostility) influencing ADHD symptoms was not supported. Another longitudinal study aimed to ascertain whether 1) maternal EE affected the course of adolescent internalising and externalising symptoms (a parent effects model); 2) the course of adolescent symptoms affected maternal EE (a child effects model); or 3) maternal EE and adolescent symptoms affected one another bidirectionally (Hale III et al., 2013). Structural equation models revealed that a parent effects model was not supported; rather, a child effects model best described the relationship.

Findings of treatment studies investigating the link between EE and clinical relapse in adult patients with psychopathology support the hypothesis that EE may play a causal role in the relapse process. Indeed, these studies have shown that patients with a range of disorders including schizophrenia (Miklowitz & Thompson, 2003), bipolar disorder (Miklowitz, George, Richards, Simoneau, & Succath, 2003) and anorexia nervosa (Eisler et al., 2000) who received family-based interventions, for example, that educate relatives about the illness and help to improve communication skills and problem solving, have significantly reduced relapse rates in comparison to patients who did not receive these interventions. However, it has been argued that caution should be taken in adopting such a potentially overly simplistic and unidirectional view of EE in relation to clinical relapse and adult psychiatric illness. Furthermore, family-based intervention studies in the adult EE literature have been used as evidence for a causal role of EE in clinical relapse; however, these studies do not indicate a causal role of EE in the onset of psychiatric disorders.

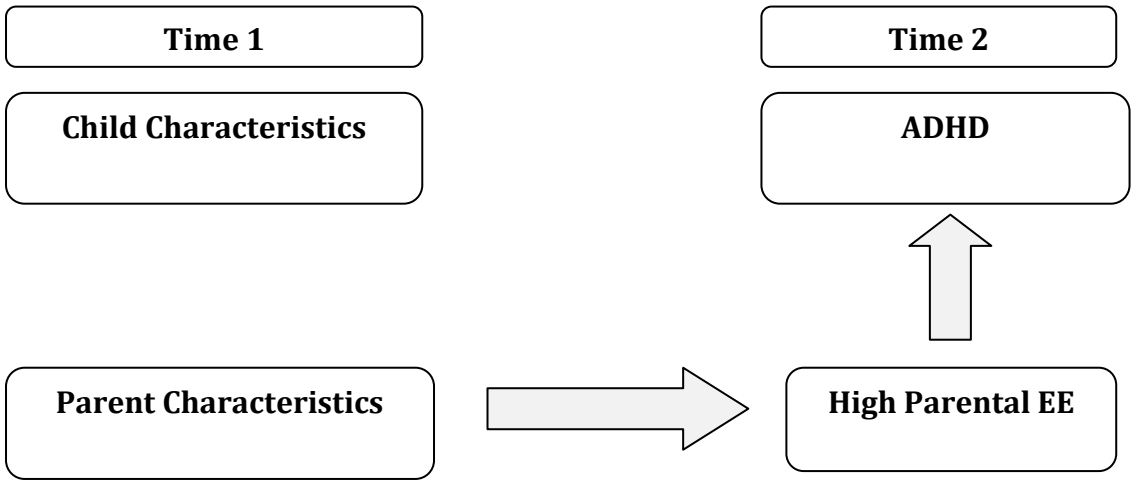

Figure 2.1 Parent Effects Model

2.9.2 Child Effects Model

In his seminal review of parent-child interaction and in an attempt to balance the child socialisation literature, Bell (1968) offered an alternative explanation to the unidirectional view of parent-child interactions. He argued that while it was accurate to infer that parents have an effect on the social interactions of their children, children also have an effect on the behaviour of their parents. From that point forward, researchers in child development were encouraged to take into account "child effects" on parent behaviour. Bell later further explained that a child is able to manipulate their environment, is an active participant in social interactions and has considerable stimulus control over adult behaviour toward the

child (Bell & Harper, 1977). In accordance with this contention of "child effects", implicit in the child effects model is the notion that high levels of EE are "provoked" by the core symptoms of ADHD which fosters a context for further negative parent-child interactions and negative behaviour from the child (see Figure 2.2).

The manner in which the inattention, overactivity and impulsivity of children with ADHD affects parent-child interaction patterns has been observed across a wide developmental range from preschool to adolescence. Observational cross-sectional studies examining differences between ADHD children and control children in their interactions with their mothers in an unstructured-play and structured task situation have illustrated that children with ADHD are less compliant to their parents' instructions and more oppositional, less responsive and able to sustain their compliance over time, show more off-task and "negative" behaviour and are more likely to request help and attention from their mothers during task accomplishment than their same-age control counterparts or children with learning disabilities (Campbell, 1973; 1975; Cunningham & Barkley, 1979). These interaction difficulties have been found to be worse and occur primarily when structured tasks are assigned to the child to perform and are less reliably observed during free play. Girls and boys with ADHD have been found to exhibit similar types of interaction discord (Befera & Barkley, 1985). Conflicted interactions have been observed in interactions with both mothers and fathers (Tallmadge & Barkley, 1983).

Cross-sectional studies have demonstrated developmental decline in these interactions, with problems being greatest in younger ADHD children, in particular 4 to5 year olds (Mash & Johnston, 1982), and declining significantly thereafter in older age groups (Barkley, Karlsson, & Pollard 1985a; Campbell, 1987). Despite that, similar age-related declines in maladaptive interaction behaviours are seen in control children to the extent that even in older ages, children with ADHD remain significantly deviant from control children in particular aspects of their parent-child interactions. In sum, observational studies have consistently demonstrated that when interacting with their parents, ADHD children, especially young children, exhibit high levels of noncompliant and negative behaviour. However, these studies have typically focussed on mother-son interactions in laboratory settings. Taken together, the results of these studies seem to suggest that it is the characteristics (e.g., inattentiveness, impulsivity, restlessness etc.) of the child with ADHD that elicit high-EE and negative reactions from parents. These studies are unable, however, to explicitly demonstrate cause and effect.

Experimental studies using stimulant medication lend more robust evidence for demonstrating how "child effects" impact on parent behaviour and induce high EE through showing the effects of child medication on parenting behaviour. It has been argued that the

use of a paradigm whereby using medication to manipulate the behaviour of an individual child and to simultaneously record the changes that occur in the reaction of parents during interactions with the medicated child offered a more direct method to assess the direction of effects in parent-child interactions (Barkley; 1981a; Kelly, 1981). If the primary symptoms of ADHD elicit negative, critical and controlling responses from parents, medication that successfully reduces the primary ADHD behavioural characteristics would produce reductions in the eliciting events for these negative parental behaviours.

The first studies to adopt this paradigm found that compared to when on placebo, medicated children with ADHD were less off-task and more compliant to their mothers' commands when medicated and their mothers' directive behaviours and use of commands decreased (Barkley, 1979; Barkley & Cunningham, 1979; Cunningham & Barkley, 1978; Humphries, Kinsbourne, & Swanson, 1978). In a succession of subsequent studies examining age-related changes in parental behaviour in response to administration of methylphenidate, Barkley and his colleagues (e.g., Barkley, 1988, 1989; Barkley, Karlsson, Strzelecki, & Murphy, 1984; Barkley et al., 1985) showed that when boys and girls with ADHD across a range of age groups in preschool and primary school were medicated, parent behaviour improves. This provided yet further support for a child-to-parent effect. However, the results of these studies were not completely consistent. The effects of child medication were often only seen during structured task interaction rather than in free play and in some cases not at all in free play. Task situations appear to foster the greatest conflicts for mother-child dyads (Mash & Barkley, 1986), especially those involving ADHD children and thus would be more likely to reveal medication effects on child and indirectly on parent behaviours. In addition, such effects were often only found when children were administered the higher medication doses.

Schachar et al. (1987) used a double-blind cross-over design to also examine the effects of methylphenidate on family functioning and relationships, including maternal expressed emotion as measured using the Parental Account of Child Symptoms (PACS; Taylor et al., 1991) in a sample boys (aged with ADHD and/or ODD/CD. In support of the child-to-parent effect, the results showed that on one hand for the 17 non-responders to medication, there were no differences in family functioning between the placebo and medication condition. On the other hand, for the 18 children who responded to medication (i.e., their behaviour improved to a clinically significant extent), the affective tone of parent-child relationships significantly improved, expressed maternal warmth increased, expressed maternal criticism decreased in the medication compared to the placebo condition. These findings were replicated when an analysis using teacher reports of child behaviour was used. The fact that improvements in family relationships were associated with active drug

treatment and not with the placebo suggest that the changes were not simply due to the expectations associated with drug treatment.

Other studies have demonstrated "child effects" using alternative methodologies to assess medication-induced changes in child behaviour and offer support for the notion that child characteristics drive high parental EE. For example, Ianna, Hallahan, and Bell (1982) observed the interactions between adult women and child confederates (school-aged children trained to portray specific behaviours) as they solved three tasks in one condition in which the child exhibited distractible behaviour and in a second condition in which the child displayed on-task behaviour. Assessment of the effects of this child behaviour manipulation on adult women revealed that women interacting with the distractible child were more controlling, made significantly more demands and more often asked the child about his performance and the demands of the task than women interacting with the on-task child. More recently, longitudinal studies have also provided support for the child effects with their findings that it was the course of externalising and internalising symptoms that affected maternal EE and not the other way around (Hale III et al., 2011).

It is possible that comorbid ODD or CD or indeed other comorbid problems (e.g., emotional problems) in children with ADHD also drive high parental EE toward children with ADHD. Studies that have examined the relationship between ADHD, ODD/CD and parental EE were reviewed earlier in this chapter and showed a mixed picture. Some studies have reported that it is the symptoms of ADHD that drive high parental EE, whilst others have shown that comorbid ODD/CD accounted for high parental EE.

In sum, studies that have manipulated child behaviour provide general support for the hypothesis that high EE is likely to be a response to characteristics of the child, most notably the core symptoms of ADHD, but also comorbid ODD/CD. The cross-sectional designs of many of these studies, however, do not permit the establishment of causal directions in the data. In addition, other experimental treatment studies have been less successful in demonstrating increases in parental positive behaviour. For example, the results from the Multimodal Treatment Study of Children with ADHD (MTA) in which children with ADHD were randomly allocated to either behavioural treatment, pharmacological treatment alone, behaviour therapy and medication combined or a community comparison group did not illustrate a clear cut relationship between improvement in ADHD symptoms in parent-child relationships (MTA Cooperative Group; 1999; Wells et al., 2000). Based on parent- and child-reports on questionnaire-based measures of parenting behaviour and family stress, the three MTA treatments were found to produce significantly greater decreases in negative parenting behaviour than did standard community treatment. However, no differences in parenting behaviour between the three

groups were revealed. In addition, no significant differences were noted between all groups on measures of family stress.

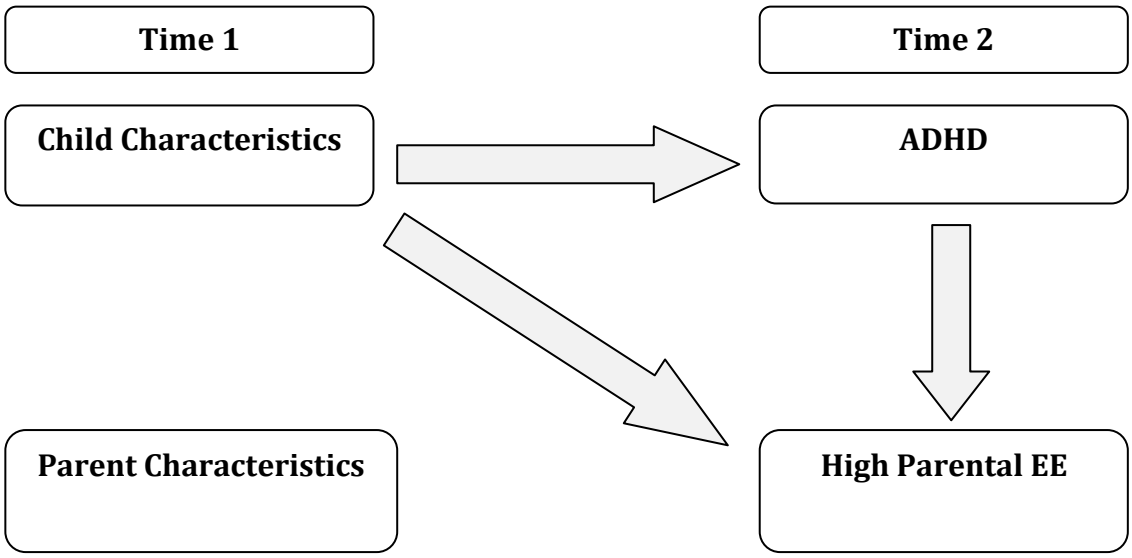

Figure 2.2 Child Effects Model

2.9.3 Parent and Child Reciprocal Effects Model

Child and family characteristics have both been found to be associated with parental EE as demonstrated in the literature reviewed earlier in this chapter. It is therefore plausible that both child and parent characteristics exert their influences on parental EE simultaneously. The design of many previous studies has limited the scope to explore these effects simultaneously. One study that attempted to achieve this found that when both child and parent psychopathology were entered in stepwise regression models, it was only child behavioural and emotional problems that predicted maternal EE in a community sample of boys (Psychogiou et al. 2007). Indeed, aside from this study, in most studies, child psychopathology has been the dependent variable and whether maternal EE and other family factors independently predict child behaviour has been the focus, rather than EE being the dependent variable and factors that predict EE being examined. In other words these studies have examined the cause-effect relationship between parental EE and ADHD in relation to whether child ADHD causes high EE or whether high EE causes ADHD or at least drives its course.

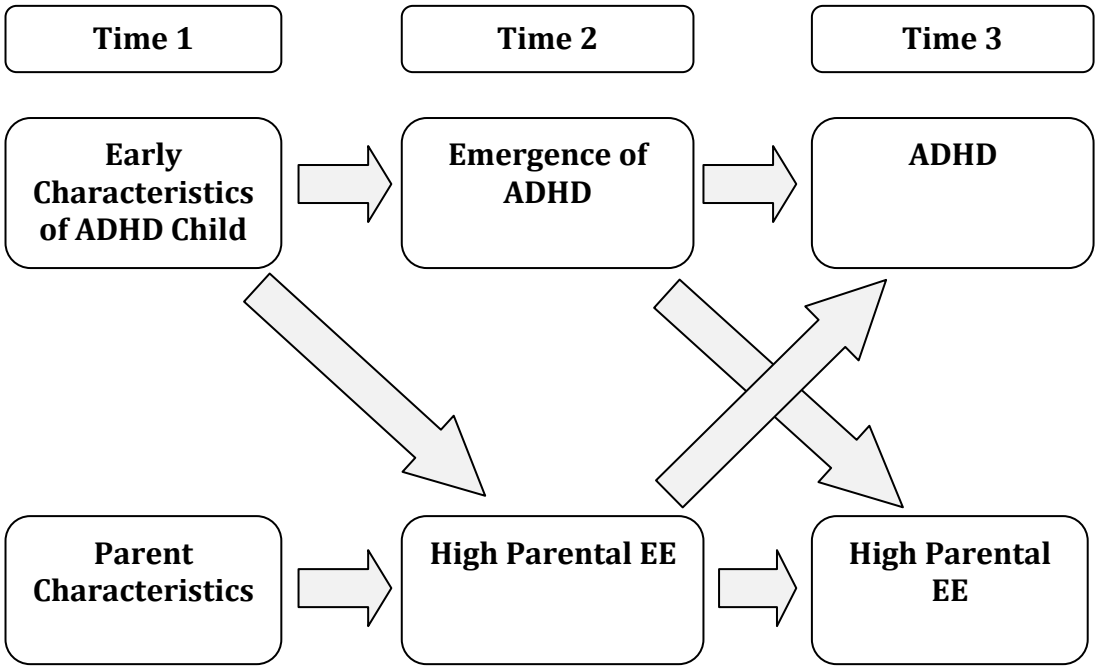

Figure 2.3 Parent and Child Reciprocal Effects Model

2.9.4 Extended Parent and Child Reciprocal Effects Model

The parent and child reciprocal effects model can be extended. Taylor (1999) hypothesised that whilst high parental EE might be driven by their child's core ADHD symptomatology rather than drive it, negative parental responses and high parental EE may contribute over time to the later development of defiance and antisocial behaviour rather than to the immediate conflicts in interactions. In other words, during development ADHD provokes high EE from parents early on in life which in turn leads to ODD/CD later on (see Figure 2.4). In the literature reviewed above, it was noted that young people with ADHD are less responsive to parental commands and parents tend to relate to them in a more controlling and critical fashion than to those without the condition (Barkley et al., 1985). It has been argued that these interactions may increase the likelihood of ADHD children developing ODD or CD during adolescence (Barkley et al., 1991). There is clear evidence for a central, even causal relationship between parenting variables and for example aggression (e.g., Patterson, 1982, Patterson, Reid, & Dishion, 1992; see also Anderson, Hinshaw, & Simmel, 1994).

Anderson et al. (1994) in a sample of boys aged 6-12 years with ADHD showed that maternal negative behaviours predicted both observed non-compliance exhibited in class and play settings and laboratory stealing. Stealing was predicted from maternal negativity even with child interactional compliance controlled. These studies demonstrate the

potential role of negative parental behaviour and high EE in predicting antisocial behaviours. Other studies have shown that negative parent-child interactions and family dysfunction (in addition to other important risk factors) mediate the increased risk for later substance abuse, criminality and antisocial spectrum disorders in adulthood (Klein & Mannuzza, 1991; Weiss & Hechtman, 1993). In addition, reductions in negative parenting during treatment have been shown to mediate reductions in children's behaviour in school Hinshaw et al., 1997). More specifically, studies were also described earlier in this chapter which reported associations between parental EE and oppositional and conduct problems in children with ADHD (Christiansen et al., 2010; Sonuga-Barke et al., 2009). However, these studies were cross-sectional thus limiting cause-effect inferences to be made.

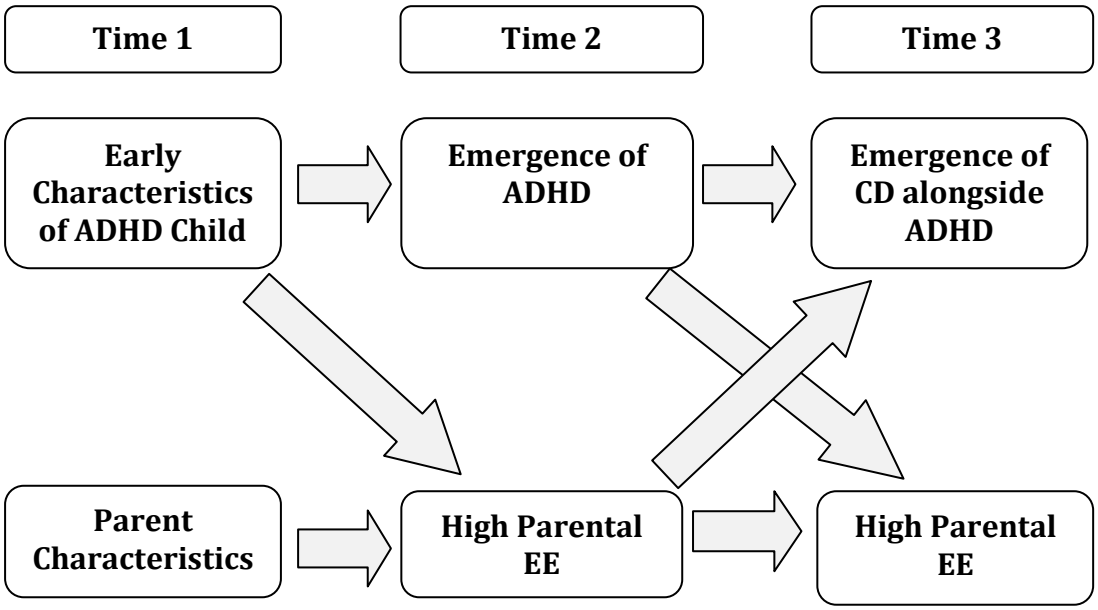

Figure 2.4 Extended Parent and Child Effects Reciprocal Effects Model

2.10 Summary of Chapter 2 and Aims of the Thesis

In summary, Chapter 2 integrated research findings relating to the relationship between parental EE and ADHD and child psychopathology more broadly. There is increasing evidence that parental EE is associated with ADHD. However, there is limited knowledge about the cause-effect relationship. The first two studies of the thesis aim to examine child and family effects on maternal EE using a within-family multilevel model design. Before moving onto these studies, the next chapter provides an overview of the key aspects of the design of this thesis and how this has allowed the aims of the thesis to be addressed.

Chapter Three

The Research Design: A Summary of Methodological Advantages

3.1 Overview of Chapter Three

Chapter 3 provides an overview of the research design employed in the studies of the thesis and the methodological advantages of this design for addressing the research aims. First, the use of a within-family/two-children-per-family (sib-pair) design will be discussed. Second, the methodological advantages of EE in comparison to other measures of the family environment will be outlined. Third, multilevel modelling (MLM) will be discussed, in particular the principles underlying it, and the benefits and utility of using this statistical technique to analyse the sib-pair data in the thesis. Fourth, the power of employing longitudinal data will be explained in brief with relevance to exploring cause-effect relationships.

3.2 Within-Family Design

This thesis aimed to provide further insight into the relationship between child ADHD and maternal EE, with particular emphasis on disentangling the direction of causation. The primary aim was to examine whether maternal EE is driven by the behaviour of the child (child-specific effects) or by family characteristics (shared family effects) that affect all children in the same family. This aim was made achievable first because a within-family/two-children-per-family design was employed. Data was obtained simultaneously for sibling pairs and their parents in families of children with ADHD. In each family the sibling pair consisted of one child with a clinical diagnosis of ADHD and in the majority of cases one child without ADHD. Before the methodological advantages of this design are discussed, child-specific effects and shared family effects are explained in relation to the literature on the shared and nonshared environment.

Children reared by the same parents within the same family encounter some of the same environmental experiences, including the same family environmental experiences.

Such shared family experiences can, for example, include socioeconomic status, parental stress, parental marital conflict, membership in a single-parent or step-family or parental psychopathology. These shared family environmental experiences are thought to influence how a parent relates to all children in the same family and are known as shared family effects. Shared family effects are therefore defined as characteristics of the parents or family context that are shared by all children in the family. Shared family influences are thought to create similarities between siblings.

Children raised by the same parents within the same family also encounter different family environmental experiences individually (e.g., one child may have a more negative relationship with their parent than their sibling) and are known as "child-specific" or "nonshared" experiences. Child-specific effects embody characteristics of the child (e.g., age, gender, temperament) that affect how the parent relates to the child (Bell & Harper, 1977; Lytton, 1990; Plomin, 1994). Twenty five years ago, Plomin and Daniels (1987) argued that these unique child-specific influences explain why siblings who are not identical and reared in the same family can differ considerably from each other, often to the extent that they are as different as children raised in different families. In the case of the current thesis, one child has ADHD while another child in the same family may not. Their argument was based primarily on the results of behavioural genetics studies which suggested that with genetic effects taken into account estimates of the nonshared environment were moderate to large across measures of personality, cognitive abilities and several forms of psychopathology. By contrast, they noted that shared or family-wide influences that create similarities between siblings had generally been found to be statistically indistinguishable from zero. This challenged long-standing and deeply held assumptions that parents had a family-wide or shared environmental effect on children's development.

The reconceptualization of environmental influences as predominantly nonshared or child specific in origin has since been accepted across developmental, personality and abnormal psychology. Indeed, a wealth of studies show that child-specific experiences whether subjective or objective are often more influential in predicting child and adolescent personality, psychological and behavioural outcomes than shared environmental experiences (Dunn & Plomin, 1990; Dunn, Stocker, & Plomin, 1990; Heatherington, Reiss, & Plomin, 1994; Plomin & Daniels, 1987; Plomin, 1994 Brody et al., 1998; Sheehan & Noller, 2002; Volling & Elins, 1998). Particular emphasis has been placed on differential parental treatment, specifically differential positivity (i.e., one child receiving more positive affect, engagement and involvement from the parent than another) and differential negativity (i.e., the parent directing more affectively negative behaviour toward one child

than toward another) as a likely candidate of the nonshared environment influencing differences between siblings. One hypothesis is that if maternal treatment is a risk factor for children's behaviour problems, child-specific maternal treatment should be associated with behavioural differences between siblings growing up within the same family. Researchers have established that children in the same family perceive differences in their parents' behaviour toward themselves and their siblings (Brody & Stoneman, 1990; Daniels et al., 1985; Koch, 1960), and that parents perceive differences in their own treatment of their children (Daniels et al., 1985). Furthermore, these differences in parental treatment within families, whether perceived or observed (Feinberg & Heatherington, 2001; Reiss et al., 1995), have been found to be predictive of child and adolescent outcomes concurrently and longitudinally (Anderson, Heatherington, Reiss, Howe 1994; Conger & Conger, 1994; Feinberg & Heatherington, 2001; Kowal, Krull, Kramer, 2004) and more negativity in the sibling relationship (Bryant & Crockenberg, 1980; Brody et al., 1987; McHale & Gamble, 1989; Stocker, Dunn, & Plomin, 1989). In recent years, Plomin and Daniel's original theory has been questioned.

In recent years, Plomin & Daniels (1987) theory that nonshared environmental influences account more for differences between siblings than shared environmental differences has been brought into question in relation to child and adolescent psychopathology for a number of reasons. First, this theory was based almost exclusively on studies of personality and cognitive ability and studies of psychopathology that were reviewed were limited to a few studies of disorders that rarely manifest before adulthood (e.g., schizophrenia). Second, the samples in behavioural genetics studies on psychopathology were often too small. Third, there were limited twin and adoption studies at the time that focused on child and adolescent psychopathology. Fourth, even in studies that have used very large samples and methodologies specifically designed to identify nonshared environmental sources of variance their efforts have failed to identify nonshared environmental influences (e.g., Reiss, Neidherhiser, Hetherington, & Plomin, 2000). Indeed, specific nonshared environmental factors typically account for 2% of the variance in outcome (Turkheimer & Waldron, 2000). More recently, some authors have proposed that rather than being a function of important and identifiable environmental influences that serve to differentiate siblings, the nonshared environment is largely composed of idiosyncratic and/or transient environmental influences with little or no long-term explanatory power (Rutter, Silber, O'Connor, & Simonoff, 1999; Turkheimer & Waldron, 2000). This school of thought is consistent with longitudinal data suggesting that nonshared environmental influences persist over time. Instead they appear largely to a given assessment period (Burt, McGue, Iacono, & Krueger, 2006; Rutter et al., 1999). Some

authors have suggested that given the collective absence of tangible results for the nonshared environment, it is time to reconsider shared environmental effects. Shared environmental influences appear to be both identifiable and persistent over time, at least prior to adulthood (Rutter et al., 1999). They also appear to persist over time (at least across childhood and adolescence). Though they may dissipate by adulthood, shared environmental influences appear to be persistent sources of individual differences in psychopathology prior to adulthood. In a recent meta-analysis of shared environmental influences on child and adolescent psychopathology, Burt (2009) found that shared family influences accounted for 10-19% of the variance within conduct disorder, oppositional defiant disorder, anxiety, depression and broad internalising and externalising disorders, regardless of their operationalization confirming , Burt's (2009) findings confirmed the hypothesis that there is mounting evidence that during childhood and adolescence, shared family influences may make an important contribution to most forms of psychopathology. Interestingly, the exception to this was ADHD which appeared to be largely genetic. Indeed, twin studies have suggested that many of the differences between siblings with and without ADHD are genetic (Bergen et al., 2007), including gene-environment interactions (Nikolas, Friderici, Waldman, Jernigan, & Nigg, 2010), but there is also a contribution of non-shared environmental effects (Larsson, Larsson, & Lichtenstein, 2004).

One contribution from behavioural genetics research for developmental researchers has been the recognition of the importance of investigating the development of all siblings in a family instead of the focus being on a single child per family. Multiple child studies make it possible to ask questions about how aspects of the nonshared (child-specific) environment unique to one child or indeed how the shared environment may lead to similar or different outcomes in the target child and his or her siblings. Traditionally, studies examining how family systems work in influencing child development have relied heavily on between-family/1-child-per-family designs in which single parent-child dyads from different families are compared with each other. Indeed this is the case with most previous studies examining the relationship between parental EE and child psychopathology. This design is based on the assumption that processes operate in similar ways for all parent-child dyads within families (McHale, Crouter, & Whiteman, 2003). However, on their own, correlations from such designs do not allow child-specific expression to be estimated. As aforementioned, the current thesis adopted a within-family/two-children-per-family (sibling pair) design to allow child-specific effects and shared family effects on maternal EE to be examined.

3.3 Prospective Longitudinal Design

A prospective longitudinal design was adopted in Studies 3 and 4 of this thesis. The advantages of longitudinal studies are well documented. Longitudinal designs encompass tracking the same type of information on the same subjects at multiple time points. Therefore, multiple or "repeated" measurements are yielded on each subject. It is a particularly important design in the field of child development as it allows how behaviour develops over time to be examined by assessing the same individuals as they age as well as identification of risk and protective factors that influence outcome to be determined. The type of longitudinal design used in the thesis is the simplest form of a prospective longitudinal study whereby the sample was followed into the future, with baseline measurements obtained at one time point (Time 1 [T1]) and follow-up measurements collected at a separate time point (Time 2 [T2]) five years later. Recall bias is minimised when collecting data prospectively. In this thesis, maternal EE as well as data on child and parent behaviour and other family variables were measured at two time points. Longitudinal data can provide information on causation, prognosis, stability and change (Rutter, 1988, cited in Sanson et al., 2002). This meant the stability and continuity of both maternal EE and child behaviour could be examined. Further, longitudinal studies allow differences in variation between individuals as well as change within them to be examined. The longitudinal design in combination with the sib-pair design allowed the effects of child-specific and family characteristics on maternal EE and child behaviour in children with ADHD to be assessed over time and therefore allow clearer inferences in the relationship between child and family characteristics and maternal EE and child behaviour to be made.

3.4 Multilevel Model Design

Multilevel modelling (MLM) was chosen as the method of statistical analysis for all studies in the current thesis. MLM is a regression based approach increasingly used across the social sciences which handles nested and clustered data. Nested data occurs when research designs include multiple measurements for each individual, and this approach allows researchers to examine how participants differ, as well as how individuals vary across measurement periods. A good example of nested data is repeated measurements taken from people over time; in this situation, the repeated measurements are nested under each person. Clustered data involves a hierarchical structure, such that individuals in the same group are hypothesised to be more similar to each other than to individuals in other groups. Here the focus is on clustered data as in the data of the current thesis sibling pairs are embedded within families. As aforementioned, in each sibling pair was one child with ADHD and in most cases one child without ADHD. MLM can be used flexibly with

different types of data structure (different numbers of levels e.g., most commonly two levels or three levels), types of design (e.g., cross-sectional, longitudinal with repeated measures, cross-classified), scales of the outcome variable (e.g., continuous, categorical) and different numbers of outcomes (e.g., univariate, multivariate) (Bell, Ferron, Kromrey, 2008). In their community study investigating predictors of between- and within-family variation in parent-child relationships, O'Connor, Dunn, Jenkins, and Rasbash (2006) used two level multilevel models and illustrated how research using multilevel designs and analytic strategies can be used to enhance understanding of family process using a large sample of families with at least two and up to three children. The advantages of MLM for addressing the aims of the thesis are discussed below.

First, MLM appropriately addresses the clustered nature of the data. Traditional standard regression (ordinary least squares, OLS) approaches or ANOVA-based techniques assume that each observation in a data set is independent. These methods of analysis are appropriate for data using between-family/single-child-per-family designs where observations can be assumed independent. The clustered nature of sibling pair data used in within-family designs violates this assumption because information obtained from siblings from the same family is likely to be more similar (i.e., expected to correlate) than that obtained from a randomly selected set of individuals. For example, genetics, shared environmental, background characteristics and interaction among siblings may in part contribute to this similarity. The use of standard regression or ANOVA based methods of statistical analysis with the data of the current thesis would mean ignoring the fact that sibling pairs are clustered within families and would increase the likelihood of making a Type 1 error to a value substantially greater than the nominal $\alpha = .05$. MLM was specifically designed to overcome this limitation. It extends multiple regression analyses to address the possible dependence of within-family data without violating the assumptions of standard multiple regression. The intra-class correlation is a critical statistic used to determine the degree of interrelatedness in the data. The ICC is calculated as the ratio of between-family variance ($\rho = \tau 00/(\tau 00 + \sigma^2)$ [residual/within-family variance]). A high ICC suggests that the assumption of independence is violated. When the ICC is high, using traditional methods such as multiple linear regression is problematic because ignoring the interdependence in the data will often yield biased results by artificially inflating the sample size in the analysis, which can lead to statistically significant findings that are not based on random sampling. In the case of the thesis, the ICC represents the extent of similarity in maternal EE between families. Put another way, the ICC represents the proportion of the total variability in maternal EE that is attributable to the families. If a family has had a large effect on maternal EE, then the variability within the family would be small relatively

speaking (i.e., maternal EE would be similar for both children in the sibling pair). In such a case, variability in maternal EE within families is minimised and the variability in EE between families would be maximised, thus the ICC would be large. In contrast, if the family has had little effect on maternal EE then it would vary a lot within families and the ICC would be small.

Second, MLM enables variance in the outcome variable (i.e., the dependent variable) to be partitioned into different levels of effect. This procedure overcomes the issue of interdependence discussed above. In the current thesis, there are two levels of effect, the first being within-family (child-specific) effects and the second being between-family (shared family/family-wide) effects. Thus, variance in maternal EE could be partitioned into child-specific (within-family) and shared family (between-family) effects. The individual children within the sibling pair comprise the lower child level (level 1) (i.e., the bottom of the hierarchy) and the families comprise the higher family level (level 2) (i.e., the top of the hierarchy). The primary aim of this thesis was to examine child-specific and shared family effects on maternal EE. Therefore in this case, child-specific (within-family) variance represents an estimate of the extent to which differences between children in their characteristics explain levels of maternal EE expressed by an individual mother. The shared family/family-wide (between-family) variance estimate is an indication of the degree to which differences between or across families can explain levels of maternal EE expressed by different mothers (i.e., the amount of variation there is between families in mean scores for maternal EE). In other words, it attempts to explore where factors that differentiate families from one another can explain variance in average EE levels expressed by mothers toward the two children in the sibling pair within families. One of the most commonly used approaches for examining multilevel data to partition the variance in the outcome into level 1 and level 2 effects is to first run an unconditional means model which is analogous to a one-way ANOVA . This model is run with no level 1 or level 2 predictor variables. This provides a baseline estimate of the level 1 and level 2 variances. It is by running this model that one can determine the ICC and assess whether a multilevel analysis is warranted (i.e., if no between-family effects are found, there is no need to use MLM). In the current thesis, this initial model was conditioned on ADHD diagnostic status and thus instead of being an unconditional model was a conditional model. This was important because of the overlap between ADHD and sibling status in the data used (all probands had a clinical diagnosis of ADHD and their siblings did not with a few exceptions). Therefore, the conditioned model provided an estimate of child-specific variance whilst taking account of the child's ADHD diagnostic status

Third, MLM allows the exploration of level 1 and level 2 variables that explain level 1 and 2 effects. Therefore, the aim of examining which child and family characteristics explained any child-specific and shared family effects found could be addressed. In any child effects found, it is possible that ADHD might explain these effects on maternal EE, but at the same time other child factors that differentiate the child with ADHD from the sibling without ADHD may be important too (e.g., oppositional/conduct problems). Likewise, if families do differ from another in levels of maternal EE, maternal psychological problems for example, may explain these shared family effects. Predictor/explanatory variables are defined as either level 1 (i.e., child-specific) or level 2 (i.e. family) predictors according to how they are assessed i.e., whether the unit of analysis is the individual child or the family. For level 1 predictors each child in the family has a unique score and for level 2 predictors, all children in the family have the same score. Multilevel model testing typically occurs in a stepwise fashion. First the above-mentioned unconditional or in this case conditioned model is run followed by models in which level 1 and level 2 predictors are added. These are described in turn below.

A random coefficients model is used to partition within-family variance as a function of a level 1 predictor. In this case, this yields the average intercept for maternal EE scores and the average slope of ADHD diagnostic status and maternal EE ratings of all sib-pairs. This approach also tests for significant differences between each sib-pair. Average intercept and slope estimates are yielded as fixed effects, while a significant p-value for the variance of the slope estimate indicates that the slope varies across sib-pairs (i.e., within-families). Fixed effects demonstrate the average relationship between the predictors and the dependent variable and can be interpreted as coefficients in a multiple regression. The coefficient is significant ($p < .05$) when $T = b/se(b) = 1.96$, $p \leq .05$ unstandardised.

A means-as-outcomes model is used to explain the variation that occurs in individual or group means as a function of a level 2 variable. For example, it was predicted that levels of maternal depressive symptoms would explain maternal EE scores for a sib-pair. By including a level 2 predictor it is hypothesised that the individual or group mean will be altered compared to the unconditional means model.

The next model includes both level 1 and level 2 predictors. This step is only completed if significant variability is accounted for by level 2 and level 2 predictors. Given that model testing occurs in a stepwise fashion, this model serves as the final step. When using full maximum likelihood estimation, the extent to which one model provides a significantly better fit than another model is evaluated by comparing the change in the log likelihood of the two models in relation to the difference in the degrees of freedom.

MLM also overcomes the drawbacks of using simple difference models and residualised models which studies of the nonshared environment have heavily relied on. Simple difference models (Plomin et al., 1996) relate sibling differences on an environmental measure to sibling differences in outcome (e.g., antisocial behaviour) or at the outcome of a single sibling. Two sibling-difference variables are used. The first variable reflects differences in the siblings' experiences (e.g., differential parenting) and the second variable reflects differences in the siblings' behavioural outcome (e.g. antisocial behaviour problems). The correlation between the two difference scores reflects the contribution of nonshared experiences to the creation of sibling outcome differences, independent of factors that differ between families (Rovine, 1994). Sibling difference scores are generally computed by subtracting one sibling's score on a measure from the other sibling's score on the same measure.

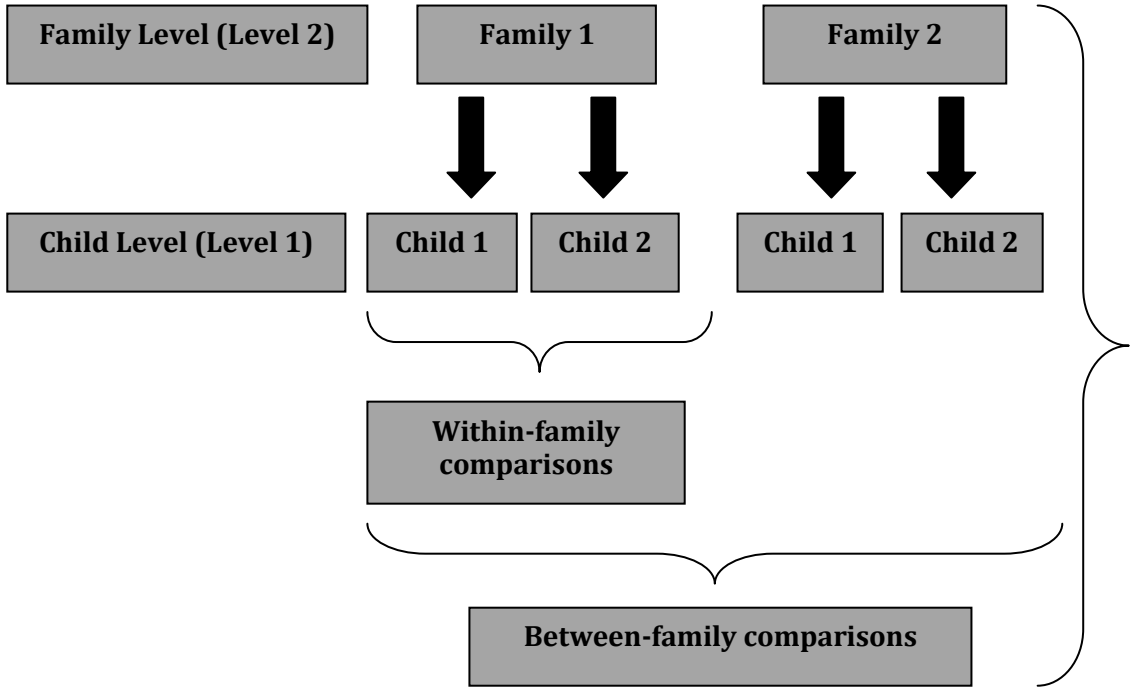

Figure 3.1 Structure of the data in the thesis

3.5 Overview of Aims and Methods of Thesis

The literature reviewed in Chapters 1 to 3 has demonstrated that gene environment interplay is important in understanding child psychopathology, and that of a multitude of environmental influences, family processes play an important role in the development and maintenance of child outcomes. In contrast to other measures of parent-child relationship quality, parental EE is unique in that it allows child-specific effects to be estimated. There is an abundance of evidence indicating associations between parental EE and child

psychopathology, including ADHD. However, the vast majority of studies have been cross-sectional in design, making it impossible to be able to make causal inferences. The first aim of this thesis was to disentangle the direction of causation in the relationship between parental EE and ADHD in relation to whether it is the behaviour of the child with ADHD (i.e., child effects) or shared characteristics of the parent or family more generally that are independent of a specific child (i.e., family effects) that predict parental EE. The literature reviewed also demonstrated that due to the high levels of comorbidity found among child psychiatric disorders, it is far from clear as to whether EE or indeed broader indices of parent-child interaction are related to specific disorders or whether they relate more generally to externalising and internalising behaviour. The second aim of the thesis, therefore, was to tease apart whether it is ADHD per se that drives high maternal EE or whether it is other comorbid problems, in particular oppositional and conduct problems. The final aim was to examine whether parental EE predicts child problems over time.

The International Multicentre ADHD Genetics (IMAGE) project, an ongoing European collaborative study which aims to examine the genetic mechanisms involved in ADHD and includes Southampton as one of its UK sites, provided a basis for this programme of research to be conducted and the aims of the thesis to be achieved. Of particular importance was the availability of data collected on children and adolescents with a clinical diagnosis of ADHD and their siblings without ADHD (in most cases). This within-family/two-children-per-family (sibling pair) design allowed child-specific effects and shared family effects on maternal EE and child behaviour to be estimated. This is explained in detail in the next chapter.

Data that had already been collected as part of the Southampton arm of the IMAGE project between 2004 and 2006 (referred to as Time 1 [T1] in the thesis] was utilised, in part, to conduct the analyses in this thesis. Written consent had been obtained from all but one of the 77 families who took part in the study to be contacted in the future about potentially participating in a follow-up study. This provided the platform for a follow-up study to be conducted and the basis for a longitudinal programme of research. I designed the follow-up study in collaboration with my supervisor, managed the project and ensured its smooth running. Specifically, this involved conceptualisation of the research questions and hypotheses, selection of the measures for the follow-up study, developing and writing the protocol, study procedures including the risk assessment, obtaining NHS and University ethics approval, recruiting 49 families into the study, conducting all of the assessments at families' homes across the areas of Southampton, Eastleigh, Winchester, Salisbury, Portsmouth and the New Forest in Hampshire, coding the Five Minute Speech Samples, entering all data and managing the database. Details of the procedure followed in

the prospective follow-up study are provided in Section 4.3.4 of the thesis. I performed all data analyses with guidance from an expert statistician in multilevel modelling and interpreted the data.

Chapter Four

Which Child and Family Characteristics Predict Maternal Expressed Emotion toward Children with ADHD?

4.1 Overview of Chapter Four

Chapter 4 presents the first two studies of the thesis. These studies used multilevel modelling (MLM) of sibling pair (sib-pair) data to cross-sectionally examine the overall contribution of child-specific (within-family) and shared family (between-family) effects in predicting maternal expressed emotion (EE) in families of children with ADHD. They also looked at which specific child and family/parent-related characteristics explained these effects. This was achieved by first using data collected from 72 children and adolescents with a clinical diagnosis of DSM-IV ADHD Combined Type (referred to as probands hereafter), their siblings and mothers who participated in the Southampton arm of the International Multicentre ADHD Genetics (IMAGE) project at Time 1 (T1), and second using data obtained from 48 of the families who took part in the follow-up study of this project at Time 2 (T2). At T1 a semi-structured psychiatric interview was used to determine child ADHD diagnostic status and at both time points mothers were administered a modified version of the Five Minute Speech Sample (FMSS) about both the proband and the sibling to measure maternal EE. They also completed standardised questionnaires about their own ADHD and depressive symptoms and both siblings' behavioural and emotional problems. The results revealed that both child-specific and shard family effects were implicated as important in predicting maternal EE. Except for the EE component warmth (WAR), child-specific effects made a stronger contribution. The picture as to which child and family characteristics explained the child and family effects found was complex with different patterns of association found for the different EE components and at the different time points. By and large, child effects were explained primarily by oppositional/conduct problems (OPP/CP), and to a lesser extent emotional problems (EP),

rather than ADHD. Overall family levels of child OPP/CP largely seemed to drive family effects in addition to but to a smaller extent maternal depressive symptoms, sib-pair average EP and ADHD symptoms. Implications of the findings and methodological limitations are discussed and the aims of Study 3 are outlined.

4.2 Introduction to Studies 1 and 2

Increasing cross-sectional and longitudinal evidence sourced from both clinic- and community-based studies indicates an association between elevated parental EE, in particular the critical dimension of the construct, and ADHD (Baker et al. 2000; Daley et al. 2003; Peris & Hinshaw, 2003; Psychogiou et al., 2007; Schwartz et al. 1990). Furthermore, it has been postulated that high parental EE toward children with ADHD may cause an increase in other aspects of child behaviour such as CD over time suggesting the relationship between elevated parental EE and ADHD may drive negative developmental trajectories associated with the disorder (Taylor, 1999). This may have important implications for clinical practice. In theory, for example, interventions that target the causes of high parental EE could foster positive developmental outcomes for children with ADHD. However, as discussed in Chapter 2, there are several issues pertaining to the interplay in the relationship between high parental EE and ADHD that remain unclear. First, little is known about what causes high parental EE toward children with ADHD. Is it the behaviour of the child that drives high parental EE (child-specific/within-family effects) or are general characteristics of the family that are independent of a specific child (between-family effects) more important? Second, with respect to child effects, how specific parental EE is to ADHD is unclear. Third, in relation to family effects, what specific family/parent factors are responsible for these? Fourth is the issue of the unitary nature of EE – are there different patterns of child and family effects on the individual components of EE? Studies 1 and 2 aimed to address these four questions using a within-family multilevel model design. As noted in Chapter 3, the within-family multilevel model design allowed child and family effects and characteristics driving these effects to be teased out.

The first aim was to examine the overall contribution of child-specific and shared family effects in predicting maternal EE toward children with ADHD. Experimental studies have shown that medication induced reductions in symptoms in boys with ADHD are associated with increases in maternal warmth and decreases in criticism (Schachar et al., 1987). One of the few studies to have examined child and parent characteristics on maternal EE simultaneously reported that child characteristics rather than maternal characteristics predicted maternal EE toward school-aged boys (Psychogiou et al., 2007). Links between parent characteristics such as maternal psychopathology and EE have also

been found (Hibbs et al., 1991; Schwartz et al., 1990). Based on these previous findings, it was hypothesised that although there would be some family effects (i.e., parental EE would differ between families as a function of family/parent characteristics), child-specific effects (i.e., parental EE would differ within families as a function of child characteristics) would be stronger. Specifically, even taking account of differences between families, parental EE would be higher toward children with ADHD than siblings without ADHD within families.

The second aim was to examine whether it is ADHD per se that drives the child effects on parental EE or whether there are other characteristics such as comorbid problems that are most important. Child characteristics examined were demographic variables and OPP/CP and EP. There is limited evidence of associations between child age and gender and parental EE. The complexity of the issue of clinical specificity of parental EE was noted in Chapter 2. The picture of the relationship between ADHD, ODD/CD and parental EE is mixed. Some studies have shown no significant differences in levels of EE between children with ADHD alone and children with ADHD plus ODD/CD (Marshall et al., 1990) or that even when parental EE was associated with ODD/CD, the significant relationship found between parental EE and ADHD withstood control of this characteristic (Peris & Hinshaw, 2003). Moreover, another study found that whilst parental EE measured at preschool predicted diagnosis of ADHD at 3rd grade, it did not predict ODD (Peris & Baker, 2000). However, more recent studies have reported that significant differences in levels of parental EE observed between clinic-referred children with ADHD and controls were removed when oppositional behaviour ratings were entered as a covariate (Christiansen et al., 2010) or that in community samples the significant effect of ADHD on maternal EE no longer remained when conduct and emotional problems were entered in the regression model (Psychogiou et al., 2007).

Based on these previous findings, it was hypothesised that both ADHD and OPP/CP would be positively associated with parental EE. However, the conflicting findings to date regarding the link between EE and comorbid disruptive problems made it difficult to make a prediction regarding the independent contribution of ADHD and OPP/CP in driving child effects (i.e., whether child effects would be accounted for by ADHD or OPP/CP or both).

The third aim was to investigate the role of general family/parent-related factors in explaining any family effects found to predict maternal EE. Specifically, family predictors examined included the gender of the sib-pair, overall family levels of child psychopathology (i.e., sib-pair average problems) and parental psychological problems including ADHD and depressive symptoms. It was hypothesised that maternal ADHD symptoms would be

negatively associated with maternal EE and that maternal depressive symptoms would be positively associated with maternal EE. These predictions are based on previous studies that have reported higher levels of child psychopathology and EE among mothers and fathers with a history of psychiatric diagnosis (Hibbs et al., 1991), depressed mothers (Bolton et al., 2003; Goodman et al., 1994) and mothers with anxiety disorder (Hirshfield et al., 1997a, b). That maternal ADHD symptoms would be negatively rather than positively associated with maternal EE was based on findings that high levels of ADHD symptoms in mothers improved the negative effects of child ADHD on parenting (Psychogiou et al., 2007).

The fourth aim was to examine whether different patterns of child and family influence are implicated in the separate domains of EE. Several studies have found that the separate categories of EE differentiate between clinical and non-clinical groups better than overall ratings of EE (Kershner et al., 1996; Baker et al., 2000). Kershner et al. reasoned that focusing more selectively on pertinent aspects of EE may strengthen applicability and possibly prognostic value of EE for general populations of children and adolescents. In addition, as noted in Chapter 2, the individual components show different patterns of association with child psychopathology. The FMSS employed in most studies of parental EE toward children consists of nine components which are used to rate high versus low CRIT and EOI. These two subtypes of EE have generally been used to examine the role of the separate EE components. Whilst this simplicity is appealing, the individual components that are used to rate CRIT and EOI may also reveal different patterns of association. For instance, Kershner and colleagues (1996) examined five of the nine components and found that mothers' initial statements differed significantly between a clinic-referred and non-referred group - mothers in the clinical group tended to express more neutral statements whereas mothers in the nonclinical group were more likely to express positive initial statements. Furthermore, in addition to IS and CRIT, positive comments (PC) also differentiated clinical and non-clinical families. These findings demonstrate the potential independent contribution of both IS and PC which are usually not examined separately, but instead used in conjunction with other components to make the more global ratings of CRIT and EOI. In relation to the finding pertaining to PC, ratings of EE generally reflect negative attitudes, but that parents vary greatly in positive expression may be an important protective factor too.

Scoring for other dimensions may have some value too. Vostanis and Nicholls (1995) who used the CFI, which also codes for warmth (WAR), found that ratings of this component differentiated between three groups of children, two clinical and one control. Based on earlier findings that found low maternal warmth is associated with child

behaviour problems in young children (Richman et al., 1982), they argued that WAR should be included in FMSS scoring. This component also taps into positive emotions.

The present studies used a modified version of the FMSS to examine child and family effects on five EE components (IS, relationship [REL], WAR, PC, CC) which denote both positive and negative emotions. It is possible that whilst child factors, including ADHD, drive the emergence of particular features of EE most directly linked to a response to challenging behaviour, critical comments for instance, parental characteristics and other family factors may be more important in other more trait-like aspects of EE such as warmth. In line with this hypothesis, in a review of children of parents with major affective disorder, maternal depression was linked to decreased warmth expressed toward children (Beardslee, Bemporad, Keller, & Kleman, 1983). More recently, there is some evidence that parental depression decreases warmth and negative parent-child relationships which in turn is linked to increased rates of externalising child behaviour (Foster, Garber, & Durlack, 2008). This different pattern of influence may be especially important to understand because parental warmth has been shown to act as a protective factor in the relationship between maternal depression and child outcomes, with children who perceived their depressed mothers as being warmer, having greater resilience to negative outcomes (Brennan, Hammen, Katz, & Le Brocque, 2002; Brennan, Le Brocque, & Hammen, 2003).

4.3 Methods for Studies 1 and 2

4.3.1 Participants

Time 1

Participants in Study 1 (i.e., T1) were 72 (of a total of 77) sibling pairs and their mothers (n = 72) who took part in the Southampton arm of the International Multicentre ADHD Genetics (IMAGE) project. Each sibling pair comprised a child with a clinical diagnosis of ADHD and a full sibling (some of whom also had ADHD) both aged between 5 and 17 years. The IMAGE project is an international collaborative study which aims to identify genes that increase the risk for ADHD. It recruited a total sample of 1,246 Caucasian children with ADHD and 1,600 Caucasian siblings of European descent from local specialist child and adolescent mental health clinics at 12 centres in eight countries including Belgium, Germany, Ireland, Israel, The Netherlands, Spain, Switzerland and the United Kingdom (Brookes et al., 2006). Children with ADHD were eligible if they had a clinical diagnosis of DSM-IV combined type ADHD, were living at home with at least one biological parent and one full sibling (for the collection of clinical information and DNA)

and had had their last medication-free period two or fewer years ago. Inclusion criteria for both probands and siblings were aged 5 to 17 years, IQ greater than 70, absence of epilepsy or any other neurological diseases or damage or any genetic or medical disorders associated with externalising behaviours that mimic ADHD. Five families were excluded in Study 1 of this thesis due to missing maternal EE data.

Fifty nine probands and 35 siblings were male and 13 probands and 37 siblings were female. The mean age of probands and siblings was 12.15 ± 2.37 years and 11.46 ± 3.17 years respectively with a large age range of 7 to 17 for probands and 6 to 17 for siblings. Twenty eight sib-pairs were male, 8 were female and 36 were mixed gender. In 47 families, the sib-pairs lived with both natural parents, in 23 families with their natural mother and in two families with their natural father.

Information pertaining to whether or not children and adolescents were taking medication was obtained. However, the quality of the data was not fit for purpose in this thesis. Data were missing for 13 families and five variables relating to whether or not youth were taking medication or nor were entered in the database and difficulty in obtaining clarity as to what each of the variable specifically represented meant that information could not be reliably reported in this thesis.

Time 2

Participants in Study 2 (i.e., T2) were 48 (of a total of 49) of the families in the Southampton arm of the IMAGE project who took part in the prospective five-year follow-up study. The response rate was 64% based on 76 families providing consent at T1 to be contacted at T2 and 49 families completing the study at T2. One family was excluded due to missing maternal EE data. Forty probands and 25 siblings were male and 8 probands and 23 siblings were female. The average age of probands and siblings was 16.81 ± 2.39 with an age range of 12 to 22 and 16.13 ± 3.46 with an age range of 9 to 22 respectively. Twenty one sib-pairs were male, 4 were female and 23 were mixed gender.

At T2, data on whether probands and siblings were on or off medication for ADHD was missing for two siblings. 45.7% (21) of probands were on medication for ADHD at the time the follow-up assessment took place, 48.9% (22) were not on medication, but had been on medication in the period between T1 and T2, 2.2% (1) had been on medication before they took part at T1, but never after and 2.2% (1) had never been treated with medication for ADHD. 4.4% (2) of siblings were on medication at the time the follow-up assessment took place, 6.7% (3) were not on medication, but had been treated with medication in the period between T1 and T2 and 84.4% (38) had never been treated with medication for ADHD.

Attrition and Retention

As described above, 77 families took part in the Southampton arm of the IMAGE project at T1 and 64% (n = 49) of this original sample were retained at T2 in the prospective follow-up study. For the cross-sectional analyses reported in this chapter, 72 families were included in the T1 analyses and 48 families were included in the T2 analyses with families excluded if maternal EE data were missing (n = 5 at T1; n = 1 at T2). The sample size utilised for the longitudinal analyses reported in the thesis was 45 families with four families excluded due to missing maternal EE data at either T1 (n = 3) or T2 (n = 1). Further details of the sample utilised in the longitudinal analyses are provided in the subsequent two chapters.

Independent *t*-tests and Pearson's chi-square tests were performed respectively to examine whether there were any significant differences in (1) mean levels of child and family/parent characteristics and (2) the proportion of children/adolescents and parents meeting clinical cut-offs on measures of behavioural and emotional problems between families who did and did not participate at both T1 and T2. Table A2.1 in Appendix 2 presents a comparison of mean scores in child and family/parent characteristics between families who did and did not take part at both time points. There were no significant differences in mean levels of child and family/parent behavioural and emotional problems between families who did and did not take part at both time points.

Independent *t*-tests run separately for probands and siblings also revealed the same pattern (see Table A2.3 in Appendix 2). In addition, child age did not differ significantly between families who did (M = 11.27, SD = 2.77) and did not (M = 11.50, SD = 2.83) participate at both time points; $t(142) = .49$, p = .63. There were also no differences in age when broken down by probands who did (M = 11.56, SD = 2.24) and did not (M = 11.81, SD = 2.57) take part at both time points; $t(70) = .45$, p = .66 and siblings who did (M = 10.98, SD = 3.21) and did not (M = 11.19, SD = 3.09) participate at both T1 and T2; $t(70) = .27$, p = .79.

Pearson's chi-square test revealed that there were no significant differences in gender between children and adolescents χ^2 (1) = .20, p = .65 who did and did not take part at both time points. The same was found when separate tests were performed for probands χ^2 (1) = .01, p = .94 and siblings χ^2 (1) = .30, p =.58. As shown in Table A2.2 in Appendix 2, Pearson's chi-square tests also demonstrated that there were no significant differences in the proportion of children and adolescents and parents meeting clinical cut-offs on measures of behavioural and emotional problems in families who did and did not take part

at both time points. This was also the case when separate tests were run for probands and siblings (see Tables A2.4 – A2.5 in Appendix 2).

4.3.2 Child Measures

Maternal Expressed Emotion

Maternal EE at both T1 and T2 was measured using the Revised Five Minute Speech Sample (RFMSS; Daley et al., 2003). The RFMSS is an adapted version of Magaña et al.'s (1986) Five Minute Speech Sample (FMSS) which comes with two comprehensive manuals – one for the collection of five minute speech samples and one for coding EE from the speech samples. This modified version was thought a more appropriate measure of parental EE in the current thesis for two reasons. First, it has been designed specifically for use with child populations. Second, unlike the FMSS, it includes a measure of warmth (WAR). Previous studies have found patterns of association between CFI-measured WAR and child psychopathology to be different from that of other EE components (Vostanis et al., 1994). Furthermore, maternal WAR has also been linked to maternal mental health (Brennan et al., 2003). Given that this study aimed to examine general family effects on maternal EE and the specific family characteristics explaining these effects, inclusion of this EE component seemed important. The RFMSS has adequate code-recode, inter-rater and test-retest reliability in addition to satisfactory construct and discriminant validity (Daley et al., 2003; cited in Yelland & Daley, 2009).

The collection of speech samples is carried out in an interview format with only the researcher and parent present. Respondents are given a set of instructions verbatim by the researcher whilst being audio-recorded. Parents are asked to talk for five minutes uninterrupted about their thoughts and feelings about the specified child and how they have been getting along with the child. Once the respondent has begun to speak, the researcher may only make one comment only after 30 seconds of silence using a standard prompt. The speech samples are then transcribed verbatim and coded directly from the audio-recordings by a trained rater on six components. The four global categories are (1) initial statement (IS), (2) relationship (REL), (3) warmth (WAR) and (4) emotional over-involvement (EOI) and the two frequency counts are (1) positive comments (PC) and (2) critical comments (CC).

In the present thesis, the component EOI was not coded. As noted in Chapter 2, many studies of the relationship between parental EE and child disorders have demonstrated that CRIT, but not EOI is associated with symptomatology. Furthermore, McCarty and colleagues (2004) found that observed behaviours did not correspond with

parental EOI and suggested the construct may lack validity with children. Some authors have suggested EOI may be more developmentally appropriate for children, especially young children, but may be more intrusive, problematic and have more of a negative effect on adolescents and adults (Psychogiou et al., 2007). In accordance with this assumption, the fact that the sample in the present thesis at follow-up was made up of a significant proportion of adolescents and young adults, EOI might have seemed an important EE component to examine. However, in adults, more often than not, high EE is not related to CRIT not EOI and criticism and EOI and CRIT are not significantly related (Hooley & Licht, 1997). Definitions of the five components and examples of how they are rated are provided below although information regarding the specific guidelines of coding is outlined in the coding manual.

Initial Statement

IS is defined as the first thought or idea expressed by the parent specifically about the child. There are two kinds of statements – those that are descriptions about the child and those that are about the relationship with the child. Statements are rated as:
Positive = 1 (e.g., "He's a really sweet boy"/"We get on really well together")
Neutral = 2 (e.g., "Billy is my youngest child"/"Billy and I get along, but he's very stubborn)
Negative = 3 (e.g., "Billy is a spiteful child"/"We always argue")

Relationship

REL is defined as the quality of relationship and joint activities undertaken between parent and child over the previous six months. This is based on the parent's reports of the relationship and the enjoyment and value the parent gets out of spending time with the child. The relationship is rated as:
Positive = 1 (e.g., "We get along really well"/"We bake a lot together and it's so much fun)
Neutral = 2 (e.g., "Our relationship is okay"/"We go to the part all the time")
Negative = (e.g., "We rarely get along")

Warmth

WAR is defined as the intensity of sentiment or feeling which parents express about the child. It relates only to warmth expressed during the speech sample and not the warmth of the parent's personality. Tone of voice, spontaneity and concern and empathy are the key elements of coding warmth. Ratings are either high = 1, moderate = 2, or negative = 3. In relation to tone of voice, high ratings are made in the presence of positive changes in tone

when the parent switches from talking about a neutral subject to talking about their child, moderate ratings if there is some evidence of this and low ratings in its absence, for example, the parent speaks about the child in a monotonic voice with no voice modulation when talking about the child. With regard to spontaneity, high ratings are made in the presence of the parent elaborating on points they are making and in doing so expressing positive feelings of appreciation, love, appreciation etc. about the child, moderate ratings are made when there is some evidence of this and low ratings are made in the absence of spontaneity where the parent is more matter of fact and just makes a statement without elaboration (e.g., "She is good at drawing"). In relation to concern and empathy, high ratings are made when the parent demonstrates concern for the child by demonstrating an ability to see things from the child's point of view or an understanding of what the child is going through, moderate ratings are made when there is some evidence of this and low ratings are made in the absence of this (e.g., "She takes toys from other children all the time, then they get upset, she doesn't seem to understand, it does my head in and really irritates me").

Positive comments

PC are positive statements made about the child's behaviour and/or personality which praise or indicate appreciation or approval for the child that are scored on the basis of tone (e.g., fluctuation to positive tone even if the content of the statement does not contain positive content) or positive phrases. For example, a positive phrase that uses descriptive words to indicate a positive trait might be "Billy is a loving boy" and a positive phrase that indicates a positive behaviour might be "Mary is great at swimming".

Critical comments

CC are statements which criticise or find fault with the child based on tone (e.g., fluctuation to a negative tone even if the statement doesn't contain critical content) and critical phrases that reflect the opinion of the parent (and not others e.g., teachers, grandparents). Critical statements include descriptive words indicative of a child's negative trait and descriptions of the child's behaviour accompanied by negative tone or indication of disapproval of the behaviour. Examples of critical phrases include "Harry bites me" or "Mary is always grumpy and bad tempered".

A counter-balanced order was used in eliciting speech samples for children with ADHD and their siblings so that in half the sample mothers' speech samples about the child with

ADHD were obtained first, followed by the sibling and in the other half, the opposite was done.

Child DSM-IV ADHD Diagnosis

The diagnosis of child DSM-IV combined type ADHD at T1 was made with the Parental Account of Childhood Symptoms (PACS; Chen & Taylor, 2006; Taylor et al., 1986) interview and the DSM-IV Total subscale of the Conners Teacher Rating Scale Revised – Long Version (CTRS-R: L; Conners, 1996). First, a symptom checklist was produced. A standardised algorithm was applied to derive each of the DSM-IV ADHD symptoms from the PACS data and this was combined with items rated two or more on the DSM-IV Total subscale of the CTRS-R: L. Second, situational pervasiveness was assessed by the presence of ADHD symptoms in more than one setting (e.g., home and school) from the PACS and scores of two or three on one or more items from the DSM-IV Total subscale of the CTRS-R: L. Third, presence of impairment was measured based on the severity of symptoms identified in the PACS. A diagnosis was made if the sufficient number of symptoms was identified to fulfil DSM-IV criteria and both impairment and situational pervasiveness were present. In cases where no CTRS-R: L data were present, situational pervasiveness was assessed with the PACS data alone. The PACS was administered to the parents of probands with a clinical diagnosis of ADHD and siblings who based on parent descriptions of their behaviour were thought to have ADHD.

The PACS is a semi-structured, standardised, investigator-based interview designed to enable an objective and accurate measure of child behavioural and emotional problems in accordance with the DSM-IV and ICD-10. The interview is administered by a trained interviewer and encompasses four sections (1 – Inattentive, Hyperactive and Impulsive Symptoms, 2- Oppositional, Defiant and Conduct problems, 3 – Emotional Problems, 4 – Comorbid Conditions) which are further divided into sub-sections. During the interview, instead of being asked for their ratings of problems, parents are asked to provide detailed descriptions of what their child has done in a range of specified situations over the previous week and in the previous year. The specified situations are defined by external events or by particular types of behaviours exhibited by the child. External events, for example, include what the child did whilst watching television, reading a book, playing and eating a meal. Examples of the different types of behaviours include crying, temper tantrums and fighting with siblings. Ratings of the severity and frequency in the previous week and year of the child's behaviour are made on four-point scale ranging from 0-3 by the interviewer according to operationalised criteria. High inter-rater agreement for pairs of interviewers has been found for each of the four sections with product-moment correlations ranging

from 0.79 to 0.96 (Taylor et al., 1986a). Adequate criterion, predictive and discriminative validity of the PACS have been found in epidemiological surveys, clinical comparison studies and treatment trials (Taylor et al., 1987, 1991, 1996).

The CTRS-R: L is a standardised 59-item teacher-rated scale designed to dimensionally screen children and adolescents aged three to 17 years for symptoms of ADHD and also provide an assessment of other behavioural and emotional problems (e.g., oppositional problems and emotional lability) at school. There are six subscales in total of which three pertain to symptoms of ADHD in accordance with the DSM-IV criteria. These three indices include DSM-IV Inattention, DSM-IV Hyperactive-Impulsive and DSM-IV Total. The DSM-IV Total subscale contains 18 items made up of the 9 items from the DSM-IV Inattention subscale and the 9 items from the DSM-IV Hyperactive-Impulsive subscale. Items are scored on a four-point Likert scale ranging from 0 ("Not true at all") to 3 ("Very much true") and indicate the extent to which each symptom applies to the specified child or adolescent. Raw scores are converted to T-Scores ranging from 39 to 90 based on age and gender specific norms. T-Scores equal to or above 60 are considered atypical with those in the range 61-65 mildly atypical, those in the range 66-69 moderately atypical and those 70 or above markedly atypical. Psychometric properties of the scale have been found to be adequate with internal consistency ranging from .77 to .96 and the test-retest reliability from .47 to .99 (Conners, Sitarenios, Parker, & Epstein, 1998).

Child ADHD Symptoms

Time 1

The DSM-IV Inattentive and DSM-IV Hyperactive-Impulsive subscales of the Conners Parent Rating Scale Revised – Long Version (CPRS-R: L, Conners, 1996) were used at T1 to dimensionally measure child ADHD symptoms. The CPRS-R: L, now superseded by Conners 3 (Conners, 2008), is a popular research and clinical instrument designed to assess ADHD and related disorders in children aged three to 17 years. The scale comprises fourteen subscales with responses to items indicating the extent to which each symptom applies to the specified child and scored on a four-point Likert scale from 0 ("not true at all") to 3 ("very much true"). The DSM-IV Inattentive and DSM-IV Hyperactive-Impulsive subscales each contain nine items which were developed in line with DSM-IV criteria. "Has difficulty organising tasks and activities" and "Fidgets with hands or feet or squirms in seat" are examples of inattentive and hyperactive-impulsive symptom items respectively.

Raw scores are converted to T-scores which are based on a large age and gender-specific normative sample. T-scores above 60 are cause for concern and have interpretive value

with T-scores between 61 and 65 interpreted as mildly atypical, between 66 and 70 as moderately atypical and above 70 as markedly atypical. The psychometric properties of the CPRS-R: L appear adequate as demonstrated by good internal reliability coefficients, high test-retest reliability and effective discriminatory power (Conners, Sitarenios, Parker, & Epstein, 1998a). The two subscales, which were highly correlated ($r = .84$, $p < .001$), were used separately to obtain descriptive statistics (e.g., means and standard deviations) and for the correlational analyses z-scores were calculated for each scale and the two scales were added together to form a composite score.

Time 2

Young person ADHD symptoms were measured with the DSM-IV-TR ADHD Inattentive and ADHD Hyperactive-Impulsive subscales of the Conners Comprehensive Behaviour Rating Scale – Parent (Conners CBRS – P; Conners, 2008). The Conners CBRS-P is a 203-item tool designed to provide a comprehensive assessment of a broad range of child behavioural and emotional problems and impairment in children aged 6 to 18 years old. Eleven DSM-IV-TR symptom subscales are included amongst a total of just over 20 other subscales. A modified version of the Conners CBRS was utilised for participants who were over the age of 18. This version was adapted with permission from the publisher, Multi-Health Systems (MHS), to make the wording of some items more age-appropriate. The wording of items of the ADHD Inattentive subscale was revised as follows:

Item 8 "Fails to complete schoolwork, chores, or tasks (even when he/she understands and is trying to cooperate)." was changed to "Fails to complete academic work or job work, chores, or tasks (even when he/she understands and is trying to cooperate)."

Item 96 "Loses things (for example, schoolwork, pencils, books, tools, or toys)." was changed to "Loses things (for example, academic work, to-do lists, pencils, books, keys, credit cards, or tools)."

Item 136 "Has trouble keeping his/her mind on work or play for long." was changed to "Has trouble keeping his/her mind on work or leisure activities for long."

For the ADHD Hyperactive-Impulsive subscale, the wording of item 148 was changed from "Is noisy and loud when playing or using free time." to "Is noisy and loud when doing leisure activities or using free time."

Revisions to the wording of items that constitute the Oppositional Defiant Disorder and Conduct Disorder subscales are described in the next section on the measurement of child OPP/CP as are adaptations to the wording of items of the Manic Episode subscale detailed in Chapter 6 in Section 6.3.2 under the heading "T1 and T2 Emotional Dysregulation Problems". Responses to items in the Conners CBRS are made on a four-point Likert scale ranging from 0 ("Not true at all") to 3 ("Very much true"). Raw scores are converted to T-scores which are based on an age and gender-specific normative sample of 3,400 young people. T-scores of 60 or above (i.e., at least one standard deviation above the mean) are usually interpreted as clinically significant. The Conners CBRS scales have been found to have good psychometric properties (Sitarenios & Wheldon, 2008). The modified version of the Conners CBRS for young adult respondents has not been validated and the lack of availability of psychometric properties for this version is discussed later in this chapter and Chapter 7 as a limitation. Given that no age and gender-specific normative data were available for the modified version, T-scores for respondents older than 18 years of age were based on age and gender norms for 18 year olds (the maximum age and gender norms are available for the Conners CBRS). The two subscales, which were highly correlated ($r = .86$, $p < .001$), were used separately to obtain descriptive statistics (e.g., means and standard deviations) and for the correlational analyses z-scores were calculated for each scale and the two scales were added together to form a composite score.

Child Oppositional/Conduct Problems

Time 1

The Oppositional scale of the CPRS-R: L (Conners, 1996), described above, and the Conduct Problems scale of the parent version of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) were used at T1 to measure child OPP/CP. The Oppositional subscale comprises 10 items (e.g., "angry and resentful"). The SDQ is a widely used 25-item brief screening measure of both positive and negative aspects of child behaviour that can be completed by parents and teachers of children aged four to 16 years. Items are rated on a three-point Likert scale ranging from 0 ("Not True") to 2 ("Certainly True"). Children are given a total score from 0 to 10 for each of the five subscales which relate to emotional symptoms, conduct problems, hyperactivity and inattention, peer relationship problems and prosocial behaviour. Total scores on the Conduct Problems scale of four or more are indicative of abnormal levels. Both satisfactory reliability and validity of the SDQ have been found (Goodman, 2001). The Oppositional scale of the CPRS-R: L and the Conduct Problems of the SDQ were highly correlated ($r = .84$, $p < .001$). In order to obtain descriptive statistics, scales were used separately. In the correlational and multilevel

model analyses, z-scores were calculated for each scale and the two scales were added together to form a composite score.

Time 2

At T2, child OPP/CP was measured with parent ratings on the DSM-IV Oppositional Defiant Disorder and DSM-IV Conduct Disorder scales of the Conners CBRS–P (Conners, 2008). The DSM-IV Oppositional Defiant Disorder and DSM-IV Conduct Disorder scales comprise eight and 15 items respectively. The wording of items of the DSM-IV Oppositional Defiant Disorder in the modified version of the Conners CBRS for young adult respondents was changed as follows:

Item 70 "Argues with adults" was changed to "Argues with adults or authority figures"

Item 127 "Actively refuses to do what adults tell him/her to do" was changed to "Actively refuses to do what adults or authority figures tell him/her to do"

The wording of item 107 of the Conduct Disorder subscale was changed from "Skips classes" to "Skips classes/lectures or work". Strong correlations were found between the two subscales ($r = .85$, $p < .001$). Descriptive statistics were obtained using the two scales separately. In the correlational and multilevel model analyses, z-scores were calculated for each scale and the two scales were added together to form a composite score.

Child Emotional Problems

Time 1

The Emotional Symptoms scale of the parent version of the SDQ and the Anxious/Shy scale of the CPRS-R: L were used at T1 to measure child EP. Total scores on the Emotional Symptoms scale of the SDQ ranging from seven to ten indicate abnormal levels of emotional symptoms. Both questionnaires have been described above. The two scales were highly correlated ($r = .74$, $p < .001$). Descriptive statistics were obtained using the two scales separately and for the correlational and multilevel model analyses, z-scores were calculated for each scale and the scales were added together to calculate a composite score.

Time 2

The DSM-IV Major Depressive Disorder and DSM-IV Generalized Anxiety Disorder subscales of the Conners Comprehensive Behaviour Rating Scale (Conners CBRS;

Conners, 2008) were used at T2 to measure child EP. The Conners CBRS has been described above. Strong correlations were found between these scales ($r = .91$, $p < .001$). Descriptive statistics were ascertained using scores from the two scales separately and for the correlational and multilevel model analyses, z-scores were calculated for each scale and the two scales were added together to form a composite score.

4.3.3 Family Measures

Overall Family Levels (Sib-Pair Mean) of Child Oppositional/Conduct Problems

Overall family levels of child OPP/CP at both T1 and T2 were measured by calculating mean OPP/CP scores for each sibling pair.

Overall Family Levels (Sib-Pair Mean) of Child Emotional Problems

Overall family levels of child EP at both T1 and T2 were measured by calculating mean EP scores for each sibling pair.

Maternal ADHD Symptoms

The self-report form of the Current Symptoms Scale (CSS-SR; Barkley & Murphy, 1998) was used to measure ADHD symptoms in mothers. The CSS-SR is an 18-item scale designed for use with adults and based on the DSM-IV definition of symptoms of ADHD including inattentiveness (e.g., "easily distracted"), hyperactivity (e.g., "feel restless") and impulsiveness (e.g., "interrupt or intrude on others"). An individual's own symptoms are rated over the past 6 months by responding to items on a four-point scale ranging from 0 ("Rarely") to 3 ("Very Often"). A score of nine symptoms or more experienced as 'often' or 'very often' is used as the standard cut-off to identify adults at risk for clinical problems. The CSS-SR has age- and gender-specific norms and good psychometric properties including satisfactory levels of internal consistency and excellent test-retest reliability (Ajcicegi, Dinn, & Harris, 2003). It also correlates with spousal, parental and cohabiting partner ratings of symptoms (Murphy & Barkley, 1996).

Maternal Depressive Symptoms

Time 1

The self-report 12-item version of the General Health Questionnaire (GHQ-12; Goldberg, 2003; Goldberg & Williams, 1988) was used to measure level of depressive symptoms in mothers. The GHQ-12 is commonly used in psychological research as a screening instrument to identify depression in the general population. This version is often favoured

due to its brevity and availability of normative data (Kalliath, O'Driscoll & Brough, 2004). Individuals are required to indicate their level of agreement with each statement over the past few weeks on a four-point scale (e.g., "less than usual", "no more than usual", "rather more than usual" or "much more than usual"). Scores from all items are summed to provide an overall score. The bimodal scoring method (0-0-1-1) which yields total scores out of 12 was used. A cut-off of three symptoms indicates clinical levels of depressive symptoms. The GHQ-12 has sound psychometric properties including high internal consistency (Winefield, Goldney, Winefield & Tiggemann, 1989) and convergent and discriminant validity.

Time 2

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was used at T2 to measure depressive symptoms in mothers. This scale was originally developed to identify clinical caseness of anxiety disorders and depression among patients in non-psychiatric hospital clinics, but has been widely used throughout the world in community settings and primary care medical practice. The scale is divided into two subscales: Anxiety (HADS-A) and Depression (HADS-A). Each subscale comprises 7 items which are intermingled to form the complete scale of 14 items and to which individuals respond to each item on a 4-point scale ranging from 0 (e.g. Not at all) to 3 (e.g. Most of the time). In the current study, the HADS will be administered to parents of young people. In a recent review of 747 studies that had used the HADS, Bjelland, Dahl, Haug, and Neckelmann, (2002) found the HADS to have good discriminant validity and internal consistency, Cronbach's alpha for HADS-A varied from .68 to .93 (mean .83) and for HADS-D from .67 to .90 (mean .82). The authors concluded: "The HADS was found to perform well in assessing severity and caseness of anxiety disorders and depression in both somatic, and psychiatric cases and [not only in hospital practice for which it was first designed] in primary care patients and the general population" (Bjelland et al., 2002).

4.3.4 Procedure for Studies 1 and 2

Ethics approval was obtained from university and National Health Service (NHS) research Ethics Committees at T1 and T2.

Time 1

Eligible probands with a clinical diagnosis of ADHD and their families were identified from four different child and adolescent mental health service (CAMHS) clinics in the county of Hampshire in the UK. Families were referred by consultant child and adolescent

psychiatrists who had conducted clinical evaluations with probands in the recent past and had explained the IMAGE project to them and their families. Families who expressed an interest in taking part in the study were sent participant information sheets (one for the parent(s) and one each for the proband and sibling), consent forms for the parent and assent forms for the children. Families who wanted to take part completed and returned the consent and assent forms to the research team.

The research team then mailed families a battery of questionnaires to complete. Parents completed eight questionnaires about each child which were measures of child behaviour and of which two (CPRS-R: L and SDQ) were used in the present thesis. They also completed a further six questionnaires about each child which were measures of parenting and pre, peri and postnatal factors in addition to five questionnaires about their own behaviour of which two (CSS-SR and GHQ-12) were used in the thesis. Children completed three questionnaires about their own behaviour. The spouse/partner/close relative or friend of the parent (i.e., main caregiver) taking part completed a questionnaire about the ADHD symptoms of the parent. At the same time, the teachers of the children were sent four questionnaires to complete about the specified child.

Families attended an appointment at which the RFMSS and PACS were administered to parents about the proband and sibling, although the PACS was only administered for siblings thought to have ADHD based on descriptions from their parents. DNA samples were also collected from parents, probands and siblings. At the same appointment as the interview assessment or at an additional separate appointment, the WISC-III (Wechsler, 1991), a measure of IQ, was administered to children along with a battery of six computer-based tasks, three of which measured inhibitory control and three that measured delay aversion. Symptom and behavior ratings were based on medication-free periods to allow for more accurate measures of the current level of symptoms and behaviours with families withdrawing stimulant medication one week prior to research assessments wherever possible. At the end of participation in the study at T1, families were asked if they would be willing to be contacted in the future about a follow-up study. Written consent was obtained from families who agreed to be contacted in the future about a follow-up study. The follow-up study was conducted five years later.

Time 2

Data at T2 was collected during the period that this thesis was undertaken. Families who had provided written informed consent to be contacted about the follow-up study were contacted at T2 via a letter of invitation. Also included with the letter was a reply slip, a participant information sheet and consent forms for parents, age-appropriate participant

information sheets and assent and consent forms for and a pre-paid envelope. Families were asked to complete and return the reply slip with their current contact details if they were interested in taking part in the study. The option of either completing and returning the assent and consent forms with the reply slip or completing the forms at the research assessment appointment was given to families. For the most part, written informed consent was obtained at the outset of the research assessment. The researcher telephoned families who returned the reply slip to arrange a research assessment appointment at their home.

Research assessments lasted between three to four hours. The RFMSS was administered to parents first, followed by a battery of three questionnaires about each child (of which the demographics questionnaire and Conners CBRS – P were used in the current study) and a battery of two questionnaires about their own behaviour (CSS-SR and HADS), both of which were used in the current study. Probands and siblings were each administered a battery of five questionnaires about their own behaviour. At the end of the research assessment, families were debriefed by the researcher in person at their home and subsequently also sent a letter.

4.4 Overview of the Analytic Strategy for Studies 1 and 2

First, descriptive statistics were obtained separately for T1 and T2 data including: 1) means and standard deviations of scores on measures of child and family/parent-related characteristics and the proportion of children and adolescents and mothers with scores meeting clinically significant levels; and 2) correlations among EE components, child and family/parent-related characteristics.

Second, cross-sectional two-level univariate multilevel models were first run in the first instance with both T1 data and T2 data separately to estimate child-specific (within-family) and shared family (between-family) effects on maternal EE, and second to examine which specific child and family/parent-related characteristics explained these effects. Using full maximum likelihood estimates, separate models were run for each EE component with the EE component entered as the dependent (outcome) variable in each model. As noted in Section 3.4 of Chapter 3, the first models for each EE component were conditioned on ADHD diagnostic status and provided a baseline estimate of how much of the variance in the maternal EE component was at the child-specific (within-family) level and how much was at the shared family (between-family) level. The intra-class correlation was calculated to yield this estimation. It was important to condition the initial models on ADHD diagnostic status due to the overlap between ADHD and sibling status in the data used. Rather than the sample being chosen at random, in each sibling pair all probands had a clinical diagnosis of ADHD and in most cases the siblings did not. Running null models in

the first step of the analysis where no child or family predictors are added to the models and therefore not conditioned on ADHD diagnostic status can lead to the assumption that there are no family effects when in fact there are family effects when ADHD diagnostic status is controlled (Sonuga-Barke et al., 2013), as demonstrated in a previous analysis of child and family effects on maternal EE with the current data (Cartwright et al., 2011).

An incremental approach to building the models was used with models built up with one predictor entered in the model at a time with child level predictors entered first followed by family level predictors. Categorical predictors at both levels were entered uncentred, continuous child level predictors were entered group mean centred and continuous family level predictors were entered grand mean centred. The contribution of individual predictors to the models was assessed using the chi-square likelihood ratio test which is used to compare the fit of the model as new predictors are added to the model. This is carried out by subtracting the log-likelihood of the new model from the value for the old model. Models were run first with all predictors kept in the model even when their addition did not significantly improve the fit of the model (i.e., they did not make an individual contribution).

The order of child-level predictors entered in the model was gender, age, child OPP/CP and child EP. The order of family level predictors entered in the model was all-boy sib-pair, all-girl sib-pair, sib-pair mean OPP/CP, sib-pair mean EP, maternal ADHD symptoms and maternal depressive symptoms. Robust standard errors were used. Maas & Hox (2005) stated that "robust standard errors turn out to be more reliable than the asymptomatic standard errors based on maximum likelihood". The models were re-run with child EP entered in the step before child OPP/CP and sib-pair EP entered in the step prior to sib-pair OPP/CP and also with maternal depressive symptoms entered prior to ADHD symptoms to check that the order of entry made no differences to the results. Child IQ was entered in all models initially after child gender and age, but dropped due to there being no significant effects.

4.5 Results for Studies 1 and 2

4.5.1 Levels of Child Behavioural and Emotional Problems

Tables 4.1 and 4.2 present the mean scores and the proportion of children with clinically significant levels of behavioural and emotional problems as a function of ADHD diagnostic status at T1 and T2 respectively. As expected, at both time points siblings with ADHD had significantly higher levels of both behavioural and emotional problems and a larger proportion had clinically significant levels of symptoms. By and large, two to three times

more siblings with ADHD had problems at clinically significant levels than siblings without ADHD. Exceptions to this were depressive symptoms at T1 for which there was little difference between the proportion of siblings with and without ADHD and conduct problems at T2 for which five times more siblings with ADHD had conduct problems than siblings without ADHD. The mean scores for conduct problems and depressive symptoms measured with the SDQ were higher in the siblings without ADHD in comparison to British children aged five to 15 in a large general population study (Meltzer, Gatward, Goodman, & Ford, 2000).

4.5.2 Levels of Maternal Problems and Average Overall Family Child Problems

Table 4.3 displays the means of behavioural and emotional problems for sib-pairs. Average levels of family child problems is an important family characteristic to examine given that it could be child behaviours at the family level rather than at the individual child level that drive high levels of maternal EE. Sib-pair mean levels of child problems were above average at both time points except for depressive symptoms at T1 which were within the normal range. Levels of maternal ADHD symptoms were higher than normal as expected as were depressive symptoms at T1, but not at T2.

Table 4.1

Mean Scores and Proportion of Children with Clinically Significant Levels of Behavioural and Emotional Problems at T1 as a Function of ADHD Diagnostic Status

	Siblings with ADHD (n = 79)	Siblings without ADHD (n = 65)	t
Inattention problems[a]			
Mean (SD)	74.37 (9.15)	53.14 (12.08)	-11.68***
% clinically significant levels (≥ 60)	94.9	23.1	
Hyperactivity/impulsivity problems[a]			
Mean (SD)	83.08 (9.81)	54.49 (14.33)	-13.66***
% clinically significant levels (≥ 60)	94.9	24.6	
Oppositional problems[a]			
Mean (SD)	76.65 (11.75)	55.85 (13.67)	-9.82***
% clinically significant levels (≥ 60)	92.4	32.3	
Conduct problems[b]			
Mean (SD)	5.73 (2.39)	2.48 (2.47)	-8.02***
% meeting clinical cut-off (≥ 4)	81.0	32.3	
Depressive symptoms[b]			
Mean (SD)	3.99 (2.69)	2.91 (2.90)	-2.31*
% meeting clinical cut-off (≥ 6)	29.1	23.1	
Anxiety symptoms[a]			
Mean (SD)	64.24 (14.85)	55.38 (14.71)	-3.58***
% clinically significant levels (≥ 60)	60.8	29.2	

Note. [a] = ratings of behaviour were made using the CPRS-R: L; [b] = ratings of behaviour were made using the parent version of the SDQ.

*p < .05. ***p < .001.

In the general British population, average scores for CP and EP (measured with the SDQ) is 1.9 (2.0) and 1.6 (1.7) (Meltzer et al., 2000).

Table 4.2

Mean Scores and Proportion of Children with Clinically Significant Levels of Behavioural and Emotional Problems at T2 as a Function of ADHD Diagnostic Status

	Probands (n = 52)	Siblings (n = 44)	t
Inattention problems			
Mean (SD)	78.75 (11.23)	55.27 (16.19)	-8.11***
% clinically significant levels (≥ 60)	94.2	29.5	
Hyperactivity/impulsivity problems			
Mean (SD)	79.98 (12.32)	56.14 (16.94)	-7.76***
% clinically significant levels (≥ 60)	90.4	29.5	
Oppositional problems			
Mean (SD)	76.81 (15.50)	56.77 (15.61)	-6.29***
% clinically significant levels (≥ 60)	80.8	34.1	
Conduct problems			
Mean (SD)	72.54 (16.69)	51.07 (11.44)	-7.44***
% clinically significant levels (≥ 60)	67.3	13.6	
Depressive symptoms			
Mean (SD)	76.79 (16.10)	60.64 (17.42)	-4.72***
% clinically significant levels (≥ 60)	78.8	40.9	
Anxiety symptoms			
Mean (SD)	79.04 (13.97)	58.52 (16.63)	-6.57***
% clinically significant levels (≥ 60)	88.5	40.9	

Note. Child behavioural and emotional problems were measured with the Conners CBRS.

***p < .001.

Table 4.3

Average Levels of Mothers' and Sib-Pairs' Problems and the Proportion of Mothers with Clinically Significant Levels of Problems at T1 and at T2

	Time 1 (n = 72)	Time 2 (n = 48)
Sib-pair inattention problems[a (T1 & T2)]		
Mean (SD)	64.78 (8.42)	67.99 (9.92)
Sib-pair hyperactivity/impulsivity problems[a (T1 & T2)]		
Mean (SD)	70.17 (8.79)	69.05 (11.26)
Sib-pair conduct problems[b (T1), a (T2)]		
Mean (SD)	4.26 (1.93)	62.70 (11.25)
Sib-pair oppositional problems[a (T1 & T2)]		
Mean (SD)	67.26 (10.13)	67.63 (12.40)
Sib-pair depressive symptoms[b (T1), a (T2)]		
Mean (SD)	3.50 (2.09)	69.39 (13.82)
Sib-pair anxiety symptoms[a (T1 & T2)]		
Mean (SD)	60.24 (11.12)	69.64 (12.17)
Sib-pair emotional dysregulation problems[a (T1 & T2)]		
Mean (SD)	66.01 (10.17)	69.73 (12.85)
Maternal ADHD symptoms[c (T1 & T2)]		
Mean (SD)	11.49 (10.70)	11.79 (10.28)
% meeting clinical cut-off	25.0 (18)	22.9 (11)
Maternal depressive symptoms[d (T1), e (T2)]		
Mean (SD)	2.49 (2.89)	4.02 (3.73)
% meeting clinical cut-off	29.2 (21)	16.7 (8)

Note. [a] = ratings were made using the CPRS-R: L; [b] = ratings were made using the parent version of the SDQ; [c] = ratings were made using the CSS-SR; [d] = ratings were made using the GHQ-12; [e] = ratings were made using the HADS.

4.5.3 Associations between EE Components, Child and Family Characteristics

Tables 4.4 and 4.5 show the correlation matrices among EE components and child characteristics at T1 and T2 respectively. Correlations between EE components, child and family characteristics were mostly significant and in the expected direction.

Intercorrelations among EE components at T1 and at T2 were in the small to moderate range and in the expected direction (i.e., IS, REL, WAR and CC were positively related to each other and PC was negatively correlated with the other EE components), but overall, associations between EE components at T2 were marginally stronger than at T1. IS, REL, WAR and CC were significantly positively correlated with all child problems at both time points except at T1 a non-significant association was found between IS and child EP. Correlations were strongest between PC, CC and child problems at both time points. However, at T1 REL was also more robustly related to child problems and at T2 WAR was more strongly associated with child problems. As expected intercorrelations among child problems were positive in the moderate to strong range at T1 and were all above .73 at T2. At T1, only moderate correlations were found between EP and other child problems whereas at T2 strong correlations were found between all child problems.

While none of the EE components were significantly correlated with maternal ADHD symptoms at T1, IS and PC were significantly although weakly associated with maternal ADHD symptoms at T2. Small, but significant correlations were found between WAR, PC and maternal depressive characteristics at both time points and between REL and mothers' depressive symptoms at T1. The intercorrelation among maternal problems was small at T1 and moderate at T2. Surprisingly, non-significant correlations were found between maternal problems and the majority of child problems at T1. For example, only small associations were found between maternal ADHD symptoms and child EP and EDP and between maternal depressive symptoms and child EP. At T2, however, as one would expect, maternal problems were significantly positively related to child problems in the moderate range.

EE components at both time points were significantly correlated with the majority of sib-pair average problems. However, non-significant correlations were found at both time points between IS, REL and one or more types of sib-pair average problems. For example, REL was not significantly associated with sib-pair mean EP at T1 or with sib-pair average ADHD, OPP/CP and EDP at T2. As expected intercorrelations among sib-pair mean problems were in the moderate to high range and in the expected direction at both time points. Sib-pair average problems were significantly positively correlated with maternal problems at T2, but at T1 sib-pair mean ADHD was not significantly related to maternal problems nor was sib-pair mean DBP to maternal depressive characteristics.

Table 4.4

Correlations between Child and Family Characteristics and EE Components at T1

	1	2	3	4	5	6	7	8	9	10	11	12
1. IS	-											
2. REL	.37***	-										
3. WAR	.26***	.49***	-									
4. PC	-.30***	-.49***	-.39***	-								
5. CC	.37***	.50***	.41***	-.24**	-							
6. Child ADHD	.27**	.47***	.21**	-.44***	.51***	-						
7. Child OPP/CP	.24**	.53***	.28***	-.46***	.52***	.82***	-					
8. Child EP	.10	.28***	.19*	-.36***	.18*	.47***	.50***	-				
9. Maternal ADHD	.06	.00	-.02	-.06	.02	.05	.10	.29***	-			
10. Maternal DEP	.06	.21**	.22**	-.16*	.04	-.05	.01	.19*	.35***	-		
11. Sib-pair ADHD	.18*	.22**	.19*	-.15*	.29***	.50***	.48***	.28***	.11	-.11	-	
12. Sib-pair OPP/CP	.17*	.32***	.26**	-.18*	.32***	.38***	.63***	.34***	.16*	.02	.77***	-
13. Sib-pair EP	.10	.13	.16*	-.18*	.08	.19*	.29***	.73***	.40***	.25**	.39***	.46***

Note. IS = initial statement; REL = relationship; WAR = warmth; PC = positive comments; CC = critical comments; ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms.

*p < .05, **p < .01, ***p < .001.

Table 4.5

Correlations between Child and Family Characteristics at and EE Components at T2

	1	2	3	4	5	6	7	8	9	10	11	12
1. IS	-											
2. REL	.39***	-										
3. WAR	.41***	.36***	-									
4. PC	-.35***	-.49***	-.47***	-								
5. CC	.47***	.55***	.58***	.37***	-							
6. Child ADHD	.18*	.30**	.30**	-.46***	.45***	-						
7. Child OPP/CP	.34***	.36***	.44***	-.45***	.66***	.78***	-					
8. Child EP	.30**	.31**	.45***	-.58***	.42***	.75***	.74***	-				
9. Maternal ADHD	.18*	.05	.07	-.17*	.09	.35***	.35***	.38***	-			
10. Maternal DEP	.09	.08	.23*	-.27**	.07	.29**	.37***	.36***	.58***	-		
11. Sib-pair ADHD	.08	.03	.20*	-.21*	.22*	.56***	.51***	.48***	.62***	.52***	-	
12. Sib-pair OPP/CP	.23*	.09	.34***	-.23*	.35***	.45***	.64***	.46***	.54***	.58***	.80***	-
13. Sib-pair EP	.17*	.17*	.36***	-.36***	.16	.39***	.42***	.70***	.55***	.51***	.68***	.66***

Note. IS = initial statement; REL = relationship; WAR = warmth; PC = positive comments; CC = critical comments; ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms.

*p < .05, **p < .01, ***p < .001.

4.5.4 Child and Family Effects on Maternal EE

Within-Family and Between-Family Variation in Maternal EE

MLM requires that the dependent variables are normally distributed. This was confirmed for IS, REL and WAR. PC and CC were log transformed which corrected the problem of non-normal distribution. The first model (Model 1) was used to cross-sectionally establish a baseline for the amount of variance in maternal EE components explained by child (within-family) and family (between-family) effects conditioned on ADHD diagnostic status at T1 and T2. In the subsequent models (i.e., Models 2-11), first, child level predictors, in the order of child gender, age, OPP/CP, EP, were entered one-at-a-time, followed by family level predictors in the order of all-boy sib-pair, all-girl sib-pair, sib-pair mean OPP/CP, sib-pair EP, maternal ADHD symptoms and lastly maternal depressive symptoms, also one-by-one.

Model 1 demonstrated that except for ADHD diagnostic status, no child-level or family-level predictor variables were entered in this initial model. Figures 4.1 and 4.2 display the proportion of variance in maternal EE explained by child and family effects at T1 and T2 respectively and Model 1 of Tables A.1 to A1.10 in Appendix 1 show the detailed information about the child-specific and shared family variance values and their level s of significance. For all EE components and at both time points there was a significant contribution of both child and family effects, except for IS at T2 for which only child effects were found (i.e., the between-family variance was not significant). The child effects were stronger for IS, REL, PC and CC at both time points. For WAR, family effects dominated, however, these effects were less strong at T2 than they were at T1; at T2 child and family effects were approximately equal.

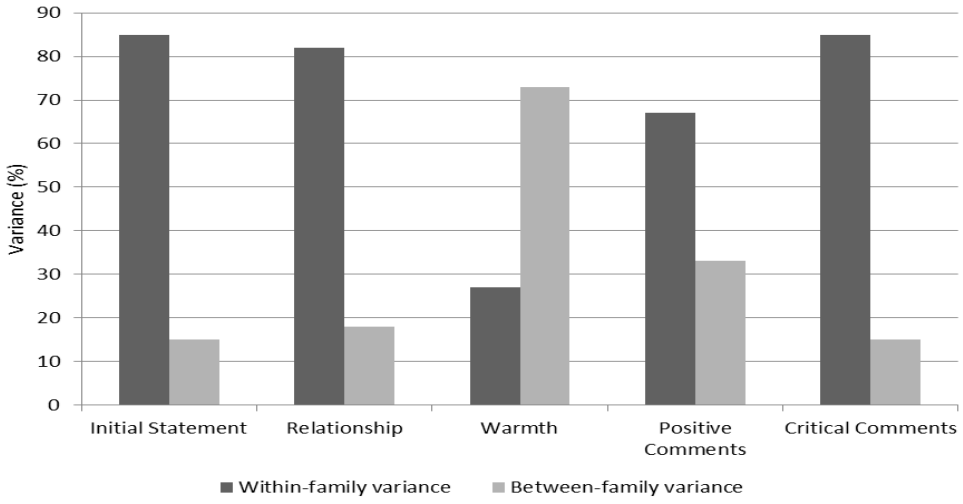

Figure 1 Proportion of variance in maternal EE at T1 explained by child and family characteristics in the initial models conditioned on ADHD diagnostic status

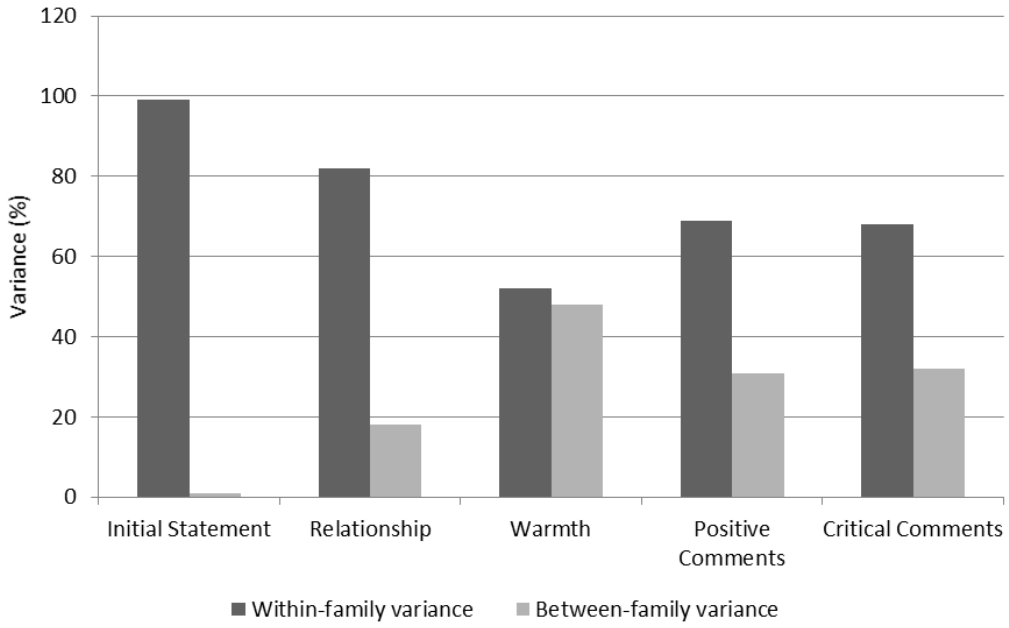

Figure 4.2 Proportion of variance in maternal EE at **T2** explained by child and family characteristics in the initial models conditioned on ADHD diagnostic status

Child Characteristics that Predicted Child Effects on Maternal EE

The effects of the specific child characteristics on maternal EE components are summarised in Tables 4.6 and 4.7 and presented in full in Tables A1.1 to A1.10 of Appendix 1. Child ADHD diagnostic status was entered in the models first. There was a significant effect of ADHD diagnostic status on all EE components at both time points, except on IS at T2 (see Model 1, Tables A1.1-A1.10 in Appendix 1). These associations were in the expected direction with ADHD diagnostic status positively related to IS, REL, WAR and CC and negatively to PC. Maternal IS (at T1 only) and REL were significantly more negative than with their children without ADHD, they expressed less warmth, fewer positive comments and a greater number of negative comments toward their children with ADHD compared to their children without ADHD. Except for IS at T1, this significant effect did not remain when other child-level and family-level predictors were entered in the model, details of which are provided below. Gender was the second child-level predictor entered in the multilevel models and age was the third. These predictors were entered in Step 2 of the stepwise regression model for IS at T2. There was a significant negative effect of gender on REL at T2 only and significantly improved the fit of the model. Mothers' relationships with boys were more negative than with girls. Except for a significant positive association found between child age and WAR at T2, there were no significant associations between child age and EE components. Mothers showed less WAR toward older children and the addition of age significantly improved the fit of the model for WAR.

Child OPP/CP were entered in the multilevel models fourth. Child OPP/CP was significantly associated with REL, PC and CC at both time points and IS and WAR at T2 and significantly improved the fit of the models. This effect held for all components except for PC at T2 when other child-level and family-level predictors were entered in the model. Mothers' initial statements and relationships were more negative and they expressed less warmth, more critical comments and less positive comments (at T1 only) the more OPP/CP their children presented with. The significant effect of ADHD diagnostic status on REL, PC and CC at both time points and WAR at T1 no longer remained when child OPP/CP was entered in the models.

Child EP were entered in the multilevel models fifth. There was a significant effect of child EP on PC at both T1 and T2 which remained when subsequent family-level predictors were entered in the model and significantly improved the fit of the models. The significant effect of child OPP/CP on PC at T2 became non-significant when EP was added. Mothers' expressed significantly less positive comments toward their children who manifested higher levels of EP.

When child OPP/CP and EP were entered in reverse order in the multilevel models, there was a marginally significant positive association between child EP and WAR at T1 and a significant positive association between child EP and CC at T2. Only in the model for CC at T2 did the addition of child EP significantly improve the fit of the model and these effects no longer remained significant when child OPP/CP were subsequently entered in the models. As noted above, when child OPP/CP were entered in the model before child EP, the association between ADHD diagnostic status and WAR at T2 became non-significant. This was also the case when child EP was entered in the model for WAR at T2 before child OPP/CP.

Table 4.6

Child Characteristics that Predicted Child Effects on Maternal EE at T1

Parameter	IS	REL	WAR	PC	CC
Child ADHD status	√* (+)	X	X	X	√* (+)
Child gender	X	X	X	X	√* (-)
Child age	X	X	√* (-)	X	X
Child OPP/CP	X	√** (+)	X	√** (-)	√** (+)
Child EP	X	X	X	√** (-)	X

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct; EP = emotional problems.

Detailed information on child characteristics explaining child effects on maternal EE is provided in Tables A1.1, A1.3, A1.5, A1.7 and A1.9 in Appendix 1.

*p < .05. **p < .01.

+ = positive association; - = negative association.

Table 4.7

Child Characteristics that Predicted Child Effects on Maternal EE at T2

Parameter	IS	REL	WAR	PC	CC
Child ADHD status	X	X	X	X	X
Child gender	X	X	X	X	X
Child age	X	X†	X	X	X
Child OPP/CP	√† (+)	√* (+)	√* (+)	X	√*** (+)
Child EP	X	X	X	√** (-)	X

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct; EP = emotional problems.

Detailed information on child characteristics explaining child effects on maternal EE is provided in Tables A1.2, A1.4, A1.6, A1.8 and A1.10 in Appendix 1.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association.

In summary, for the majority of EE components, child OPP/CP predominated in significantly predicting child (within-family) effects found to influence maternal EE components. IS was explained by ADHD diagnostic status at T1, but by child OPP/CP at T2. Child effects on REL at both time points was predicted by child OPP/CP and by gender at T1. Child age played a role in predicting child effects on WAR at T1 and at T2 child OPP/CP drove the child effects on WAR. Child effects on PC were predicted by child EP at both time points and also by child OPP/CP at T1. Child OPP/CP drove the child effects on CC at T1 and T2.

Family Characteristics that Predicted Family Effects on Maternal EE

The effects of the specific family/parent-related characteristics on maternal EE components are summarised in Tables 4.8 and 4.9 and presented in full in Tables A1.1 to A1.10 in Appendix 1. Sib-pair gender was entered dummy coded as two variables, all-boy sib-pair (0 = all-girl or mixed gender sib-pair, 1 = all-boy sib-pair) and all-girl sib-pair (0 = all-boy or mixed gender sib-pair, 1 = all-girl sib-pair), which were entered in turn as the first and second family-level predictors in the models. There were no significant effects of sib-pair gender on any of the EE components (except marginally for all-boy sib-pair on T1 CC) when they were initially entered in the models and their addition did not significantly improve the fit of the models. The exception to this was for IS at T2, for which a negative effect of all-girl sib-pair was found. However, in the final models in which all child- and family-level predictors had been entered, all-boy sib-pair was positively significantly and marginally significantly associated with T1 CC and T2 CC respectively and all-girl sib-pair

was negatively significantly related to T2 CC. When sib-pair mean EP and maternal depressive symptoms were added to the model for T1 CC, all-boy sib-pair became significant. This perhaps suggests interactions between these family-level predictors in predicting CC at T1 such that mothers were more critical in families of all-boy sib-pairs with high levels of EP and if they themselves had high levels of depressive symptoms. Similarly, although sib-pair gender was not significantly associated with CC at T2 and its addition did not significantly improve the fit of the model, all-boy sib-pair became marginally positively associated with CC at T2 when sib-pair OPP/CP was entered and there was a significant effect of all-girl sib-pair when sib-pair mean EP was entered. This suggests that mothers may be more critical in families of all-boy sib-pairs with high levels of OPP/CP.

The third family-level predictor entered in the model was sib-pair mean OPP/CP. This family characteristic had a significant effect on all EE components and significantly improved the fit of the models except for REL at T2. This effect remained significant in the final model for WAR and CC at both time points (although only marginally for WAR at T2) after all other family-level predictors had been entered, but not for IS at T1 nor PC at both time points. For the latter components, the effect was lost when overall family levels of child EP were entered in the models. It must be noted that the effect of sib-pair mean OPP/CP on WAR at T1 varied depending on the other family-level predictors entered in the model. The effect on WAR at T1 became marginally significant when sib-pair EP was entered, but became significant again when maternal depressive symptoms were added. Similarly, the significant effect of sib-pair mean OPP/CP on WAR at T2 became non-significant when sib-pair mean EP were entered, but became significant again when maternal ADHD symptoms were added.

Overall family levels of child EP, the fourth family-level predictor entered in the model, did not significantly predict EE at T1. There was a significant effect of sib-pair mean EP on PC at T2 which significantly improved the fit of the model and remained in the final model when all other child and family-level predictors had been entered (see Model 9, Table 4.13). Sib-pair EP was marginally significantly associated with WAR at T2 when initially entered in the model, although did not significantly improve the fit of the model. It became significant when maternal ADHD symptoms were added and remained significant in the final model (see Models 9-11, Table 4.11).

When sib-pair average OPP/CP and EP were entered in reverse order, there was a significant effect of sib-pair mean EP on WAR at T2, PC at both time points and CC at T1 and its addition significantly improved the fit of the models. Sib-pair EP was also marginally significantly associated with REL at both time points and WAR at T1, but it did not significantly improve the fit of the model. Except for WAR and PC at T2, the effect of

sib-pair EP (whether significant or marginally significant) was lost when overall family levels of child OPP/CP were entered, although WAR at T2 also went from significant to marginally.

Maternal ADHD symptoms, the fifth family-level predictor added to the models, was significantly negatively associated with WAR at T2 and remained so in the final model with all child- and family-level predictors included (see Model 10, Table 4.11). In families of mothers with high ADHD symptoms, mothers expressed more WAR toward their children. There was also a marginal significant positive effect of this parent characteristic on PC at T2 (see Model 10, Table 4.13) with mothers with elevated ADHD symptoms expressing more positive comments about their children. Whilst maternal ADHD symptoms were not significantly related to REL at T1, the association became marginally significant when maternal depressive characteristics were entered in the model.

Maternal depressive symptoms, the final family-level predictor entered in the models, made a significant independent contribution in predicting REL and WAR at T1 (but not at T2) with mothers with high levels of maternal depressive characteristics indicating less warmth for their children and more negative relationships. Maternal depressive characteristics were marginally positively associated with PC at both time points, but did not significantly improve the fit of the model.

When maternal ADHD and depressive symptoms were entered in reverse order, maternal depressive characteristics made an independent significant contribution in predicting REL, WAR and PC at T1. The significant effect on PC at T1 became marginal when maternal ADHD symptoms were added in the final model.

Table 4.8

Family Characteristics that Predicted Family Effects on Maternal EE at T1

Parameter	IS	REL	WAR	PC	CC
All-boy sib-pair	X	X	X	X	√* (+)
All-girl sib-pair	X	X	X	X	X
Sib-pair mean OPP/CP	X	√*** (+)	√* (+)	X	√*** (+)
Sib-pair mean EP	X	X	X	X	X
Maternal ADHD	X	√† (-)	X	X	X
Maternal DEP	X	√** (+)	√* (+)	√† (-)	X

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct; EP = emotional problems; DEP = depressive symptoms.

Detailed information on child characteristics explaining child effects on maternal EE is provided in Tables A1.1, A1.3, A1.5, A1.7 and A1.9 in Appendix 1.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association.

Table 4.9

Family Characteristics that Predicted Family Effects on Maternal EE at T2

Parameter	IS	REL	WAR	PC	CC
All-boy sib-pair	X	X	X	√† (-)	√† (+)
All-girl sib-pair	√** (-)	X	X	X	√** (-)
Sib-pair mean OPP/CP	√** (+)	X	√† (+)	X	√*** (+)
Sib-pair mean EP	X	X	√* (+)	√** (-)	X
Maternal ADHD	X	X	√* (-)	√* (+)	X
Maternal DEP	X	X	X	√† (-)	X

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct; EP = emotional problems; DEP = depressive symptoms.

Detailed information on family characteristics explaining family effects on maternal EE is provided in Tables A1.2, A1.4, A1.6, A1.8 and A1.10 in Appendix 1.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association.

The multilevel models were also re-run with sib-pair average problems and maternal psychopathology reversed in their order of entry. First, in relation to maternal ADHD symptoms, as noted above, in the final models maternal ADHD symptoms significantly predicted WAR at T2 and PC at T2 and had a marginal significant effect on REL at T1. However, it was only for WAR at T2 that this parent characteristic had a significant effect when the predictor was initially entered in the model. When maternal ADHD symptoms were instead entered in the model after all-girl sib-pair (i.e., prior to sib-pair problems), they did not significantly predict family effects on any EE components when initially entered and did not improve the fit of the models. For WAR and PC at T2, the significant effect of maternal ADHD symptoms was found when sib-pair EP was entered in the model. For REL at T1, maternal ADHD symptoms had a marginal significant effect once sib-pair OPP/CP were added to the model. Second, with regard to maternal DEP, no differences were found when the order of entry was changed bar some minor exceptions. There was a significant effect of maternal DEP on PC at T2 when entered prior to overall family levels of child problems, but this effect became marginal when sib-pair mean OPP/CP were entered in the model. In addition, there was a marginal significant effect of maternal depressive symptoms on WAR at T2 when it was entered before overall family levels of child problems, but this effect did not remain when overall family levels of child OPP/CP were added. Third, in relation to sib-pair OPP/CP, the pattern was the same when the order of entry was reversed except minimally for PC at both time points. Whereas a significant effect was found on this component when sib-pair mean OPP/CP was entered

before parent problems, this effect was only marginal when entered after parent psychological symptoms. Whereas sib-pair mean OPP/CP had a significant effect on IS when entered before maternal psychopathology, this effect is only marginal when entered after maternal psychopathology.

In summary, in the final models estimated with all child- and family-level predictors entered, family effects on REL at T1 were driven by sib-pair mean OPP/CP and maternal DEP, but at T2 no specific family/parent characteristics explained these effects on this component. The same pattern was found for WAR at T1 as was found for REL at T1. Family effects on WAR at T2 were explained by sib-pair mean EP and maternal ADHD symptoms. There were no specific family/parent characteristics found to predict family effects on PC at T1 except for a marginal effect of maternal DEP. Family effects on PC at T2 were explained by sib-pair mean EP. Family effects on CC at both time points were driven by sib-pair mean OPP/CP.

4.6 Discussion

The main aim of the present studies was to use a within-family (sib-pair) design and multilevel modelling to cross-sectionally test whether child-specific (within-family) effects (i.e., differences between siblings that have ADHD and those who do not in the same family) rather than general family (between-family) effects predict maternal EE and whether these effects displayed different patterns of influence on the individual components of maternal EE. Of particular interest was whether the same pattern of effects would arise for maternal WAR as other EE components given the closer link between this component and parental personality and its potential importance as a protective feature for children with ADHD.

The hypothesis that both child and family effects would be implicated in predicting maternal EE was confirmed with the exception that no significant family effects were found on IS at T2. Moreover, as predicted, strong and significant child effects were found on all EE components at both time points and for IS, REL, PC and CC these effects were stronger than family effects. WAR was to some extent different from the other components as family effects were stronger than child effects. However, whereas at T1 family effects dominated, at T2 the proportion of variance in child and family effects was near equal. This suggests that although family effects may have a more influential role than child effects in predicting WAR, child effects still make a salient contribution to high levels of this EE component. Although child effects, for the most part, dominated, because family effects were also implicated this pattern of results supports the notion of a child and parent effects model. Previous studies have supported a child effects model. For instance,

Psychogiou et al. (2007) showed that associations between maternal psychopathology and maternal criticism did not remain significant after controlling for child symptoms. Further support comes from treatment studies that showed that symptom improvement in ADHD children on stimulant medication lead to displays of more warmth and support and less negativity by their mothers (Schachar et al., 1987). More generally, Lifford et al. (2009) found that child ADHD symptoms impacted on mother-son hostility (albeit not EE per se) both concurrently and longitudinally, but there were no effects in the other direction. In the broader context of child psychopathology, a Dutch longitudinal study concluded that a child effect model best described the relationship between mothers' EE perceptions and the course of adolescent externalising and internalising symptoms in light of their findings that the course of child behaviour predicted maternal EE rather than the other way around (Hale et al., 2011). Although the findings of the present study support a child and parent effects model, particularly in relation to WAR, they are to some extent consistent with other studies that support a child effects model given that the majority of EE components were driven largely by child effects.

The finding that family effects had a stronger influence on WAR is novel in the literature. As previously noted, in their treatment study, Schachar et al. (1987) found that maternal warmth increased when boys' ADHD symptoms improved on stimulant medication. As noted above, these previous findings support a high maternal EE as a "child effect". However, this novel finding may be due to the methodological perspective taken in the study. The first relates to the inclusion of WAR in the FMSS measure. The original version of the FMSS which the vast majority of childhood studies have used to measure EE and its constituent parts does not include WAR. In EE studies in the adult literature that have predominantly used the CFI, which does score WAR, most have used a high versus low EE approach in which case the components were not considered individually. Why? Two child studies, one cross-sectional and one longitudinal, have used the CFI to measure EE. The first study found that WAR distinguished between school-aged children with CD, EP and controls, with maternal warmth being lowest in the CD group (Vostanis et al., 1994). Using the same sample, in the second study, Vostanis & Nicholls (1995) showed that low levels of maternal warmth at the initial assessment predicted presence of conduct disorder, as measured using the CBCL, at the 9-month follow-up assessment. Many studies have examined the relationship between maternal warmth and child psychopathology, but not using the specific EE construct. For instance, Alizadeh, Applequist, and Coolidge (2007) used parenting scales and demonstrated that the parents of children with ADHD show less warmth and involvement with their children compared to control children. Indeed, Schachar et al.'s (1987) experimental study did not use a standardised EE measure of WAR.

Second, although many studies have explored associations between child and family characteristics and parental EE, the between-family designs of most studies (i.e., single parent-child dyads from different families are compared with each other), has precluded child and family effects to be teased out. Some authors have questioned whether the contribution of the shared family environment has been overlooked (Burt, 2009; Jenkins, Rasbash, & O'Connor, 2003). They speculated that rather than shared family effects influencing children directly, instead, they exert indirect effects via their influence on nonshared aspects of the environment such as differential parental treatment. In other words, the family context may increase or decrease the likelihood that children will be treated differently by their parents.

Examination of which specific child characteristics appeared to be driving the child effects for these components revealed that they were, for the most part, accounted for by child OPP/CP. There was a significant effect of ADHD when it was entered alone – the global components of EE (i.e., IS, REL, WAR) were rated as more negative or low and mothers expressed more critical comments and fewer positive comments toward their children with ADHD as compared with their children without ADHD within families.

However, when OPP/CP was entered it overshadowed this effect except for IS and CC at T1. This seems to suggest that it was child OPP/CP, rather than ADHD, that 'provoked' the negative and critical response from mothers. This finding adds to a rather mixed picture with regard to the relative role of ADHD and CP or OPP in "driving" high EE. The results are consistent with previous cross-sectional studies that have demonstrated that the significant association between parental EE and ADHD did not hold when CP was entered in a community sample (Psychogiou et al. 2007) and in a clinic-referred sample when OPP were entered (Christiansen et al., 2010). In the latter study, the link between parental EE and OPP was mediated by stress-related cortisol. In contrast, earlier cross-sectional studies showed no differences in levels of EE between children with ADHD alone and children with ADHD plus ODD/CD (Marshall et al., 1990) and the association between parental EE and ADHD withstood control of ODD/CD (Peris & Hinshaw, 2003).

A longitudinal study showed that parental EE predicted diagnosis of ADHD, but not ODD, at follow-up four years after the initial assessment (Peris & Hinshaw, 2000). Psychogiou (2004) demonstrated that child ADHD symptoms as rated by their mothers on an ADHD rating scale, were associated with maternal CRIT and EOI, but these factors were not significant after controlling for other factors including and also were not replicated when fathers and teachers reported on the child's ADHD in a large community sample of children with a mean age of 8 (only the effect of child ADHD on EOI remained significant based on maternal reports of child ADHD). The lack of significant effects between different reporters may have been due to small sample size of fathers and teachers. In an Australian

clinic-referred sample of male and female children and adolescents with and without ADHD, Rey, Walter, Plapp, and Denshire (2000) found there was no differences in family environment, as measured by the Global Family Environment Scale (GFES; Rey et al., 1997) between the ADHD and non-problem group. Instead family environment was worst amongst children and adolescents with ADHD and comorbid conduct and oppositional disorders.

Results from parenting studies are also mixed with some consistently showing that in ADHD populations disruptive behaviour exacerbates and is more predictive of less effective parenting practices than ADHD (McLaughlin, & Harrison, 2006; Johnston, 1996) and others finding little support for this trend (Chronis-Tuscano et al., 2008). Despite this compelling evidence for the role of OPP/CP in high EE in ADHD the relationship between ADHD, OPP/CP and EE needs further enquiry. Taylor (1999) suggested that during development ADHD provokes high EE from parents early on in life which leads to ODD/CD later on. However, testing this hypothesis was beyond the scope of these studies due to their cross-sectional design. Furthermore, certain ADHD children might be especially vulnerable to the effects of parental EE. For instance, Sonuga-Barke et al. (2009) have shown that children with ADHD carrying a specific genotype of the dopamine (DAT1) and the serotonin transporter (5-HTTLPR) were more likely to have CP. Sensitivity to the effects of parental EE on CP was moderated by variants of the DAT1 and 5HHTT genes. Furthermore, in a recent study by Sonuga-Barke et al. (2010) ADHD children whose parents were highly critical and lacked warmth had significantly higher levels of ODD and CD. The study also found gene-environment interactions involving EE and polymorphisms of the serotonin and dopamine transporters suggesting that maternal expressions of warmth and hostility may act together with genetic factors in altering severity of ADHD (Sonuga-Barke et al., 2008).

Maternal PC seemed to stand out from the rest of the components. Child effects on this component appeared to be accounted for by both OPP/CP and EP at T1 and only EP at T2. Few previous studies have examined the role of PC. Kershner et al. (1996) found that PC was among three components that differentiated clinical and non-clinical families.

The only component for which child effects were explained by ADHD was IS. Little is known about child and family influences on this EE component. This is because although the FMSS comprises nine components, nearly all studies that report that they have examined individual components of FMSS measured EE, have collapsed these nine components into two components, notably CRIT and EOI. CRIT is derived from a negative initial statement, a negative description of the relationship, or one or more criticisms. In a study which examined five of the nine components, Kershner et al. (1996) found that IS ratings differed significantly between a clinic-referred and non-referred group

–mothers in the clinical group tended to express more neutral statements whereas mothers in the nonclinical group were more likely to express positive initial statements. Furthermore, IS, CRIT and positive comments were the three components that differentiated clinical and non-clinical families.

High levels of EE may have serious implications for the development of children with ADHD. Criticism has been found to have negative consequences on recipient's feelings about themselves (Coyne, Downey, & Boergers, 1992). Criticism has also been found to have a negative effect on the way families deal with conflict and negative feelings (Baron, 1988). Goodman et al. (1994) found that mother's high levels of critical attitudes mediated the relationship between mother's depression and decreased self-esteem.

Consistent with previous findings, for the most part, child demographic characteristics were not associated with maternal EE minus a few exceptions. Child age independently predicted maternal warmth with mothers expressing less warmth toward older children within families. In contrast, there was also a trend toward an association between younger child age and negative REL at T2; however this effect changed from being non-significant when initially entered to marginal after sibling pair male gender was entered in the model. Similarly, there was a trend toward an association between male gender and REL at T2, but this effect disappeared when sibling pair male gender was entered. Female gender was associated with CC in the final model atT1, even though this effect was not significant when it was first entered in the model. This suggests that gender may interact with other child and family factors to exert its effect on maternal EE. This finding is consistent with one of the few studies that found a link between child gender and parental EE. Hirshfield et al. (1997) found that child female gender was associated with maternal CRIT controlling for child age, behavioural inhibition and maternal anxiety, although the effect fell to trend significance when the number of mood and behaviour disorders were controlled. However, it contradicts the findings of another study whereby mothers were more critical toward boys in comparison to girls and mothers expressed more positive remarks for girls than boys (Psychogiou, 2004). Entry of age and gender did not alter independent associations found between other child characteristics (e.g., ADHD, OPP/CP or EP) and maternal EE.

Exploration of the specific factors that might account for family effects revealed a more complex pattern. First, overall levels of child OPP/CP accounted for differences in between families in REL, although only at T1, WAR and CC. This is a novel finding, again perhaps due to the design of the study. The inclusion of more than one mother-child dyad per family enabled differences in maternal EE within and between families to be estimated. This finding suggests that it is not only the behaviour of the child with ADHD that may 'provoke' negative parent-child relationships, low warmth and criticism. It also

has important clinical implications for the treatment of ADHD. First, that OPP/CP of siblings may also contribute to 'provoking' high levels of maternal EE means that if ignored the behaviour of siblings may impinge on therapeutic processes aimed at reducing negative EE in families of children with ADHD. Second, therefore, it stresses the potential importance of assessing EE toward the siblings of children with ADHD and the behavioural problems of the siblings. This may help to identify siblings who have problems and need referring for treatment or who are at risk of developing further problems and are suitable for prevention and it may help in the process of reducing high levels of EE in families of children with ADHD. This, in turn, may prevent exacerbation of existing problems or the development of other problems over time in both the child with ADHD and their sibling(s).

Second, maternal depressive symptoms seemed to be an important predictor of EE components REL and WAR at T1 and marginally of PC at both time points. The discrepant findings for REL and WAR between T1 and T2 data may be attributable to the different measures of depression used at the different time points. Whereas at T1 mothers completed the GHQ-12, at T2 they were administered the HADS. Congruent with this finding, studies in the child development literature generally have indicated that parental depression is associated with decreased warmth expressed toward children and negative parent-child relationships and that this contributes to increased rates of externalising behaviours in children (Beardslee et al., 1983; Foster et al., 2008).

Few studies using the EE measure, however, have examined the relationship between domains of parental EE and depression in ADHD populations and those that have contradict this finding. One study of girls with ADHD found that maternal depressive symptoms as measured with the Beck Depression Inventory (BDI; Beck et al., 1988) was not associated with maternal EE and the relationship between ADHD and maternal EE withstood control of it. In the broader context of child psychopathology, maternal depressive symptoms, also measured with the GHQ-12, were not associated with maternal CRIT or EOI in a community sample of school-aged boys (Psychogiou et al., 2007). However, recently, in a sample of 8 to 12 year-olds at-risk for depression, FMSS maternal EE was associated with history of maternal depression in the child's lifetime and both these variables were related to children's own reports of their depressive symptoms and alongside current maternal depression were also associated with total and externalising problems on the CBCL (Tompson et al., 2010). Schwartz et al. (1990) also found a link between maternal criticism and depressive symptoms and the increased risk of psychiatric diagnosis in children of high EE mothers' was not independent of mothers' affective disorder. In a large sample recruited from an ongoing study of the health of mothers and their children that commenced during pregnancy, structural equation models revealed that EE CRIT and

degree of maternal depression both independently predicted adolescents' externalising symptoms and that EE CRIT intervenes in the association between maternal depression and adolescent externalising symptoms (Nelson et al., 2003). Despite these the mixed picture of previous research, this finding suggests that children in ADHD families in which mothers are depressed may be more vulnerable to low levels of warmth. Family characteristics, in particular high levels of warmth, are an important source of support, that buffer against stress and act as a protective factor in the relation between maternal depression and child outcomes as demonstrated in Brennan et al.'s work (2003). In their study, children were more resilient to negative outcomes if they perceived their depressed mothers as being warmer.

With respect to maternal psychopathology, there was also a significant effect of maternal ADHD on WAR and PC at T2 even after controlling for maternal depression. There was also trend significance for REL at T1. On the face of it these effects seemed to be in the opposite direction to that expected intuitively – mothers' ADHD was associated with higher WAR, more positive REL and an increase in PC. It is well documented in parenting studies that parental ADHD is associated with increased negligence and tolerance, lower levels of involvement and positive parenting and increased levels of inconsistent and permissive parenting (Arnold, O'Leary, & Edwards, 1997; Chronis-Tuscano et al., 2008; Harvey, Danforth, McKee, Ulsazek, & Friedman, 2003; Murray & Johnston, 2006). These findings were echoed by Psychogiou et al. (2007). However, in their study, high levels of ADHD symptoms in mothers of children with ADHD led to higher WAR. The authors speculated that high-ADHD mothers may have increased empathy and tolerance for the high-ADHD child. Further evidence to support these findings is limited due to the lack of investigations of the relationship between mothers' EE and ADHD symptoms has received little attention. Psychogiou (2004) found that CRIT was positively correlated with ADHD symptoms, conduct problems, and child emotional symptoms. EOI was positively correlated with maternal ADHD and maternal anti-social characteristics. EOI was also positively correlated with child ADHD symptoms and conduct problems. Mothers with high maternal ADHD symptoms expressed more CRIT for the child with high ADHD symptoms. Other studies in which the EE measure per se was not used have demonstrated that high parental impulsivity was associated with increased in criticism over time (Griggs & Mikami, 2011).

The findings pertaining to the effect of maternal ADHD symptoms should be treated with caution for several reasons. First, further examination of these effects revealed that maternal ADHD symptoms seemed to only exert their influence on WAR at T2 in conjunction with overall family levels of EP. When the order of entry of family characteristics was reversed, there was not a significant effect of maternal ADHD

symptoms until overall family levels of child EP were entered. Indeed, in the original order of entry, overall family levels of child EP became significantly associated with WAR at T2 only when maternal ADHD symptoms were entered in the model. A similar pattern emerged for PC at T2, but also the marginal effect of maternal ADHD symptoms became significant when maternal depressive symptoms were entered. Second, for REL at T1, the effect of maternal ADHD symptoms reached marginal levels of significance once maternal depressive symptoms were entered in the model. Furthermore, when the order of entry of family characteristics was reversed, the effect became significant when overall family levels of OPP/CP were entered. Therefore, the results with regard to maternal ADHD symptoms are not clear-cut and seem to suggest that this family characteristic may interact with other family characteristics to exert its influence on maternal EE.

It is also possible that genetics play a role as possible drivers of family effects through passive genotype-environment correlations (association between the genotype a child inherits from his or her parents and the environment in which the child is raised [Jaffee & Price, 2008]) linked to parent mental health and child behaviour. For example, previous studies using genetically sensitive research designs (i.e., in vitro fertilization and adoption research designs) found that for genetically related and unrelated mothers, conflict influenced child antisocial behaviour through mother-to-child hostility (Harold, Elam, Lewis, Rice, & Thapar, 2012; Harold et al., 2013). Evocative rGEs cannot also be ruled out.

Limitations

There are several methodological limitations of the present studies that should be borne in mind. First, like many previous studies of EE in child populations (Hibbs et al., 1991; Marshall et al., 1990; Peris & Hinshaw, 2003; Psychogiou et al., 2007; Stubbe et al., 1993), the data were cross-sectional in nature. The hypothesis in the literature generally is that a reciprocal interaction exists between child and parental characteristics. Indeed, in the current studies both child and family effects were implicated in predicting maternal EE. However, to clarify such an issue, a prospective longitudinal study is needed to allow the cause-effect relationship to be examined.

Second, EE was assessed in mothers only. In dual-parent households, maternal EE alone may not reflect *parental or family EE* as it does not take into account fathers' EE and the similarities and differences in maternal and paternal EE. Furthermore, the inclusion of only one parent's EE and the exclusion of all other family members' EE is also unlikely to capture EE as it was intended, that is, as a broader index of the family environment. Families are made up of multiple subsystems. A number of authors who have explored EE

in these have highlighted the importance of assessing expressed emotion attitudes in all family members (e.g., parents', child's EE and sibling's EE). Bullock, Bank and Burraston (2002) found that sibling critical expressed emotion was associated with younger brother concurrent and future disruptive behaviour, substance use, and increased criminal activity. It may also be that EE in single-parent families differs from dual-parent families and is a family effect worth considering in future research of this nature. However, most studies to date have not found an association between single parent status and EE (Asarnow et al., 1993; Hibbs et al., 1993; McCleary & Sanford, 2002).

Third, although teacher ratings of child EP and ODD/CD were obtained for the original sample, these ratings were not used in the multi-level analysis due to missing data for these variables. In addition, mothers reported on their own ADHD and mood symptoms. Mothers may have over-reported their own and their child's symptoms.

Fourth, no measure of personality was included in the analysis. Findings in relation to EE in adult psychiatric patients have found that personality is an important factor contributing to high-EE, more so than psychopathology of the relative or parent. High-EE relatives tend to have a more internal locus of control for their own behaviour than low-EE relatives and therefore prefer to take control in managing their own life problems and difficulties (Hooley, 1998). In addition, high-EE relatives have been shown to be more conventional in their attitudes and behaviour, to feel less capable and optimistic about their lives, to be less flexible, tolerant and lower in empathy and achievement by independence compared to low-EE relatives (Hooley & Hiller, 2000).

Fifth, medication status was not taken into account in the analyses as a child level predictor and should have been controlled for. Reliable data on whether or not children and adolescents were on medication for ADHD at T1 was not available to be included in the analysis as a control child level variable. At T2 just under 50% of probands were on medication at the time the assessment took place. This means that symptoms reported may have been less severe, particularly in relation to externalising symptoms, and therefore child outcomes may have been worse than depicted when these youth were not on medication. Mothers' EE scores may also have been lower toward children who were on medication.

Sixth, the age range in the sample was fairly large (5-17 years and 9-22 years at T1 and T2 respectively) with a substantial proportion (43%) of children at T2 no longer children, but young adults. Further discussion of this is provided in Section 7.3 of the thesis.

Seventh, as previously mentioned in Chapter 4, the possibility of evocative rGEs cannot be ruled out. The results seem to fit with the notion of evocative rGEs that genetically influenced child characteristics (in this case child ADHD or

oppositional/conduct problems) may have evoked negativity from parents (Ge et al., 1996), but this could not be tested with a sib-pair design. Instead, an adoptee or twin design would be needed.

Lastly, the study did not look at other family-specific effects on MEE such as the role of single-parent families vs. dual-parent families, socio-economic status of the family or the number of other siblings in the family. A semi-structured interview was used at T1, but not at T2, to determine ADHD diagnostic status.

4.7 Conclusions

In conclusion, the results of Studies 1 and 2 indicate that both child and family effects are implicated in cross-sectionally predicting maternal EE toward children with ADHD. While child effects predominated for IS, REL, PC and CC, family effects made a stronger contribution in predicting WAR. Except for IS, child effects were largely explained by child OPP/CP, rather than ADHD. Maternal depressive and to a lesser extent ADHD symptoms, overall family levels of child OPP/CP and EP drove family effects. However, the cross-sectional nature of these results limits the direction of causation to be established. Study 3 exploited a longitudinal design to extend the findings of Studies 1 and 2 and facilitate a better understanding of the cause-effect relationship between child and family effects and maternal EE toward children with ADHD. The overall aim of the study was to examine T1 child-specific (within-family) and shared family (between-family) effects on T2 maternal EE, which T1 child and family characteristics explained these effects and whether different patterns of T1 child and family effects were found on the individual domains of maternal EE.

Chapter Five

Do Child or Family Characteristics Predict Maternal EE over Time?

5.1 Overview of Chapter Five

Chapter 5 presents the third study of the thesis which employed longitudinal data to extend the findings of Studies 1 and 2. Multilevel models and a within-family design were again used to examine T1 child and family effects on T2 maternal EE in a subsample of 45 children with a clinical diagnosis of ADHD Combined Type, their siblings (n = 45) and mothers (n = 45) who took part in the Southampton arm of the International Multicentre ADHD Genetics (IMAGE) project at T1 and T2. The results demonstrated that child effects made a stronger contribution than family effects in predicting maternal EE over time, except for warmth (WAR) for which child and family effects contributed equally. After controlling for T1 EE additional child effects were longitudinally driven by child oppositional and conduct problems (OPP/CP), although to a lesser extent than was found in Studies 1 and 2, and emotional problems (EP) rather than ADHD per se. Average family levels of child OPP/CP largely accounted for longitudinal family effects on maternal EE. Interestingly, although maternal depressive symptoms predicted family effects over time on maternal warmth (WAR), the effect was in the opposite direction to what was expected. These analyses suggest that child and family factors continue to operate between T1 and T2 to determine EE levels. Implications of the findings and methodological limitations are discussed and the aims of Study 4 are outlined.

5.2 Introduction to Study 3

Findings that have demonstrated a link between parental EE and the presence and course of ADHD emphasise the importance for clinical practice of understanding child and family/parent factors that underpin the relationship. Studies 1 and 2 examined child-specific (within-family) and shared family (between-family) effects on maternal EE (indexed by its individual components) and the specific child and family characteristics

which explained these effects. The findings demonstrated that both child and family effects predicted maternal EE, but child effects made a stronger contribution. Specifically, child effects were driven by child OPP/CP (and to a lesser extent child EP), rather than ADHD, and family effects were driven predominantly by overall family levels of child OPP/CP and maternal depressive symptoms. Although these studies are unique in that they are the first studies to use a within-family multilevel model design to examine the relationship between parental EE and ADHD in clinic-referred children and adolescents, they bore a number of limitations. In particular, they were cross-sectional in design making it impossible to make causal inferences regarding the relationship between child and family effects and parental EE.

Psychogiou et al.'s (2007) community study of school-aged boys which similarly aimed to simultaneously examine child and parent characteristics on maternal EE was limited due to its use of cross-sectional data too. Indeed in the broader context of prior studies examining associations between parental EE and child psychopathology, most have suffered from the same methodological drawback (i.e., they have conducted all measurements at one time point or conducted one measure of EE at Time 1 to predict a future measurement of child adjustment). Indeed, there are four notable exceptions. In these longitudinal EE studies all measured EE twice, two with the FMSS (Hastings, Daley, Burns, & Beck, 2006; Peris & Baker, 2000), one with the CFI (Vostanis & Nicholls, 1995) and one with the LEE questionnaire (Hale III et al., 2011). These studies yielded conflicting results. In the earliest and most specific to ADHD, Peris and Baker found that preschool measured parental CRIT predicted a diagnosis of ADHD in 3rd grade, thus supporting Marshall et al.'s (1990) suggestion that parental EE may independently influence the long-term outcome of ADHD. In contrast, Hale III et al. (2011) found that over a one-year period adolescent externalising and internalising symptoms predicted high levels of parental EE rather than the other way around. In a sample of mothers of children with intellectual disability, Hastings et al. (2006) did not find a longitudinal relationship between maternal criticism and child and adolescent externalising symptoms over a two-year period. However, none of these longitudinal studies were conducted in samples of children or adolescents with ADHD at entry to the study and their focus was by and large testing whether parental EE drives child outcomes. In other words, these studies aimed to examine the direction of effect as to whether parental EE causes child problems or the other way around.

Study 3 aimed to extend Studies 1 and 2, addressing the same issues, but with the advantage of a longitudinal design with EE measured at two time points over a five-year period, to test whether child-specific or shared family effects predict maternal EE over time. Specifically, Study 3 had the following aims. First, it aimed to examine the continuity of

the separate EE components over a five year period. Extant evidence with respect to the stability of EE in paediatric populations is mixed. In a clinic-based sample, CFI maternal critical comments decreased and warmth increased from the first assessment to the nine-month follow-up assessment in children with conduct disorders, but remained stable for children with emotional disorders (Vostanis & Nicholls, 1995). In families of adolescents and adults with autism, considerable stability was found in overall EE measured with the FMSS over an 18-month period (Greenberg, Seltzer, Hong, & Orsmond, 2006). Peris and Baker (2000) found good stability over a two-year period (average 28.7 months) from pre-school to first grade in a community sample on the overall rating of FMSS-EE, but levels of stability for the individual components criticism and EOI were lower. McGuire and Earls (1994) administered the FMSS to the families of 29 disadvantaged children and found that EE ratings remained moderately stable when the FMSS was administered 5 weeks later. Based on these findings, it was hypothesised that the individual components of maternal EE would not significantly change over a five-year period. Stability is of particular interest given the rapidly changing behavioural profiles exhibited by children of different ages.

Second, the study aimed to examine the overall contribution of T1 child-specific or shared family effects on T2 maternal EE. It was predicted that both child-specific and family effects would longitudinally predict maternal EE, but T1 child-specific effects would make a stronger contribution than shared family effects. This prediction was based on the findings of Studies 1 and 2 and from studies that have demonstrated that when examined simultaneously child characteristics rather than maternal characteristics predicted maternal EE (Psychogiou et al., 2007) and that medication induced reductions in symptoms in children with ADHD are associated with improvements on measures of parent-child interaction including maternal EE (Schachar et al., 1987).

The third aim was to examine which T1 child characteristics explained T1 general child effects onT2 maternal EE. It was predicted that T1 child ADHD diagnostic status and child OPP/CP would be associated with T2 maternal, but that child OPP/CP would overshadow the effects of T1 ADHD diagnostic status. This is based on the findings of Studies 1 and 2 and previous studies described in the previous chapter (e.g., Christiansen et al., 2010; Psychogiou et al., 2007; Sonuga-Barke et al., 2008, 2009).

The fourth aim was to examine which T1 family characteristics explained T1 family effects on T2 maternal EE. Based on the findings of Studies 1 and 2, it was predicted that T1 overall family levels of child OPP/CP and maternal depressive characteristics would be associated with T2 maternal EE.

Fifth, the study aimed to examine whether T1 child and family effects displayed different patterns of association with the different T2 EE components. Based on the findings of the previous studies, it was hypothesised that although there would be some

family effects, child-effects would predominate for all EE components, except for WAR, for which family effects would be stronger.

5.3 Method

5.3.1 Participants

Participants included in the present longitudinal analysis were 45 sib-pairs and their mothers (n = 45) who took part in the Southampton arm of the IMAGE project both at T1 and T2. In each sib-pair, one child had a clinical diagnosis of DSM-IV ADHD Combined Type and in most, but not all families the other child did in the sib-pair did not have a diagnosis of ADHD. The two waves of data were collected five years apart. Four families were excluded from the total sample of 49 families who took part at both time points due to missing maternal EE data at either T1 (n = 3 families) or T2 (n = 1 family). Thirty seven probands were male and 8 were female and 23 siblings were male and 22 were female. Twenty two sibling pairs were made up of one boy and one girl, 19 were made up two boys and four were made up of two girls. The mean age of probands at T1 was 12.04 ± 2.29 years (7.75 – 16.91) and 16.69 ± 2.40 years (12 – 22) at T2. The mean age of siblings at T1 was 11.37 ± 3.20 years (6 – 17.08) and 15.93 ± 3.46 (9 – 22) at T2. Information on attrition and retention has been provided in Chapter 4.

5.3.2 Child Measures

T1 and T2 Maternal Expressed Emotion

Maternal EE at T1 and T2 was measured using the Revised Five Minute Speech Sample (RFMSS; Daley et al., 2003) as described in section 4.3.2 of Chapter 4.

T1 Child ADHD Diagnosis Status

The diagnosis of DSM-IV ADHD Combined Type at T1 was made using the Parental Account of Childhood Symptoms (PACS; Chen & Taylor, 2006; Taylor et al., 1986a) and the DSM-IV Total subscale of the Conners Teacher Rating Scale Revised – Long Version (CTRS-R: L; Conners, 1996) as described in section 4.3.2 of Chapter 4.

T1 Child ADHD Symptoms

The DSM-IV Inattentive and DSM-IV Hyperactive-Impulsive subscales of the Conners Parent Rating Scale Revised – Long Version (CPRS-R: L; Conners, 1996) were used to dimensionally measure ADHD symptoms in the correlational analyses with z-scores

calculated for each subscale and the two subscales added to together to form a composite score, as described in Chapter 4.

T1 Child Oppositional/Conduct Problems

The Oppositional subscale of the CPRS-R: L (Conners, 1996) and the Conduct Problems subscale of the parent version of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) were used to measure child OPP/CP at T1. Z-scores were calculated for each subscale and the two subscales were added together to form a composite score as described in section 4.3.2 of Chapter 4.

T1 Child Emotional Problems

The Anxious/Shy subscale of the CPRS-R: L and parent reports on the Emotional Symptoms subscale of the SDQ were used to measure child EP at T1. Z-scores were calculated for each subscale and the two subscales were added together to form a composite score as described in section 4.3.2 of Chapter 4.

5.3.3 Family Measures

T1 Overall Family Levels of Child Oppositional/Conduct Problems

Overall family levels of child OPP/CP at both T1 and T2 were measured by calculating mean OPP/CP scores for each sibling pair as described in section 4.3.3 of Chapter 4.

T1 Overall Family Levels of Child Emotional Problems

Overall family levels of child EP at both T1 and T2 were measured by calculating mean EP scores for each sibling pair in section 4.3.3 of Chapter 4.

T1 Maternal ADHD Symptoms

The Current Symptoms Scale- Self Report Form (CSS-SR; Barkley & Murphy, 1998) was used to measure maternal ADHD symptoms at T1 as described in section 4.3.3 of Chapter 4.

T1 Maternal Depressive Symptoms

The General Health Questionnaire – 12-item Version (GHQ-12; Goldberg, 2003; Goldberg & Williams, 1988) was used to measure maternal depressive symptoms at T1 as described in section 4.4.3.4 of Chapter 4.

5.3.4 Procedure

The procedures employed at T1 and T2 have been described in full in section 4.3.4.

5.4 Analytic Strategy

First, descriptive statistics were obtained including the means and standard deviations and frequencies of EE component scores to illustrate change in EE components over time, followed by correlations among T2 EE components, T1 EE components and T1 child and family/parent characteristics.

Second, longitudinal two-level univariate multilevel models were run separately for each EE component using the same analytic strategy adopted in Studies 1 and 2 (see section 3.4 of Chapter 4 for a full description) with a few exceptions. This time, given the longitudinal design of the study, the T1 and T2 data combined was used first to estimate *T1* child-specific (within-family) and shared family (between-family) effects on *T2* maternal EE and second to examine which specific T1 child and family/parent characteristics explained these effects longitudinally. The first model was conditioned on T1 ADHD diagnostic status *and* T1 EE to provide a baseline longitudinal estimate of how much of the variance in maternal EE was at the child-specific (within-family) level and how much was at the shared family (between-family) level. This means that any effects seen for specific child or family variables in these models will be over and above those cross sectional effects seen at T1. The order in which the T1 child level and family level predictors were entered in the model remained the same as in Studies 1 and 2 (i.e., child gender, T1 age, T1 child OPP/CP, etc.).

5.5 Results

5.5.1 Continuity of Maternal EE over Time

Table 5.1 shows the distribution of IS, REL and WAR at T1 and T2 as a function of ADHD diagnostic status and Table 5.2 presents the mean scores for the separate EE components at T1 and T2 as a function of ADHD diagnostic status. There was considerable stability in the separate components of EE over a five-year period. The intercorrelations presented in Table 5.3 also show there was strong continuity between T1 and T2 WAR, PC and CC (respectively r = .59, r = .50, r = .42), but only moderate correlations were observed between T1 and T2 IS and REL (respectively r = .18, r = .27).

Table 5.1

Distribution of EE Components at T1 and T2 as a Function of ADHD Diagnostic Status

	Siblings with ADHD (n = 49)				Siblings without ADHD (n = 41)			
	T1		T2		T1		T2	
Maternal EE component	*n*	%	*n*	%	*n*	%	*n*	%
Initial statement								
Positive	10	20.4	9	18.4	11	26.8	9	22.0
Neutral	31	63.3	33	67.3	30	73.2	31	75.6
Negative	8	16.3	7	14.3	0	0.00	1	2.4
Relationship								
Positive	14	28.6	9	18.4	21	51.2	20	48.8
Neutral	25	51.0	36	73.5	17	41.5	20	48.8
Negative	10	20.4	4	8.2	3	7.3	1	2.4
Warmth								
High	19	38.8	23	46.9	22	53.7	26	63.4
Moderate	21	42.9	18	36.7	16	39.0	13	31.7
Low	9	18.4	8	16.3	3	7.3	2	4.9

Table 5.2

Mean Scores of EE Components at T1 and T2 as a Function of ADHD Diagnostic Status

	Siblings with ADHD (n = 49)			Siblings without ADHD (n = 41)		
	T1	T2		T1	T2	
Maternal EE component	Mean (SD)	Mean (SD)	t	Mean (SD)	Mean (SD)	t
Initial statement	1.96 (.61)	1.96 (.58)	.00	1.73 (.45)	1.80 (.46)	-.73
Relationship	1.92 (.70)	1.90 (.51)	.17	1.56 (.63)	1.54 (.55)	.19
Warmth	1.80 (.74)	1.69 (.68)	.68	1.54 (.64)	1.41 (.59)	.90
Positive Comments	2.37 (2.62)	2.39 (2.17)	-.55	4.24 (3.15)	4.93 (4.42)	-.34
Critical Comments	3.16 (2.54)	4.29 (3.55)	-1.32	1.10 (1.45)	1.49 (2.57)	-.32

5.5.2 Associations between Child and Parent Characteristics over Time

Pearson's correlations between T2 EE components, T1 EE components and T1 child and family characteristics are presented in Table 5.3. Most correlations between T1 child and family characteristics and T2 EE components were significant and in the expected direction. Intercorrelations among T2 EE components were moderate and in the expected direction. T1 EE components were weakly to moderately correlated with T2 EE components except the relationship between T1 IS and T2 IS was not significant and nor was the association between T1 IS and T2 PC. Small to medium correlations were found between T1 child behavioural and emotional problems and T2 EE, although T1 child emotional problems were not significantly associated with T2 REL.

On one hand, T1 maternal ADHD did not appear to be an important influence on maternal EE as it was not significantly correlated with T2 EE components or with T1 EE components. On the other hand, mothers' depressive symptoms seemed to have a significant effect on EE – this characteristic was positively associated with T1 and T2 REL, T2 IS, T1 WAR and CC and negatively correlated with T2 PC. T1 sib-pair average behavioural and emotional problems were weakly correlated with some, but not all T2 EE components. All T1 sib-pair mean behavioural and emotional difficulties were significantly associated with T2 PC and CC in the anticipated direction. In addition, T1 sib-pair mean ADHD symptoms, EP and EDP were positively related to T2 WAR, T1 sib-pair average OPP/CP was positively correlated with T2 IS as was T1 EDP. Significant associations were found between almost all T1 sib-pair average behavioural and emotional problems and T2 EE components. The exceptions were the relationships between T1 sib-pair mean EP and T1 IS and T1 CC, between T1 sib-pair mean EDP and T1 IS and between T1 sib-pair average ADHD symptoms and WAR for which non-significant correlations were found.

Correlations among T1 child problems were moderate to high with the highest correlations between ADHD symptoms, OPP/CP and EDP. Likewise, T1 maternal depressive characteristics and ADHD symptoms were moderately positively correlated.

In contrast to what one might expect, T1 child problems were not significantly correlated with T1 maternal problems except T1 child EP was positively associated with maternal ADHD symptoms. Moderate positive correlations were found between T1 sib-pair average problems and T1 maternal ADHD symptoms, but T1 sib-pair average problems were not significantly related to T1 maternal depressive characteristics.

Table 5.3

Correlations between Child and Family Characteristics and Maternal EE over Time

	1	2	3	4	5	6	7	8	9	10	11	12	13	15	16	17	18
1. T1 IS	-																
2. T1 REL	.47***	-															
3. T1 WAR	.31**	.62***	-														
4. T1 PC	-.34***	-.53***	-.44***	-													
5. T1 CC	.38***	.55***	.44***	-.17	-												
6. T1 child ADHD	.22*	.40***	.25**	-.42***	.44***	-											
7. T1 child OPP/CP	.28**	.55***	.35***	-.46***	.47***	.80***	-										
8. T1 child EP	.13	.26**	.30***	-.38***	.14	.47***	.51***	-									
9. T1 mother ADHD	.09	.05	.15	-.06	.08	.10	.15	.23*	-								
10. T1 mother DEP	.24*	.25**	.26**	-.15	.19*	.01	.08	.09	.43***	-							
12. T1 sib-pair ADHD	.21*	.25**	.17	-.18*	.15	.50***	.47***	.40***	.20*	.03	-						
13. T1 sib-pair OPP/CP	.27**	.42***	.34**	-.22*	.24*	.37***	.63***	.44***	.24*	.13	.75***	-					
14. T1 sib-pair EP	.17	.23*	.29**	-.21*	.08	.27**	.37***	.75***	.30*	.12	.54***	.59***	-				
15. T2 IS	.18*	.20*	.27**	-.24*	.18*	.21*	.31*	.31**	.16	.11	.12	.24*	.12	-			
16. T2 REL	.17	.27**	.27**	-.26*	.19*	.28**	.36***	.11	.07	.20*	-.01	.12	.12	.39***	-		
17. T2 WAR	.22*	.46***	.59***	-.33**	.39***	.34**	.45***	.37***	.04	-.03	.31**	.40	.27**	.39***	.34**	-	
18. T2 PC	-.15	-.45***	-.53***	.50***	-.32**	-.45***	-.50***	-.41***	-.14	-.24*	-.25**	-.34***	-.30**	-.35***	-.48***	-.45***	-
19. T2 CC	.19*	.33**	.30**	-.24*	.42***	.44***	.62***	.30**	.07	-.02	.25**	.37***	.22*	.45***	.57***	.57***	-.37***

Note. IS = initial statement; REL = relationship; WAR = warmth; PC = positive comments; CC = critical comments; ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms

*p < .05, **p < .01, ***p < .001.

5.5.3 Child and Family Effects on Maternal EE over Time

Within-Family and Between-Family Variation in T2 Maternal EE

The first model run was used to longitudinally establish a baseline for the amount of variance in T2 maternal EE explained by T1 child (within-family) and family (between-family) effects conditioned on ADHD diagnostic status and T1 EE. Except for ADHD diagnostic status and T1 EE, no predictor variables were entered in this initial model. Figure 5.1 shows the proportion of within-family and between-family variance in maternal EE at T2. Column 1 of Tables A3.1 to A3.5 in Appendix 3 shows the within-family and between-family variance values and their levels of significance for T2 WAR, PC and CC. These initial models were similar to the initial models of the cross-sectional analyses of T2 data presented in Chapter 4, but in contrast, due to the longitudinal design of these models, they have been conditioned on T1 ADHD diagnostic status *and* T1 maternal EE as opposed to just being conditioned on T1 ADHD diagnostic status. The within-family variance was .25 and the between-family variance was .01 (χ^2(df = 44) = 48.88, p = .283) for T2 IS and for T2 REL the within-family variance was .23 and the between-family variance was .03 (χ^2(df = 44) = 56.54, p = .09). There was a significant longitudinal contribution of both T1 child and family effects on T2 WAR, PC and CC. Only significant T1 child effects on T2 IS and REL were found. Except for WAR, T1 child effects on T2 EE made a stronger longitudinal contribution than T1 family effects, although the overall longitudinal contribution of family effects on WAR was only marginally stronger than child effects.

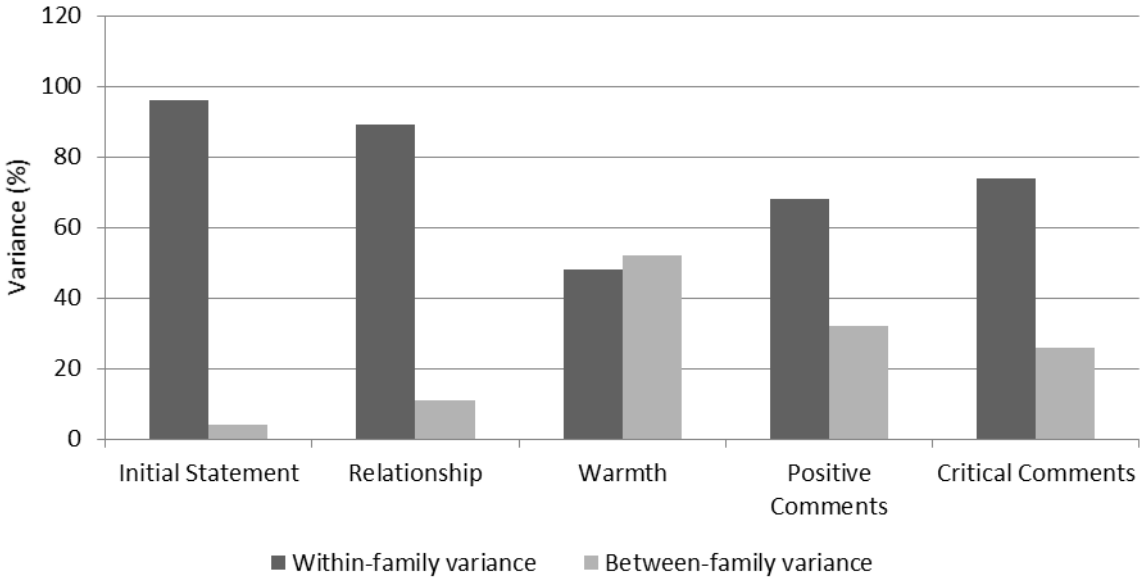

Figure 5.1. The proportion of within-family and between-family variance in the initial model conditioned on T1 ADHD diagnostic status and T2 maternal EE

T1 Child Characteristics that Predicted T1 Child Effects on T2 maternal EE

Table 5.4 summarises the specific child characteristics that predicted T1 child effects on T2 EE components and Tables A3.1 to A3.5 in Appendix 3 present full information regarding the multilevel models. T1 ADHD diagnostic status was entered in the multilevel models first with T1 EE to control for T1 EE components and therefore allow effects observed of child and family factors on T2 EE that were over and above those relations observed at T1 to be examined. There was a non-significant association between T1 and T2 IS. T1 and T2 WAR and T1 and T2 PC were significantly associated each other and T1 and T2 REL and T1 and T2 CC were marginally significantly associated with each other. The significant associations between T1 and T2 WAR and T1 and T2 PC remained significant when all child- and family-level predictors had been entered in the model. The marginally significant associations between T1 and T2 REL and T1 and T2 CC became non-significant after child OPP/CP were entered in Step 3 of the stepwise multiple regression model and fourth in the multilevel model respectively. T1 ADHD diagnostic status was significantly and positively associated with T2 WAR and T2 CC and significantly and negatively associated with T2 PC. There was a positive and marginally significant association between T1 ADHD diagnostic status and T2 REL. The associations between T1 ADHD diagnostic status and T2 WAR and PC remained significant after all child- and family-level predictors were entered in the model. The associations between T1 ADHD diagnostic status and T2 REL and CC became non-significant after T1 child OPP/CP was entered. There was a non-significant association between T1 ADHD diagnostic status and T2 IS.

Child gender and T1 child age were entered in second and third respectively in the multilevel models. There were no significant effects of T1 child age on any of the individual T2 EE components. Child gender was significantly and positively associated with T2 IS and negatively with T2 REL and CC. The association held for T2 IS and T2 REL after all child predictors had been entered, but did not remain for T2 CC after T1 child OPP/CP were entered.

T1 child OPP/CP was entered fourth in the multilevel models. There was a significant and positive association between T1 child OPP/CP and T2 CC. This association remained after all child- and family-level predictors were entered. T1 child OPP/CP and T2 were marginally significantly and positively associated with T2 WAR, but the association did not remain after T1 child EP was entered (see Model 5, Table 5.6).

T1 child EP was entered fifth in the multilevel models. There was a significant and positive association between T1 child EP and T2 IS, an effect that remained in the final model when all child and family level predictors had been entered. T1 child EP was not significantly associated with the other individual components.

Table 5.4

T1 Child Characteristics that Predicted T1 Child Effects on T2 Maternal EE Components

Parameter	T2 IS	T2 REL	T2 WAR	T2 PC	T2 CC
T1 child ADHD status	X	X	X	X	X
T1 EE	X	X	√*** (+)	√*** (+)	X
Child gender	√* (+)	√** (-)	X	X	X
T1 child age	X	X	√† (+)	X	X
T1 child OPP/CP	X	X	†* (+)	X	√*** (+)
T1 child EP	√† (+)	X	X	X	X

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct; EP = emotional problems. Full information on T1 child characteristics that predicted T2 child effects over and above the cross-sectional effects seen at T1 are provided in Tables A3.1 to A3.5 in Appendix 3.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association.

T1 Family Characteristics that Predicted T1 Family Effects on T2 Maternal EE

Tables 5.5 presents a summary of family effects that accounted for T2 maternal EE components and Tables A3.1 to A3.5 in Appendix 3 display full details of the multilevel models of the specific family/parent characteristics that explained T1 family effects on T2 EE components. The first and second T1 family-level predictor entered in the model was sib-pair gender, dummy coded as all-boy sib-pair and all-girl sib-pair and entered in that order. All-boy sib-pair was significantly and positively associated with T2 IS and negatively with T2 PC, but there were no significant associations between this family characteristic and the remaining components. These associations remained in the final model with all predictors entered.

The third T1 family-level predictor entered in the model was T1 sib-pair average OPP/CP. There was a significant and positive association between sib-pair mean OPP/CP and all EE components except for T2 REL. This association remained for IS, PC and CC with all child- and family-level predictors entered, but not for T2 WAR after T1 sib-pair average EP had been entered.

T1 overall family levels of child EP, the fourth T1 family-level predictor entered in the model, were not significantly associated with any of the T2 individual components of EE. When T1 sib-pair EP and OPP/CP were entered in reverse order, there was a positive and significant association between T1 sib-pair mean EP and T2 CC, but the association did not remain after sib-pair OPP/CP was entered.

T1 maternal ADHD symptoms were the fifth family-level characteristic entered. There were no significant associations between T1 maternal ADHD symptoms and the

individual T2 EE components. There was a significant and negative association between maternal depressive symptoms, the last family-level predictor entered, and T2 WAR which is in the opposite direction to what would be expected. Maternal depressive symptoms were not significantly associated with the other two components.

Table 5.5

T1 Family Characteristics that Predicted T1 Family Effects on T2 Maternal EE Components

Parameter	T1 IS	T2 REL	T2 WAR	T2 PC	T2 CC
All-boy sib-pair	X	X	X	√* (-)	X
All-girl sib-pair	X	X	√† (-)	X	X
T1 sib-pair mean OPP/CP	√* (+)	X	√* (+)	√* (-)	√* (+)
T1 sib-pair mean EP	X	X	X	X	X
T1 maternal ADHD	X	X	X	X	X
T1 maternal DEP	X	X	√* (-)	X	X

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct; EP = emotional problems; DEP = depressive symptoms.

Full information on T1 family characteristics that predict T2 family effects over and above the cross-sectional effects seen at T1 are provided in Tables A3.1 to A3.5 in Appendix 3.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association.

5.6 Discussion

The primary aim of the present study was to extend the findings of Studies 1 and 2 using a within-family multilevel modelling design, but also a longitudinal, instead of a cross-sectional design, to test whether child or family effects drive maternal EE over time in families of clinic-referred children with ADHD between T1 and T2. Specifically, it examined: (1) the continuity of maternal EE over a five-year period; (2) T1 child and family effects on T2 maternal EE (3) which T1 child and family characteristics explained any longitudinal child and family effects found and (4) whether T1 child and family effects showed different patterns of association with T2 EE components. Crucially T1 EE was controlled in all the MLMs so effects observed of child and family factors on T2 EE were over and above those relations observed at T1. Of particular interest was whether the same pattern of effects would arise longitudinally as they had cross-sectionally in Studies 1 and 2.

As predicted, this study demonstrated continuity in the individual components of EE over a five year period. This finding is quite compelling given how much children and families change over a span of 5 years. The stability of maternal EE over a 5-year period provided evidence for the validity of the construct with regard to ADHD and childhood disorder more generally. This finding is in accordance with previous studies which have demonstrated moderate stability in parental EE over a period of 5 weeks (McGuire &

Earls, 1994), 9-months toward children with emotional disorders (Vostanis & Nicholls, 1995), 18 months in adolescents and adults with autism (Greenberg et al., 2006) and over a two-year period from preschool to first grade in a community sample of high-risk children (Peris & Baker, 2000). One question that remains debated in the EE literature is whether EE is a 'state' or 'trait'. In a sample of adult patients with chronic schizophrenia, their well sibling and parents, Schreiber, Breier, and Pickar (1995) found that parents showed significantly more EOI with the child with schizophrenia, significantly more WAR toward the well child and that the mean score for CC was higher towards the well child, but this effect did not reach a level of statistical significance. In accordance with their hypothesis, they concluded that EOI and WAR are related to the state of the child because the level of response was different between the two siblings and that CC was a parental personality trait because the level of response was similar. Longitudinal studies that have measured EE at two or more time points have concluded that because the EE measure was stable over time that it should be conceptualised as a consistent feature of parental attitudes (i.e., a personality trait) rather than a transient state or reaction to a stressor (e.g. child behaviour). The findings of the current study demonstrate on the one hand strong within-family differences in EE (i.e., levels of EE response differ between siblings) which would seem to support the hypothesis that EE is state. On the other hand, the stability of EE found over 5 years suggests that it is a trait.

The hypothesis that T1 child effects would be stronger than T1 family effects in predicting T2 maternal EE was confirmed. In fact, there were no significant family effects implicated in predicting IS and REL over time (i.e., the variance in shared family effects were not significant). For IS and REL, the results do not support the hypothesis that both child and family effects would predict maternal EE over time. These findings are partially consistent with the cross-sectional results of Studies 1 and 2. In accordance with the T2 cross-sectional findings, the between-family variance in IS was not significant; however, in contrast to Studies 1 and 2, shared family effects on REL over time were not significant and naturally the proportion of between-family variance was less. Family effects on WAR, PC and CC over time were equally as strong as family effects observed cross-sectionally in Studies 1 and 2. Whereas the longitudinal findings pertaining to IS and REL in the present study support the notion that for these components EE is predominantly a "child effect", the results regarding WAR, PC and CC support a child and parent effects model with child effects playing a stronger role for PC and CC and child and family effects almost equally driving WAR over time.

This pattern of results in relation to IS and REL are congruent with previous cross-sectional studies that have simultaneously examined the influence of child and family characteristics on maternal EE in a community sample of school-aged boys (Psychogiou et

al., 2007), treatment trials (Schachar et al., 1987) and longitudinal studies that have shown child effects in that boys' ADHD symptoms had an impact on mother-son hostility, but no parent effects (i.e., the effects were not found in the other direction). Furthermore, Peris & Hinshaw (2003) found an association between maternal CRIT and diagnosis of ADHD, but no relationship between maternal EE and maternal depression, suggesting only the operation of child effects in predicting maternal EE. There are no previous longitudinal studies that have been specifically designed to examine child and family effects on maternal EE toward children with ADHD. In addition, longitudinal studies in the broader context of the field of EE have aimed to tackle whether it is parental EE causes child disorders or whether it is the other way around. However, these studies still provide some evidence of a child effects model. Hale III et al.'s (2011) found that adolescent externalising symptoms predicted high levels of parental EE rather than the other way around.

In examining which specific child characteristics explained child effects found over time, at T2 mothers had more negative relationships, made less positive comments and more critical comments about their children who at T1 were diagnosed with ADHD. This finding is consistent with Studies 1 and 2 and previous cross-sectional studies in community samples of preschool (Daley et al., 2003) and school-aged (Marshall et al., 1990; Psychogiou et al., 2007) children and longitudinal studies in the same aged population (Peris & Baker, 2000) that have established an association between parental EE and ADHD in childhood. However, this association became non-significant after T1 child oppositional/conduct problems were entered in the regression and multilevel models. This pattern was also found in Studies 1 and 2. In contrast to the results of Study 1, but in line with the findings of Study 2, T1 ADHD diagnostic status did not predict T2 IS. Furthermore, there was no association between T1 ADHD diagnostic status and T2 REL. Interestingly, it appears, therefore, that there is no longitudinal association between ADHD and IS or REL. Maternal FMSS-CRIT is rated in the presence of a negative REL rating, a negative IS rating and one or more critical comments and in their study Hastings et al. (2006) did not find a longitudinal relationship between maternal CRIT and child and adolescent externalising symptoms over a two-year period.

Whilst for WAR, PC and CC the significant effect of ADHD was lost when T1 child OPP/CP was entered, there was only a significant effect of OPP/CP on CC over time when all child and family characteristics were entered in the model including the control of T1 CC. Given the longitudinal design of the current study, these findings provide more robust evidence that comorbid problems, rather than ADHD, may provoke high levels of maternal EE. In line with Taylor's (1999) hypothesis, it could still also mean that early in childhood the behaviour of the child with ADHD provoked high levels of EE, which in turn increased comorbid OPP/CP. The analyses still do not allow conclusions to be made

about causation. However, unlike the results of Studies 1 and 2 which showed a consistent pattern of child OPP/CP concurrently predicting the majority of the individual EE components, the findings of the present study demonstrated that it was only for one component that this association held true. T1 child OPP/CP problems did predict T2 WAR, but this association did not remain significant when child emotional problems were entered.

Numerous preceding studies have demonstrated that parents of children with conduct and/or oppositional problems have higher levels of EE, in particular CRIT (Hibbs et al., 1991, 1993; Peris & Hinshaw, 2003; Psychogiou et al., 2007; Stubbe et al., 1993). The picture as to whether it is comorbid oppositional/conduct problems that account for much of the link between ADHD and high parental EE is less clear. As previously mentioned Psychogiou et al. (2007) and Christiansen et al. (2010) found the association between ADHD symptoms and maternal FMSS-EE did not remain when conduct problems were entered. One study found no association between comorbid ODD or CD in boys with ADHD (Marshall et al., 1990) and another study in girls did find an association, but the link between ADHD and FMSS-EE withstood control of comorbid aggression (Peris & Hinshaw, 2003).

Interestingly, child EP did not drive PC changes between T1 and T2 as was found concurrently in Studies 1 and 2. Instead, T1 child EP predicted T2 WAR and there was a trend toward T2 IS. In general, maternal warmth has been associated with children's better regulation of positive affect (Davidov & Grusec, 2006). This finding, however, is inconsistent with a previous study which found that WAR was higher in children with conduct disorders than children with emotional disorders (Vostanis et al., 1994) and that WAR in both clinical groups was higher than controls. Other than the marginal effect of child EP on IS over time, no other child characteristics accounted for child effects on IS over time (not even T1 IS). A similar pattern was found for PC; only T1 PC had a significant effect on T2 PC.

Child demographic characteristics, specifically male gender, explained child effects on REL over time. Indeed, this was the only T1 child characteristic to predict T2 REL. Child problems were not associated with REL over time, thus the results did not support the prediction that REL would be associated with OPP/CP. Cook et al., 1989) identified higher levels of EE in mother-son dyads than in mother-daughter dyads in a sample of adolescents with psychiatric problems. However, by and large, most studies have not found associations between child demographic characteristics and parental EE. In one of the few other studies that has found an effect of gender, the opposite was found with female gender associated with maternal CRIT (Hirshfield et al., 1997).

By and large, in relation to child effects, there are two important differences between the cross-sectional results of Studies 1 and 2 and the longitudinal findings of the present study. First, although in Studies 1 and 2, child psychopathology, whether that be a diagnosis of ADHD, OPP/CP or EP, predicted all the individual components, in the present study, child psychopathology, specifically child OPP/CP predicted only one component (i.e., CC) over time. Second, whilst in Studies 1 and 2 there was a clear pattern pertaining to child characteristics that predicted the individual components (i.e., child OPP/CP predicted the majority of components and child EP predicted PC), the opposite was true in the current study with the exception of CC. Indeed, child EP accounted for child effects on WAR and IS over time.

Upon investigating specific T1 family characteristics that might explain family effects found to predict WAR, PC and CC over time, as predicted T1 overall levels of child OPP/CP explained differences between families in average levels of these components at T2. This finding is partially supported by the results of Studies 1 and 2 which showed that sib-pair mean OPP/CP were concurrently associated with WAR and CC at both time points; PC at T2 was instead predicted by sib-pair mean EP (and the significant effect of sib-pair OPP/CP on T1 PC no longer remained after sib-pair average EP was entered). The findings of the present study further emphasise the clinical implications of the association between overall family levels of child OPP/CP and maternal EE, an issue raised in the discussion of the previous chapter.

Maternal depressive symptoms at T1 accounted for differences between parents' average levels of WAR at T2. Surprisingly, and contrary to what was predicted and what was found in Study 1, this association was negative (i.e., mothers' average levels of WAR at T2 were lower in families of mothers who at T1 had higher levels of depressive symptoms). This finding was echoed in a community study of child and family effects on maternal EE which found maternal depressive symptoms were negatively associated with criticism (Psychogiou et al., 2007). The authors of this study suggested that mothers with low mood may be less talkative and thus may make fewer comments in general about their children. However, WAR is rated on tone of voice, to warmth expressed only during the speech sample and when it is known that the parent has depression it is discounted based on the notion that depressed people are capable of expressing warmth. Still ratings may be more difficult if parents speak little during the speech sample. The authors suggested their results may also be due to their use of a community sample. Families in the present study were drawn from a clinical sample and thus one might expect higher levels of depressive symptoms in parents. This finding is congruent with the results of Study 1 only as far as to say that there was an association between maternal depressive symptoms and WAR at T1, but the effect was in the opposite direction. Unlike in Study 1 no effects of maternal

depressive symptoms were found on REL or PC. As discussed in Chapter 4, findings in relation to the relationship between parental depressive symptoms and depressive symptoms are mixed.

Limitations

The present study was unique in that it used a within-family multilevel modelling design and longitudinal data to examine child and family effects on maternal EE over time. However, there are several methodological limitations that should be taken into account. The majority of these are carried over from Studies 1 and 2 given that the same data in Study 3 were used. These are described in brief here given that further explanation was provided in Chapter 4 and will also be given in Chapter 7. First, the use of maternal EE alone and lack of data on paternal EE may be unlikely to capture EE as it was intended, that is, as a broader index of the family environment and difference patterns of child and family effects might be found for maternal and paternal EE. Second is the lack of proxy reports on measures of child and parent psychopathology. Third, in relation to specific family/parent-related factors some studies have implicated parental personality as a factor contributing to high levels of EE. Fourth, at T2 only parent reports on whether or not the target child had a diagnosis of ADHD were available and administration of a standardised psychiatric interview was not conducted. Fifth, five minute speech samples were coded by different raters at T1 than T2. Due to the lack of degrees of freedom, cross-level interactions in the multilevel models could not be conducted. The study included only two time points and to be able to disentangle the cause-effect relationship three or more time points are required.

5.7 Conclusions

In conclusion, the longitudinal analysis demonstrated the stability of maternal EE over time and provides preliminary evidence for a potentially causal role of child effects and to a much lesser extent family effects in driving negative maternal EE. Consistent with Studies 1 and 2, child effects were stronger than family effects in predicting EE components over time, except for WAR for which case child and family effects contributed equally. Longitudinal child effects were accounted for by child OPP/CP and EP, rather than ADHD per se, but in contrast to Studies 1 and 2, child OPP/CP only explained child effects on EE between T1 and T2 for CC. There was a clear pattern of average family levels of child OPP/CP predicting all but one (REL) EE components over time in addition to maternal depressive symptoms for WAR, but in the opposite direction to what was expected.

Studies 1, 2 and 3 have revealed the complexity of the relationship between child and family characteristics and maternal EE toward children with ADHD. It takes a step further in allowing the drawing of causal inference on the role of some specific child and family effects in determining maternal EE. This issue is also relevant to the extent to which maternal EE at T1 drives the emergence of child problems by T2 – over and above the associations seen at T1. Study 4 addresses this question using additional longitudinal analysis.

Chapter Six

Does Maternal Expressed Emotion Drive the Development of Child Problems over Time in Families of Children with ADHD?

6.1 Overview of Chapter Six

Chapter 6 presents the fourth and final study of the thesis. This study extended the longitudinal analysis of Study 3 using the same sample of 45 sibling pairs and multilevel models to primarily examine whether high maternal EE increases the risk of the development of child behavioural and emotional problems over time in children with ADHD. The results demonstrated continuity in problems over the five year period in both siblings with and without ADHD. As expected for siblings with ADHD, hyperactive-impulsive symptoms marginally significantly decreased and inattention symptoms significantly increased. Whilst in both ADHD and non-ADHD siblings CD and ODD did not significantly increase over time, internalising symptoms did. Despite stability being high and disruptive behaviour not increasing, there was still some change to be explained and analyses indicated some predictors of change. After controlling for T1 problems changes in child problems appeared to be driven by both child and family effects over time, with an equal contribution of effects in predicting oppositional and conduct problems (OPP/CP) and emotional problems (EP) and stronger child effects in influencing emotional dysregulation problems (EDP). More specifically increase in overall maternal EE and both T1 warmth (WAR) and decrease in WAR significantly predicted changes in OPP/CP by T2, (i.e., after controlling for OPP/CP at T1). There was also a role for CC. T1 maternal ADHD dominated longitudinal family effects on OPP/CP, EP and EDP as did overall family levels of T1 EE, except for EDP. Implications of the findings and limitations of the study are discussed.

6.2 Introduction to Study 6

It has consistently been found in cross-sectional and longitudinal studies that parents of children and adolescents with ADHD often exhibit higher levels of EE including increased criticism and less warmth (Daley et al., 2003; Marshall et al., 1990; Peris & Hinshaw, 2003; Psychogiou et al., 2007). This finding was supported in the first three studies of this thesis. However, the findings of these studies also showed that the effect of ADHD was overshadowed by other comorbid child problems especially OPP/CP which is convergent with previous research (Christiansen et al., 2010). Furthermore, concurrent results of the first three studies support the notion of a child and parent effects model to best explain the relationship between child ADHD and parental EE given that both child and family effects were implicated in predicting maternal EE. The same pattern was found longitudinally; however, for IS and REL, the results suggested these components are a "child effect". Indeed, although child and family effects were found to cross-sectionally and longitudinally predict maternal EE, except for WAR, child effects were stronger than family effects. Previous studies have found that high parental EE toward children with ADHD is most likely a "child effect" (i.e., it is the behaviour of the child with ADHD that drives high parental EE) (Psychogiou et al., 2007; Schachar et al., 1987). The first three studies of this thesis provide further insight into the cause-effect relationship between maternal EE and child ADHD with respect to whether it is child or family effects that drive maternal EE. With respect to the cause-effect relationship, there is also the issue of whether maternal EE drives negative developmental trajectories in children with ADHD.

Marshall and colleagues (1990) suggested that negative family environment as reflected in measures of EE may independently determine the long-term outcome of ADHD. Some researchers have shown parental EE to have predictive validity. For instance, Peris & Baker (2000) showed that high EE at preschool predicted diagnosis of ADHD at third grade four years later. Furthermore, some authors have suggested that high parental EE might be a risk factor likely to drive the development of comorbid problems in children with ADHD, in particular oppositional and conduct problems. In the broader context of child psychopathology, Patterson's coercion theory proposes that parental negativity during parent-child interaction may escalate pre-existing child problems thus playing a key role in the development of conduct problems in ADHD.

Researchers have theorised that the highly heritable, early challenging behaviour associated with ADHD may set in motion a chain of events in which the child's difficult behaviour elicits negative reactions (e.g., parental distress and maladaptive parenting) from the environment, which may foster the development of conduct problems (Lahey, Miller,

Gordon, & Riley, 1999; Patterson, DeGarmo, & Knutson, 2000). It follows that EE may be an important risk or protective factor in developmental outcomes for children with ADHD, particularly with regard to the course of concurrent conduct problems (Johnston & Mash, 2001).

In accordance with this view, but more specific to ADHD, Taylor (1999) postulated that while family dysfunction does not initiate ADHD, it could still lead to the development of conduct disorder in people with ADHD; in the case of EE specifically, it is likely that the early symptoms of inattention, hyperactivity and impulsivity and associated hard-to-manage behaviour may elicit high levels of EE which in turn may exacerbate existing developmental risk and lead to the development of comorbid conduct disorder. Particular aspects of parental EE may also act in disrupting risky pathways and improve prognosis. For example, parental warmth seems to be a protective factor. A decreased risk for the development of ADHD in low birth weight children was found when mothers showed high levels of warmth (Tully, Arseneault, Caspi, Moffitt, & Morgan, 2004).This is of particular clinical importance especially given that children with concurring problems have poorer outcomes than children with ADHD alone.

Study 4 extended the longitudinal analysis of Study 3 and aimed to examine child-specific (within-family) and shared family (between-family) effects on child problems over time, including OPP/CP, EP and also emotional dysregulation problems (EDP). Specifically, in examining which specific child characteristics accounted for child effects, the primary aim was to test the hypothesis that high maternal EE at T1 and change in EE from T1 to T2 would predict the development of child problems over time especially child OPP/CP. As noted in Chapter 2, several recent studies have provided support for the hypothesis that high parental EE contributes to the development of OPP/CP over time. First, in a large sample of clinic-referred children with ADHD who participated in the IMAGE project , Sonuga-Barke et al. (2009) found an association between both mothers' and fathers' negative EE (indexed by high levels of criticism and low levels of warmth) and the presence of conduct problems. Second, also in a subsample of families recruited into the IMAGE project, Christiansen et *al.* (2010) found that while parents of children with ADHD were more hostile and critical towards their children than parents of healthy controls, this effect was removed when children's oppositional problems were entered in the ANOVA model as a covariate. Further analysis of the individual components of EE demonstrated that only low parental WAR was associated with oppositional problems in ADHD with high WAR predicting low scores of oppositional behaviour. These findings suggest that WAR may be a protective factor that buffers resilience in children with ADHD to developing later oppositional and conduct problems. Longitudinal research in an epidemiological sample of monozygotic twins found that the twin that received more

negativity and less warmth had more antisocial behaviour problems (Caspi et al., 2004). Based on previous findings, it was predicted that T1 maternal EE would be positively associated with OPP/CP at T2. Furthermore, T1 WAR would predict T2 OPP/CP.

6.3 Method

6.3.1 Participants

Participants were 45 children with ADHD, their siblings (n = 45) and mothers (n = 45) who took part in the Southampton arm of the IMAGE project at T1 and T2 as described in section 5.3.1 of Chapter 5.

6.3.2 Child Measures

T1 Child ADHD Symptoms

The DSM-IV Inattentive and DSM-IV Hyperactive-Impulsive subscales of the Conners Parent Rating Scale Revised – Long Version (CPRS-R: L; Conners, 1996) were used to dimensionally measure ADHD symptoms in the correlational analyses with z-scores calculated for each subscale and the two subscales added to together to form a composite score, as described in Chapter 4.

T1 and T2 Oppositional/Conduct Problems

At T1, the Oppositional subscale of the Conners Parent Rating Scale Revised – Long Version (CPRS-R: L; Conners, 1996) and the Conduct Problems subscale of the parent version of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997) were used to measure child OPP/CP. At T2, the DSM-IV Oppositional Defiant Disorder and DSM-IV Conduct Disorder subscales of the Conners Comprehensive Behaviour Rating Scale (Conners CBRS; 2008) were used to measure T2 child OPP/CP. Z-scores were calculated for each subscale and the two subscales for T1 were added together as were the two subscales for T2 to form a composite scores for T1 and T2 as described in Section 4.3.2 of Chapter 4.

T1 and T2 Emotional Problems

At T1, the Anxious/Shy subscale of the CPRS-R: L and parent reports on the Emotional Symptoms subscale of the SDQ were used to measure child EP. At T2, emotional problems (EP) were measured using the DSM-IV Major Depressive Disorder and DSM-IV Generalised Anxiety Disorder subscales of the Conners CBRS (Conners, 2008). Z-scores

were calculated for each subscale and the two subscales for T1 were added together as were the two subscales for T2 to form composite scores for T1 and T2 as described in Section 4.3.2 of Chapter 4.

T1 and T2 Emotional Dysregulation Problems

T1 emotional dysregulation problems (EDP) were measured using the Conners' Global Index: Emotional Lability scale of the CPRS-R: L (Conners, 1996) and the DSM-IV Manic Episode scale of the Conners CBRS (Conners, 2008) at T2. These questionnaires have been described in full in Section 4.3.2 of Chapter 4.

T1 Child ADHD Diagnostic Status

The diagnosis of DSM-IV ADHD Combined Type at T1 was made using the Parental Account of Childhood Symptoms (PACS; Chen & Taylor, 2006; Taylor et al., 1986a) and the DSM-IV Total subscale of the Conners Teacher Rating Scale Revised – Long Version (CTRS-R: L; Conners, 1996) as described in Section 4.3.2.2 of Chapter 4.

T1 Maternal Expressed Emotion and Change in Maternal Expressed Emotion

T1 maternal EE was measured using a modified version of the Five Minute Speech Sample (Daley et al., 2003) as described in Section 4.3.2.1 of Chapter 4. Maternal EE was also dichotomised into high EE and low EE. High EE is coded when at least one global category (i.e., IS, REL, WAR) is rated as negative or low and when the respondent has made more critical comments than positive comments. Change in overall EE was measured by rating whether EE had increased over time (i.e., the rating had changed from low to high), stayed the same, or decreased over time (i.e., the rating had changed from high to low) so that 1 = decreased, 2 = stayed the same, 3 = increased. Similarly, change in IS, REL and WAR was measured by rating each of these components on a scale from 1 to 5 according to whether the rating had decreased by 1 level = 1 (i.e., the rating changed from negative/low at T1 to neutral at T2 or from neutral at T1 to positive/high at T2), decreased by 2 levels = 2 (i.e., the rating changed from negative/low at T1 to positive/high at T2), stayed the same = 3, increased by 1 level = 4 (i.e., the rating changed from positive/high at T1 to neutral at T2 or from neutral at T1 to negative/low at T2), or increased by 2 levels = 5 (i.e., the rating went from positive/high at T1 to negative/low at T2). Change in PC and CC was measured using a difference score.

6.3.3 Family Measures

T1 Overall Family Levels of Maternal EE and Change in Overall Family Levels of Maternal EE from T1 to T2

T1 overall family levels of overall maternal EE was measured by coding whether EE ratings toward both siblings were low = 1, toward 1 sibling was high, but toward the other were low = 1, or toward both siblings were high = 2. Change in overall family levels of overall maternal EE were measured by coding whether from T1 to T2 EE ratings toward both siblings decreased = 1, toward 1 sibling decreased, but toward the other sibling stayed the same = 2, toward both siblings stayed the same or toward 1 sibling decreased, but toward the other sibling increased = 3, toward 1 sibling increased, but toward the other sibling stayed the same = 4, or toward both siblings increased = 5. T1 overall family levels of IS, REL and WAR were measured by coding whether EE ratings toward both siblings were positive/high = 1, toward 1 sibling were positive/high, but toward the other sibling were neutral = 2, toward both siblings were neutral or toward 1 sibling were positive/high, but toward the other sibling were negative/low = 3, toward 1 sibling were negative/low, but toward the other sibling were neutral = 4, or toward both siblings were negative/low. Change in overall family levels of IS, REL and WAR were measured by coding whether from T1 to T2 EE ratings toward siblings pairs had decreased = 1, stayed the same = 2, or increased = 3. An average score of the two siblings in each pair was calculated for T1 overall family levels of PC and CC and a difference score was used to measure change in overall family levels of PC and CC.

T1 Maternal ADHD Symptoms

The Current Symptoms Scale- Self Report Form (CSS-SR; Barkley & Murphy, 1998) was used to measure maternal ADHD symptoms at T1 reported in Section 4.3.3 of Chapter 4.

T1 Maternal Depressive Symptoms

The General Health Questionnaire – 12-item Version (GHQ-12; Goldberg, 2003; Goldberg & Williams, 1988) was used to measure maternal depressive symptoms at T1 outlined in Section 4.3.3 of Chapter 4.

6.3.4 Procedure

The procedures employed at T1 and T2 are outlined in full in Section 4.3.4 of Chapter 4.

6.4 Overview of Analytic Strategy

First, descriptive statistics were obtained including a comparison of the means and standard deviations of scores on measures of child behavioural and emotional problems between T1 and T2 and Pearson's correlations among T2 behavioural and emotional problems, T1 EE components and T1 family/parent characteristics. Second, two-level multilevel models using full maximum likelihood estimates were run to first estimate the overall longitudinal contribution of T1 within- and between-family effects on T2 behavioural and emotional problems and second to investigate whether T1 maternal EE and change in maternal EE over time toward the individual child explained any child effects on T2 behavioural and emotional problems and also whether sib-pair EE and change in sib-pair EE over time explained any family effects on T2 behavioural and emotional problems. Separate models were run for each behavioural and emotional problem – OPP/CP, EP and EDP with child and family level predictors entered in the model one at a time as was done in the previous studies. First the effects of overall maternal EE (i.e., high versus low EE) were examined and for behavioural problems for which overall maternal EE was found to predict child problems over time, the individual components of EE that might account for child effects were examined. The order of entry for child-level variables was T1 ADHD diagnostic status, T1 child problems, gender, T1 age, T1 overall maternal EE and change in maternal EE from T1 to T2. The order of entry of family-level variables was all-boy sib-pair gender, all-girl sib-pair gender, overall family levels of T1 maternal EE, change in overall family levels of maternal EE, maternal ADHD symptoms and maternal depressive symptoms.

6.5 Results

6.5.1 Continuity and Change in ADHD Symptoms and Other Child Problems over Time

Table 6.1 presents change in mean scores of inattention and hyperactive-impulsive symptoms and other externalising and internalising symptoms from T1 to T2 for siblings with and without ADHD. Overall, externalising problems demonstrated continuity, but did not significantly increase over the five year period in siblings with ADHD or siblings without ADHD. However, internalising problems did. Dependent t-tests revealed that for siblings with ADHD inattentive symptoms, anxiety symptoms and emotional dysregulation problems significantly increased from T1 to T2. In addition, there was a marginally significant increase in hyperactive-impulsive and depressive symptoms over time. In contrast to what was expected, there was no change in conduct problems and although there was a small increase in oppositional problems over time, the change was not

statistically significant. Only depressive symptoms significantly increased in siblings without ADHD over the five year period.

6.5.2 Correlations among T1 and T2 Child Characteristics

Table 6.2 displays correlations among T2 child behavioural and emotional problems and T1 EE components and other T1 child characteristics. T1 EE components and T1 child behaviour were almost all significantly related to each other and to T2 child behaviour in the expected direction and in the moderate to high range.

Intercorrelations between T2 child problems were high and positive as expected. A similar pattern was found among T1 child problems, but the correlations were weaker at T2 as they were predominantly in the moderate range. Moderate to high correlations were found between T1 and T2 child problems in the anticipated direction suggesting high levels of continuity but at the same time that there was some change between T1 and T2 to explain.

Significant correlations in the weak to moderate range were found among T2 child problems and all T1 EE components except the relationship between T2 EDP and T1 IS was non-significant. A similar pattern was found in terms of the associations between T1 child problems and T1 EE components although there were more non-significant correlations including the relationship between T1 IS and T1 child EP and EDP and between T1 CC and T1 EP. Correlations among T1 EE components in the longitudinal data set have been discussed in section 5.6.2 of chapter 5.

Table 6.1

Mean Scores on Measures of Child Problems by Sibling Status and Time Point

	Siblings with ADHD (n = 49)					Siblings without ADHD (n = 44)				
	T1		T2			T1		T2		
Child Problems	Mean	SD	Mean	SD	*t*	Mean	SD	Mean	SD	*t*
Inattentive symptoms[a]	74.27	9.22	78.73	11.02	-3.06**	53.68	11.71	55.34	16.22	-1.07
Hyperactive-impulsive symptoms[a]	82.55	10.28	79.65	12.60	1.87†	55.46	15.16	56.34	17.43	-.42
Conduct problems[b,c]	.50	.79	.54	.92	-.41	-.60	.90	-.65	.65	.49
Oppositional problems[a]	76.57	11.47	76.37	15.81	.12	56.59	13.09	57.10	15.94	-.32
Depressive symptoms[b,c]	.14	.95	.38	.87	-1.83†	-.16	1.05	-.46	.95	2.14*
Anxiety symptoms[a]	64.51	15.08	78.73	14.18	-5.80***	55.80	15.61	58.73	16.96	-.13
Emotional dysregulation problems[a]	72.94	12.40	79.45	14.09	-3.24**	57.88	14.97	58.20	19.18	-1.12

Note. [a] = symptoms/problems were measured using the CPRS-R: L at T1 and the Conners CBRS at T2; [b] = symptoms/problems were measured using the SDQ at T1 and the Conners CBRS at T2; [c] = scores have been standardised due to different scales used at T1 and T2.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

6.5.3 Correlations among T1 Family Characteristics and T2 Child Characteristics

As shown in Table 6.3, most, but not all family characteristics including family average levels of EE at baseline were significantly related to each other and to child behaviour at follow-up. Specifically, maternal ADHD symptoms at T1 were significantly positively associated with all T2 child problems, but T1 maternal depressive symptoms were only significantly correlated with T2 child emotional problems. Intercorrelations among T1 maternal problems have been discussed previously in Section 5.6.2 of chapter 5.

T2 child problems were significantly related to some, but not all sib-pair mean EE components. Small correlations were found between T2 DBP, T2 EP and T1 sib-pair IS, REL and WAR and between T2 ADHD and T1 sib-pair REL. The pattern pertaining to which T1 child problems were related to which specific T1 sib-pair EE components was similar in some respects. T1 DBP, EP and EDP were all significantly correlated with T1 sib-pair REL and WAR, but DBP and EDP were also significantly associated with CC and DBP with IS. Intercorrelations among T1 sib-pair EE ranged from small to moderate in the expected direction, although surprisingly T1 sib-pair CC was not significantly related to T1 sib-pair PC. As expected, EE components at T1 were for the most part significantly correlated with T1 sib-pair EE components. These correlations ranged from weak to very strong. Non-significant associations, however, were found between T1 sib-pair PC and T1 CC and between T1 sib-pair CC and T1 PC.

Table 6.2

Correlations among T1 and T2 Child Characteristics

	1	2	3	4	5	6	7	8	9	10	11	12
1. T1 IS	-											
2. T1 REL	.47***	-										
3. T1 WAR	.31**	.62***	-									
4. T1 PC	-.34***	-.53***	-.44***	-								
5. T1 CC	.38***	.55***	.44***	-.17	-							
6. T1 ADHD	.22*	.40***	.25*	-.42***	.44***	-						
7. T1 OPP/CP	.28**	.55***	.35***	-.46***	.47***	.80***	-					
8. T1 EP	.13	.26**	.30**	-.38***	.14	.47***	.51***	-				
9. T1 EDP	.15	.47***	.33**	-.42***	.43***	.70***	.82***	.60***	-			
10. T2 ADHD	.20*	.41***	.22*	-.42***	.36***	.85***	.73***	.49***	.63***	-		
11. T2 OPP/CP	.25**	.47***	.33**	-.43***	.38***	.66***	.83***	.46***	.70***	.77***	-	
12. T2 EP	.23*	.48***	.41***	-.46***	.35***	.63***	.66***	.56***	.66***	.75***	.75***	-
13. T2 EDP	.09	.35***	.21*	-.29**	.28**	.71***	.56***	.54***	.65***	.85***	.79***	.81***

Note. IS = initial statement; REL = relationship; WAR = warmth; PC = positive comments; CC = critical comments; ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; EDP = emotional dysregulation problems.

*p < .05, **p < .01, ***p < .001.

Table 6.3

Correlations among T1 Family Characteristics and T2 Child Characteristics

	1	2	3	4	5	6	7	8	9	10
1. T1 maternal ADHD	-									
2. T1 maternal DEP	.43***	-								
3. T1 sib-pair IS	.11	.30**	-							
4. T1 sib-pair REL	.09	.24*	.44***	-						
5. T1 sib-pair WAR	.16	.29**	.41***	.67***	-					
6. T1 sib-pair PC	-.08	-.19*	-.48***	-.42***	-.42***	-				
7. T1 sib-pair CC	.13	.28**	.40***	.32**	.46***	.14	-			
8. T2 ADHD	.32**	.12	.17	.20*	.10	-.15	.10	-		
9. T2 OPP/CP	.32**	.16	.20*	.27**	.20*	-.15	.15	.77***	-	
10. T2 EP	.35***	.21*	.22*	.28**	.30**	-.19	.10	.75***	.75***	-
11. T2 EDP	.33**	.13	.09	.16	.08	-.04	.08	.85***	.79***	.81***

Note. IS = initial statement; REL = relationship; WAR = warmth; PC = positive comments; CC = critical comments; ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; EDP = emotional dysregulation problems; DEP = depressive symptoms.

*p < .05. **p < .01. ***p < .001.

6.5.4 Child and Family Effects on Child Behaviour over Time

Child-Specific and Shared Family Variation in T2 Child Behaviour

The first multilevel model run was used to establish a baseline for the proportion of variance in T2 child problems explained by T1 child (within-family) and T1 family (between-family) effects conditioned on T1 ADHD diagnostic status and T1 child problems as shown in Figure 6.1. Column 1 of Tables A4.1 to A4.5 in Appendix 4 shows the child-specific and shared family variance values and their levels of significance in the initial model. There was both significant child-specific (within-family) and shared family (between-family) variation in T2 OPP/CP, EP and EDP. Whilst the proportion of T1 child and family effects on T2 child OPP/CP and EP was equally strong, the contribution of T1 child effects in predicting T2 EDP was stronger than family effects.

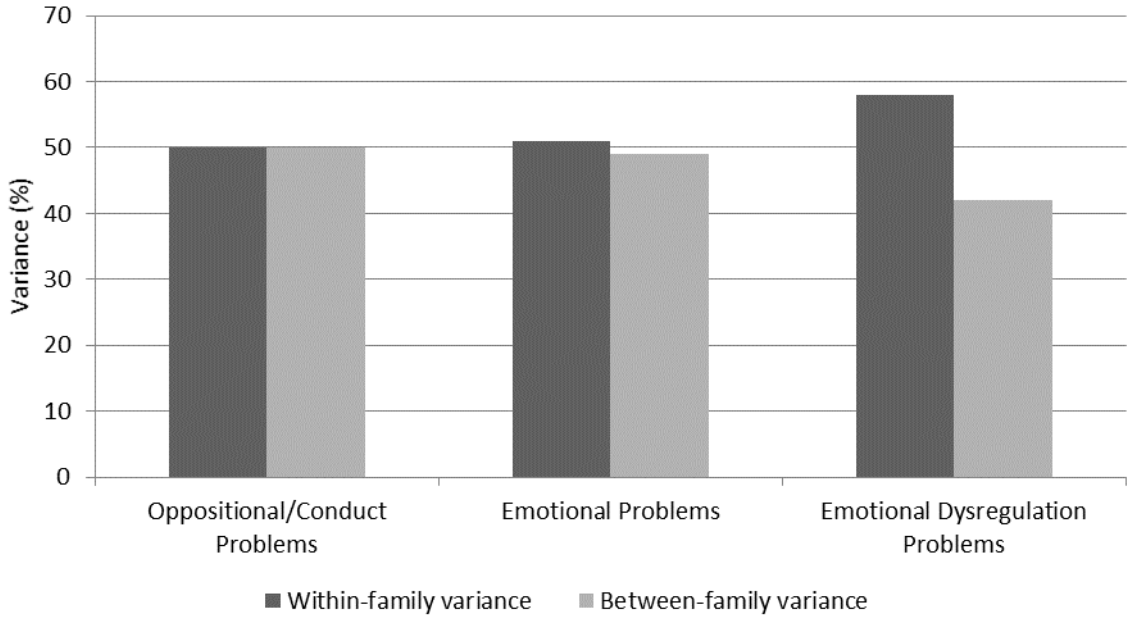

Figure 6.1 Proportion of T2 child problems explained by T1 child and family factors in the initial models conditioned on T1 ADHD diagnostic status and T1 child problems

T1 Child Characteristics Associated with T2 OPP/CP

Table 6.4 presents a summary of the specific T1 child characteristics that significantly predicted T2 child OPP/CP. These results are presented in full in Tables A4.1 to A4.5 in Appendix 4. In the first model, T1 ADHD diagnostic status and T1 child OPP/CP were entered. The results revealed significant and positive associations between T1 ADHD diagnostic status, T1 child OPP/CP and T2 OPP/CP. These associations remained in the final model after all child- and family-level predictors were entered. There were no

significant effects of gender or T1 age on T2 OPP/CP when entered in the model second and third respectively.

Did T1 Overall Maternal EE Predict T2 OPP/CP?

There was no effect of T1 overall maternal EE on T2 OPP/CP after controlling for T1 levels of OPP/CP. When multilevel models were run separately for T2 oppositional problems (OPP) and conduct problems (CP), the same pattern of results was revealed.

Are there Specific T1 EE Components that Predict T2 OPP/CP?

Further analysis to test whether there are specific EE components that drive T2 OPP/CP in which the individual EE components were entered in separate models instead of overall maternal EE revealed that T1 WAR was significantly and positively associated with T2 OPP/CP and significantly improved the fit of the model. The effect dropped to marginal significant when overall family levels of maternal EE were entered. There was no significant association between T1 CC and T2 OPP/CP, but when change in T1 CC was added to the model, T1 CC became significantly positively associated with T2 OPP/CP. There was no significant association between T1 CC and T2 OPP/CP, but when change in T1 CC was added to the model, T1 CC became significantly positively associated with T2 OPP/CP. T1 IS, REL nor PC accounted for child effects on T2 child OPP/CP.

Does Change in Overall Maternal EE Over Time Predict T2 OPP/CP?

Increase in overall EE over time from low at T1 to high at T2 was significantly and positively associated with T2 OPP/CP and significantly improved the fit of the model. This association remained in the final model after all child- and family-level predictors were entered (see Model 11, Table 6.4). When multilevel models were run separately for T2 oppositional problems (OPP) and conduct problems (CP), the same pattern of results was revealed with the exception that T1 ADHD diagnostic status was not significantly associated with T2 OPP.

Does Change in Maternal EE Components Over Time Predict T2 OPP/CP?

Increase in WAR was also significantly positively associated with T2 OPP/CP and this effect remained when all child- and family-level predictors were added. Contrary to what was expected, decrease in CC over time was significantly associated with T2 OPP/CP and this effect in addition to the positive association between T1 CC remained significant in the final model with all child- and family-level predictors entered in the model.

T1 Child Characteristics that Predicted T2 EP

As shown in Table A4.4 in Appendix 4, T1 ADHD diagnostic status was significantly and positively associated with T2 EP when entered in the model; this association remained in the final model after all child- and family-level variables were entered. Surprisingly, there was no association between T1 and T2 EP. Child effects were not explained by gender, T1 age, T1 maternal EE or change in maternal EE over time.

T1 Child Characteristics that Predicted T2 EDP

Table 6.6 and A4.5 in Appendix 4 displays T1 child characteristics that account for child effects on T2 EDP. T1 ADHD diagnostic status and T1 EDP were significantly and positively associated with T2 EDP; however there were no effects of gender, T1 age, maternal EE or change in maternal EE over time.

Table 6.4

T1 Child Characteristics that Predicted T1 Child Effects on T2 Child OPP/CP

		T2 OPP/CP	
Parameter	Overall EE	WAR	CC
T1 child ADHD status	√* (+)	X	X
T1 child OPP/CP	√*** (+)	√*** (+)	√
Child gender	X	X	X
T1 child age	X	X	X
T1 maternal EE	X	√† (+)	√** (+)
T1-T2 change in maternal EE	√** (+)	√* (+)	√*** (-)

Note. OPP/CP = oppositional and conduct; EE = expressed emotion; ADHD = attention-deficit hyperactivity disorder.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association

Table 6.5

T1 Family Characteristics that Predicted T1 Family Effects on T2 Child OPP/CP

	T2 OPP/CP		
Parameter	Overall EE	WAR	CC
All-boy sib-pair	X	X	X
All-girl sib-pair	X	X	X
T1 family maternal EE	√**	X	X
T1-T2 change in family maternal EE	X	x	X
Maternal ADHD	√*** (+)	√*** (+)	√*** (+)
Maternal DEP	X	X	X

Note. OPP/CP = oppositional and conduct; EE = expressed emotion; ADHD = attention-deficit/hyperactivity disorder; DEP = depressive symptoms.

[a] = T1 OPP/CP; [b] = T1 EP; [c] = T1 EDP.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association

T1 Family Characteristics that Predicted T2 Child Problems

All-boy sib-pair and all-girl sib-pair were the first and second family-level predictors to be entered after all child-level predictors. As summarised in Tables 6.5 and 6.7 and shown in more detail in Tables A4.1 to A4.5 in Appendix 4, the results revealed significant and negative associations between all-girl sib-pair and both T2 child EP and EDP, associations that remained after all child- and family-level predictors were entered in the models. T1 overall family levels of EE and change in overall family levels of EE over time were the fourth and fifth family-level predictors entered respectively. Significant and positive associations were found between T1 overall family levels of EE and both T2 OPP/CP and EP, but not EDP and addition of this family characteristic significantly improved the fit of the models. These associations remained when all child- and family-level predictors were entered in the models. There were no significant effects of change in overall family levels of EE and T2 child problems. Interestingly, T1 maternal ADHD symptoms, the fifth family-level predictor entered in the models, were significantly and positively associated with all T2 child problems and these associations remained in the final model. In addition, improvement in model fit was substantial. However, there were no significant effects of T1 maternal depressive symptoms on T2 child problems.

Table 6.6

T1 Child Characteristics that Predicted T1 Child Effects on T2 Child EP and EDP

Parameter	T2 EP	T2 EDP
T1 child ADHD status	√*** (+)	√** (+)
T1 child problems[a, b]	X	√** (+)
Child gender	X	X
T1 child age	X	X
T1 overall maternal EE	X	X
T1-T2 change in overall maternal EE	X	X

Note. EP = emotional problems; EDP = emotional dysregulation problems; ADHD = attention-deficit hyperactivity disorder; EE = expressed emotion.

[a] = T1 EP; [b] = T1 EDP.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association

Table 6.7

T1 Family Characteristics that Predicted T1 Family Effects on T2 Child EP and EDP

Parameter	T2 EP	T2 EDP
All-boy sib-pair	X	X
All-girl sib-pair	√** (-)	X
T1 overall family maternal EE	√*	X
T1-T2 change in overall family maternal EE	X	X
Maternal ADHD	√*** (+)	√*** (+)
Maternal DEP	X	X

Note. EP = emotional problems; EDP = emotional dysregulation problems; EE = expressed emotion; ADHD = attention-deficit hyperactivity disorder; DEP = depressive symptoms.

[a] = T1 EP; [b] = T1 EDP.

*p < .05. **p < .01. ***p < .001. † ≤ .10 (marginally significant).

+ = positive association; - = negative association

6.6 Discussion

The present study extended the longitudinal analysis of Study 3 and examined T1 child-specific (within-family) and shared family (between-family) effects on T2 child problems and the specific child and family characteristics that explain these effects. First, the study demonstrated continuity in child problems over time. As expected and in accordance with previous longitudinal studies (Biederman et al., 2006), in siblings with ADHD, inattentive symptoms significantly increased and hyperactive-impulsive symptoms decreased. However, conduct and oppositional problems did not significantly increase over five years in siblings with ADHD. Rather, internalising symptoms, including depression and anxiety increased in both ADHD and non-ADHD siblings.

Both child and family effects were implicated in predicting child OPP/CP over time. They contributed equally in predicting OPP/CP and EP, but for EDP child effects were stronger than family effects. The main aim of the study was to test the hypothesis that maternal EE and change in EE from T1 to T2 predicted child problems over time. Average levels of OPP/CP were stable in both ADHD and non-ADHD siblings, but did not increase over time. The study provided partial support for this hypothesis. T1 overall maternal EE did not predict T2 OPP/CP, but increase in overall maternal EE did, also controlling for T1 OPP/CP. Furthermore, in relation to the individual components of EE, T1 WAR and decrease in WAR seemed to predict increases in OPP/CP over time. There was also a role for CC, but in the opposite direction to that expected. In contrast, overall maternal EE or the specific components were not predictive of later child EP and EDP. Given that the overall prevalence of OPP/CP remained stable over time, it seems that increase in negative EE plays a role in maintaining levels of OPP/CP in children with ADHD over time. There was no effect of maternal EE on EP or EDP over time which begs the question: if not maternal EE, what is it that is driving an increase in these problems over time?

In accordance with previous cross-sectional studies, the longitudinal finding in the present study that increase in overall maternal EE over time predicted T2 OPP/CP supports Patterson's theory of coercive family process and more specifically Taylor's hypothesis that in the developing link between ODD/CD and EE it remains possible that during development ADHD provokes high EE from parents early in life which later on leads to ODD/CD. In the broader context of parent-child interaction, high levels of poor supervision and inconsistent discipline and low levels of warmth and involvement have been found to predict conduct problems (Frick et al., 1992; Haapasalo & Tremblay, 1994). Similarly, EE studies have demonstrated that high levels of CRIT (Hibbs et al., 1991; Peris & Hinshaw, 2003; Pyschogiou et al., 2007; Stubbe et al. 1993) and low levels of warmth

(Vostanis et al., 1994) are associated with conduct and oppositional problems in community and clinical samples of children.

More specifically in relation to the link between ADHD, ODD/CD and parental EE, in an Australian study of girls and boys with and without ADHD, family environment, as measured by the Global Family Environment Scale (GFES; Rey et al., 1997), was worst amongst children and adolescents with ADHD and comorbid conduct and oppositional problems (Rey et al., 2000). Furthermore, also in a clinical sample and more recently, the high levels of hostility and criticism shown by parents toward their children with ADHD were significantly influenced by children's oppositional problems, as measured using parent and teacher reports, to the extent that the significant relationship between ADHD and parental EE was removed when oppositional problems were entered as a covariate (Christiansen et al., 2010). A similar pattern of results was found by Sonuga-Barke et al. (2009) who reported significantly higher levels of conduct problems in children with ADHD whose parents were highly critical and lacked warmth. This study also found gene-environment interactions involving EE and polymorphisms of the serotonin and dopamine transporters suggesting that maternal expressions of warmth and hostility may act together with genetic factors in altering severity of ADHD.

Of the individual components of EE, the results suggest that maternal warmth plays a particularly important role in the later development of OPP/CP. In the present study low WAR predicted high levels of OPP/CP five years later. This finding highlights the potential protective role of parental warmth and is consistent with some of the above-mentioned studies (e.g., Christiansen et al., 2010; Sonuga-Barke et al., 2009; Vostanis et al., 1994). For instance, in Christiansen et al.'s (2010) study, a step-wise linear regression conducted with the individual domains of FMSS revealed that only the component low parental warmth was associated with oppositional problems in ADHD with high warmth predicting low scores of oppositional behaviour. Further evidence comes from longitudinal studies. Albeit not in a sample of children with ADHD, a behavioural genetics study by Caspi et al. (2004) showed that within MZ twin pairs, the twin receiving more maternal negativity and less warmth at age 5 had more antisocial problems at age 7 even after genetic influences on child behaviour problems were controlled. Positive aspects of parenting including praise, positive affect and physical positive, measured in a parent-child interaction task, have also been found to predict the future course of conduct problems in children diagnosed with ADHD between the ages of 4 and 7 years (Chronis et al., 2007). This has important clinical implications as it seems that positive aspects of parental attitudes towards children may serve to buffer children with ADHD from negative outcomes.

The number of critical comments mothers expressed about their children was also predictive of later OPP/CP; however, the results in relation to this EE component presented a somewhat ambiguous picture. Specifically, contrary to what was expected, decrease in critical comments predicted higher scores of OPP/CP at T2. Moreover, although there was no significant association between T1 CC and T2 OPP/CP when T1 CC was initially entered in the model, it became significant when change in CC was entered in the model and remained so in the final model when all child-level and family-level predictors were entered in the model. This emphasises that maternal criticism may be a risk factor which increases the vulnerability of children with ADHD for negative developmental outcomes which in this case is OPP/CP. This finding is also in line with previous studies with the exception of Christiansen et al.'s (2010) study. In their study high criticism did not predict high oppositional behaviour.

Interestingly, maternal EE was not predictive of emotional problems or emotional dysregulation problems at five-year follow-up. This suggests that the emotional climate of the family may only be a risk factor for externalising symptoms rather than internalising symptoms in children with ADHD. However, several studies have shown parental EE to be predictive of depression in children and adolescents (Asarnow et al., 2001). Although anxiety and emotional dysregulation problems increased in siblings with ADHD and depression increased in siblings without ADHD, it is unclear what is driving this increase in internalising symptoms.

In examining family effects, one of the most striking findings was that that overall family levels of T1 EE (i.e., EE toward both siblings combined) predicted OPP/CP and EP over time. This is important clinically as it highlights the importance of assessing parental EE toward not only the child with ADHD, but also other siblings in the family. ADHD symptoms independently predicted later child OPP/CP, EP and EDP. The familial basis of ADHD is well-recognised with studies reporting that children with the disorder are more likely to have a parent with ADHD than other children (Smalley et al., 2000). For instance, a family study of children with ADHD found that 15% to 20% of mothers and 20% to 30% or fathers have ADHD (Biederman et al., 1992). There are no studies to the best of the author's knowledge that have examined the effects of parent ADHD on long-term outcomes of children with ADHD and thus knowledge is limited. Studies have shown that parental ADHD is associated with increased negligence and intolerance (Arnold et al., 1997), less effective parental monitoring and consistent implementation of constructive management techniques (Evans , Vallano, & Pelham, 1994), lax discipline (Harvey et al., 2003; Murray & Johnston, 2006) and inconsistent discipline, lower levels of involvement and positive parenting (Chronis-Tuscano et al., 2008). The behaviour of the child with ADHD may make parents with ADHD especially vulnerable to heightened levels of stress,

poor parenting practices and increased EE which in turn exacerbates existing comorbid problems in children with ADHD or increases their risk of developing problems later on.

Limitations

There are a number of methodological limitations that should be taken into account, the majority of which have been described elsewhere in Section 4.7.3 of Chapter 4 given that the same data, design and methodology was used in the current study. Furthermore, a full review of the methodological limitations of all studies of the thesis is provided in the next chapter. One limitation specific to this study should be considered when interpreting the results. This study was longitudinal to the extent that measurements of EE and child and parent characteristics were obtained at two time points. If Taylor's (1999) hypothesis is to be tested more rigorously, future studies would need to measure EE and child and family characteristics at three or more time points, with the first wave of measurements taken at an early stage of the child's development, to disentangle the cause-effect relationship as to whether it is the difficult behaviour of the child with ADHD that drives high levels of parental EE and whether in turn high levels of parental EE increase the risk of the emergence of OPP/CP later on. Unfortunately, the first wave of measurement taken in this study was when children were between the ages of 5 and 17 years and to obtain measurements at a third time point was not possible to carry out within the timeframe of the thesis.

6.7 Conclusions

In conclusion, the results of this longitudinal study demonstrated that internalising rather than externalising symptoms increased over time in both siblings with ADHD and siblings without ADHD and that child behaviour is driven by both child and family effects. Increase in overall negative maternal EE, decrease in WAR and T1 WAR predicted child OPP/CP over five years. Family effects were mostly driven by mothers' ADHD symptoms. The findings suggest that although negative maternal EE does not appear to play a role in worsening aggressive and oppositional behaviour in children with ADHD, it still seems to influence its maintenance which may contribute to negative developmental trajectories, but low WAR may be a protective factor.

Chapter Seven

General Discussion and Implications of the Findings

7.1 Overview of Chapter 7

The final chapter of the thesis attempts to integrate the findings from all the studies presented. First, the cross-sectional and longitudinal findings relating to child and family effects on maternal EE are summarised followed by the longitudinal results pertaining to T1 child, in particular maternal EE, and family effects on T2 child problems. Second, the methodological limitations of the studies and ways in which they could be overcome in future studies are discussed. Third, implications of the findings for clinical practice are discussed. Lastly, possible directions for future research are presented.

7.2 Summary of the Main Findings

This thesis revealed a complex pattern for the role of child and family effects in predicting maternal EE and the role of maternal EE in driving the emergence and persistence of childhood problems. The key findings are summarised below.

Determinants of Parent EE

Child and Family Level Effects Both Predict Maternal EE

In Studies 1 and 2, both child and family effects were found to cross-sectionally predict all maternal EE components with the exception of T2 IS for which child effects only were found. However, whereas WAR was predominated by family effects, child effects were stronger in predicting the other EE components. The longitudinal analysis in Study 3 showed that both child and family effects were important in predicting maternal EE, but this wasn't the case for all components. Specifically, there were no significant longitudinal family effects on IS and REL. In contrast to the cross-sectional findings, the longitudinal analysis showed that family effects were not stronger than child effects; rather, the effects were near equal in their contribution.

Oppositional and Conduct Problems Make A Significant Contribution to Child Effects on Maternal EE

In terms of specific child characteristics, OPP/CP and EP, rather than ADHD diagnostic status seemed to cross-sectionally and longitudinally drive child effects found. In fact, ADHD diagnostic status only drove child effects on IS in the cross-sectional analyses of Studies 1 and 2. ADHD diagnostic status was cross-sectionally significantly positively related to IS, REL, WAR and CC and negatively with PC at both time points when entered in the initial models, but this association no longer remained significant when OPP/CP or EP were entered. This precise pattern was found longitudinally in Study 3 for PC and CC. However, in the initial longitudinal models T1 ADHD diagnostic status was not significantly related to T2 IS and was only marginally significantly associated with T2 REL and WAR.

OPP/CP significantly cross-sectionally and longitudinally predicted CC and cross-sectionally predicted REL at both time points, PC at T1 and WAR at T2 controlling for other child and family characteristics. In relation to PC at T2, although child OPP/CP did not cross-sectionally significantly predict child effects on this component, addition of this characteristic to the model removed the significant effect of ADHD diagnostic status. Numerous previous studies have established that parental EE is higher toward children with conduct problems compared to children without conduct problems (Hibbs et al., 1991; Peris & Baker, 2000; Stubbe et al., 1993; Vostanis & Nicholls, 1995). Research has also shown a link between parental EE, ADHD and comorbid oppositional and conduct problems (Christiansen et al., 2010; Sonuga-Barke et al., 2009).

In Studies 1 and 2, EP cross-sectionally drove child effects on PC at both time points with mothers expressing fewer PC toward their children the more EP they had. This was not the case in the longitudinal analysis of Study 3; neither EP nor OPP/CP longitudinally drove child effects on PC. This pattern was also found for REL. It was only gender that explained longitudinal child effects on REL with more negative relationships between mothers and boys. Interestingly, EP longitudinally predicted IS and WAR. Results from earlier studies demonstrate associations between parental EE and childhood depression (Asarnow et al., 1993; Asarnow et al., 2001; Bolton et al., 2003; McCleary & Sanford, 2002; Silk et al., 2009), anxiety (Kwon et al., 2006; Stubbe et al., 1993), emotional problems measured more broadly (Vostanis et al., 1994; Vostanis & Nicholls, 1995) and self-injurious behaviour (Wedig & Nock, 2007). Daley et al. (2003) found warmth to predict child emotional adjustment in a sample of pre-school children. Indeed, in a study examining whether parental EE is a specific risk factor for depression or a nonspecific correlate of child psychopathology, FMSS-EE showed some specificity as a risk factor for depression as rates of critical EE were significantly higher among mothers of children and

adolescents with depression as compared to mothers of nondepressed children and adolescents with ADHD or mothers of controls (Asarnow et al., 2001). These findings suggest that EP may be more important in predicting maternal EE than ADHD. However, in the current thesis EP was associated with lack of positive comments, but not criticism.

Overall, the finding that EP and OPP/CP were more important than ADHD is consistent with the work of Psychogiou et al. (2007) who found that the significant association between mothers' criticism and elevated ADHD in school-aged boys became non-significant when child emotional symptoms and conduct problems were entered in the multiple regression model. The findings are, however, incongruent with a less recent longitudinal study in school-aged girls that showed linkages with EE were stronger for ADHD than for aggression and withstood control of comorbid aggression, the converse was not true (Peris & Hinshaw, 2003). Whereas the pattern of child effects pertaining to CC was clear cut and consistent across the cross-sectional and longitudinal analyses (i.e., child effects were driven both cross-sectionally and longitudinally by OPP/CP) this was not the case for the other components. There are two differences that stand out. First, cross-sectionally it seemed that OPP/CP was the most important child characteristic predicting child effects, but longitudinally it was EP driving these effects. Second, the EE components, namely IS and WAR, that were not predicted cross-sectionally by EP or OPP/CP were the two components longitudinally predicted by EP.

Family Levels of Child OPP/CP Predict Family Effects on Maternal EE

The pattern with regard to family effects was more complex. However, overall family levels of child OPP/CP seemed to be the most important characteristic in explaining family effects. Specifically, family effects on CC were driven by this family characteristic in both the cross-sectional models of Studies 1 and 2 and the longitudinal model of Study 3. In addition, sibling pair average levels of OPP/CP cross-sectionally explained family effects on REL and WAR at T1, but not at T2 and longitudinally predicted WAR and PC. Parental psychopathology was important too. Maternal depressive symptoms were found to significantly positively be associated with REL and WAR at T1, but not at T2 in the cross-sectional models and also WAR in the longitudinal models. Surprisingly, maternal ADHD symptoms cross-sectionally drove family effects on T2 WAR, but no other components. Furthermore, there were no longitudinal associations between maternal ADHD symptoms and maternal EE. As aforementioned, although average levels of sibling pair OPP/CP explained family effects on REL and WAR at T1 in the cross-sectional analyses, this was not the case at T2. Instead, family effects for these components at T2 were driven by overall family levels of child EP. In the cross-sectional analyses, sibling pair average EP was marginally significantly associated with REL at T1 and T2, and WAR at

T1 and significantly related to PC at T1, but for these components these links disappeared when sibling pair mean OPP/CP was entered in the model.

While Overall Levels of Maternal EE Components are Stable over Time, EE is not Fixed by T1 and Continues to be Affected by Child and Family Effects during a Five year Follow-up

Study 3 also examined the continuity of EE over a five-year period and in accordance with other studies that have examined the stability of EE over periods ranging from 5 weeks to 2 years (Greenberg et al., 2006; McGuire & Earls, 1994; Peris & Baker, 2000), found little change from T1 to T2.

Increase in Negative Maternal EE between T1 and T2 Predicts Changes in Child OPP/CP Levels over Time

The thesis also examined whether maternal EE predicts child problems over time. Study 4 showed that both child and family effects longitudinally predicted child problems. Child and family effects contributed equally for OPP/CP, near equally for EP with child effects slightly stronger than family effects and for EDP child effects were stronger. In terms of the specific child effects, consistent with Taylor's (1999) hypothesis, increase in overall maternal EE over time significantly predicted child OPP/CP at T2. Exploration of the specific EE components that might drive this effect revealed that T1 WAR and decrease in WAR predicted T2 OPP/CP. This is consistent with previous findings showing an association between ADHD, oppositional problems and parental EE with high levels of WAR predicting low scores of oppositional behaviour (Christiansen et al., 2010). This finding provides further evidence for the notion that WAR may act as a protective factor by increasing positive outcomes in children with ADHD. T1 overall family levels of EE also predicted T2 OPP/CP and EP, suggesting that from a clinical perspective examining EE toward siblings of children with ADHD is important too. Interestingly, maternal ADHD also seemed to drive family effects on all types of child problems over time. It is possible that mothers of children with ADHD may be particularly vulnerable to characteristics of the child (e.g., ADHD symptoms) which in turn leads to high levels of EE and as a consequence the emergence of further problems in the child later on.

Parental ADHD is a Major Risk Factor for Poor Outcomes

The second longitudinal analysis demonstrated that parental ADHD seems to be a major risk factor for poor outcomes and appeared to explain the majority of family effects on the emergence of problems.

7.3 Implications for Models of EE and Development

By and large, the findings of this thesis are most consistent with the child and parent reciprocal effects model. Both child and family effects were implicated in predicting maternal EE in both the cross-sectional and longitudinal analyses. Further, increase in EE predicted child OPP/CP over a period of five years. Child OPP/CP and EP rather than ADHD were also found to predict negative maternal EE. In line with Taylor's (1999), it is possible that high EE is initially provoked in the early years of a child's life by their ADHD symptoms, which in turn increases high parental EE and consequently the development of conduct and oppositional problems. Due to their only being two time points in the data for this thesis and that children were not assessed from a young age, it is difficult to ascertain whether ADHD symptoms provoked high maternal EE at an early age which consequently increased OPP/CP and that in this sample the relationship between OPP/CP and maternal EE is a reflection of these processes having already occurred and that as OPP/CP continues to account for child effects on maternal EE, maternal EE persists in maintaining OPP/CP.

It might also be argued that a child effects model is more plausible given the small contribution of family effects found. This is consistent with previous findings indicating that maternal EE levels decrease when children's symptoms of ADHD reduced when on stimulant medication and that when child and maternal psychopathology were examined simultaneously, only child characteristics predicted maternal EE (Psychogiou et al., 2007). Furthermore, in the broader context of the relationship between parental EE and child psychopathology, evidence points toward a child effects model. For example, Vostanis & Nicholls (1995) demonstrated that only ratings of child behaviour at the first assessment and not initial EE levels strongly predicted symptomatic changes over a period of nine months. Hale et al. (2011) also found that the course of adolescent internalising and externalising symptoms affects maternal EE, and not the other way around. The results of the current thesis suggest that OPP/CP and EP rather than ADHD provoke high maternal EE.

Using the current design we cannot rule out the possibility of passive gene environment correlations as explanations of the family effects on the emergence of behaviour problems over time and the child effects on changes in maternal EE. With regard to the former, it is possible that parental EE and behavioural and emotional problems emerging in adolescence are controlled by shared genes, genes which may also be implicated in the relationship between T1 parent ADHD and the emergence of behaviour problems. With regard to the latter, childhood behaviour problems and emerging parents

EE might be controlled by the same genes. In order to tease these issues out, a more genetically informative design is required (Harold et al., 2013).

7.4 Limitations

The findings of this thesis have provided some important insights. However, there are numerous methodological limitations that need to be borne in mind when interpreting the findings of this thesis. These are each described in turn. First, the use of a clinic-referred and predominantly White sample limits the generalizability of the findings. Second, the studies of the current thesis join a long line of other studies exploring the relationship between parental EE and child psychopathology (e.g., Asarnow et al., 1993; Baker et al., 2000; Hale et al., 2011; Peris & Hinshaw, 2003; Psychogiou et al., 2007; Schwartz et al., 1990; Stubbe et al., 1993; Vostanis et al., 1994) in that they have used maternal EE data only. Paternal EE data were available at T2 only and for only 16 fathers and thus not included in the analyses. In dual-parent households, maternal EE alone may not reflect *parental* EE as it does not take into account father's EE and the similarities and differences in maternal and paternal EE. Maternal and paternal EE may be associated with each other, but determinants of EE may differ for mothers and fathers. For instance, with respect to family characteristics, Hibbs et al. (1991) found that in the association between parental psychopathology and EE whereas for fathers no specific psychiatric diagnosis was predictive of high EE status for mothers high EE status was more common in mothers with a mood disorder. It is possible that child characteristics may be related differently to maternal and paternal EE too.

Following on from this, the inclusion of maternal EE only may also limit the ability to capture EE as it was intended, that is, as a broader index of the family environment. Families are made up of multiple subsystems, not just one member. In the adult EE literature, a number of authors have highlighted the importance of assessing EE attitudes in all family members. Indeed, child and family factors may have different patterns of influence on children's emotional attitudes toward their parents or indeed their siblings. Marshall et al. (1990) found a considerable degree of reciprocity between parental and child EE and suggested that children with ADHD and parents mirror the affective attitudes held by one another. Yelland and Daley (2009) found that in a community sample of boys aged 7 to 11 years and their siblings a modified version of the EE measure was a valid and reliable measure of child EE (toward child in sibling relationships). Furthermore, Bullock et al. (2002) found that sibling CRIT was associated with younger brother concurrent and future disruptive behaviour, substance use and increased criminal activity. This is an especially important finding in light of the findings of this thesis as it may not only be

maternal EE that increases the risk of children with ADHD later on developing oppositional and/or conduct problems. Future studies, especially those exploring this particular issue, would benefit from examining both parents' EE toward their children, but also children's EE toward their parents and siblings' EE toward each other. It may also be important to examine parent-to-parent EE.

Third, maternal EE was assessed with a modified version of the FMSS and not the CFI. The CFI is considered the most comprehensive assessment of EE. Calam and Peters (2006) argued knowledge of agreement between CFI-EE and FMSS-EE ratings is limited, especially in child populations. They compared EE ratings made using a modified version of the CFI made age-appropriate (and approved by one of the originators of the method) with ratings made using the FMSS in a sample of children referred for behavioural difficulties. Levels of FMSS-EE were exceptionally high and therefore compared to previous studies that found either good levels of agreement (Magaña et al., 1986) or a trend towards underreporting in the FMSS (Malla et al., 1991; Van Humbeeck et al., 2002), the FMSS had a lower rather than a higher threshold for EE. Concordance between the modified version of the FMSS used in the current thesis and the CFI has not yet been undertaken. However, this version of the FMSS used in the current thesis offered the advantage that contrary to the CFI and original FMSS it has been designed especially for use with children it has been validated in child populations. Furthermore, given that it takes considerably less time to collect and code the data using the FMSS, it was more practical to use this measure, particularly in terms of inconveniencing families as little as possible. Use of the CFI would have been beyond the scope of the thesis.

Fourth, the longitudinal design of the study, in particular that the first and second waves of data collection were five years apart, meant that EE ratings were made by different raters at the different time points. In addition, at both time points, the rater was not blind to diagnosis of the child. In future studies, the rater should remain blind to diagnosis.

Fifth, a particularly important limitation that must be acknowledged across the four studies was that all data obtained about child and parent characteristics were based on maternal reports alone. Mothers completed the CPRS-R: L and SDQ at T1 and the Conner's CBRS at T2 as reports of their children's behavioural and emotional problems and the CSS-SR at T1 and T2, the GHQ-12 at T1 and the HADS at T2 as measures of their own ADHD and depressive symptoms. Reliance solely on maternal reports both of their own and their children's behaviour increases the likelihood of inflated correlations between the child and parent measures. Second, mothers may have over-reported their own and their child's symptoms. Mother's responses might have been influenced by systematic personal biases associated with factors such as expectations and attributions about child

difficult behaviour or other factors such as parental depression (Eddy, Dishion, & Stoolmiller, 1998). In their overview of methodological and conceptual issues underlying the assessment of change during psychotherapy with children and families, Eddy et al. 1998 draw on the work of Fergusson et al. (1993) who hypothesised that maternal depression biased mothers toward perceiving the world as hostile and this inflated the negativity of ratings on topics such as child antisocial behaviour. It is possible that mothers with symptoms of psychopathology were biased and negative, and they tended to report more problems in themselves and their children (Richters, 1992). Moreover, given the longitudinal design of two studies in this thesis, the use of single-agent (i.e., parent only) measures of outcomes may have presented a confound in terms of change. It is unclear whether the change is due to the property of the measure or to actual psychological change. Future studies would benefit from the inclusion of measurements of child and parent behaviour from multiple informants such as teachers with respect to child behaviour and spouses/partners or close friends or relatives in relation to mothers' behaviour.

At T1 DSM-IV diagnoses of ADHD Combined Type were made using the PACS. However, no such assessment was repeated at T2 of the study.

Teacher ratings of child behaviour on the SDQ and CRS-R: L at T1 and the Conner's CBRS at T2 (i.e., the subscales used in the current thesis to measure child behaviour) were obtained at both time points. However, there was a considerable amount of missing data at T2 which prohibited its utility in the T2 cross-sectional analysis and the longitudinal analysis of Study 4. For the purposes of the current thesis teacher ratings were therefore not used. Replication of the T1 cross-sectional analysis and longitudinal analysis of Study 3 with teacher ratings of child behaviour may be explored in future studies conducted beyond the present thesis. With regard to missing teacher data at T1, for probands, 17% of the data on the Oppositional and Anxious/Shy subscales of the CRS-R: L and the Conduct Problems subscale of the SDQ and 15% on the Emotional Symptoms subscale of the SDQ was missing. For siblings, 19% of the data on both used subscales of the SDQ and 21% on the three used subscales of the CRS-R: L was missing. In terms of teacher ratings on the Conner's CBRS at T2, for probands, 68% of the data on the DSM-IV Conduct Disorder subscale, 65% on the DSM-IV Major Depressive Disorder subscale and 62% on the DSM-IV Oppositional Defiant Disorder, DSM-IV Manic Episode and DSM-IV Generalised Anxiety Disorder subscales was missing. For siblings, 61% of the data was missing on all subscales except for the DSM-IV Conduct Disorder subscale for which 62% was missing.

Nonresponse, despite the mailing of reminders, from teachers played a part in the substantial amount of missing data at T2 and of course applies at T1 too. However, key to this problem was that the majority of children at follow-up were in secondary school, college or university, in employment or unemployed. Indeed, the maximum age of children

at T2 was 22 and the mean age of children selected for the T2 cross-sectional and longitudinal analysis of Study 4 was 16.47 years and 16.31 years respectively. In primary school students are assigned to one teacher for all subjects which affords teachers the opportunity to get to know their students well. In secondary school, college and university students have different teachers for different subjects and the degree to how well teachers know their students varies, particularly in college and university. Teachers commented that they felt unable to answer particular questions either because they did not know the young person well enough or because they thought the question was not relevant. Use of the teacher version of the Conner's CBRS was not relevant for adolescents and young adults at T2 who were in employment or unemployed. An alternative approach to obtaining alternative informant ratings for adolescents' and young adults' behaviour would be to ask another relative (other than the parent e.g., grandparent), close friend or girlfriend/boyfriend of the individual. In previous studies, researchers have approached employers to provide information about the behaviour of participants. This, however, may raise ethical concerns and participants may not feel comfortable with this approach. Self-report measures of child and behavioural problems were obtained at T2, but were not used in the current thesis given this data was only available at one time point.

At T1 ratings of mothers ADHD symptoms were obtained from the husbands or partners of 59 out of 72 mothers selected in Study 1. The collection of such data at T2 was not practical due to the time constraints of the full research assessment for each family. Daley, Fearon, Birchwood and Hoeger found very high response rates and high correlations between the CSS-SR and the version of the CSS completed by a friend. In conclusion, while it would be ideal to have an objective informant of adults' ADHD symptomatology as it was mentioned elsewhere in the thesis adults seem able to give a relatively accurate account of their ADHD symptoms (Murphy & Schachar, 2000) and most importantly their account of their ADHD symptoms appears not to be influenced by having a child with ADHD (Faraone et al., 1997).

Sixth, the age range in the sample was fairly large (5-17 years and 9-22 years at T1 and T2 respectively) with a substantial proportion (43%) of children at T2 no longer children, but young adults. Older age significantly predicted maternal low WAR in Study 1; however, age was not associated with levels of maternal WAR in Study 2 or the other EE components in Studies 1, 2 or 3 and T1 child age did not predict T2 child problems in the longitudinal analysis of Study 4. A modified version of the Conner's CBRS – P which was made age-appropriate was used to measure child behavioural and emotional problems in young people aged 18 or older. This was approved by the publisher. However, this modified version has not been validated in young adults and T-scores were based on the age and gender norms for an 18-year old. The opportunity to divide the sample into

children and adults and thus obtain separate measures of behaviour for these two groups and run separate analyses for the two groups was limited by the sample size. Therefore, the Conner's CBRS was selected as the measure that could best capture behaviour across this wide age range and that could measure a broad range of child psychopathology based on the DSM-IV criteria. Consequently different measures were used at T1 and T2 to measure child behaviour. For example, with respect to OPP/CP, at T1 they were measured with the Conduct Problems scale of the SDQ and the Oppositional scale of the CPRS-R: L and at T2 with the DSM-IV Oppositional Defiant Disorder and Conduct Problems scales of the Conner's CBRS. The SDQ was not administered to parents at T2 given that it was designed for use in children up to 16 years of age and because of the large proportion of children above that age at T2. The fact that both the Conner's CBRS and the CPRS-R: L were developed by the same author and in accordance with each other made the scales between the two questionnaires more comparable. However, SDQ, CPRS-R: L and Conner's CBRS scales used in this thesis were moderately to highly correlated with each other.

A proportion of young people aged 18 or older had moved out of home and were therefore no longer living with their mothers. In addition, the degree of contact between young people who had moved away from home and their mothers varied. Given that EE is intended as a measure of the emotional climate of the home and is assumed to reflect the way that they interact with their child in everyday life, it may have been useful to include whether or not the young person was living at home as a child demographic characteristic in the analyses of Studies 1, 2 and 3 to examine whether it had an effect on levels of maternal EE.

Seventh, unlike many studies of ADHD that include samples of boys only, the sample in this thesis consisted of both males and females. However, whilst for siblings without ADHD, the number of males and females was near equal, for siblings with ADHD there were more than four times as many boys as girls. This is in accordance with prevalence rates of males to females with ADHD in populations at large (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Ford, Goodman, & Meitzer, 2003), but male gender was significantly associated with REL in the longitudinal analysis in Study 3. Nevertheless, in the remaining multilevel model analyses in Studies 1, 2 and 3, gender did not affect levels of the individual components of EE or levels of child problems in the longitudinal analysis of Study 4.

Eighth, Studies 3 and 4 addressed the methodological limitation of the cross-sectional design of Studies 1 and 2, and indeed of many previous studies of parental EE in child populations, by employing a longitudinal design. However, maternal EE and measures of child and family characteristics were measured at only two time points. This presented a number of issues specifically for Study 4. First, development as a transient or continuous

process can be only crudely be indexed by data from two assessment points as information is available only at the beginning and the end of some interval, in this case 5 years. This allows only a linear trend to be estimated. At least three time points are required to estimate the precision of a trend. Second, increase in the precision of statistical estimates of change parameters can be quite dramatic when multiple data assessments are used (Willet, 1989). Third, effect size and power is improved with multiple wave designs (Kraemer, 1991; Kraemer & Thieman, 1987). However, collection of data at multiple time points was beyond the scope the thesis. It would be better in future studies examining Taylor's (1999) hypothesis to have three or more waves of assessment with the first conducted early in childhood.

Ninth, sibling-pair data (i.e., two-children-per-family) meant that there were not enough degrees of freedom to perform cross-level interactions in the multilevel modelling analyses conducted in this thesis. If it had have been possible to conduct cross-level interactions, it would have afforded the opportunity to explore, for example, whether family characteristics such as maternal depressive symptoms show a greater EE discrepancy as a function of the child's ADHD status or indeed the level of oppositional and conduct problems they have.

Tenth, it was noted in Chapter 2 that few studies have shown associations between parental EE and family demographic factors. Family demographic factors such as SES, maternal age, single versus dual parent families were not examined in the first three studies of the thesis to establish whether this holds true in the sample used in this thesis and supports these previous findings.

Lastly, as previously mentioned in Chapter 4, the possibility of evocative rGEs cannot be ruled out. The results seem to fit with the notion of evocative rGEs that genetically influenced child characteristics (in this case child ADHD or oppositional/conduct problems) may have evoked negativity from parents (Ge et al., 1996), but this could not be tested with a sib-pair design. Instead, an adoptee or twin design would be needed.

7.4 Clinical Implications of the Findings

The complex pattern of results found in this thesis demonstrates that further work is still needed to advance understanding of the processes underlying the relationship between parental EE and ADHD, but there are several findings of this thesis that are of particular clinical importance. First, both child *and* family characteristics were found to influence maternal EE. Second, it appeared that child effects were explained by comorbid child problems, particularly OPP/CP, but also EP, rather than ADHD per se. Third, overall

family levels of OPP/CP most reliably accounted for family effects, but maternal depressive symptoms and to lesser extent maternal ADHD symptoms were important too. Fourth, increases in EE over time seemed to drive high levels of child OPP/CP at follow-up. Furthermore, two EE components in particular played an important role in the development of OPP/CP later on. Low WAR and greater CC predicted high OPP/CP scores.

In light of these findings it may be useful for routine screening of high levels of EE in the parents of children with ADHD upon initial assessment when families are referred to child and adolescent mental health services or from a preventative perspective even earlier when children are first identified, for example, by family support workers or health visitors, as manifesting behavioural problems. Given that overall family levels of OPP/CP were found to influence maternal EE components both concurrently and longitudinally, it may be important to measure parental EE toward the child with ADHD and their siblings or at least toward the sibling or siblings also manifesting behavioural problems, in particular aggressive-type behaviour. In screening parents for high levels of EE, it may also be pertinent to identify if there are specific aspects of EE that are particularly high in parents, for example, a parent may be rated as low in warmth, but may not have a high score on other components.

Upon identification of high EE in parents of children with ADHD, interventions that aim to reduce parental EE may be an important part of treatment plans for families of children with ADHD. A number of intervention trials with the families of adults with psychiatric disorder that aimed to lessen EE in the home have proven effective in reducing relapse across a number of different disorders (Falloon et al., 1985; Fristad et al., 1998; Goldstein and Miklowitz, 1995; Hogarty et al., 1991; Holder & Anderson, 1990; Kim & Miklowitz, 2004; Leff et al., 1990; Pharoah, Mari, Rathbone, & Wang, 2012). Specifically, such interventions may benefit from focusing on reducing negative aspects of EE such as criticism which may lessen the ADHD child's vulnerability for negative developmental outcomes, especially the exacerbation or later emergence of OPP/CP, and on increasing positive facets of EE such as warmth to promote positive adjustment in the ADHD child (i.e., greater resilience to OPP/CP later on). The findings of this thesis have demonstrated that maternal criticism, indexed in this thesis by the number of critical comments expressed by mothers about their children, may be a particularly important aspect of EE to target in prevention and intervention given that increase in CC over time predicted high levels of child OPP and CP at T2.

That child effects were stronger and largely accounted for by OPP/CP and EP, but also that family effects were consistently driven by overall family levels of child OPP/CP highlights that the therapeutic approach as well as focusing on reducing EE may also

require the delivery of disruptive/aggressive behaviour management techniques. The finding that family effects were also driven by maternal ADHD symptoms, and to a lesser extent by ADHD symptoms, raises the issue that prior to the provision of an EE-reducing intervention, parents may also need to be screened for mental health problems. In cases where parents are found to be experiencing mental health difficulties, treatment of these problems may need to be addressed first in order for the intervention to be effective or indeed the intervention may need to be tailored to account for these difficulties and the impact they are having. However, the link between parental psychiatric illness and EE still remains unclear. For example, is EE a reflection of parental psychopathology or do they both independently predict child psychopathology? In terms of prevention, generic parenting programmes for parents of young children at risk of behavioural and emotional problems may benefit from the inclusion of an element that specifically addresses EE in the home of the child.

7.5 Future Directions

As noted in the previous section, the findings of this thesis have added to the literature on the relationship between parental EE and ADHD. They have furthered understanding of the cause-effect relationship as to whether it is child or family effects that contribute to high levels of maternal EE toward children with ADHD and whether maternal EE increases the risk of the development of problems, especially OPP/CP, in children with ADHD over time. In examining child and family effects the findings also provided further clarification of the specificity of parental EE to ADHD and which specific family characteristics explain family effects on maternal EE.

The findings of this thesis need replicating across different samples and different methodologies. The sample in this thesis like the majority of other EE studies in child populations was predominantly White. Further studies are also required to examine whether the results found in thesis are replicated across samples of different ethnic background as cultural factors may play an important role in influencing parental EE toward their children.

In this thesis, mothers reported on their children's and their own psychopathology. Future studies using the same within-family and multilevel modelling design are needed in which teacher and parent reports of children's behaviour and proxy reports (e.g., partner/spouse/close relative or friend) of parents' behaviour are obtained to examine whether use of alternative reports of child and parent behaviour yield similar results. Older child and adolescent self-reported ratings of their own behaviour may also be useful. The use of multi-reports of children's behaviour is especially important with regard to

determining whether the results that OPP/CP and EP rather than ADHD drive maternal EE would be replicated and whether the same family characteristics would be implicated in predicting family effects.

Future research is also required in which paternal EE as well as maternal EE is examined to determine whether the same or different child and family characteristics are implicated in predicting fathers' EE. The determinants, for example in mothers may be more complex due to it being more usual for them to have greater daily and intense contact with their children. Increased interaction may allow particular characteristics of mothers, for example their mental health, and/or the child's actual behaviour to have a greater role in shaping their reaction to them. As discussed in the limitations, future work should measure child EE toward parents and their siblings also to enable *family* EE and its relationship with ADHD to be examined which could provide a rationale basis for assessment and interventions for high-EE children as well as parents within the family system in clinical practice. In a previous study of depressed mothers, in which EE directed toward the spouse, self and child was assessed, toddlers were more likely to be exposed to criticism between parents as well as that from their mothers (Rogosch et al., 2004).

It is assumed that the way that parents talk about their children reflects the way that they interact with their children. The relationship between parent-child interaction measures and EE scores has been explored with adults (Miklowitz et al., 1984). In child populations, two previous studies demonstrated that parental EE ratings corresponded with ratings of parent-child interaction as measured using observations of parents and children during structured tasks (Marshall et al., 1990; McCarty et al., 1994). Further studies are required in which observational measures of parent-child interaction are included to determine whether EE classifications reflect the actual home environment. This is particularly important for clinical intervention. For instance, if the way parents talk about their child (i.e., EE ratings) does not correspond with the actual behaviour of the parent toward their child, interventions aimed at reducing parental EE may not be effective in improving actual parent-child interaction.

Another issue that requires further exploration involves the extent to which parental EE status is influenced by other child and family characteristics. First, in relation to child characteristics, this thesis examined whether diagnosis of ADHD explained child effects on maternal EE. However, the effect of severity and chronicity of ADHD diagnosis on parental EE may also be important and worthy of examination in future studies. It was noted in Chapter 1 that as well ADHD being a highly comorbid disorder, children with ADHD also experience problems in a range of functional domains. In a large sample of adolescents of mothers with varying histories of depression or who were not depressed, EE criticism and degree of maternal depression were both independently associated with

adolescent externalising symptoms and functional impairment experienced in close friendships, social life, academic performance and school behaviour (Nelson et al., 2003). It is of particular clinical importance that future studies examine whether functional impairment accounts for child effects on parental EE toward children with ADHD. The research literature underscores that measures of parental affection are particularly sensitive to genetic characteristics of the child. For instance, the results of a recent study showed that maternal warmth and hostility may act together with genetic factors in altering severity of ADHD. Future work is needed to examine the role gene-environment interactions involving EE and genes in children with ADHD.

It was mentioned in the limitations that family demographic characteristics other than sib-pair gender were not examined in this thesis. Although in general, it has been found that such family characteristics are not related to parental EE or at least that the relationship between parental EE and child psychopathology withstands control of these factors, future studies should still examine whether the results of this thesis are replicated controlling for other family factors such as dual-parent versus single parent status, step-parent household, SES, maternal age and number of siblings in the family. Another family characteristic that should be explored in future research is the personality of parents. Findings in relation to EE in adult psychiatric patients have found that personality is an important factor contributing to high-EE, more so than psychopathology of the relative or parent. High-EE relatives tend to have a more internal locus of control for their own behaviour than low-EE relatives and therefore prefer to take control in managing their own life problems and difficulties (Hooley, 1998). In addition, high-EE relatives have been shown to be more conventional in their attitudes and behaviour, to feel less capable and optimistic about their lives, to be less flexible, tolerant and lower in empathy and achievement by independence compared to low-EE relatives (Hooley & Hiller, 2000). One other aspect of parental psychopathology that was not examined in this thesis that may be of particular important to explore in future studies is parent antisocial characteristics, particularly in fathers. Fathers of children with ADHD who themselves have ADHD have been found to have high levels of conduct problems and antisocial personality disorder (Rhee & Waldman, 2002). Furthermore, research has shown that paternal antisocial behaviours, alcoholism and substance use are associated with externalising behaviour problems in children, particularly among clinical samples (Phares, 1996; Phares & Compas, 1992). Returning to the point that different child and family characteristics may be implicated in predicting maternal and paternal EE, fathers' antisocial characteristics is a potential parent-related factor that might be particularly important in predicting high EE in fathers in particular. In a parenting study, Psychogiou (2004) found that fathers' antisocial

characteristics were negatively associated with involvement and mothers' antisocial characteristics were positively associated with inconsistent discipline.

Continuing with discussion of family characteristics, further work is needed to determine whether parental EE toward children with ADHD has independent explanatory power beyond measures of family stress and adjustment and parental psychopathology. Whilst fewer in number than studies showing that maternal EE has predictive validity after accounting for other family characteristics such as maternal stress and psychopathology, some studies have shown that maternal stress was a better predictor of child externalising behaviours (Baker et al., 1998). From a clinical perspective, if other measures of the family environment, such as parenting stress, are found to have as much predictive power or more predictive power than EE in their relationship with child ADHD, these other measures which often can be more quickly completed and scored may have greater utility for service providers to seek understanding of children with ADHD.

7.6 Concluding Remarks

In summary, the results of this thesis revealed a complex picture with both child and family effects implicated as being important in determining maternal EE *and* child behaviour in families of children with ADHD. There are a number of findings in particular that stand out. First, for the majority of EE components (except WAR), both the cross-sectional and longitudinal contribution of child-specific (within-family) effects were stronger than between-family effects. Second, comorbid child OPP/CP (and to a lesser extent EP) seemed more important than ADHD in driving the child effects found to predict maternal EE. Third, maternal mental health, particularly maternal depressive symptoms, and overall family levels of child OPP/CP both played an important role in cross-sectionally and longitudinally explaining general family effects found. Fourth, increase in overall maternal EE over time seemed to predict later child OPP/CP. Specifically, low WAR at T1 was associated with higher OPP/CP scores at follow-up. Therefore, high maternal WAR may serve as a protective factor in preventing or reducing the escalation or emergence of OPP/CP over time in children with ADHD. This finding in particular has important implications for clinical practice.

Appendix 1: Cross-Sectional Multilevel Models Examining Child and Family Effects on Maternal EE Components (Chapter 4, Section 4.5.4)

List of Tables

Table A1.1

Multilevel Models Examining Child and Family Effects on IS at T1

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.75 (.06)	1.82 (.08)	1.82 (.08)	1.82 (.07)
Child level				
Child ADHD status	.26 (.08)**	.22 (.09)*	.22 (.09)*	.24 (.09)**
Child gender		-.14 (.12)	-.14 (.12)	-.14 (.12)
Child age			.00 (.02)	.00 (.02)
Child OPP/CP				-.01 (.82)
Variance estimates				
Child effects	.25 (.04)***	.25 (.04)***	.25 (.04)***	.25 (.04)***
Family effects	.05 (.04)*	.05 (.04)*	.05 (.04)*	.05 (.04)*
-2* log likelihood	232.838	230.991	230.988	230.946
Change in model fit (χ^2)		1.847	0.003	0.042
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A1.1 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.81 (.07)	1.77 (.11)	1.78 (.11)	1.80 (.11)
Child level				
Child ADHD status	.24 (.09)**	.25 (.09)**	.22 (.10)*	.19 (.10)†
Child gender	-.14 (.12)	-.10 (.15)	-.19 (.15)	-.20 (.15)
Child age	-.00 (.02)	-.00 (.02)	-.00 (.02)	.00 (.02)
Child OPP/CP	.01 (.04)	.01 (.04)	.01 (.04)	.01 (.04)
Child EP	-.04 (.04)	-.04 (.04)	-.04 (.04)	-.04 (.04)
Family level				
All-boy sib-pair		.07 (.12)	.07 (.12)	.07 (.11)
All-girl sib-pair			.27 (.22)	.25 (.22)
Sib-pair mean OPP/CP				.05 (.02)*
Variance estimates				
Child effects	.24 (.04)***	.24 (.04)***	.24 (.04)***	.24 (.04)***
Shared-family effects	.05 (.04)**	.05 (.04)**	.05 (.03)*	.04 (.03)*
-2* log likelihood	229.975	229.690	227.419	223.977
Change in model fit (χ^2)	0.971	.285	2.271	3.442
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.1 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.78 (.11)	1.79 (.11)	1.78 (.11)
Child level			
Child ADHD status	.21 (.10)*	.21 (.10)*	.20 (.10)*
Child gender	-.19 (.15)	-.19 (.15)	-.19 (.15)
Child age	-.00 (.02)	-.00 (.02)	-.00 (.02)
Child OPP/CP	.01 (.04)	.01 (.04)	.01 (.04)
Child EP	-.04 (.04)	-.04 (.04)	-.04 (.04)
Family level			
All-boy sib-pair	.08 (.11)	.08 (.11)	.08 (.11)
All-girl sib-pair	.27 (.22)	.27 (.22)	.28 (.22)
Sib-pair mean OPP/CP	.04 (.02)	.02 (.03)	.02 (.03)
Sib-pair mean EP	.02 (.03)	.00 (.00)	-.00 (.00)
Maternal ADHD		.04 (.02)	.04 (.03)
Maternal DEP			.01 (.02)
Variance estimates			
Child effects	.24 (.04)***	.24 (.04)***	.24 (.04)***
Family effects	.04 (.03)*	.04 (.03)*	.04 (.03)*
-2* log likelihood	223.389	223.375	222.829
Change in model fit (χ^2) from prior to present; *dfs*	0.588	0.014	.546
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. ***p < .001.

Table A1.2

Multilevel Models Examining Child and Family Effects on IS at T2

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.80 (.07)	1.83 (.10)	1.81 (.10)	1.93 (.11)
Child level				
Child ADHD status	.17 (.11)	.15 (.13)	.18 (.13)	-.09 (.16)
Child gender		-.07 (.13)	-.06 (.13)	-.01 (.12)
Child age			-.05 (.03)	-.05 (.03)
Child OPP/CP				.12 (.06)*
Variance estimates				
Child effects	.28 (.06)***	.28 (.06)***	.27 (.06)***	.25 (.05)***
Family effects	.00 (.04)	.00 (.04)	.00 (.04)	.01 (.04)
-2* log likelihood	150.957	150.634	148.318	143.792
Change in model fit (χ^2)		0.323	2.316	4.526*
from prior to present; *dfs*				
		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. ***p < .001.

Table A1.2 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.97 (.12)	1.82 (.16)	1.72 (.14)	1.71 (.14)
Child level				
Child ADHD status	-.14 (.18)	-.10 (.18)	.04 (.15)	.04 (.15)
Child gender	-.02 (.12)	.12 (.15)	.30 (.14)*	.30 (.14)*
Child age	-.05 (.03)†	-.05 (.02)†	-.05 (.02)*	-.05 (.02)*
Child OPP/CP	.09 (.06)	.10 (.07)	.11 (.07)	.11 (.07)
Child EP	.06 (.08)	.05 (.08)	.02 (.08)	.02 (.08)
Family level				
All-boy sib-pair		.18 (.14)	.21 (.14)	.23 (.13)
All-girl sib-pair			-.55 (.21)*	-.53 (.18)**
Sib-pair mean OPP/CP				.10 (.03)**
Variance estimates				
Child effects	.25 (.05)***	.24 (.05)***	.23 (.05)***	.22 (.05)***
Shared-family effects	.01 (.04)	.02 (.04)	.01 (.03)	.00 (.03)
-2* log likelihood	143.086	141.422	135.323	129.340
Change in model fit (χ^2)	0.706	1.664	6.099*	5.983*
from prior to present; *dfs*				
	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.2 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.72 (.14)	1.72 (.14)	1.72 (.14)
Child level			
Child ADHD status	.04 (.15)	.03 (.15)	.03 (.15)
Child gender	.30 (.14)*	.30 (.14)*	.30 (.14)*
Child age	-.05 (.02)*	-.05 (.02)*	-.05 (.02)*
Child OPP/CP	.11 (.07)	.11 (.07)	.11 (.07)
Child EP	.02 (.08)	.02 (.08)	.02 (.08)
Family level			
All-boy sib-pair	.22 (.14)†	.22 (.13)†	.23 (.13)†
All-girl sib-pair	-.56 (.18)**	-.55 (.19)**	-.57 (.18)**
Sib-pair mean OPP/CP	.12 (.05)*	.11 (.05)*	.13 (.05)*
Sib-pair mean EP	-.03 (.05)	-.03 (.05)	-.02 (.05)
Maternal ADHD		.00 (.01)	.00 (.01)
Maternal DEP			-.02 (.02)
Variance estimates			
Child effects	.22 (.05)***	.22 (.05)***	.22 (.05)***
Family effects	.00 (.03)	.00 (.03)	.00 (.03)
-2* log likelihood	129.096	129.036	127.611
Change in model fit (χ^2) from prior to present; *dfs*	0.244	0.060	1.425
	1	1	1

Note. Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.3

Multilevel Models Examining Child and Family Effects on REL at T1

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.50 (.08)	1.51 (.10)	1.52 (.11)	1.69 (.10)
Child level				
Child ADHD status	.49 (.10)***	.48 (.11)***	.47 (.11)***	.11 (.12)
Child gender		-.03 (.13)	-.03 (.13)	.03 (.12)
Child age			.01 (.03)	.01 (.02)
Child OPP/CP				.17 (.04)***
Variance estimates				
Child effects	.34 (.06)***	.34 (.06)***	.34 (.06)***	.30 (.05)***
Family effects	.08 (.05)**	.08 (.05)**	.08 (.05)**	.09 (.05)**
-2* log likelihood	280.697	280.635	280.412	268.325
Change in model fit (χ^2)		0.062	0.223	12.087**
from prior to present; *dfs*				
		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

**p < .01. ***p < .001.

Table A1.3 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.70 (.10)	1.69 (.12)	1.69 (.12)	1.74 (.13)
Child level				
Child ADHD status	.11 (.12)	.11 (.12)	.11 (.11)	.03 (.12)
Child gender	.04 (.12)	.04 (.15)	.03 (.15)	.01 (.15)
Child age	.01 (.02)	.01 (.02)	.01 (.02)	.01 (.03)
Child OPP/CP	.16 (.05)**	.16 (.05)**	.16 (.05)**	.17 (.05)**
Child EP	.03 (.05)	.03 (.05)	.03 (.05)	.03 (.05)
Family level				
All-boy sib-pair		.00 (.15)	.00 (.15)	.01 (.15)
All-girl sib-pair			.03 (.22)	-.01 (.18)
Sib-pair mean OPP/CP				.12 (.03)***
Variance estimates				
Child effects	.30 (.05)***	.30 (.05)***	.30 (.05)***	.30 (.05)***
Family effects	.09 (.05)**	.09 (.05)**	.09 (.05)**	.04 (.04)*
-2* log likelihood	267.897	267.897	267.883	251.579
Change in model fit (χ^2)	0.428	0.000	0.014	16.304**
from prior to present; *dfs*				
	1	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems. Standard errors are in parentheses.

**p < .01. †p ≤ .10 (marginally significant).

Table A1.3 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.74 (.12)	1.74 (.12)	1.74 (.12)
Child level			
Child ADHD status	.03 (.12)	.03 (.12)	.01 (.13)
Child gender	.01 (.15)	.01 (.15)	.00 (.15)
Child age	.01 (.03)	.01 (.03)	.01 (.03)
Child EP	.03 (.05)	.03 (.05)	.03 (.05)
Child OPP/CP	.17 (.05)**	.17 (.05)**	.18 (.05)**
Family level			
All-boy sib-pair	.01 (.14)	.02 (.14)	.04 (.13)
All-girl sib-pair	-.01 (.18)	-.02 (.18)	.04 (.19)
Sib-pair mean OPP/CP	.12 (.03)***	.12 (.03)***	.13 (.03)***
Sib-pair mean EP	-.01 (.03)	.00 (.03)	-.02 (.03)
Maternal ADHD		-.00 (.01)	-.01 (.00)†
Maternal DEP			.06 (.02)**
Variance estimates			
Child effects	.30 (.05)***	.30 (.05)***	.30 (.05)***
Family effects	.04 (.04)*	.04 (.04)*	.01 (.04)
-2* log likelihood	251.537	251.010	239.507
Change in model fit (χ^2) from prior to present; *dfs*	0.042	.526	11.503**
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A1.4

Multilevel Models Examining Child and Family Effects on REL at T2

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.54 (.08)	1.70 (.09)	1.69 (.09)	1.80 (.12)
Child level				
Child ADHD status	.39 (.10)***	.29 (.12)*	.31 (.12)*	.08 (.18)
Child gender		-.34 (.12)**	-.33 (.12)**	-.29 (.12)*
Child age			-.03 (.03)	-.03 (.03)
Child OPP/CP				.10 (.05)†
Variance estimates				
Child effects	.23 (.05)***	.22 (.04)***	.21 (.04)***	.20 (.04)***
Family effects	.05 (.04)*	.03 (04)†	.04 (.04)*	.04 (.04)*
-2* log likelihood	147.700	139.518	137.975	133.839
Change in model fit (χ^2)		8.182**	1.543	4.136*
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.4 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.74 (.13)	1.69 (.17)	1.65 (.16)	1.64 (.16)
Child level				
Child ADHD status	.18 (.20)	.19 (.20)	.26 (.20)	.26 (.20)
Child gender	-.26 (.12)*	-.22 (.14)	-.13 (.15)	-.13 (.15)
Child age	-.04 (.02)	-.04 (.02)†	-.04 (.02)†	-.04 (.02)†
Child OPP/CP	.15 (.07)*	.15 (.07)*	.16 (.07)*	.16 (.07)*
Child EP	-.09 (.07)	-.09 (.07)	-.11 (.07)	-.11 (.07)
Family level				
All-boy sib-pair		.07 (.12)	.07 (.12)	.08 (.12)
All-girl sib-pair			-.33 (.24)	-.33 (.23)
Sib-pair mean OPP/CP				.04 (.03)
Variance estimates				
Child effects	.18 (.04)***	.18 (.04)***	.18 (.04)***	.18 (.04)***
Family effects	.05 (.03)**	.05 (.04)**	.05 (.03)**	.05 (.03)**
-2* log likelihood	131.537	131.274	129.050	128.159
Change in model fit (χ^2)	2.302	.263	2.224	0.891
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.4 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.63 (.16)	1.62 (.16)	1.62 (.16)
Child level			
Child ADHD status	.26 (.20)	.28 (.19)	.28 (.19)
Child gender	-.13 (.15)	-.13 (.15)	-.13 (.15)
Child age	-.04 (.02)†	-.04 (.02)†	-.04 (.02)†
Child OPP/CP	.16 (.07)*	.15 (.07)*	.15 (.07)*
Child EP	-.11 (.07)	-.11 (.07)	-.11 (.07)
Family level			
All-boy sib-pair	.08 (.11)	.09 (.11)	.09 (.11)
All-girl sib-pair	-.25 (.26)	-.28 (.25)	-.28 (.26)
Sib-pair mean OPP/CP	.00 (.05)	.02 (.06)	.02 (.06)
Sib-pair mean EP	.06 (.05)	.07 (.06)	.07 (.06)
Maternal ADHD		-.01 (.01)	-.01 (.01)
Maternal DEP			.00 (.02)
Variance estimates			
Child effects	.18 (.04)***	.18 (.04)***	.18 (.04)***
Family effects	.04 (.03)**	.04 (.03)**	.04 (.03)**
-2* log likelihood	127.144	126.258	126.257
Change in model fit (χ^2) from prior to present; *dfs*	1.015	.886	.001
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01, ***p < .001. †p ≤ .01 (marginally significant).

Table A1.5

Multilevel Models Examining Child and Family Effects on WAR at T1

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.67 (.08)	1.57 (.11)	1.59 (.10)	1.64 (.10)
Child level				
Child ADHD status	.23 (.06)**	.29 (.09)**	.26 (.08)**	.17 (.10)†
Child gender		.17 (.11)	.16 (.10)	.18 (.11)
Child age			.04 (.02)*	.04 (.02)*
Child OPP/CP				.04 (.04)
Variance estimates				
Child effects	.13 (.02)***	.12 (.02)***	.12 (.02)***	.11 (.02)***
Family effects	.36 (.07)***	.36 (.07)***	.36 (.07)***	.36 (.07)***
-2* log likelihood	249.043	245.777	240.064	238.003
Change in model fit (χ^2)		3.266	5.713*	2.061
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.5 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.64 (.10)	1.65 (.13)	1.63 (.13)	1.63 (.13)
Child level				
Child ADHD status	.16 (.09)†	.16 (.09)†	.16 (.09)†	.15 (.09)
Child gender	.18 (.11)	.18 (.12)	.16 (.12)	.16 (.12)
Child age	.04 (.02)*	.04 (.02)*	.04 (.02)*	.04 (.02)*
Child OPP/CP	.03 (.04)	.03 (.04)	.03 (.04)	.03 (.04)
Child EP	.03 (.02)	.03 (.02)	.03 (.02)	.03 (.02)
Family level				
All-boy sib-pair		-0.02 (.17)	.01 (.17)	.02 (.17)
All-girl sib-pair			.17 (.29)	.14 (.27)
Sib-pair mean OPP/CP				.09 (.04)*
Variance estimates				
Child effects	.11 (.02)***	.11 (.02)***	.11 (.02)***	.11 (.02)***
Family effects	.36 (.07)***	.36 (.07)***	.36 (.07)***	.33 (.06)***
-2* log likelihood	237.009	236.996	236.543	231.341
Change in model fit (χ^2)	.994	.013	.453	5.202*
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.5 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.62 (.14)	1.61 (.13)	1.60 (.13)
Child level			
Child ADHD status	.15 (.09)	.15 (.09)	.15 (.09)
Child gender	.16 (.12)	.16 (.12)	.16 (.12)
Child age	.04 (.02)*	.04 (.02)*	.04 (.02)*
Child OPP/CP	.03 (.04)	.03 (.04)	.03 (.04)
Child EP	.03 (.02)	.03 (.02)	.03 (.02)
Family level			
All-boy sib-pair	.04 (.17)	.05 (.16)	.08 (.16)
All-girl sib-pair	.16 (.26)	.16 (.26)	.22 (.27)
Sib-pair mean OPP/CP	.08 (.04)†	.08 (.04)†	.09 (.04)*
Sib-pair mean EP	.03 (.04)	.04 (.05)	.03 (.04)
Maternal ADHD		-.01 (.01)	-.01 (.01)
Maternal DEP			.07 (.03)*
Variance estimates			
Child effects	.11 (.02)***	.11 (.02)***	.11 (.02)***
Family effects	.33 (.06)***	.32 (.06)***	.29 (.06)***
-2* log likelihood	230.928	230.303	224.226
Change in model fit (χ^2) from prior to present; *dfs*	0.413	.625	6.077**
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.6

Multilevel Models Examining Child and Family Effects on WAR at T2

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.40 (.09)	1.36 (.11)	1.38 (.11)	1.51 (.14)
Child level				
Child ADHD status	.35 (.10)**	.38 (.12)**	.35 (.10)**	.09 (.16)
Child gender		.07 (.12)	.06 (.12)	.11 (.12)
Child age			.04 (.03)	.04 (.03)
Child OPP/CP				.12 (.05)*
Variance estimates				
Child effects	.23 (.05)***	.23 (.05)***	.22 (.04)***	.20 (.04)***
Family effects	.21 (07)***	.22 (.07)***	.23 (.07)***	.23 (.07)***
-2* log likelihood	183.076	182.817	180.387	175.575
Change in model fit (χ^2)		.259	2.430	4.812*
from prior to present; *dfs*				
		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A1.6 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.55 (.14)	1.40 (.18)	1.40 (.18)	1.39 (.18)
Child level				
Child ADHD status	.03 (.18)	.07 (.17)	.11 (.17)	.10 (.16)
Child gender	.09 (.12)	.18 (.14)	.22 (.14)	.22 (.14)
Child age	.05 (.03)	.04 (.03)	.04 (.03)	.04 (.03)
Child OPP/CP	.09 (.05)†	.10 (.05)†	.10 (.05)†	.10 (.05)*
Child EP	.05 (.06)	.04 (.06)	.03 (.06)	.03 (.06)
Family level				
All-boy sib-pair		.23 (.19)	.21 (.19)	.24 (.18)
All-girl sib-pair			-.33 (.27)	-.28 (.26)
Sib-pair mean OPP/CP				.19 (.05)**
Variance estimates				
Child effects	.20 (.04)***	.20 (.04)***	.20 (.04)***	.20 (.04)***
Family effects	.22 (.07)***	.22 (.07)***	.22 (.07)***	.16 (.06)***
-2* log likelihood	175.031	173.480	172.450	162.957
Change in model fit (χ^2)	0.544	1.551	1.030	9.493**
from prior to present; *dfs*				
	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.6 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.37 (.17)	1.35 (.17)	1.35 (.17)
Child level			
Child ADHD status	.09 (.16)	.12 (.16)	.12 (.16)
Child gender	.22 (.14)	.23 (.14)	.23 (.14)
Child age	.04 (.03)	.04 (.03)	.04 (.03)
Child OPP/CP	.10 (.05)*	.10 (.05)*	.10 (.05)*
Child EP	.03 (.06)	.03 (.06)	.03 (.06)
Family level			
All-boy sib-pair	.25 (.17)	.27 (.17)	.26 (.17)
All-girl sib-pair	-.11 (.25)	-.21 (.27)	-.19 (.26)
Sib-pair mean OPP/CP	.11 (.08)	.16 (.08)*	.15 (.08)†
Sib-pair mean EP	.12 (.07)†	.16 (.07)*	.16 (.07)*
Maternal ADHD		-.02 (.01)*	-.02 (.01)*
Maternal DEP			.01 (.03)
Variance estimates			
Child effects	.20 (.04)***	.20 (.04)***	.20 (.04)***
Family effects	.15 (.05)***	.12 (.05)***	.12 (.05)***
-2* log likelihood	160.360	155.825	155.479
Change in model fit (χ^2) from prior to present; *dfs*	2.597	4.535*	.346
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.7

Multilevel Models Examining Child and Family Effects on PC at T1

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.44 (.09)	1.45 (.11)	1.45 (.11)	1.25 (.12)
Child level				
Child ADHD status	-.61 (.10)***	-.61 (.11)***	-.61 (.11)***	-.20 (.15)
Child gender		-.02 (.12)	-.02 (.13)	-.09 (.12)
Child age			-.01 (.03)	-.00 (.03)
Child OPP/CP				-.19 (.05)***
Variance estimates				
Child effects	.33 (.06)***	.33 (.06)***	.33 (.06)***	.28 (.05)***
Family effects	.19 (.07)***	.19 (.07)***	.19 (.07)***	.21 (.06)***
-2* log likelihood	304.818	304.798	304.611	289.199
Change in model fit (χ^2)		0.020	0.187	15.412**
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

**p < .01. ***p < .001.

Table A1.7 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.25 (.12)	1.24 (.16)	1.24 (.16)	1.23 (.16)
Child level				
Child ADHD status	-.19 (.15)	-.19 (.15)	-.19 (.15)	-.17 (.15)
Child gender	-.10 (.11)	-.10 (.13)	-.11 (.14)	-.11 (.14)
Child age	-.01 (.02)	-.01 (.02)	-.01 (.02)	-.01(.02)
Child OPP/CP	-.14 (.05)**	-.14 (.05)**	-.14 (.04)**	-.14 (.05)**
Child EP	-.11 (.03)**	-.11 (.03)**	-.11 (.03)**	-.11 (.03)**
Family level				
All-boy sib-pair		.01 (.17)	.01 (.17)	.00 (.17)
All-girl sib-pair			.06 (.20)	.09 (.20)
Sib-pair mean OPP/CP				-.07 (.03)*
Variance estimates				
Child effects	.25 (.04)***	.25 (.04)***	.25 (.04)***	.25 (.04)***
Family effects	.22 (.06)***	.22 (.06)***	.22 (.06)***	.20 (.06)***
-2* log likelihood	281.818	281.816	281.745	278.320
Change in model fit (χ^2)	7.381**	.002	0.071	3.425
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A1.7 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.25 (.16)	1.25 (.16)	1.26 (.16)
Child level			
Child ADHD status	-.19 (.15)	-.19 (.15)	-.18 (.15)
Child gender	-.11 (.14)	-.11 (.14)	-.11 (.14)
Child age	-.01 (.02)	-.01 (.02)	-.01 (.02)
Child OPP/CP	-.14 (.05)**	-.14 (.05)**	-.14 (.05)**
Child EP	-.11 (.03)**	-.11 (.03)**	-.11 (.03)**
Family level			
All-boy sib-pair	-.02 (.16)	-.02 (.16)	-.03 (.16)
All-girl sib-pair	.05 (.20)	.05 (.20)	.01 (.21)
Sib-pair mean OPP/CP	-.05 (.04)	-.05 (.04)	-.05 (.04)
Sib-pair mean EP	-.05 (.04)	-.04 (.04)	-.03 (.04)
Maternal ADHD		-.00 (.01)	.00 (.04)
Maternal DEP			-.04 (.02)†
Variance estimates			
Child effects	.25 (.04)***	.25 (.04)***	.25 (.04)***
Family effects	.20 (.06)***	.20 (.06)***	.19 (.06)***
-2* log likelihood	277.060	277.023	274.082
Change in model fit (χ^2) from prior to present; *dfs*	1.260	0.037	2.941
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

**p < .01. ***p < .001. †p ≤ .01(marginally significant).

Table A1.8

Multilevel Models Examining Child and Family Effects on PC at T2

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.50 (.11)	1.48 (.12)	1.48 (.12)	1.33 (.14)
Child level				
Child ADHD status	-.49 (.11)***	-.47 (.12)***	-.48 (.12)***	-.17 (.19)
Child gender		.05 (.15)	.05 (.15)	-.01 (.15)
Child age			.01 (.03)	.01 (.03)
Child OPP/CP				-.14 (.07)*
Variance estimates				
Child effects	.32 (.07)***	.33 (.07)***	.32 (.07)***	.29 (.06)***
Family effects	.15 (.07)***	.14 (.07)***	.14 (.07)***	.16 (.07)***
-2* log likelihood	194.756	194.641	194.499	189.577
Change in model fit (χ^2)		0.115	0.142	4.922*
from prior to present; *dfs*				
		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. ***p < .001.

Table A1.8 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.17 (.13)	1.31 (.16)	1.31 (.16)	1.33 (.16)
Child level				
Child ADHD status	.10 (.17)	.06 (.17)	.05 (.17)	.05 (.17)
Child gender	.05 (.13)	-.04 (.12)	-.05 (.14)	-.06 (.14)
Child age	.00 (.03)	.00 (.03)	.00 (.03)	.00 (.03)
Child OPP/CP	-.02 (.07)	-.03 (.08)	-.03 (.08)	-.03 (.08)
Child EP	-.24 (.08)**	-.23 (.08)**	-.23 (.08)**	-.23 (.08)**
Family level				
All-boy sib-pair		-.21 (.16)	-.21 (.16)	-.24 (.16)
All-girl sib-pair			.06 (.30)	.03 (.29)
Sib-pair mean OPP/CP				-.17 (.05)**
Variance estimates				
Child effects	.23 (.05)***	.23 (.05)***	.23 (.05)***	.23 (.05)***
Family effects	.19 (.07)***	.18 (.07)***	.18 (.06)***	.14 (.06)***
-2* log likelihood	178.018	176.670	176.631	169.571
Change in model fit (χ^2)	11.559**	1.348	0.039	7.060**
from prior to present; *dfs*				
	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems. Standard errors are in parentheses.

**p < .01. ***p < .001.

Table A1.8 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.38 (.14)	1.40 (.13)	1.40 (.13)
Child level			
Child ADHD status	.03 (.16)	.00 (.16)	-.00 (.16)
Child gender	-.06 (.14)	-.07 (.14)	-.07 (.14)
Child age	.00 (.03)	.01 (.03)	.01 (.03)
Child OPP/CP	-.02 (.08)	-.02 (.08)	-.02 (.08)
Child EP	-.22 (.08)**	-.22 (.08)**	-.22 (.08)**
Family level			
All-boy sib-pair	-.26 (.14)†	-.27 (.14)†	-.27 (.14)†
All-girl sib-pair	-.28 (.28)	-.21 (.28)	-.25 (.28)
Sib-pair mean OPP/CP	-.00 (.07)	-.04 (.07)	.01 (.08)
Sib-pair mean EP	-.22 (.07)**	-.25 (.07)**	-.24 (.07)**
Maternal ADHD		.01 (.01)†	.02 (.01)*
Maternal DEP			-.04 (.02)†
Variance estimates			
Child effects	.23 (.05)***	.23 (.05)***	.23 (.05)***
Family effects	.10 (.05)***	.09 (.05)***	.07 (.05)**
-2* log likelihood	160.380	158.357	154.986
Change in model fit (χ^2) from prior to present; *dfs*	9.191**	2.023	3.371
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05, **p < .01, ***p < .001. †p ≤ .10 (marginally significant).

Table A1.9

Multilevel Models Examining Child and Family Effects on CC at T1

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	.54 (.08)	.47 (.10)	.48 (.10)	.62 (.12)
Child level				
Child ADHD status	.67 (.10)***	.72 (.11)***	.71 (.11)***	.40 (.16)*
Child gender		.13 (.11)	.13 (.11)	.20 (.11)†
Child age			.02 (.03)	.01 (.03)
Child OPP/CP				.15 (.05)**
Variance estimates				
Child effects	.40 (.07)***	.39 (.06)***	.38 (.06)***	.34 (.06)***
Family effects	.06 (.05)*	.07 (.05)*	.07 (.05)*	.09 (.05)**
-2* log likelihood	293.826	292.741	292.390	284.586
Change in model fit (χ^2)		1.085	.351	7.804**
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ 1.0 (marginally significant).

Table A1.9 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	.62 (.12)	.42 (.17)	.42 (.17)	.46 (.16)
Child level				
Child ADHD status	.40 (.16)*	.45 (.16)**	.46 (.15)**	.39 (.15)**
Child gender	.20 (.11)†	.37 (.14)*	.39 (.15)*	.38 (.15)*
Child age	.01 (.03)	.01 (.03)	.01 (.03)	.01 (.03)
Child OPP/CP	.15 (.06)*	.16 (.06)*	.16 (.06)*	.17 (.06)**
Child EP	-.01 (.05)	-.00 (.05)	-.00 (.05)	-.00 (.05)
Family level				
All-boy sib-pair		.28 (.16)†	.28 (.16)†	.30 (.15)†
All-girl sib-pair			-.09 (.18)	-.13 (.15)
Sib-pair mean OPP/CP				.14 (.03)***
Variance estimates				
Child effects	.34 (.06)***	.33 (.06)***	.33 (.06)***	.33 (.06)***
Family effects	.09 (.05)**	.09 (.05)**	.09 (.05)**	.02 (.04)
-2* log likelihood	284.568	281.165	281.003	259.194
Change in model fit (χ^2)	0.018	3.403	.162	21.809**
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.9 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	.46 (.16)	.46 (.16)	.46 (.16)
Child level			
Child ADHD status	.39 (.15)*	.39 (.15)*	.39 (.15)*
Child gender	.37 (.15)	.38 (.15)*	.37 (.15)*
Child age	.01 (.03)	.01 (.03)	.01 (.03)
Child OPP/CP	.17 (.06)**	.17 (.06)**	.17 (.06)**
Child EP	-.00 (.05)	-.00 (.05)	-.00 (.05)
Family level			
All-boy sib-pair	.30 (.15)†	.30 (.15)†	.30 (.15)*
All-girl sib-pair	-.13 (.15)	-.13 (.15)	-.11 (.15)
Sib-pair mean OPP/CP	.14 (.03)***	.14 (.03)***	.14 (.03)***
Sib-pair mean EP	-.00 (.03)	-.00 (.03)	-.01 (.03)
Maternal ADHD		-.00 (.01)	-.00 (.01)
Maternal DEP			.02 (.02)
Variance estimates			
Child effects	.33 (.06)***	.33 (.06)***	.33 (.06)***
Family effects	.02 (.04)	.02 (.04)	.02 (.04)
-2* log likelihood	259.190	259.183	257.996
Change in model fit (χ^2)	0.004	.007	1.187
from prior to present; *dfs*	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional and conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.10

Multilevel Models Examining Child and Family Effects on CC at T2

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	.56 (.10)	.70 (.15)	.70 (.15)	1.02 (.15)
Child level				
Child ADHD status	.90 (.12)***	.81 (.15)***	.80 (.15)***	.12 (.17)
Child gender		-.27 (.17)	-.27 (.17)	-.10 (.13)
Child age			.01 (.04)	.00 (.03)
Child OPP/CP				.31 (.05)***
Variance estimates				
Child effects	.36 (.07)***	.36 (.07)***	.36 (.07)***	.21 (.04)***
Family effects	.17 (.08)***	.14 (.08)**	.14 (.08)**	.22 (.07)***
-2* log likelihood	205.208	202.551	202.512	175.880
Change in model fit (χ^2)		2.657	0.039	26.632**
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

**p < .01. ***p < .001.

Table A1.10 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.03 (.15)	.85 (.16)	.84 (.16	.82 (.15)
Child level				
Child ADHD status	.10 (.17)	.14 (.16)	.18 (.16)	.19 (.15)
Child gender	-.11 (.13)	.00 (.14)	.05 (.15)	.05 (.15)
Child age	.01 (.03)	.00 (.03)	.00 (.03)	.00 (.03)
Child OPP/CP	.30 (.06)***	.31 (.06)***	.31 (.06)***	.31 (.06)***
Child EP	.02 (.06)	.01 (.06)	-.00 (.06)	-.00 (.06)
Family level				
All-boy sib-pair		.28 (.18)	.26 (.18)	.30 (.16)†
All-girl sib-pair			-.30 (.27)	-.25 (.16)
Sib-pair mean OPP/CP				.25 (.05)***
Variance estimates				
Child effects	.21 (.04)***	.20 (.04)***	.20 (.04)***	.20 (.04)***
Family effects	.22 (.07)***	.21 (.07)***	.20 (.07)***	.11 (.05)***
-2* log likelihood	175.813	173.537	172.637	154.460
Change in model fit (χ^2)	0.067	2.276	0.900	18.177**
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A1.10 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	.84 (.15)	.82 (.15)	.82 (.14)
Child level			
Child ADHD status	.18 (.15)	.22 (.15)	.22 (.15)
Child gender	.05 (.15)	.06 (.15)	.06 (.15)
Child age	.00 (.03)	.00 (.03)	.00 (.03)
Child OPP/CP	.31 (.06)***	.31 (.06)***	.31 (.06)***
Child EP	-.00 (.06)	-.01 (.06)	-.01 (.06)
Family level			
All-boy sib-pair	.29 (.15)†	.30 (.16)†	.31 (.15)†
All-girl sib-pair	-.43 (.18)*	-.51 (.17)**	-.55 (.16)**
Sib-pair mean OPP/CP	.34 (.07)***	.39 (.06)***	.43 (.07)***
Sib-pair mean EP	-.13 (.07)	-.10 (.07)	-.09 (.06)
Maternal ADHD		-.01 (.01)	-.01 (.01)
Maternal DEP			-.04 (.03)
Variance estimates			
Child effects	.20 (.04)***	.20 (.04)***	.20 (.04)***
Family effects	.09 (.05)***	.08 (.04)***	.07 (.04)***
-2* log likelihood	151.143	147.676	144.993
Change in model fit (χ^2) from prior to present; *dfs*	3.317**	3.467	2.683
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Appendix 2: Comparison of Child and Family Characteristics between Families Who Did and Did Not Participate at T2 (Chapter 4, Section 4.3.1)

List of Tables

Table A2.1

Mean Scores on Measures of Child Behaviour as a Function of Families Who Did and Did Not Take Part at T2

Child behaviour	Participated at both T1 and T2 (n = 90)	Did not participate at both T1 and T2 (n = 54)	*t*
Inattentive symptoms[a]	64.89 (14.62)	64.61 (15.61)	-.11
Hyperactive-impulsive symptoms[a]	70.21 (18.56)	70.11 (19.00)	-.03
Conduct problems[b]	4.52 (2.96)	3.83 (2.81)	-1.38
Oppositional problems[a]	67.47 (15.75)	66.91 (17.41)	-.20
Depressive symptoms[b]	3.62 (2.83)	3.30 (2.85)	-.67
Anxiety symptoms[a]	60.54 (15.85)	59.74 (14.72)	-.30
Emotional dysregulation problems[a]	66.08 (15.51)	65.91 (16.42)	-.06
Family/parent problems	**Participated at both T1 and T2 (n = 45)**	**Did not participate at both T1 and T2 (n = 27)**	***t***
Maternal ADHD symptoms[c]	10.09 (9.53)	13.81 (12.25)	1.35
Maternal depressive symptoms[d]	2.47 (2.97)	2.52 (2.81)	.07
Sib-pair inattentive symptoms[a]	64.89 (7.83)	64.61 (9.47)	-.14
Sib-pair hyperactive-impulsive symptoms[a]	70.21 (9.06)	70.11 (8.48)	-.05
Sib-pair conduct problems[b]	4.52 (1.99)	3.83 (1.77)	-1.48
Sib-pair oppositional problems[a]	67.47 (9.72)	66.91 (10.97)	-.23
Sib-pair depressive symptoms[b]	3.62 (2.19)	3.30 (1.95)	-.64
Sib-pair anxiety symptoms[a]	60.54 (11.36)	59.74 (10.91)	-.30
Sib-pair emotional dysregulation problems[a]	66.08 (10.55)	65.91 (9.71)	-.07

Note. [a] = symptoms/problems were measured using the CPRS-R: L; [b] = symptoms/problems were measured using the SDQ; [c] = symptoms were measured using the CSS-SR; [d] = symptoms were measured using the GHQ-12.

Table A2.2

Crosstabulations of Children's Participation at T1 and T2 and whether Clinical Cut-Offs Met on Scores of Child Behaviour

	Inattentive Symptoms				Hyperactive-Impulsive Symptoms				Conduct Problems				Oppositional Problems			
	Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met			
Participated at T1 and T2	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ
Yes (n = 90)	55 (-.40)	35 (.40)	.20	-.04	56 (-.30)	34 (.30)	.10	-.03	57 (1.4)	33 (-1.4)	1.84	.11	60 (.50)	30 (31.3)	.20	.04
No (n = 54)	35 (.40)	19 (-.40)			35 (.30)	19 (-.30)			28 (-1.4)	26 (1.4)			34 (-.50)	20 (.50)		

Note. Adjusted standardised residuals appear in parentheses next to group frequencies.

Table A2.2 (continued)

	Depressive Symptoms				Anxiety Symptoms				Emotional Dysregulation Problems			
	Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met			
Participated at T1 and T2	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ
Yes (n = 90)	25 (.50)	65 (-.50)	.24	.04	42 (.00)	48 (.00)	.00	.00	57 (-.20)	33 (.20)	.03	-.02
No (n = 54)	13 (-.50)	41 (.50)			25 (.00)	29 (.00)			35 (.20)	19 (-.20)		

Note. Adjusted standardized residuals appear in parentheses next to group frequencies

Table A2.3

Mean Scores on Measures of Child Behaviour as a Function of Sibling and Who Did and Did Not Take Part at Both T1 and T2

	Probands					Siblings				
	Participated at both T1 and T2					Participated at both T1 and T2				
	Yes (n = 45)		No (n = 27)			Yes (n = 45)		No (n = 27)		
Child Problems	Mean	SD	Mean	SD	*t*	Mean	SD	Mean	SD	*t*
Inattentive symptoms[a]	74.93	8.56	74.48	9.17	-.21	54.84	12.36	54.74	14.49	-.03
Hyperactive-impulsive symptoms[a]	83.87	9.23	83.63	9.52	-.10	56.56	15.16	56.59	16.31	.01
Conduct problems[b]	6.18	2.32	5.07	2.32	-1.96	2.87	2.60	2.59	2.74	-.43
Oppositional problems[a]	77.38	11.46	76.11	12.55	-.44	16.85	13.03	57.70	57.56	.04
Depressive symptoms[b]	4.20	2.69	4.07	2.79	-.19	3.04	2.88	2.52	2.74	-.77
Anxiety symptoms[a]	65.87	14.90	64.52	15.33	-.37	55.22	15.10	54.96	12.61	-.08
Emotional dysregulation problems[a]	74.18	11.93	74.33	13.11	.05	57.98	14.49	57.48	15.17	-.14

Note. [a] = symptoms/problems were measured using the CPRS-R: L; [b] = symptoms/problems were measured using the SDQ.

Table A2.4

Crosstabulations of Probands' Participation at Both T1 and T2 and Clinical Cut-Offs Met on Scores of Child Behaviour

	Inattentive Symptoms				Hyperactive-Impulsive Symptoms				Conduct Problems				Oppositional Problems			
	Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met			
Participated at T1 and T2	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ
Yes (n = 45)	44 (.40)	1 (.40)	.14	.04	43 (-.20)	2 (.20)	.02	-.02	39 (1.3)	6 (-1.3)	1.81	.16	41 (-.20)	4 (.20)	.05	-.03
No (n = 27)	26 (-.40)	1 (.40)			26 (.20)	1 (-.20)			20 (-1.30)	7 (1.30)			25 (.20)	2 (-.20)		

Note. Adjusted standardised residuals appear in parentheses next to group frequencies.

Table A2.4 (continued)

	Depressive Symptoms				Anxiety Symptoms				Emotional Dysregulation Problems			
	Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met			
Participated at T1 and T2	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ
Yes (n = 45)	14 (.10)	31 (-.10)	.02	.02	29 (-.20)	16 (.20)	.04	-.02	39 (-.30)	6 (.30)	.08	-.03
No (n = 27)	8 (-.10)	19 (.10)			18 (.20)	9 (-.20)			24 (.30)	3 (-.30)		

Note. Adjusted standardized residuals appear in parentheses next to group frequencies

Table A2.5

Crosstabulations of Siblings' Participation at Both T1 and T2 and Clinical Cut-Offs Met on Scores of Child Behaviour

	Inattentive Symptoms				Hyperactive-Impulsive Symptoms				Conduct Problems				Oppositional Problems			
	Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met			
Participated at T1 and T2	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ
Yes (n = 45)	11 (-.80)	34 (.80)	.67	-.10	13 (-.40)	32 (.40)	.16	-.05	18 (.90)	27 (-.90)	.79	.11	19 (.70)	26 (-.70)	.56	.09
No (n = 27)	9 (.80)	18 (-.80)			9 (.40)	18 (-.40)			8 (-.90)	19 (.90)			9 (-.70)	18 (.70)		

Note. Adjusted standardised residuals appear in parentheses next to group frequencies.

Table A2.5 (continued)

	Depressive Symptoms				Anxiety Symptoms				Emotional Dysregulation Problems			
	Clinical Cut-Off Met				Clinical Cut-Off Met				Clinical Cut-Off Met			
Participated at T1 and T2	Yes	No	χ^2	φ	Yes	No	χ^2	φ	Yes	No	χ^2	φ
Yes (n = 45)	11 (.60)	34 (-.60)	.34	.07	13 (.30)	32 (-.30)	.07	.03	18 (-.10)	27 (.10)	.00	-.01
No (n = 27)	5 (-.60)	22 (.60)			7 (-.30)	20 (.30)			11 (.10)	16 (-.10)		

Note. Adjusted standardized residuals appear in parentheses next to group frequencies

Table A2.6

Mean Scores on Measures of Child Behaviour as a Function of ADHD Status and Who Did and Did Not Take Part at Both T1 and T2

	Siblings with ADHD					Siblings without ADHD				
	Participated at both T1 and T2					Participated at both T1 and T2				
	Yes (n = 49)		No (n = 30)			Yes (n = 41)		No (n = 24)		
Child Problems	Mean	SD	Mean	SD	*t*	Mean	SD	Mean	SD	*t*
Inattentive symptoms[a]	73.69	9.34	74.57	9.15	.41	54.20	11.64	52.33	12.68	-.60
Hyperactive-impulsive symptoms[a]	82.10	10.47	83.93	9.10	.79	56.22	15.02	53.17	12.80	-.83
Conduct problems[b]	5.96	2.33	5.30	2.44	-1.19	2.74	2.68	2.00	2.09	-1.16
Oppositional problems[a]	76.71	11.27	76.90	12.41	.07	56.61	13.03	54.75	14.73	-.53
Depressive symptoms[b]	3.92	2.62	3.97	2.75	.08	2.78	2.62	2.46	2.80	-.46
Anxiety symptoms[a]	63.96	14.58	63.77	14.73	-.06	56.22	15.38	54.75	13.25	-.39
Emotional dysregulation problems[a]	73.06	11.73	74.90	12.61	.65	58.12	14.99	54.58	13.64	-.95

Note. [a] = symptoms/problems were measured using the CPRS-R: L; [b] = symptoms/problems were measured using the SDQ.

Appendix 3: Longitudinal Multilevel Models Examining Child and Family Effects on Maternal EE Components (Chapter 5, Section 5.5.3)

List of Tables

Table A3.1

Multilevel models Examining T1 Child and Family Effects on T2 IS

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.56 (.17)	1.60 (.18)	1.59 (.18)	1.67 (.21)
Child level				
Child ADHD status	.13 (.12)	.10 (.13)	.12 (.13)	-.05 (.19)
T1 IS	.14 (.09)	.14 (.09)	.14 (.09)	.14 (.09)
Child gender		-.08 (.13)	-.08 (.13)	-.05 (.12)
T1 child age			-.03 (.03)	-.02 (.03)
T1 child OPP/CP				.08 (.06)
Variance estimates				
Child effects	.25 (.05)***	.26 (.06)***	.26 (.05)***	.25 (.05)***
Family effects	.01 (.04)	.00 (.04)	.00 (.04)	.01 (.04)
-2* log likelihood	135.657	135.256	134.527	132.910
Change in model fit (χ^2)		0.401	0.729	1.617
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; IS = initial statement; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

***p < .001.

Table A3.1 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.64 (.20)	1.51 (.20)	1.45 (.20)	1.53 (.21)
Child level				
T1 child ADHD status	-.09 (.17)	-.06 (.16)	.04 (.14)	.06 (.14)
T1 IS	.16 (.09)	.15 (.09)†	.15 (.08)†	.10 (.08)
Child gender	.00 (.13)	.14 (.15)	.29 (.14)*	.29 (.14)*
T1 child age	-.02 (.02)	-.02 (.02)	-.03 (.02)	-.03 (.02)
T1 child OPP/CP	.04 (.06)	.05 (.06)	.04 (.05)	.04 (.05)
T1 child EP	.13 (.04)**	.13 (.04)**	.13 (.04)**	.13 (.04)**
Family level				
T1 all-boy sib-pair		.21 (.14)	.21 (.14)	.23 (.13)†
T1 all-girl sib-pair			-.55 (.21)*	-.55 (.22)*
T1 sib-pair mean OPP/CP				.09 (.03)**
Variance estimates				
Child effects	.21 (.04)***	.19 (.04)***	.19 (.04)***	.19 (.04)***
Family effects	.03 (.04)	.04 (.04)*	.02 (.03)†	.01 (.03)
-2* log likelihood	125.005	122.826	116.211	111.491
Change in model fit (χ^2)	7.905**	2.179	6.615*	4.720*
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; WAR = warmth; OPP/CP = oppositional/conduct problems; EP = emotional problems.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.1 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.54 (.21)	1.54 (.21)	1.53 (.22)
Child level			
T1 child ADHD status	.04 (.14)	.04 (.13)	.04 (.14)
T1 IS	.10 (.08)	.10 (.08)	.11 (.08)
Child gender	.29 (.14)*	.28 (.14)*	.28 (.14)*
T1 child age	-.03 (.02)	-.03 (.02)	-.03 (.02)
T1 child OPP/CP	.04 (.05)	.04 (.05)	.04 (.05)
T1 child EP	.13 (.04)**	.13 (.04)**	.13 (.04)**
Family level			
T1 all-boy sib-pair	.22 (.13)†	.22 (.12)†	.23 (.13)†
T1 all-girl sib-pair	-.60 (.23)*	-.58 (.23)*	-.59 (.24)*
T1 sib-pair mean OPP/CP	.12 (.05)*	.12 (.05)*	.12 (.05)*
T1 sib-pair mean EP	-.04 (,05)	-.05 (.05)	-.05 (.05)
T1 maternal ADHD		.00 (.00)	.00 (.00)
T1 maternal DEP			-.01 (.01)
Variance estimates			
Child effects	.19 (.04)***	.19 (.04)***	.19 (.04)***
Family effects	.01 (.03)	.01 (.03)	.01 (.03)†
-2* log likelihood	110.635	109.910	109.797
Change in model fit (χ^2) from prior to present; *dfs*	0.856	0.725	0.113
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; IS = initial statement; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05, **p < .01, ***p < .001. †p ≤ .10 (marginally significant).

Table A3.2

Multilevel models Examining T1 Child and Family Effects on T2 REL

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	1.30 (.15)	1.48 (.15)	1.46 (.15)	1.56 (.18)
Child level				
Child ADHD status	.31 (.11)**	.21 (.12)†	.23 (.12)†	.12 (.19)
T1 REL	.15 (.08)†	.14 (.07)*	.15 (.07)*	.13 (.07)†
Child gender		-.35 (.12)**	-.35 (.12)**	-.33 (.12)**
T1 child age			-.03 (.03)	-.03 (.03)
T1 child OPP/CP				.05 (.06)
Variance estimates				
Child effects	.23 (.05)***	.22 (.05)***	.22 (.05)***	.21 (.04)***
Family effects	.03 (.04)†	.01 (.04)	.02 (.03)	.02 (.03)
-2* log likelihood	134.755	125.878	124.382	123.573
Change in model fit (χ^2)		8.877**	1.496	0.809
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; REL = relationship; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.2 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	1.55 (.17)	1.52 (.18)	1.51 (.18)	1.56 (.19)
Child level				
T1 child ADHD status	.15 (.19)	.16 (.19)	.20 (.20)	.20 (.20)
T1 REL	.12 (.06)†	.12 (.06)†	.11 (.06)†	.08 (.07)
Child gender	-.34 (.11)**	-.30 (.12)*	-.24 (.12)*	-.24 (.12)*
T1 child age	-.03 (.03)	-.03 (.03)	-.03 (.03)	-.03 (.03)
T1 child OPP/CP	.08 (.05)	.08 (.05)	.07 (.05)	.08 (.05)
T1 child EP	-.08 (.05)†	-.08 (.05)†	-.08 (.05)†	-.08 (.05)†
Family level				
T1 all-boy sib-pair		.05 (.11)	.06 (.11)	.07 (.11)
T1 all-girl sib-pair			-.22 (.23)	-.22 (.22)
T1 sib-pair mean OPP/CP				.04 (.04)
Variance estimates				
Child effects	.19 (.04)***	.19 (.04)***	.19 (.04)***	.19 (.04)***
Family effects	.03 (.03)†	.03 (.03)†	.03 (.03)†	.03 (.03)†
-2* log likelihood	119.751	119.612	118.553	117.828
Change in model fit (χ^2)	3.822	0.139	1.059	0.725
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; REL = relationship; OPP/CP = oppositional/conduct problems; EP = emotional problems.

**p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.2 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.55 (.20)	1.55 (.20)	1.61 (.22)
Child level			
T1 child ADHD status	.20 (.20)	.20 (.20)	.18 (.20)
T1 REL	.08 (.07)	.08 (.07)	.05 (.08)
Child gender	-.24 (.12)*	-.24 (.12)*	-.23 (.11)*
T1 child age	-.03 (.03)	-.03 (.03)	-.03 (.03)
T1 child OPP/CP	.08 (.05)	.08 (.05)	.09 (.05)
T1 child EP	-.08 (.05)	-.08 (.05)	-.08 (.05)
Family level			
T1 all-boy sib-pair	.07 (.11)	.07 (.11)	.06 (.12)
T1 all-girl sib-pair	-.21 (.23)	-.20 (.23)	-.17 (.22)
T1 sib-pair mean OPP/CP	.03 (.06)	.03 (.06)	.03 (.06)
T1 sib-pair mean EP	.01 (.05)	.01 (.05)	.01 (.05)
T1 maternal ADHD		.00 (.00)	-.00 (.00)
T1 maternal DEP			.02 (.02)
Variance estimates			
Child effects	.19 (.04)***	.19 (.04)***	.19 (.04)***
Family effects	.03 (.03)*	.03 (.03)*	.02 (.03)*
-2* log likelihood	117.769	117.682	116.588
Change in model fit (χ^2) from prior to present; *dfs*	0.059	0.087	1.094
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; REL = relationship; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. ***p < .001.

Table A3.3

Multilevel models Examining T1 Child and Family Effects on T2 WAR

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	.47 (.16)	.48 (.16)	.53 (.16)	.57 (.11)
Child level				
Child ADHD status	16 (.09)†	.15 (.10)	.14 (.09)	-.05 (.13)
T1 WAR	.60 (.11)***	.60 (.11)***	.58 (.11)***	.57 (.11)***
Child gender		-.02 (.11)	-.02 (.11)	.01 (.11)
T1 child age			.03 (.02)	.04 (.02)
T1 child OPP/CP				.08 (.05)†
Variance estimates				
Child effects	.15 (.03)***	.15 (.03)***	.14 (.03)***	.13 (.03)***
Family effects	15 (.05)***	.15 (.05)***	.16 (.05)***	.16 (.05)***
-2* log likelihood	133.077	133.035	130.904	127.785
Change in model fit (χ^2)		0.042	2.131	3.119
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; WAR = warmth; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

***p < .001. †p ≤ .10 (marginally significant).

Table A3.3 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	.66 (.17)	.64 (.21)	.65 (.21)	.74 (.22)
Child level				
T1 child ADHD status	-.09 (.14)	-.07 (.13)	-.05 (.13)	-.03 (.13)
T1 WAR	.56 (.11)***	.55 (.10)***	.55 (.10)***	.48 (.11)***
Child gender	.03 (.10)	.05 (.13)	.08 (.12)	.10 (.12)
T1 child age	.04 (.02)†	.04 (.02)†	.04 (.02)†	.04 (.02)†
T1 child OPP/CP	.06 (.04)	.06 (.04)	.06 (.04)	.06 (.04)
T1 child EP	.08 (.03)*	.08 (.03)*	.08 (.03)*	.08 (.03)*
Family level				
T1 all-boy sib-pair		.06 (.16)	.03 (.17)	.08 (.15)
T1 all-girl sib-pair			-.29 (.21)	-.28 (.23)
T1 sib-pair mean OPP/CP				.09 (.04)*
Variance estimates				
Child effects	.12 (.02)***	.12 (.02)***	.12 (.02)***	.12 (.02)***
Family effects	.16 (.05)***	.16 (.05)***	.16 (.05)***	.13 (.04)***
-2* log likelihood	122.144	122.013	120.821	115.408
Change in model fit (χ^2)	5.641*	0.131	1.192	5.413*
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; WAR = warmth; OPP/CP = oppositional/conduct problems; EP = emotional problems.

*p < .05. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.3 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	.74 (.21)	.73 (.21)	.67 (.19)
Child level			
T1 child ADHD status	-.04 (.13)	-.03 (.13)	-.03 (.13)
T1 WAR	.49 (.11)***	.49 (.11)***	.53 (.09)***
Child gender	.10 (.12)	.10 (.12)	.09 (.12)
T1 child age	.04 (.02)†	.04 (.02)†	.04 (.02)†
T1 child OPP/CP	.08 (.03)*	.08 (.03)*	.08 (.03)*
T1 child EP	.06 (.04)	.06 (.04)	.06 (.04)
Family level			
T1 all-boy sib-pair	.06 (.16)	.06 (.16)	.08 (.15)
T1 all-girl sib-pair	-.33 (.26)	-.36 (.25)	-.43 (.24)†
T1 sib-pair mean OPP/CP	.10 (.05)*	.11 (.05)*	.11 (.05)*
T1 sib-pair mean EP	-.03 (.05)	-.02 (.05)	-.03 (.05)
T1 maternal ADHD		-.01 (.01)	-.00 (.01)
T1 maternal DEP			-.05 (.02)*
Variance estimates			
Child effects	.12 (.02)	.12 (.02)	.12 (.02)
Family effects	.13 (.04)***	.13 (.04)***	.11 (.04)
-2* log likelihood	115.029	113.844	109.355
Change in model fit (χ^2) from prior to present; *dfs*	0.379	1.185	4.489*
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; WAR = warmth; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.4

Multilevel Models Examining T1 Child and Family Effects on T2 PC

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	.84 (.17)	.78 (.17)	.78 (.17)	.76 (.16)
Child level				
Child ADHD status	-.25 (.11)*	-.21 (.12)†	-.20 (.11)†	-.12 (.15)
T1 PC	.45 (.07)***	.46 (.07)***	.46 (.07)***	.44 (.08)***
Child gender		.10 (.15)	.10 (.15)	.08 (.15)
T1 child age			-.02 (.03)	-.02 (.03)
T1 child OPP/CP				-.04 (.07)
Variance estimates				
Child effects	.26 (.05)***	.26 (.06)***	.26 (.06)***	..26 (.05)***
Family effects	.12 (.06)***	.11 (.06)***	.11 (.06)***	.12 (.06)***
-2* log likelihood	163.759	163.250	162.920	162.571
Change in model fit (χ^2)		0.509	0.330	0.349
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; PC = positive comments; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.4 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	.79 (.17)	1.00 (.18)	1.01 (.18)	1.09 (.16)
Child level				
T1 child ADHD status	-.10 (.16)	-.15 (.14)	-.16 (.14)	-.17 (.14)
T1 PC	.42 (.09)***	.42 (.08)***	.42 (.08)***	.37 (.07)***
Child gender	.07 (.16)	-.10 (.17)	-.11 (.19)	-.13 (.18)
T1 child age	-.02 (.03)	-.02 (.02)	-.02 (.02)	-.02 (.02)
T1 child OPP/CP	-.03 (.07)	-.04 (.06)	-.04 (.06)	-.04 (.06)
T1 child EP	-.04 (.05)	-.05 (.05)	-.05 (.05)	-.05 (.05)
Family level				
T1 all-boy sib-pair		-.33 (.15)*	-.33 (.15)*	-.36 (.15)*
T1 all-girl sib-pair			.07 (.28)	.08 (.23)
T1 sib-pair mean OPP/CP				-.12 (.03)**
Variance estimates				
Child effects	.25 (.05)***	.25 (.05)***	.25 (.05)***	.24 (.05)***
Family effects	.12 (.06)***	.11 (.06)***	.11 (.06)***	.07 (.05)**
-2* log likelihood	161.827	158.237	158.173	148.384
Change in model fit (χ^2)	0.744	3.590	0.064	9.789**
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; PC = positive comments; OPP/CP = oppositional /conduct problems; EP = emotional problems.

*p < .05. **p < .01. ***p < .001.

Table A3.4 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	1.12 (.17)	1.12 (.17)	1.13 (.17)
Child level			
T1 child ADHD status	-.18 (.14)	-.19 (.14)	-.17 (.14)
T1 PC	.36 (.07)***	.36 (.07)***	.34 (.08)***
Child gender	-.13 (.18)	-.13 (.18)	-.14 (.18)
T1 child age	-.02 (.02)	-.02 (.02)	-.02 (.02)
T1 child EP	-.05 (.05)	-.05 (.05)	-.06 (.05)
T1 child OPP/CP	-.04 (.06)	-.04 (.06)	-.05 (.06)
Family level			
T1 all-boy sib-pair	-.37 (.15)*	-.37 (.15)*	-.36 (.15)*
T1 all-girl sib-pair	.02 (.25)	.03 (.26)	-.01 (.26)
T1 sib-pair mean OPP/CP	-.10 (.04)*	-.10 (.04)*	-.09 (.04)*
T1 sib-pair mean EP	-.04 (.05)	-.04 (.05)	-.04 (.05)
T1 maternal ADHD		.00 (.01)	.01 (.01)
T1 maternal DEP			-.03 (.02)
Variance estimates			
Child effects	.24 (.05)***	.24 (.05)***	.24 (.05)***
Family effects	.06 (.05)**	.06 (.05)**	.06 (.06)**
-2* log likelihood	147.751	147.636	146.367
Change in model fit (χ^2) from prior to present; *dfs*	0.633	0.115	1.269
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; PC = positive comments; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A3.5

Multilevel Models Examining T1 Child and Family Effects onT2 CC

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	.44 (.12)	.59 (.16)	.60 (.15)	.93 (.17)
Child level				
Child ADHD status	.66 (.16)***	.53 (.17)**	.52 (.17)**	.02 (.19)
T1 CC	.26 (.14)†	.30 (.13)*	.30 (.13)*	.18 (.12)
Child gender		-.33 (.16)*	-.33 (.16)*	-.18 (.14)
T1 child age			.02 (.03)	.03 (.03)
T1 child OPP/CP				.25 (.06)***
Variance estimates				
Child effects	.36 (.08)***	.37 (.08)***	.36 (.08)***	.26 (.05)***
Family effects	.13 (.08)**	.10 (.07)**	.10 (.07)**	.17 (.07)**
-2* log likelihood	188.563	184.522	184.301	171.194
Change in model fit (χ^2)		4.041*	0.221	13.107**
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; CC = critical comments; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A3.5 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	.93 (.17)	.82 (.19)	.82 (.19)	.82 (.18)
Child level				
T1 child ADHD status	.02 (.20)	.05 (.20)	.09 (.20)	.13 (.18)
T1 CC	.18 (.12)	.15 (.13)	.14 (.12)	.07 (.12)
T1 child gender	-.18 (.14)	-.08 (.16)	-.04 (.16)	-.00 (.16)
T1 child age	.03 (.03)	.03 (.03)	.03 (.03)	.03 (.03)
T1 child OPP/CP	.25 (.06)***	.26 (.05)***	.25 (.05)***	.25 (.05)***
T1 child CP	.01 (.04)	.01 (.04)	.01 (.04)	.01 (.04)
Family level				
T1 all-boy sib-pair		.20 (.21)	.19 (.21)	.26 (.18)
T1 all-girl sib-pair			-.24 (.25)	-.26 (.21)
Sib-pair mean OPP/CP				.16 (.04)**
Variance estimates				
Child effects	.26 (.05)***	.25 (.05)***	.25 (.05)***	.24 (.05)***
Family effects	.17 (.07)***	.17 (.07)***	.17 (.07)***	.10 (.05)***
-2* log likelihood	171.159	170.156	169.603	154.680
Change in model fit (χ^2)	0.035	1.003	0.553	14.923**
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; cc = critical comments; OPP/CP = oppositional/conduct problems; EP = emotional problems.

**p < .01. ***p < .001.

Table A3.5 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	.81 (.19)	.81 (.19)	.79 (.19)
Child level			
T1 child ADHD status	.14 (.19)	.14 (.19)	.15 (.19)
T1 CC	.07 (.12)	.07 (.12)	.10 (.12)
T1 child gender	-.00 (.16)	-.00 (.16)	-.01 (.16)
T1 child age	.03 (.03)	.03 (.03)	.03 (.03)
T1 child EP	.01 (.04)	.01 (.04)	.01 (.04)
T1 child OPP/CP	.26 (.05)***	.26 (.05)***	.25 (.05)***
Family level			
T1 all-boy sib-pair	.26 (.19)	.26 (.19)	.27 (.19)
T1 all-girl sib-pair	-.22 (.23)	-.24 (.23)	-.28 (.23)
T1 sib-pair mean OPP/CP	.15 (.06)*	.15 (.06)*	.15 (.06)*
T1 sib-pair mean EP	.02 (.05)	.03 (.05)	.02 (.05)
T1 maternal ADHD		-.01 (.01)	-.00 (.01)
T1 maternal DEP			-.03 (.02)
Variance estimates			
Child effects	.24 (.05)***	.24 (.05)***	.24 (.05)***
Family effects	.10 (.05)***	.10 (.05)***	.09 (.05)***
-2* log likelihood	154.526	154.053	152.737
Change in model fit (χ^2) from prior to present; *dfs*	0.154	0.473	1.316
	1	1	1

Note. ADHD = attention-deficit hyperactivity disorder; cc = critical comments; OPP/CP = oppositional/conduct problems; EP = emotional problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. ***p < .001.

Appendix 4: Longitudinal Multilevel Models Examining Child and Family Effects on Child Behaviour (Chapter 6, Section 6.5.4)

List of Tables

Table A4.1

Multilevel Models of T1 Child and Family Effects on T2 Child OPP/CP (with Overall EE)

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	-.41 (.24)	-.36 (.27)	-.35 (.27)	-.35 (.26)
Child-level predictors				
T1 child ADHD	.75 (.37)*	.73 (.36)*	.71 (.35)*	.80 (.38)*
T1 child OPP/CP	.65 (.10)***	.64 (.10)***	.64 (.10)***	.66 (.09)***
Child gender		-.11 (.32)	-.11 (.32)	-.04 (.33)
T1 age			.07 (.14)	.11 (.15)
T1 maternal EE				-.40 (.42)
Variance estimates				
Child effects	1.03 (.22)***	1.02 (.22)***	1.02 (.21)***	.95 (.20)***
Family effects	1.03 (.34)***	1.04 (.34)***	1.04 (.34)***	1.16 (.36)***
-2* log likelihood	307.334	307.201	307.014	306.108
Change in model fit (χ^2)		0.133	0.187	0.906
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; EE = expressed emotion.

*p < .05. ***p < .001.

Table A4.1 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	-1.68 (.40)	-1.51 (.45)	-1.50 (.45)	-1.82 (.43)
Child-level predictors				
T1 child ADHD	.69 (.39)†	.62 (.39)	.63 (.40)	.83 (.35)*
T1 child OPP/CP	.62 (.10)***	.61 (.10)***	.61 (.11)***	.65 (.09)***
Child gender	.01 (.32)	-.15 (.39)	-.14 (.40)	.11 (.36)
T1 age	.05 (.14)	.04 (.15)	.04 (.15)	.12 (.15)
T1 maternal EE	.45 (.52)	.61 (.53)	.60 (.53)	-.26 (.51)
Change in maternal EE	1.23 (.29)***	1.28 (.30)***	1.28 (.30)***	1.20 (.30)***
Family-level predictors				
All-boy sib-pair		-.38 (.44)	-.38 (.44)	-.47 (.41)
All-girl sib-pair			-.07 (.71)	-.45 (.67)
T1 sib-pair EE				1.15 (.34)**
Variance estimates				
Child effects	.86 (.18)***	.87 (.18)***	.87 (.18)***	.81 (.17)***
Family effects	.99 (.31)***	.93 (.30)***	.93 (.30)***	.80 (.27)***
-2* log likelihood	295.302	294.516	294.505	285.668
Change in model fit (χ^2)	10.806**	0.786	0.011	8.837**
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A4.1 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	-2.15 (.50)	-2.04 (.50)	-2.05 (.51)
Child-level predictors			
T1 child ADHD	.85 (.36)*	.75 (.38)*	.75 (.37)*
T1 child OPP/CP	.66 (.09)***	.67 (.10)***	.67 (.10)***
Child gender	.12 (.36)	.09 (.37)	.09 (.36)
T1 age	.13 (.15)	.13 (.15)	.13 (.15)
T1 maternal EE	-.40 (.51)	-.30 (.53)	-.30 (.52)
Change in maternal EE	1.02 (.33)**	1.11 (.36)**	1.11 (.36)**
Family-level predictors			
All-boy sib-pair	-.52 (.41)	-.48 (.36)	-.48 (.36)
All-girl sib-pair	-.32 (.72)	-.02 (.69)	-.00 (.72)
T1 sib-pair EE	1.38 (.38)**	1.27 (.40)**	1.27 (.40)**
Change in sib-pair EE	.44 (.35)	.29 (.33)	.30 (.32)
Maternal ADHD symptoms		.06 (.01)***	.06 (.01)***
Maternal DEP symptoms			.00 (.06)
Variance estimates			
Child effects	.80 (.17)***	.81 (.17)***	.81 (.17)***
Family effects	.80 (.27)***	.47 (.20)***	.47 (.20)***
-2* log likelihood	284.713	270.913	270.909
Change in model fit (χ^2) from prior to present; *dfs*	0.955	13.800	0.004
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A4.2

Multilevel Models of T1 Child and Family Effects on T2 Child OPP/CP (with WAR)

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	-.41 (.24)	-.36 (.27)	-.35 (.27)	-1.17 (.42)
Child-level predictors				
T1 child ADHD	.75 (.37)*	.73 (.36)*	.71 (.35)*	.57 (.35)
T1 child OPP/CP	.65 (.10)***	.64 (.10)***	.64 (.10)***	.63 (.10)***
Child gender		-.11 (.32)	-.11 (.32)	-.19 (.31)
T1 age			.07 (.14)	-.00 (.05)
T1 maternal WAR				.55 (.27)*
Variance estimates				
Child effects	1.03 (.22)***	1.02 (.22)***	1.02 (.21)***	1.00 (.21)***
Family effects	1.03 (.34)***	1.04 (.34)***	1.04 (.34)***	.90 (.31)***
-2* log likelihood	307.334	307.201	307.014	301.814
Change in model fit (χ^2)		0.133	0.187	5.200*
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; WAR = warmth. Standard errors are in parentheses.

*p < .05. ***p < .001.

Table A4.2 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	-3.43 (1.22)	-3.23 (1.27)	-3.21 (1.29)	-3.28 (1.29)
Child-level predictors				
T1 child ADHD	.61 (.36)†	.54 (.35)	.55 (.36)	.62 (.36)
T1 child OPP/CP	.58 (.10)***	.57 (.10)***	.57 (.10)***	.57 (.10)***
Child gender	-.17 (.31)	-.03 (.06)	-.39 (.42)	-.33 (.45)
T1 age	-.03 (.06)	-.03 (.06)	-.03 (.06)	-.02 (.06)
T1 maternal WAR	.82 (.28)**	.88 (.28)**	.88 (.28)**	.67 (.42)†
Change in maternal WAR	.61 (.30)*	.63 (.30)*	.62 (.31)*	.63 (.31)*
Family-level predictors				
All-boy sib-pair		-.54 (.43)	-.54 (.43)	-.53 (.44)
All-girl sib-pair			-.13 (.61)	-.18 (.63)
T1 sib-pair WAR				.14 (.21)
Variance estimates				
Child effects	1.00 (.21)***	1.00 (.21)***	1.00 (.21)***	.99 (.21)***
Family effects	.75 (.28)***	.71 (.28)***	.71 (.28)***	.71 (.27)***
-2* log likelihood	296.847	294.994	294.953	294.653
Change in model fit (χ^2)	4.967*	1.853	0.041	0.300
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; WAR = warmth. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A4.2 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	-3.28 (1.30)	-3.51 (1.14)	-3.58 (1.13)
Child-level predictors			
T1 child ADHD	.62 (.36)	.54 (.36)	.52 (.36)
T1 child OPP/CP	.57 (.10)***	.58 (.11)***	.58 (.11)***
Child gender	-.33 (.45)	-.36 (.45)	-.37 (.45)
T1 age	-.02 (.06)	-.02 (.06)	-.02 (.06)
T1 maternal WAR	.69 (.42)†	.76 (.43)†	.78 (.43)†
Change in maternal WAR	.68 (.31)*	.77 (.31)*	.81 (.31)*
Family-level predictors			
All-boy sib-pair	-.52 (.44)	-.47 (.40)	-.48 (.40)
All-girl sib-pair	-.17 (.63)	.22 (.61)	.28 (.61)
T1 sib-pair WAR	.13 (.22)	.04 (.23)	.02 (.23)
Change in sib-pair WAR	-.07 (.29)	-.04 (.26)	-.06 (.26)
Maternal ADHD symptoms		.06 (.01)***	.06 (.01)***
Maternal DEP symptoms			.03 (.05)
Variance estimates			
Child effects	1.00 (.21)***	1.01 (.21)***	1.01 (.21)***
Family effects	.70 (.27)***	.38 (.21)***	.36 (.21)***
-2* log likelihood	294.612	281.225	280.920
Change in model fit (χ^2) from prior to present; *dfs*	0.041	13.387**	0.305
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; WAR = warmth; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ 1.00 (marginally significant).

Table A4.3

Multilevel Models of T1 Child and Family Effects on T2 Child OPP/CP (with CC)

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	-.41 (.24)	-.36 (.27)	-.35 (.27)	-.50 (.29)
Child-level predictors				
T1 child ADHD	.75 (.37)*	.73 (.36)*	.71 (.35)*	.57 (.36)
T1 child OPP/CP	.65 (.10)***	.64 (.10)***	.64 (.10)***	.61 (.10)***
Child gender		-.11 (.32)	-.11 (.32)	-.19 (.33)
T1 age			.07 (.14)	.02 (.05)
T1 maternal CC				.28 (.20)
Variance estimates				
Child effects	1.03 (.22)***	1.02 (.22)***	1.02 (.21)***	1.02 (.22)***
Family effects	1.03 (.34)***	1.04 (.34)***	1.04 (.34)***	.97 (.33)***
-2* log likelihood	307.334	307.201	307.014	305.393
Change in model fit (χ^2)		0.133	0.187	1.621
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; CC = critical comments. Standard errors are in parentheses.

*p < .05. ***p < .001.

Table A4.3 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	-.88 (.32)	-.87 (.37)	-.88 (.37)	-.88 (.45)
Child-level predictors				
T1 child ADHD	.56 (.34)	.47 (.35)	.45 (.36)	.46 (.36)
T1 child OPP/CP	.45 (.12)***	.39 (.11)**	.39 (.11)**	.39 (.12)**
Child gender	-.19 (.31)	-.35 (.36)	-.37 (.38)	-.37 (.39)
T1 age	-.00 (.05)	-.01 (.05)	-.01 (.05)	-.01 (.05)
T1 maternal CC	.70 (.21)**	.89 (.20)***	.90 (.20)***	.90 (.23)***
Change in maternal CC	-.19 (.04)***	-.23 (.04)***	-.23 (.04)***	-.23 (.04)***
Family-level predictors				
All-boy sib-pair		-.64 (.41)	-.64 (.40)	-.64 (.40)
All-girl sib-pair			.14 (.57)	.14 (.61)
T1 sib-pair CC				.01 (.36)
Variance estimates				
Child effects	.77 (.16)***	.78 (.16)***	.78 (.16)***	.78 (.16)***
Family effects	1.06 (.31)***	.64 (.23)***	.64 (.23)***	.64 (.23)***
-2* log likelihood	291.040	276.554	276.500	276.500
Change in model fit (χ^2)	14.353**	14.486**	0.054	0.000
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; CC = critical comments. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

Table A4.3 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	-1.03 (.46)	-.86 (.46)	-.74 (.47)
Child-level predictors			
T1 child ADHD	.48 (.35)	.40 (.36)	.36 (.34)
T1 child OPP/CP	.43 (.12)**	.45 (.12)**	.45 (.12)***
Child gender	-.36 (.39)	-.37 (.39)	-.38 (.40)
T1 age	-.00 (.05)	-.00 (.05)	-.00 (.05)
T1 maternal CC	.79 (.24)**	.78 (.24)**	.80 (.24)**
Change in maternal CC	-.19 (.05)***	-.19 (.05)***	-.19 (.05)***
Family-level predictors			
All-boy sib-pair	-.68 (.40)	-.62 (.35)	-.66 (.36)
All-girl sib-pair	.19 (.61)	.52 (.58)	.67 (.58)
T1 sib-pair CC	.18 (.37)	.01 (.36)	-.09 (.37)
Change in sib-pair WAR	-.12 (.10)	-.11 (.08)	-.14 (.08)
Maternal ADHD symptoms		.06 (.01)***	.05 (.01)***
Maternal DEP symptoms			.08 (.05)
Variance estimates			
Child effects	.76 (.16)***	.77 (.16)***	.77 (.16)***
Family effects	.62 (.23)***	.35 (.17)***	.31 (.17)***
-2* log likelihood	274.703	260.574	258.555
Change in model fit (χ^2) from prior to present; *dfs*	1.797	14.129**	2.019
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; OPP/CP = oppositional/conduct problems; CC = critical comments. Standard errors are in parentheses.

**p < .01. ***p < .001.

Table A4.4

Multilevel Models of T1 Child and Family Effects on T2 Child Emotional Problems

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	-1.10 (.30)	-1.10 (.36)	-1.10 (.36)	-1.06 (.36)
Child-level predictors				
T1 child ADHD	2.03 (.33)***	2.03 (.36)***	2.02 (.37)***	1.83 (.42)***
T1 child EP	.09 (.13)	.09 (.13)	.09 (.13)	.10 (.14)
Child gender		-.00 (.38)	-.00 (.38)	-.12 (.40)
T1 age			.03 (.21)	-.02 (.22)
T1 maternal EE				.55 (.41)
Variance estimates				
Child effects	1.44 (.30)***	1.44 (.30)***	1.44 (.30)***	1.50 (.32)***
Family effects	1.40 (.47)***	1.40 (.47)***	1.40 (.47)***	1.22 (.45)***
-2* log likelihood	336.941	336.941	336.924	335.635
Change in model fit (χ^2)		0.00	0.017	1.289
from prior to present; *dfs*		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; EP = emotional problems; EE = expressed emotion. Standard errors are in parentheses.

***p < .001.

Table A4.4 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	-1.55 (.57)	-1.49 (.59)	-1.33 (.59)	-1.66 (.59)
Child-level predictors				
T1 child ADHD	1.75 (.42)***	1.72 (.45)***	1.93 (.42)***	2.23 (.40)***
T1 child EP	.09 (.14)	.09 (.14)	.09 (.14)	.09 (.13)
Child gender	-.11 (.39)	-.16 (.44)	.19 (.42)	.44 (.40)
T1 age	-.05 (.23)	-.05 (.23)	-.05 (.23)	.03 (.21)
T1 maternal EE	.88 (.57)	.93 (.60)	.81 (.60)	-.08 (.70)
Change in maternal EE	.47 (.33)	.48 (.34)	.34 (.35)	.27 (.35)
Family-level predictors				
All-boy sib-pair		-.10 (.46)	-.19 (.46)	-.23 (.46)
All-girl sib-pair			-2.07 (.58)**	-2.43 (.56)***
Overall family EE				.98 (.42)*
Variance estimates				
Child effects	1.53 (.32)***	1.54 (.33)***	1.49 (.31)***	1.40 (.30)***
Family effects	1.14 (.43)***	1.11 (.43)***	.92 (.38)***	.89 (.37)***
-2* log likelihood	334.656	334.617	327.415	322.805
Change in model fit (χ^2)	0.979	0.039	7.202**	4.610*
from prior to present; *dfs*	1	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; EP = emotional problems; EE = expressed emotion. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A4.4 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	-1.88 (.63)	-1.75 (.67)	-1.64 (.57)
Child-level predictors			
T1 child ADHD	2.26 (.39)***	2.14 (.41)***	2.17 (.42)***
T1 child EP	.09 (.13)	.10 (.13)	.10 (.13)
Child gender	.44 (.40)	.38 (.41)	.39 (.41)
T1 age	.04 (.21)	.03 (.21)	.04 (.21)
T1 maternal EE	-.20 (.73)	-.05 (.75)	-.09 (.77)
Change in maternal EE	.10 (.49)	.20 (.50)	.17 (.51)
Family-level predictors			
All-boy sib-pair	-.27 (.45)	-.24 (.42)	-.22 (.42)
All-girl sib-pair	-2.34 (.63)**	-2.01 (.61)**	-2.22 (.66)**
Overall family EE	1.15 (.46)*	1.02 (.47)*	1.13 (.49)*
Change in overall family EE	.32 (.58)	.17 (.57)	.04 (.56)
Maternal ADHD symptoms		.06 (.02)**	.07 (.02)***
Maternal DEP symptoms			-.07 (.05)
Variance estimates			
Child effects	1.39 (.29)***	1.41 (.30)***	1.40 (.30)***
Family effects	.89 (.37)***	.54 (.30)***	.52 (.30)***
-2* log likelihood	322.436	311.721	310.760
Change in model fit (χ^2) from prior to present; *dfs*	0.369	10.715**	0.961
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; EP = emotional problems; EE = expressed emotion. Standard errors are in parentheses.

*p < .05. **p < .01. ***p < .001.

Table A4.5

Multilevel Models of T1 Child and Family Effects on T2 Child Emotional Dysregulation Problems

Parameter	Model 1	Model 2	Model 3	Model 4
Fixed effects estimates				
Intercept	61.47 *3.43*	62.52 *4.31*	62.48 *4.27*	62.49 *4.26*
Child-level predictors				
T1 child ADHD	15.25 *4.30***	14.67 4.82**	14.64 *4.76***	14.37 *5.04***
T1 child EDP	.43 *.17**	.42 *.17**	.45 *.17**	.45 *.17***
Child gender		-2.23 *4.00*	-2.05 *3.95*	-2.22 *4.10*
T1 age			-.74 *.67*	-.77 *.72*
T1 maternal EE				1.00 *4.40*
Variance estimates				
Child effects	146.75 *30.94****	148.57 *31.32****	145.19 *30.61****	145.66 *30.71****
Family effects	107.64 *41.18*	102.93 *40.51*	104.82 *40.41*	103.81 *40.28*
-2* log likelihood	745.031	744.634	743.646	743.596
Change in model fit (χ^2) from prior to present; *dfs*		0.397	0.988	0.050
		1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; EDP = emotional dysregulation problems. *Standard errors* are in italics.

*p < .01, **p < .01. ***p < .001.

Table A4.5 (continued)

Parameter	Model 5	Model 6	Model 7	Model 8
Fixed effects estimates				
Intercept	56.47 *5.93*	56.34 *6.37*	57.13 *6.02*	56.17 *6.14*
Child-level predictors				
T1 child ADHD	13.84 *5.04***	13.90 *5.25**	15.71 *4.98***	16.54 *5.09***
T1 child EDP	.43 *.17**	.43 *.17**	.42 *.16**	.43 *.16**
Child gender	-2.11 *3.96*	-1.98 *4.51*	.71 *4.58*	1.57 *4.82*
T1 age	-.85 *.77*	-.85 *.76*	-.84 *.76*	-.75 *.76*
T1 maternal EE	4.86 *5.88*	4.76 *5.98*	3.66 *6.07*	.60 *7.57*
Change in maternal EE	5.61 *3.91*	5.57 *3.96*	4.52 *4.10*	4.22 *4.07*
Family-level predictors				
All-boy sib-pair		.26 *4.49*	-.14 *4.43*	-.23 *4.45*
All-girl sib-pair			-14.04 *5.69**	-15.21 *6.22**
Overall family EE				3.24 *5.19*
Variance estimates				
Child effects	150.24 *31.67****	150.05 *31.63****	146.41 *30.86****	144.98 *30.56****
Family effects	90.93 *38.42*	91.23 *38.45*	84.22 *36.60*	84.70 *36.49*
-2* log likelihood	742.206	742.203	738.639	738.134
Change in model fit (χ^2) from prior to present; *dfs*	1.390	0.003	3.564	0.505
	1	1	1	1

Note. See above.

Table A4.5 (continued)

Parameter	Model 9	Model 10	Model 11
Fixed effects estimates			
Intercept	51.71 (6.70)	52.79 (6.27)	53.27 (6.22)
Child-level predictors			
T1 child ADHD	16.98 (5.08)**	15.89 (5.31)**	16.04 (5.31)**
T1 child EDP	.45 (.16)**	.46 (.16)**	.46 (.16)**
Child gender	1.78 (4.81)	1.33 (4.88)	1.39 (4.84)
T1 age	-.69 (.74)	-.72 (.75)	-.72 (.75)
T1 maternal EE	-2.24 (7.90)	-.10 (8.08)	-1.18 (8.12)
Change in maternal EE	.52 (5.54)	1.48 (5.66)	1.33 (5.68)
Family-level predictors			
All-boy sib-pair	-.97 (4.51)	-.60 (3.96)	-.51 (3.95)
All-girl sib-pair	-13.22 (6.76)†	-10.18 (6.88)	-11.09 (7.10)
Overall family EE		5.85 (5.73)	6.33 (5.72)
Change in overall family EE		5.55 (5.52)	5.03 (5.46)
Maternal ADHD symptoms		.59 (.13)***	.63 (.15)***
Maternal DEP symptoms			-.32 (.70)
Variance estimates			
Child effects	141.91 (29.92)***	143.03 (30.15)***	142.83 (30.11)***
Family effects	83.50 (35.83)***	51.69 (30.03)***	51.48 (29.97)***
-2* log likelihood	736.379	726.566	726.393
Change in model fit (χ^2) from prior to present; *dfs*	1.755	9.813**	0.173
	1	1	1

Note. ADHD = attention-deficit/hyperactivity disorder; EDP = emotional dysregulation problems; DEP = depressive symptoms. Standard errors are in parentheses.

*p < .01, **p < .01. ***p < .001. †p ≤ .10 (marginally significant).

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