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The Influence of Parental Depression, Interparental Conflict and Parent-Child Hostility on the Development of Psychopathology in Children and Adolescents

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Declaration

I hereby declare that this thesis has not been and will not be submitted in whole or in part to another University for the award of any other degree.

Kate Arnold

17th March 2016

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Summary

Parental depression has been highlighted as a major risk factor for the development of psychopathology in children and adolescents (Mars et al, 2012; Sellers et al, 2013). Garber & Martin (2002) suggest that the primary environmental stressor that children living with depressed parents are exposed to relates to the impact of maternal depression on marital conflict. Interparental conflict has been demonstrated to have negative effects on various aspects of family functioning and relationships. Strong associations between conflict levels in parental relationships and negative parent-child relationships support this theory (Erel & Burmann, 1995). The parent- child relationship is viewed as a central mechanism for the transmission of psychopathology across generations of a family (Rutter et al., 2010).

This thesis examines the relationship between parental depression and family processes (specifically interparental and parent-child relationships) on the development of depressive and aggressive symptoms in children and adolescents. Four data sets examine these associations: a high risk sample of parents with recurrent depression and their adolescent children (Early Prediction of Adolescent Depression, EPADS), a community sample of low-risk adolescents and their families (Welsh Family Study, WFS), a sample of families who have conceived children using Artificial Reproductive Technologies (the Cardiff IVF Study, C-IVF) and a longitudinal adoption study (Early Growth and Development Study, EGDS). Findings from this thesis presents evidence that a process exists by which parental depressive symptoms contribute to the development of psychopathology in children and adolescents through disrupted interparental and parent-child relationships. Parental depression was consistently associated with interparental conflict, which in turn was associated with higher levels of parent-child hostility. For child outcomes, the most consistent finding was the

association between parent-child hostility and child and adolescent symptoms of aggression. This was observed in all four data sets, in a variety of samples and across a range of developmental periods. The association was observed where adolescents were classed as being at either a high or low-risk for the development of psychopathology, and for those families where parents were rearing genetically related and genetically unrelated children.

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Chapter 1: General Introduction

The current thesis will examine associations of parental depression, interparental conflict and hostile parent-child relationships with the development of depressive and aggressive symptoms in children and adolescents. This introductory chapter will first discuss the presence of psychopathology in children and adolescents, establishing why it is important to examine risk factors for the development of mental health problems. Biological and environmental explanations and theories for the development of child and adolescent psychopathology will then be reviewed, with a detailed focus on the associations of parental depression, interparental conflict and parent-child hostility with the development of depressive and aggressive symptoms in children and adolescents. Methodological issues relating to previous research into the influence of indices of family relationships (interparental conflict and parent-child relationships) on the development of negative child outcomes will be discussed, providing a review of a range of existing research designs that have examined these associations. A summary will be provided to highlight what we have learnt from the current literature on the associations of parental depression, interparental conflict and parent-child hostility with depressive and aggressive symptoms in children and adolescents. Finally, this chapter will outline the studies which will be presented in this thesis.

Child and adolescent psychopathology

It is increasingly common for children and adolescents to experience problems with their mental health. Conservative estimates for the prevalence of psychopathology in this age group estimate that 1 in 10 children will experience a mental health problem that involves a level of distress or social impairment that is likely to warrant treatment (Tamsin Ford, Goodman, & Meltzer, 2003). More recent reviews suggest that as many

as 1 in 5 young people, under the age of 18 are affected by mental health problems (Costello, Copeland, & Angold, 2011; Costello, Egger, Copeland, Erkanli & Angold, 2011). The presence of psychopathology in children and adolescents is an international concern, as demonstrated by the number of countries that have attempted to provide prevalence rates, including the United Kingdom (Green, McGinnity, Meltzer, Ford, & Goodman, 2005), the United States (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012), New Zealand (Fergusson & Horwood, 2001), Germany (Beesdo-Baum, Knappe, Asselmann, Zimmerman, et al., 2015) and Canada (Romano, Tremblay, Vitaro, Zoccolillo, & Pagani, 2001), as well as meta-anlayses of studies that have considered the global prevalence rates of psychopathology in children and adolescents (Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015). Estimates for prevalence rates from these studies range between 12% (Green et al., 2005) to 38% (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) of children and adolescents experiencing a mental health problem through the course of their childhood. Variance in estimates for the prevalence of mental health problems in young people might be a result of studies focusing on different age groups or stages of childhood, and/or using a variety of assessments and different definitions of what constitutes a mental health problem (Polanczyk et al., 2015).

The presence of psychopathology in children and adolescents has been well documented within empirical literature and research (Collishaw, Maughan, Goodman, & Pickles, 2004; Rubin & Pepler, 2013). There are a range of mental health problems that this age group might experience including depression (Lewis, Collishaw, Thapar, & Harold, 2014; Rudolph & Lambert, 2009), self-harm (Hawton & James, 2005), generalised anxiety disorder (Mondin et al., 2013), post-traumatic stress disorder

(Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012), conduct disorders (Kim-Cohen et al., 2005), Attention Deficit Hyperactivity Disorder (Daniels, MacLure, & Youdell, 2015), eating disorders (Frank, Hagman, & Solomon, 2015) and developmental disabilities such as Autism Spectrum Disorders (Simonoff et al., 2008). The most commonly reported mental health problems in adolescence are behavioural disorders, anxiety disorders, and depression (Costello et al., 2011; Kessler et al., 2011; Rutter, 2007), with UK prevalence estimates of 7.0%, 4.4%, and 1.3% respectively (Green, McGinnity, Meltzer, Ford, & Goodman, 2004). Research into child and adolescent mental health problems attempt to explore why some children develop psychopathologies and which factors might increase or decrease a child's risk of developing these symptoms or disorders.

Distinctions in child psychopathology are typically made between internalising (emotional) and externalising (behavioural) problems, both of which have behavioural and affective components (Cummings & Davies, 1994; Grych & Fincham, 2001; Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). An overview of these problems will be provided before more specific descriptions of the outcomes assessed in the current thesis: depressive and aggressive symptoms in children and adolescents. Following this, an outline of possible risk factors and mechanisms will be examined.

Internalising problems

Internalising problems are often conceptualised as 'emotional' difficulties (Reynolds, Houlston, Coleman, & Harold, 2014) but also feature behaviours including withdrawal, fearfulness and inhibition (Eisenberg et al., 2001). Individuals who experience internalising problems appear to direct negative emotions inwardly rather than at others (Roeser, Eccles, & Sameroff, 1998) and appear to experience strong self-regulation (Causadias, Salvatore, & Sroufe, 2012). Internalising problems that children

and adolescents are affected by are related to fearfulness, anxiousness, shyness, sadness and low self-esteem (Ollendick & King, 1994; Zahn-Waxler et al., 2000). As children develop through childhood and adolescence, the presence of these problems increases (Kessler et al., 2012; Merikangas et al., 2003).

Characteristics associated with anxiety disorders include an excessive amount of worry, fatigue, restlessness, irritability and problems with focussing and concentrating (American Psychological Association, 2013). Common anxiety disorders among children and adolescents are often related to problematic attachments formed with caregivers, family members and peers (American Psychological Association, 2013). The presence of anxiety disorders in children and adolescents has been well documented within empirical literature since the late 19th century (Costello, Egger, Copeland, Erkanli, Angold, 2011; Klein & Last, 1989).

Anxiety disorders appear more frequently in children than depression or behavioural disorders and have thus been frequently examined (Cartwright-Hatton, McNicol, & Doubleday, 2006; Silverman & Field, 2011). Conservative estimates for the prevalence rates of anxiety disorders state that around 3% of children experience an anxiety disorder at some point during their pre-adolescent period. Some community studies consider anxiety disorders to be even more common in children and adolescents, with prevalence estimates ranging between 9% to 32% (Essau & Ollendick, 2013; Fox, Halpern, & Forsyth, 2008; Merikangas et al., 2003). Anxiety disorders increase during adolescence with a review of findings from the Christchurch Health and Development Study (Fergusson & Horwood, 2001) reporting that at age 15, 18% of the female sample had an anxiety disorder (which increased to over 22% by 18), and almost 7% of the male sample experienced anxiety problems at age 15 (which increased to almost 12% by age 18), although types of anxiety may differ across different developmental periods.

There is also evidence that anxiety disorders in children and adolescents persist into adulthood and might influence maladaptive behaviours that can lead to the increase of psychopathology over time (Kasen, Cohen, Skodol, Johnson, & Brook, 1999). Adult anxiety disorders can not only be seriously debilitating but are often associated with substance abuse disorders, depression and suicidality (Crum & Pratt, 2001; Grant et al., 2004; Placidi et al., 2000). It is therefore important to identify anxiety symptoms and disorders in children so that support can be offered to the child and family and to reduce the risks associated with the continuation of anxiety disorders into adulthood (Beesdo, Knappe, & Pine, 2009).

The term depression refers to an overall lowering of functioning (National Collaborating Centre for Mental Health, 2005). It is characterised by sadness, low mood, loss of interest in activities and decreased energy (Cieza et al., 2004). Although instances of depression in childhood seem to have been documented since the 17th century (Rao et al., 2009), early researchers and practitioners considered it to be more of an 'adult disease'. Depression in children is now more accepted and our understanding of it has progressed from simply applying theories developed for depression in adults to children, to recognising the unique aetiology and prevalence of depression in childhood and adolescence.

There is a distinction between depressed moods which can be measured by symptoms on a continuous scale, and depressive disorders which are formal categorical measurements of depressive symptoms (Flett, Vredenburg, & Krames, 1997; Garber & Horowitz, 2008). Within a clinical context, depressive disorders refer to a group of behaviours and symptoms that cluster around three changes of experience: changes in mood, changes in cognitions/thinking and changes in activity that result in significantly impaired functioning in social, occupational, or educational contexts (National

Collaborating Centre for Mental Health, 2005). A range of negative outcomes and diminished social and psychological functioning have been associated with even moderate levels of depressive symptoms (Lewinsohn, Solomon, Seeley, & Zeiss, 2000). The current thesis will be focusing on symptoms of depression in children and adolescents, as opposed to considering instances of depressive disorders within the samples.

Children and adolescents who suffer from depression and display depressive symptomology are characterised as having low moods (Lau & Eley, 2010), a lack of enthusiasm (Huberty, 2012) and as being frequently socially withdrawn from family members and peers (Witvliet, Brendgen, van Lier, Koot, & Vitaro, 2010). Depression in children and adolescence is also associated with a number of harmful and destructive behaviours, including substance abuse, cigarette smoking, risky sexual behaviours, physical health problems, and increases in suicidal ideation with a thirty-fold increased risk of suicide completion (Birmaher et al., 1996; Brent et al., 1998; Stolberg, Clark, & Bongar, 2002.; Thapar, Collishaw, Pine, & Thapar, 2012). Children who experience depression are also likely to suffer impairments in several educational areas (Kovacs & Devlin, 1998) such as basic academic skills, language development, executive functioning abilities, memory and coordination (Jacobs, Reinecke, Gollan, & Kane, 2008). They are also more likely to have lower levels of self-esteem (Orth, Robins, & Widaman, 2012) and increased incidences of social anxiety (Wright, Banerjee, Hoek, Rieffe, & Novin, 2010).

Children identified as having early onset depression are at a higher risk of later depressive episodes in adolescence and adulthood (Emslie et al., 1997; Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Maria Kovacs & Devlin, 1998; Lewinsohn, Rohde, Klein, & Seeley, 1999; Rao, Hammen, & Daley, 1999), with evidence that these

early signs and symptoms of depression show strong continuities with clinical depression and anxiety disorders in adulthood (Fombonne et al., 2001; Pine, Cohen, Cohen, & Brook, 1999). Depression in childhood can lead to serious functional and developmental problems (Rao & Chen, 2009). The prevalence of depression in adolescents aged between 13 years to 18 years is 11.2%, and it is thus viewed as one of the most prevalent disorders experienced by this age group (Merikangas et al., 2010). Depressive symptoms within adolescent populations are associated with long term maladjustment and interpersonal problems (Aalto-Setälä, Marttunen, Tuulio-Henriksson, Poikolainen, & Lönnqvist, 2002) and these problems might be more severe in instances where depressive symptoms co-occur with conduct problems (McCarty et al., 2013). Associations between adolescents who struggle with these issues and problems as they enter adulthood have been found: they are likely to experience higher levels of unemployment, lower incomes, have greater problems securing stable housing arrangements and are less likely to cohabit properties (Fombonne et al., 2001). These factors all seem to be related to a social drift that, according to Fombonne and colleagues, is a direct consequence of childhood psychopathology.

Evidence has demonstrated that paediatric depression negatively impacts society in conjunction with the individuals and families directly affected by it. Children and adolescents who experience depression have a heightened use of medical services (Angold, Costello, Farmer, Burns, & Erkanli, 1999; Lynch & Clarke, 2006) which can place an increased strain on health care facilities. The recognition of the individual and societal burden of depression has resulted in an increased interest in research associated with the aetiology of depression in children and adolescence (Horowitz & Garber, 2006). To effectively tackle the growing rates of depression, it is prudent to understand how depressive symptoms develop.

Historically, developmental psychologists considered children too developmentally immature to experience symptoms of depression (Luby, 2010). Since the 1980s however, studies began to demonstrate that children as young as 6 years could display depressive symptoms which might even be severe enough to manifest as a clinical diagnosis (Carlson & Cantwell, 1980). Although our understanding of depression within this age group had progressed, there still seem to be gaps in our knowledge of how it might develop and manifest. More recent studies examining the presence of depressive symptoms in children aged between 3 years and 6 years have demonstrated that even children within this developmental period can experience depression (Luby, 2010; Stalets & Luby, 2006). However, depressive symptoms within this age group are far less frequently reported than within adolescents. Fuhrmann et al (2013) assessed the prevalence of depressive symptoms in a large, population-based sample of preschool children (N = 653, Mean Age = 6.2 years). They found that 5.7% of the sample presented with depressive symptoms with no gender differences between girls and boys. Depressive symptoms show a strong continuity to depressive disorders (Rice, Harold, & Thapar, 2002) and it is widely accepted that a continuum exists on which both symptoms and clinical diagnosis of depression show both concurrent and future impairment (Costello et al., 2003).

Externalising problems

Externalising problems are characterised by harmful, disruptive and disinhibited behaviours that can include aggression, rule-breaking, and oppositional behaviours (Ford et al., 2003; Kovacs & Devlin, 1998). Increased incidences of aggressive and antisocial behaviour problems in children and adolescents have been reported globally (Collishaw et al., 2004). These behaviours typically manifest as an individual's outward behaviour to their external environment (Campbell, Shaw, & Gilliom, 2000). Common

externalising problems in children are aggression, hostility, antisocial behavioural issues, non-compliant behaviours, delinquency and vandalism (Erath & Bierman, 2006). These are often characterised as disruptive, hyperactive and aggressive behaviours within empirical literature (Hinshaw, 1987). Children and adolescents with externalising problems often direct negative emotions, that manifest as anger, aggression and frustration towards others (Roeser et al., 1998; Zahn–Waxler, Klimes-Dougan, & Slattery, Marcia, 2000). Externalising problems are highly prevalent amongst children and adolescents (Liu, 2004), with estimates indicating that between 10 to 20 % of young people experience externalising behaviours (Belfer, 2008; World Health Organization, 2001). Males are more likely to exhibit externalising behaviour problems or disorders than females (Costello et al., 2003; Rescorla et al., 2007).

Children can experience difficulties as a result of their problematic behaviours; children who exhibit externalising behaviours often experience peer victimisation (van Lier et al., 2012), academic difficulties (Masten et al., 2005) and higher incidences of conflictual relationships with their parents (Burt, McGue, Krueger, & Iacono, 2005). Young people who experience externalising problems are also at a greater risk for developing internalising problems (Boylan, Georgiades, & Szatmari, 2010). Externalising disorders in childhood present a major risk factor for delinquency, crime and violence in adulthood (Betz, 2009; Farrington, 1991; Moffitt, 1993).

Externalising problems are often associated with antisocial behaviours (Liu, 2004). Within children however, the term externalising behaviour is more commonly used to refer to less severe, disruptive and destructive behaviours (Shaw & Winslow, 1997). A distinction between internalising and externalising problems is the presence of hyperactivity in externalising problems (Liu, 2004). Problems with attention and hyperactivity are a specific subtype of externalising problems that the American

Psychological Association (2013) refer to as Attention Deficit/Hyperactivity Disorder (ADHD). ADHD is characterised as a pattern of behaviours related to the frequent occurrence of inattention, hyperactivity or impulsivity and is primarily identified as a neurodevelopmental disorder (Thapar et al., 2012), though the symptoms that are used to define its presence are consistent with those that are also used to identify externalising problems (Reynolds & Kamphaus, 2004; Reynolds, 2004). Estimates for the prevalence of ADHD in children and adolescents are high; it is estimated that 5% of children in the United Kingdom have ADHD (McCarthy et al., 2012) with higher instances reported in the United States, where five million children under the age of 17 years (8.8% of the population) are diagnosed with ADHD (Visser et al., 2014). Externalising problems and antisocial behaviours increase during adolescence (Rutter, 2007), with males being more likely to be aggressive and antisocial than female adolescents (Lewinsohn et al., 1993; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004; McDermott, 1996).

The presentation of aggression can vary across different developmental periods. Aggression typically refers to verbal and physical behaviours that are either threatening or deliberately intended to cause harm towards others (Nagin & Tremblay, 1999). In young children however, the intent to hurt individuals might not be a component of the behaviour (Wakschlag, Tolan, & Leventhal, 2010). Aggressive behaviours in younger children might be a result of fear or anger in specific situations, or a result of underdeveloped self-regulation skills or frustration at unmet needs (Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996; Wakschlag et al., 2010).

Aggression as a behavioural response might not always be an abnormal reaction to specific situations. Aggressive behaviours are observed in most species and, in certain contexts and situations, these behaviours might be viewed as a relatively normal

reaction to ensure individual survival (Haller, Mikics, Halász, & Tóth, 2005). The normality of aggression appears to be constricted by specific moral rules and official laws which children must be taught to allow them to fully understand the appropriateness of their responses. Abnormal aggressive behaviours are defined by their inappropriateness, frequency, prolonged expression and by the burden placed on families and peers involved with the negative behaviours (Haller et al., 2005). It is thus very common for children to exhibit aggressive behaviours; in fact it is rarer for children under the age of three years to not exhibit physical aggression (Buchmann, Hohmann, Brandeis, Banaschewski, & Poustka, 2014). However, persistent aggression has been associated with several negative outcomes for young people as they transition into adulthood including higher levels of unemployment, greater instances of criminal behaviours and negative consequences on socioeconomic status (Buchmann et al., 2014).

A child displaying severe symptoms of aggressive behaviours might be diagnosed with conduct disorders (CD), or oppositional defiant disorders (ODD) (Frick, 2012). A young person that has been identified as having a CD displays behaviours that infringe on the rights of others; seriously violate societal rules or norms; involve aggressive behaviours directed towards people or animals; lead to the destruction of property; and commonly involve deceitfulness and theft (American Psychological Association, 2013). During childhood, the prevalence of conduct disorders might be as high as 9.5% (Nock, Kazdin, Hiripi, & Kessler, 2007), though general population studies report rates ranging from less than 1% to more than 10% (American Psychological Association, 2013) with a higher percentage of these children being identified as male (Nock et al., 2007). Children with ODD will display negative, hostile and defiant behaviours, which are typically directed toward a child's parents or teachers

(Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). This disorder usually becomes evident before the age of 8 years, and rarely later than early adolescence (American Psychological Association, 2013). It represents a pattern of behaviour that lasts at least six months where children lose their temper, are argumentative with adults, appear easily annoyed yet frequently and deliberately annoy others, and also includes behaviours that can be viewed as spiteful or vindictive. The prevalence rate for oppositional defiant disorder is 10.2% (Nock et al., 2007). Oppositional defiant disorder is regarded by some as less serious than aggressive and delinquent disorders in children as it appears to be more related to defying or refusing to comply with adult's requests than using physical aggression. Moffitt's developmental taxonomy (1993) proposes that antisocial acts are committed by two distinct groups of people: the first is a persistent group who have early onsets of antisocial behaviour which continues throughout their life and often lead to individuals becoming life-long offenders. The second group is considered to be much larger and has been described as the 'adolescent limited' group, who might offend during their teenage years but mostly reform once they mature into adults. In terms of developmental pathways, early incidences of externalising problems might be predictive of the more problematic 'persistent' offenders as children who display delinquent behaviours prior to adolescence are two to three times more likely to become chronic violent offenders compared to those with a later onset (Loeber & Farrington, 2001).

Theories for the development of child and adolescent psychopathology

The current thesis will be specifically focussing on the associations of family socialisation and parental depression with depressive and aggressive symptoms in children and adolescents, as these have been identified as two commonly occurring mental health problems within in this age group (Costello, Copeland, & Angold, 2011b;

Kessler et al., 2005; Merikangas et al., 2010). Empirical evidence has offered a representation of the prevalence of mental health problems present in childhood and adolescence, which have hence been associated with not only concurrent, but also with future impairments. It is therefore important to study the development of mental health problems within these age groups and developmental periods to better understand the aetiology of psychopathology.

Several vulnerabilities might influence the emergence of depressive and aggressive symptoms within children and adolescents, including biological vulnerabilities e.g. genetic factors and pubertal hormones (Hyde, Mezulis, & Abramson, 2008; Sisk & Zehr, 2005), affective vulnerabilities e.g. temperament (Elovainio et al., 2015; Jessee, Mangelsdorf, Shigeto, & Wong, 2012; Lemery, Essex, & Smider, 2002), cognitive vulnerabilities e.g. negative cognitive styles (Dunbar et al., 2013) and negative life events (Johnson, Whisman, Corley, Hewitt, & Rhee, 2012).

Within the current thesis, the development of mental health problems in children and adolescents will be examined using a developmental psychopathology approach, which proposes that to understand human development, developmental processes such as biological, psychological, and environmental mechanisms must be integrated to fully understand the complexity involved in the aetiology of these problems (Cicchetti & Toth, 1998). Both biological and environmental explanations for the development of depressive and aggressive symptoms in children and adolescents will now be briefly reviewed, although in contemporary literature it is more commonly accepted that multiple factors contribute to the development of depressive and aggressive symptoms in children, involving an interaction between genes and the environment. It can still, however, be beneficial to review biological and environmental factors in isolation, to better inform our understanding of how they might interact with one another.

Theoretical review of biological explanations for the development of psychopathology

Biological explanations for the development of psychopathology in children and adolescents include biochemical and neuropsychological factors, functional and anatomical brain differences, genetic predispositions or vulnerabilities and hormonal changes during puberty (Hyde et al., 2008). An overview of biological explanations for child and adolescent psychopathology will be provided, including an examination of the associations of psychopathologies with early biological risk factors, neurotransmitters, structural brain differences, physiological associations, genetic factors associated with psychopathology and an overview of the intergenerational transmission of psychopathology.

Early biological risk factors associated with child and adolescent psychopathology

Biological explanations for the development of psychopathology can include pre/perinatal risk factors (Liu, 2004). Factors associated with early risk can include the intra-uterine environment such as maternal physical and emotional health (O'Hara, 2009), maternal use of both prescribed and non-prescribed drugs during pregnancy (Behnke & Eyler, 2009) and any genetic or maternal factors that might inhibit the growth and development of the foetus (Liu, 2004). Some factors that have been specifically associated with the development of externalising problems in children include maternal malnutrition, illness during pregnancy, and smoking or consuming drugs and alcohol during pregnancy (Brennan, Grekin, & Mednick, 1999; Orlebeke, Knol, & Verhulst, 2010).

Birth complications have also been linked with increased risks in the development of psychopathologies in children and adolescents (Stoff, Breiling, &

Maser, 1997). Obstetric complications have been associated with problems related to oxygen deprivation in the infant brain which have in turn, been associated with the development of psychopathology in children and adolescents including schizophrenia (Cannon, Jones, & Murray, 2002), depression, anxiety and mood disorders (Vocisano, Klein, Keefe, Dienst, & Kincaid, 1996), attention deficit disorders, and conduct and aggressive problems (Guth, Jones, & Murray, 1993). Birth complications combined with problematic parenting techniques have been directly associated with higher instances of a child being predisposed to developing externalising and antisocial behaviours (Gardner, 2000). The implication here is that biological (birth complications) and environmental risks (parenting) in combination might be important factors to consider for the development of psychopathology in children and adolescents.

Neurotransmitters associated with child and adolescent psychopathology

Adults who experience mood disorders such as anxiety, depression and depressive symptoms appear to have a dysregulation in their neurochemistry, specifically relating to rates of serotonin, norepinephrine and acetylcholine (Thase, Jindal, & Howland, 2002). Children and adolescents who experience depression appear to demonstrate a hyposecretion of melatonin and reduced cortisol responses when compared with children who do not experience depression (Cavallo, Holt, Hejazi, Richards, & Meyer, 1987; Ryan & Dahl, 1993). Some empirical evidence points towards a serotonergic system marker for depression as children who have never been depressed, but have a high number of first degree relatives who have had depression show a similar pattern of results. Furthermore, Selective Serotonin Reuptake Inhibitors (SSRIs) have proved to be effective in reducing depressive symptoms in children (Emslie et al., 1997).

Although there is some evidence that dopamine might also be involved with the development of aggression (Baker, Bezdjian, & Raine, 2006) the neurochemical that has been studied and scrutinised the most intensely is serotonin (Moore, Scarpa, & Raine, 2002; Preski & Shelton, 2001; Raine, 2002). It is thought that imbalances in serotonin, specifically lower levels of the neurochemical, are likely to increase impulsive behaviours which might increase the likelihood of a young person engaging in risky and antisocial behaviours including acting out in an aggressive manner.

Functional and anatomical brain differences associated with child psychopathology

Specific abnormalities in functioning of areas of the brain that have been associated with depression in adults include the dysregulated functioning of the prefrontal cortex-limbic-striatal regions, reduced prefrontal volume, hippocampal abnormalities and resting frontal brain asymmetry (Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Tomarkenand & Keener, 2010). Studies which have compared children of depressed mothers with children of non-depressed mothers have shown evidence of localised brain asymmetry in the left frontal hypoactivation in infants and adolescents of depressed mothers (Dawson, Frey, Panagiotides, Osterling, & HessI, 1997; Field, Fox, Pickens, & Nawrocki, 1995; Tomarken, Dichter, Garber, & Simien, 2004). A recent study has also found associations between early instances of externalising behaviour during infancy and adolescent brain structure (Caldwell et al., 2015). The study specifically found that males, but not females, who had demonstrated increased levels of externalising behaviours early in childhood, had smaller amygdala volumes during adolescence.

Genetic factors associated with child psychopathology

Several researchers stress the importance of investigating and identifying susceptibility genes for depression to better understand the increased vulnerability of developing depression in children who might inherit these gene markers. Although there appears to be some support for genes associated with major depression, mainly CREB1 (Levinson, 2006) and 5HTTLPR (Risch et al., 2009), there have been no specific genes identified as being definitively associated with genetic transmission of depression between generations, and there has been a lack of replication of findings for specific genetic variants. Although there appears to be a genetic vulnerability that might contribute to the development of depression in children and adolescents, the exact mechanism underlying a genetic risk for depression is still unclear. It might be that genes influence the risk for depression through endophenotypes such as temperament, cognitive style, along with hormone and neurotransmitter levels (Thapar & Rice, 2006). The contribution of genetic effects for major depression in adults has been estimated to account for between 26% to 42% of variance (Sullivan, Neale, & Kendler, 2000). An estimate of genetic effects on depression in children and adolescents varies based on severity of symptoms, maternal versus child reports of depressive symptomology and behaviours, and children's age and gender (Goodman, 2007). Adolescent depressive disorders in females have a heritability estimate of 40% (Glowinski, Madden, Bucholz, Lynskey, & Heath, 2003). However, some estimates show adolescent depressive symptoms and disorders to have similar levels of heritability to adult depressive symptoms and depression (Rice, 2010).

Genes which might increase child and adolescent vulnerability for the development of aggressive behaviours have also been identified. Genetic variables that have been associated with aggressive symptoms and disorders primarily focus on

functional polymorphisms in the dopamine receptor gene (DRD4) and serotonin transporter gene (5-HTT) (Simons et al., 2011). Molecular genetic studies of specific disorders such as conduct disorders have not been consistent (Monuteaux, Biederman, Doyle, Mick, & Faraone, 2009) as although some studies found associations between functional polymorphism (5HTTLPR) of the serotonin transporter gene (SLC6A4) and the risk for Conduct Disorder (Malmberg, Wargelius, Lichtenstein, Oreland, & Larsson, 2008; Sakai et al., 2006), others have failed to detect this association (Cadoret et al., 2003).

Emotional and behavioural models of motivation and child psychopathology

Difficulties in the regulation of emotions are often associated with child and adolescent psychopathology. Emotions that a young person might experience (including anxiety, fear, sadness or anger) might become dysfunctional in situations where the young person misidentifies their own emotions, misdirects an emotional reaction or their emotional arousal is unpredictable or extreme (Schwenck et al., 2014).

Dysregulation of emotions might impact on child and adolescent psychopathology.

Explanations for this can be found in behavioural models of motivation: a behavioural approach system (BAS) which regulates motives towards desirable goals and a behavioural avoidance (or inhibition) system (BIS) which regulates aversive motives to avoid unpleasant stimuli or situations (Carver & White, 1994).

Anxiety within children appears to be very closely tied to exaggerated and sometimes inappropriate fear responses (Britton, Lissek, Grillon, Norcross, & Pine, 2011). Carver & White (1994) describe how during stressful or challenging situations, a child's reaction is consistent with the behavioural avoidance system (BIS). This reaction manifests neurophysiologically by increasing the activation of the amygdala which promotes avoidance and withdrawal behaviours along with elevated levels of cortisol.

Depression in children might be associated with a weakening of BAS in that the child is less likely to be motivated to engage in pleasurable activities or seek rewards.

Decreased neural activity in the striatum and medial prefrontal cortex and decreased left frontal activity has been observed in depressed children and adolescents, as has elevated levels of cortisol. Disrupted, aggressive and antisocial behaviours in children appear physiologically to be directly opposite to the biology of anxiety problems with a weakening of the BIS and domination of the BAS so the child or adolescent appears ready for confrontation. Looking at the neurophysiology of antisocial children reveals an overall pattern of reduced activation in brain regions that guide emotion regulation and cognitive control (Schneider, 2014).

Intergenerational transmission of psychopathology

Intergenerational transmission describes the transfer of individual abilities, behaviours, traits and outcomes from parents to their children (Lochner, 2008). One of the major risk factors for the development of psychopathology in children and adolescents is the presence of a parent who has a mental health disorder. Children of depressed parents are estimated to be three times more likely to develop depression (Weissman, Wickramaratne, Nomura, Warner, Pilowsky, & Verdeli, 2006).

Intergenerational transmission of psychopathology appears to not be phenotype specific as parental depression is a risk factor not only for depressive symptoms in children but it also associated with a range of negative psychological outcomes, including other mood disorders and conduct disorders (Thapar et al., 2015). As discussed in the previous section, parents might genetically pass on risks for the development of psychopathology through shared or common genes but they also might pass on these risks through elevated environmental risks associated with children being exposed to behaviours related to parental psychopathology. The current thesis will look at the

intergenerational transmission of psychopathology, specifically, the development of depressive and aggressive symptoms in children and adolescents exposed to parental depression.

Environmental risk factors for the development of child psychopathology

A range of environmental factors have been associated with the development of depressive and aggressive symptoms. Some literature suggests that risk factors may overlap; a child might be exposed to multiple risk factors that have been associated with the development of both depressive and aggressive symptoms (Gjone & Stevenson, 1997). One such risk factor is acute or chronic economic strain, which has previously been identified as influencing the development of mental health problems in children and adolescents (Conger, Ge, Elder, Lorenz, & Simons, 1994; McLoyd, & Wilson, 1991). However, evidence suggests that in some circumstances, economic strain associated with poverty impacts children indirectly through negative parenting practices (Conger & Conger, 2007). Other researchers have concluded that in early childhood, economic deprivation and being exposed to less nurturing and engaged parenting by those with less economic resources, are negatively associated with child well-being both separately and collectively (Kiernan & Huerta, 2008). The specific effects of parenting on child psychopathology will be explicitly outlined later within this chapter.

Child and adolescent psychopathology have also been associated with factors outside of the family. The association between child and adolescent psychopathology and negative peer experiences has been examined. Various studies have shown that children who experience depressive symptoms report problems with interpersonal relationships within school settings, have poorer friendships and more negative self-evaluations with relation to their friendships and peer interactions, and lower levels of perceived social competence (Goodyer, Wright, & Altham, 1990; Rudolph & Lambert,

2009). Third parties also rate children with psychopathologies as being more rejected by their peers (Rudolph, Hammen, & Burge, 1994).

Past research has identified that exposure to physical and sexual abuse can have severe and prolonged negative effects on child and adolescent mental health. Exposure to childhood sexual abuse has been associated with increased risks for depression, anxiety, suicidal behaviours, conduct disorders and substance-use disorders (Fergusson, Horwood, & Lynskey, 1996; Fergusson, Woodward, & Horwood, 2000). These effects are sustained even when confounding factors such as lower social economic status, have been controlled for (Fergusson, Woodward & Horwood, 2000). This demonstrates that exposure to sexual abuse or sexual trauma in childhood can contribute to an individual's vulnerability for developing psychopathologies. These complex associations are very important in considering the development of psychopathologies in children and adolescents, but are beyond the scope of this thesis.

The current thesis examines risk mechanisms for the development of psychopathology (specifically depressive and aggressive symptoms) in children and adolescents. It will primarily examine the associations between parental depression, negative parent-child relationships (Erel & Burman, 1995), and negative interparental relationships (Grych & Fincham, 1990; Harold et al., 1997, 2002, 2007, 2012).

Parental depression

Adult depression is a common mental health problem with prevalence estimates ranging between 6% to 17% (Blazer, Kessler, McGonagle, & Swartz, 1994). Over 15 million children living in the United States are believed to be living with at least one depressed parent (Sim & England, 2009). Parental depression has been associated with several negative outcomes in children and adolescents including poor academic achievement (Hay et al., 2001), social problems (Sim & England, 2009), problems with

physical health (Bartlett et al., 2001; 2004), as well as problems with psychological or mental health outcomes (Goodman et al., 2011). Parental depression is viewed as a major risk factor for child and adolescent well-being (Luoma et al., 2001).

Parental depression appears to negatively influence children's cognitive and academic development even from an early age. Murray and colleagues (2006) found associations between maternal postnatal depression and male and female children's IQ scores at age 5, as well as negative associations between postnatal depression and boy's performance on their General Certificate of Secondary Education (GCSE) (Murray et al., 2010). Significant associations have also been observed between early parental depression during infant childhood and child IQ scores at 11 years (Hay et al., 2001). Children of depressed mothers are also more likely to struggle with attention, numeracy skills, and tend to have higher incidences of receiving statements for Special Educational Needs than control participants. Research into the effects of parental depression on cognitive outcomes in children and adolescents, including academic achievement, intellectual levels, IQ scores, and language and cognitive developmental outcomes, does however appear to be limited and is primarily focused on maternal depression (Sanger, Iles, Andrew, & Ramchandani, 2015). This accumulation of research provides evidence for the association between parental depression in the early years of a child's life and negative outcomes. However, other studies suggest associations between postnatal depression and child outcomes that are explained by the continuity of maternal depression across childhood (Fihrer, McMahon, & Taylor, 2009). It might not necessarily be the specific presence of postnatal depression that so negatively influences children, but the presence of parental depression at any time during childhood.

Physical health outcomes in children have also been associated with parental depression (Bartlett et al., 2001; 2004) including increased presentations of headaches, stomach aches, and indigestion problems, along with higher incidents of colds and asthma (Timko, Cronkite, Berg, & Moos, 2002). Doctors have reported that toddlers of depressed mothers are more likely to be rated as being in poor health and more likely to be seen for somatic symptoms than children of non-depressed parents (Sim & England, 2009). Furthermore, these children are less likely to be vaccinated and have a higher rate of emergency room visits (Minkovitz et al., 2005). Children of depressed parents might even be more likely to be involved in trauma related to traffic accidents, due to the association between parental depression and improper use of car seats for infants (McLennan & Kotelchuck, 2000). These observed health problems have been reported to be sustained as children of depressed transition into the adolescent developmental period and even as the child enters middle age (Weissman et al., 2006). The previously described studies have demonstrated the existence of a relationship between parental depression and negative physical health outcomes for children, but there might also be indirect effects of parental depression on children's physical health problems.

The current thesis focuses on the association between parental depression with the development of depressive and aggressive symptoms in children and adolescents. It is therefore important to consider the associations between parental depression and child psychopathology. Indeed, children of depressed parents are more likely to suffer from a range of psychological problems, including depression, which is in itself associated with increased physical health problems for children and adolescents (Fergusson & Woodward, 2002). Parental depression is understood to be a major risk factor for the development of internalising problems (including depressive symptoms) and externalising problems (including aggressive symptoms) in children and adolescents

(Goodman, 2007; Goodman et al., 2011). Children are three times more likely to have an affective disorder if they have a parent who has major depressive disorder (Weissman et al., 2006). Higher rates of anxiety symptoms and disorders have also been reported in children of depressed parents compared to children of non-depressed parents (England & Sim, 2009). These symptoms appear to present in children of depressed parents earlier on in life, and for longer durations with greater functional impairments than individuals who experience depression who have not had a parent with depression (Keyes & Goodman, 2006).

Associations between parental depression and externalising problems in children and adolescents have also been observed (Connell & Goodman, 2002; England & Sim, 2009; Goodman et al., 2011). Higher levels of disruptive, defiant, aggressive and oppositional behaviours are detected in children of depressed parents (Biederman et al., 2001). The relationship between parental depression and externalising problems in children might be a result of shared genetic vulnerability or environmental mechanisms, including parenting disruptions as a result of parental depression (Silberg & Rutter, 2002). Parental depression has been associated with hostile parent-child relationships and inconsistent parenting (Lovejoy, Graczyk, O'Hare, & Neuman, 2000) which in turn has been associated with child externalising problems (Patterson, Reid, & Dishion, 1992). In a study that examined the impact of parental depression and economic disadvantage on child outcomes, Reising and colleagues (2013) found that disrupted parenting accounted for the association between parental depressive symptoms and economic disadvantage with children's externalising symptoms.

Research into the direction of association between parental depression and child psychopathology is somewhat limited. An association that has been more commonly examined is that of parental depression with early child temperament. Temperament can

be defined as "constitutionally based differences in reactivity and self-regulation, in the domains of affect, activity and attention" (Rothbart & Bates, 2006). Although early research considered temperament to be genetically determined (Thomas, Chess, Birch, Hertzig, & Korn, 1963) it has more recently been recognised that although temperament can be stable over time, multiple factors including experience and early environmental exposure, alongside genetic factors, can contribute to child temperament (Hanington, Ramchandani, & Stein, 2010). Children of depressed parents are more likely to exhibit temperamental qualities that are considered to be difficult (Carr, 2015; Laurent et al., 2013). Difficult temperament in early childhood has been associated with later manifestations of depression in children. Studies that have considered the direction of effects between parental depression and difficult child temperament (a potential precursor for child depressive symptoms) have shown significant parent-to-child effects, with less evidence of child to parent effects (Hanington et al., 2010). Some research examining the association between parental depression and child psychopathology has however found that child psychopathology can impact on parental depressive symptoms in general population samples (Guo & Slesnick, 2011; Kouros & Garber, 2010; Nicholson, Deboeck, Farris, Boker, & Borkowski, 2011). Although some studies have examined the associations between trajectories of adolescent and maternal depressive symptoms (Feske et al., 2001; Perloe, Esposito-Smythers, Curby, & Renshaw, 2014), few have examined direct associations between maternal and child depressive symptoms. One specific study that used a high-risk sample of adolescents with mothers who experienced recurrent clinical depression, examined the association between adolescent depressive symptoms and the course of depressive symptoms in mothers (Sellers et al., 2016). The authors concluded that child depressive symptoms can impact on the recurrence of maternal depressive episodes The researchers also

found that depression symptoms in daughters, but not sons, predicted an increase in maternal depressive symptoms across time which might indicate that the direction of the association could be influenced by child gender.

Few researchers have studied the direction of association between parental depression and child aggressive symptoms (Choe, Shaw, Hyde, & Forbes, 2014). Some studies show that higher levels of parental depressive symptoms (particularly maternal depression) increased the risk of commonly occurring disruptive child behaviour developing into more serious aggressive symptoms in later childhood and adolescence (Campbell, et al., 2000; Goodman et al., 2011; Shaw, Gilliom, Ingoldsby, & Nagin, 2003; Weinfield, Ingerski, & Moreau, 2008). However, it is also clear that children's behavioural problems and aggressive symptoms might exacerbate negative emotions, including depression in parents (Allen, Manning, & Meyer, 2010). This has stimulated the need for researchers to try to disentangle and fully understand the associations between child aggressive symptoms and parental depression (Shaw, Gross & Moilanen, 2009). The current thesis will examine the impact of parental depressive symptoms on children and adolescents and how these symptoms of child psychopathology are associated with the parent-child relationship.

The aforementioned studies have primarily focused on the association between maternal (as opposed to paternal) depression and symptoms of psychopathology in children and adolescents. The focus on the impact of maternal depression might be due to the increased prevalence of depression in women (Albert, 2015) with estimates that 1 in 4 women will require treatment for depression, compared with 1 in 10 men (National Institute for Health and Care Excellence, 2009). Other reasons might include expectancies for fathers to not be as involved in child rearing as mothers (Russell & Russell, 1987) and the difficulties often faced by researchers in recruiting fathers for

research on child development (Cassano, Adrian, Veits, & Zeman, 2006; Mitchell et al., 2007).

Researchers who have examined the influence of paternal depression have demonstrated that depression in fathers is associated with: a greater level of father-to-child hostility (Harold et al., 2011), and negative effects on children's general psychological functioning (LeFrançois, 2011; 2012) as well as increased risk for child depressive and anxiety symptoms when maternal depressive symptoms have been controlled for (Reeb et al., 2015). Paternal depression also appears to negatively impact depressive symptoms in mothers (Laurent et al., 2013).

Some evidence suggests that compared to paternal depression, maternal depression is associated with a greater risk for negative outcomes in children (Connell & Goodman, 2002), and more severe episodes of child depression (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005). There are however inconsistencies in research assessing the impact of maternal versus paternal depression on children, with some researchers showing comparatively similar effects of maternal and paternal depression on negative child outcomes (Jacob & Johnson, 1997; Marchand & Hock, 1998). Furthermore the effects of paternal and maternal depression appear to independently influence child outcomes (Cummings, Schermerhorn, Keller, & Davies, 2008). It is therefore important that research examines the processes through which both maternal and paternal depression may influence the risk for the development of child psychopathology, specifically depressive symptoms and aggressive behaviours. Research should also consider the role of fathers as this may increase or decrease the risk of negative child outcomes associated with parental depression. If the father presents with no psychopathology, this may be a protective factor for children of depressed mothers (Brennan, Hammen, Katz, & Le Brocque, 2002). In families where the father has

depression, risk for child psychopathology may increase through genetic or environmental mechanisms (Goodman & Gotlib, 1999; Klein et al., 2005; Marmorstein, Malone & Iacono, 2004). Children who are exposed to two parents with depression appear to be at a significantly greater risks for developing psychopathology (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002; Weissman, et al., 2006) compared to children with one parent with depression.

As discussed, only a limited number of studies have examined the influence of paternal depression on child outcomes, but even fewer have considered paternal depression separately from measures of maternal depression. Where studies have examined the influence of paternal depression, depression in fathers was associated with adverse emotional and behavioural outcomes in young children (aged 3-5 years) even when controlling for maternal postnatal depression (Ramchandani, Stein, Evans, & O'Connor, 2005). Children whose fathers are more chronically depressed appear to be at a higher risk of negative emotional and behavioural outcomes in middle childhood as well (Ramchandani et al., 2008).

The association between parental depression and the development of psychopathology

A range of mechanisms can explain how parental depression might negatively influence child and adolescent development, and more specifically, the development of depressive and aggressive symptoms. Psychopathology in children might be transmitted through genetic, neuroregulatory, and environmental factors (Beardslee, Gladstone, & O'Connor, 2011).

When a family has a depressed member, the home environment is likely to be characterised by more conflict and less support which ultimately might lead to family dysfunction and increased children's risk of developing depressive and aggressive

symptoms (Garber & Horowitz, 2008). The social support that a parent has access to might also influence the extent to which parental depression affects children and adolescents. Low levels of social support reported by parents with depressive symptoms are associated with impaired parenting behaviours; better outcomes are reported where depressed parents have higher levels of social support, which leads to improved outcomes in children (Gunlicks & Weissman, 2008).

The current thesis will focus on family socialisation processes that parental depression might influence, which might influence the development of depressive and aggressive symptoms in children and adolescents. Specifically, the thesis will assess the direct association between parental depression and child mental health outcomes, and also the indirect influence parental depression might have on the presence of depressive and aggressive symptoms in children through indices of family socialisation, specifically the parent-child relationship and the interparental relationship.

Parental depression and the parent-child relationship

The quality of the parent-child relationship is likely to be impaired by parental depression (Barry, Kochanska, & Philibert, 2008; Kaufman et al., 2004) which suggests that parenting and the parent-child relationship might be a possible environmental mechanism through which parental depression has a negative impact on children (Wilson & Durbin, 2010). This is supported by ecological models of parenting, which propose that parenting is influenced by behavioural variables, including parental depressive symptoms (Abidin, 1992; Belsky, 1984).

Parental depression has been associated with less optimal parenting qualities including withdrawn, harsh and inconsistent parenting, less open parent-child communication, as well as increases in parental hostility and irritability towards their child (Beardselee, Versage, & Giadstone, 1998; Goodman & Gotlib, 1999; Lovejoy et

al., 2000; Ponnet et al., 2013). Disruptions in the parent-child relationship are not only evident for the parent with depression, but might also impact on their partner's relationship with their child (Ponnet et al., 2013). This puts children of depressed parents at a greater risk of negative outcomes through disrupted parenting from both parents. An additional risk might be due to sustained impairments in the parent-child relationship where a parent has had a history of depression; in situations where parents no longer report symptoms of depression, parent-child interactions appear to remain disrupted (Jaser et al., 2005).

Although studies have primarily focused on the impact of maternal depression on parent-child relationships (Beardselee et al., 1998; Goodman & Gotlib, 1999; Goodman et al., 2011; Lovejoy et al., 2000), some researchers have also recognised the need to assess how parenting and parent-child relationships might be affected by paternal depression (Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000; Cummings, Goeke-Morey, & Raymond, 2004; Jacobs, Talati, Wickramaratne, & Warner, 2015; Ponnet et al., 2013). In a meta-analytic review of empirical literature focusing on the association between parental depression and parenting, Wilson and Durbin (2010) concluded that the effect sizes of the relationship between maternal depression, paternal depression and negative parenting behaviours were comparable.

Parental depression and the interparental relationship

It has been demonstrated that depression not only negatively impacts on the individual experiencing depressive symptoms but can significantly impair a couples' relationship quality (Reich, 2003). Several negative associations have been observed between an individuals' depressive symptoms and their relationship with their partner, including poorer martial adjustment, decreased relationship satisfaction, greater marital discord, and greater problems establishing and maintaining intimacy compared to

relationships where neither partner experiences depressive symptoms (Basco, Prager, Pita, Tamir, & Stephens, 1992; Jacob & Johnson, 1997; Whisman & Beach, 2012; Whisman, Uebelacker, & Weinstock, 2004). Although not all relationships which include a depressed partner experience problems (Gabriel, Beach, & Bodenmann, 2010), it seems probable that this association between depression and couples' overall relationship quality exists (Barbato & D'Avanzo, 2008; Doohan, Carrère, & Riggs, 2010).

Although not the focus of the current thesis, it is important to acknowledge that the direction of the association between depression and relationship discord remains unclear, e.g. whether depressive symptoms negatively impact on partner relationships, or whether hostility within a relationship influences depressive symptoms. Some studies point to the negative impact that distress or discord in relationships has on the development, maintenance, severity and intensity of depressive symptoms experienced by an individual (Gabriel et al., 2010; Jacob & Johnson, 1997; Whisman & Beach, 2012), whereas a longitudinal study suggests that depression might be a risk factor for, and predict discord in marital relationships (Gilliam & Cottone, 2005). Fincham and colleagues (1997) examined the association between depressive symptoms and marital satisfaction in couples; for women it appeared that reductions in marital satisfaction led to depressive symptoms – an association which was reversed for men in the study. It seems most likely that there is a bi-directional relationship between depression and relationship discord (Doohan et al., 2010) and that this association might be stronger for females than males (Reich, 2003).

The association between family relationships and the development of depressive and aggressive symptoms in children and adolescents

Several factors that contribute to the development of children's mental health and general well-being have previously been identified and have historically been categorised into environmental factors (social economic status, housing, parental employment and income, education), biological factors (abnormalities of the central nervous system, poor nutrition, low birth weight, prenatal exposure to alcohol or drugs) and interpersonal relationships (child attachment styles, parenting and social networks for parents and children) (Ford, Mitrofan, & Wolpert, 2013). Parental depression and family relationships (specifically the parent-child and interparental relationship) have been shown to influence the development of depressive and aggressive symptoms in children and will be the primary focus of the thesis. This body of work will look at the associations of specific indices of the family environment with the development of depressive and aggressive symptoms in children and adolescents.

The influence of the family environment on child development is a well-established area of interest. It became a prominent area of research in the 1960's and 1980's in particular as researchers began examining the importance of a child's family in accounting for individual differences in child development (Steinberg & Steinberg, 1987). Family mechanisms that might explain negative child and adolescent outcomes also became a specific area of research interest (Davies & Cummings, 1995; Ge, Conger, Lorenz, Elder, & Simons, 1994). At this time, the social landscape that children were being reared in was changing, with an increase in the number of marriages ending, carrying with it an increase in the number of children of divorce. Researchers and policy makers recognised the importance in understanding the outcomes for such children.

Amato and Keith (1991) compared children from intact families with children from

divorced families finding a small yet significant difference between the groups, with poorer adjustments (including academic achievement, conduct problems, psychological adjustment, self-concept, social adjustment, mother-child relations and father-child relations) for children whose parents had divorced.

Studies on the children of divorce appeared to take a slightly different path of examination in the early 90's. Researchers began to explore the specific mechanisms surrounding the dissolution of marriage that might be negatively impacting on children and started to consider that it might not be the actual event of the divorce that was having detrimental impacts on children, but the family conditions pre-dating the divorce (Cummings, Vogel, Cummings, & El-Sheikh, 1989; Cummings, Ballard, El-Sheikh, & Lake, 1991; Grych & Fincham, 1990). Shaw and colleagues (1993) identified the importance of acknowledging the presence of conflict within a couple's relationship (interparental conflict) and how it might negatively impact on children. Interparental conflict both prior to, and following a divorce, was a better predictor of children's adjustment than the marital status of their parents. The stress that interparental conflict put on the household appeared to be detrimental to all family members' mental health (Bradbury & Fincham, 1990; Gotlib & Hammen, 1992; Grych & Fincham, 1990). Cummings and colleagues (1989, 1991) identified associations between marital conflict and child adjustment across a range of child development stages, with children as young as six months showing signs of distress as a result of interparental conflict (Crockenberg, Leerkes, & Lekka, 2007; Cummings & Davies, 1994; Harold, Shelton, Goeke-Morey, & Cummings, 2004; Reynolds, Harold, & Pryor, 2001). These findings highlight the importance of examining interparental conflict and child mental health outcomes across a range of child developmental stages, which this thesis intends to do.

The impact of interparental conflict on children and adolescents

Previous empirical literature appears to have struggled to provide a universal definition for what interparental conflict is. Early researchers focussed on examining conflict as an attempt to influence another individual to accept their point of view (Miller, Danager & Forbes, 1986) or as a challenge to the maintenance of interpersonal harmony (Kerig, 1996). Some research has focused on the operationalisation of conflict e.g. defining the process of conflict, its content and its duration (Emery, 1982). A common way of measuring marital conflict is to examine the amount of hostility within a spousal relationship (Harold, Shelton, Goeke-Morey & Cummings, 2004; Harold et al., 2013; Rhoades et al., 2012; Sellers et al, 2014; Shelton & Harold, 2008;) which is typically defined by displays of anger and hostility (Sturge-Apple, Davies & Cummings, 2006). Although the term interparental conflict will be used relatively broadly within the thesis, the majority of the empirical studies will operationalise interparental conflict using measures of hostility expressed by partners in the marital relationship. The exception to this will be Chapter 4 (Study 3) which will measure interparental conflict with multiple assessments that aim to capture individual's beliefs about their partners behaviours (The Behaviour Affect Rating Scale; Melby et al., 1995), a measure of marital instability (Booth, Johnson & Edwards, 1983) and an observational measure of couple relationship quality (Marital Interaction Task, Dogan, Lei, Milne-Kahn & Pong, 2005).

Children living in homes where parental conflict is frequent and poorly resolved have been repeatedly associated with increased levels of negative child outcomes (Grych & Fincham, 2001). Approaches to obtaining information on child reactions to interparental conflict have included interviews and questionnaires with children, their parents and teachers (Cummings, Goeke-Morey, & Papp, 2003), diary accounts and

observational studies (Crockenberg et al., 2007) as well as more naturalistic approaches where children watch recordings of adults involved in conflict (Davies, Myers, Cummings, & Heindel, 1999).

Negative outcomes that have been frequently associated with children exposed to interparental conflict include child internalising and externalising problems, such as depressive and aggressive symptoms (Buehler et al., 1997; Cummings & Davies, 1994; Forehand, Neighbors, Devine, & Armistead, 1994; Grych & Fincham, 1990; Jouriles, Farris, & McDonald, 1991), impaired academic achievement (Ghazarian & Buehler, 2010; Harold, Aitken, & Shelton, 2007), and problems with cognitive functioning (Hinnant, El-Sheikh, Keiley, & Buckhalt, 2013). Children who have been exposed to interparental conflict also experience greater difficulties within social situations; they are more likely to be withdrawn (Grych & Fincham, 1990) and have difficulties with social competence (Paley, Conger, & Harold, 2000).

Significant associations have also been observed between interparental conflict and children's psychobiological functioning, especially for female children. Associations between exposure to interparental conflict and early pubertal maturation have been observed in young females (Ellis & Garber, 2000). Early pubertal maturation in females is associated with negative health and psychosocial outcomes such as an increased risk of breast cancer (Kampert, Whittemore, & Paffenbarger, 1988), obesity (Prentice & Viner, 2013), and higher levels of criminality, substance abuse problems, social isolation and early sexual behaviours (Copeland et al., 2010).

The primary child and adolescent outcomes that will be the focus within the current thesis are internalising problems, specifically depressive symptoms, and externalising problems, specifically aggressive behaviours, as these are recognised as two child outcomes associated with interparental conflict (Cummings & Davies, 1994;

Grych & Fincham, 2001; Reynolds et al., 2014). The aim of this thesis is to extend the understanding of the association between interparental conflict on the development of depressive and aggressive symptoms in children and adolescents whilst simultaneously considering parental depressive symptoms and hostile parenting practices.

Mechanisms which explain the impact of interparental conflict on children

Given the well-established associations observed between interparental conflict and negative outcomes in children, it is important to consider *how* conflict between parents affects children. There is some debate as to whether children are directly affected by interparental conflict, or if they are indirectly affected by the conflict through some other part of the family environment (e.g. disrupted parent-child relations) (Fauber & Long, 1991; Grych & Fincham, 1993). Theories and research that attempt to provide explanations for this association will now be examined.

Theories and research that assess the direct impact of interparental conflict on children focus on the underlying psychological processes (e.g. cognitions, emotions) engendered in children living in households where they are exposed to hostile and conflicted relations between their parents. Research which focuses on the direct effects of conflict on children takes into consideration characteristics of the parental disputes by highlighting that the specific forms of conflict that are most disruptive to children are ones that are intense, frequent and unresolved (Cummings & Cummings, 1988; Grych & Fincham, 1993). The cognitive contextual framework (Grych & Fincham, 1990) and emotional security hypothesis (Davies & Cummings, 1994) will now be summarised.

The cognitive contextual framework (Grych & Fincham, 1990) proposes that the attributions that children give to their parents' conflict could account for effects on their well-being. Within this framework, children's responses to conflict are believed to occur through cognitive processing of conflict. The impact of interparental conflict on

children operates on two levels: (1) how the conflict is expressed by the parents, and (2) how the child interprets the meaning of the conflict. During primary processing, a child becomes aware of a conflict and experiences an initial level of arousal. Characteristics of the conflict (frequency, intensity and resolution potential) and contextual factors (quality of parent-child relationship, child temperament, child gender and history of exposure to conflict) influence the initial primary stage of appraisal. During the secondary stage of processing, a child attempts to understand the conflict by determining why the conflict is occurring and what their response should be. Children that view the conflict as threatening, or who feel unable to cope effectively, are more likely to experience anxiety and helplessness. Whereas children who blame themselves for the parental argument have been shown to exhibit feelings of guilt, shame, and sadness. If the conflict is frequent, intense and poorly resolved, these attributes are believed to increase children's risk of emotional and behavioural problems, which can include depressive and aggressive symptoms (Grych, Harold, & Miles, 2003). The attributions that children make about parental conflict can affect how negatively it impacts them. Depressive and aggressive symptoms are associated with children who attribute threat and self-blame towards interparental conflict. For girls, threat-based attributions of interparental conflict exacerbated their symptoms of depression, more so than for boys, whose attributions of self-blame and responsibility exacerbated their aggressive symptoms (Grych et al., 2003).

The emotional security hypothesis (Davies & Cummings, 1994) considers the importance of the attachment processes and highlights the role of children's emotional security in assessing their individual differences in well-being and emotional and behavioural outcomes to conflict, including depressive and aggressive symptoms. When parents argue, a child's emotional security is threatened, as a result of which, feelings of

emotional reactivity might be affected and the child might feel anger, sadness or fear. A child's representation of family relationships might also be affected, in that they might expect conflict to transfer to other dyads within the family system (e.g. the parent-child relationship). A child might feel motivated to regulate their exposure to conflict in a direct way, whereby a child intervenes during the conflict, or the child might actively withdraw from the hostile episode. The impact of the interparental conflict has on children might be explained by the extent to which one or more of these aspects of emotional security is adversely affected, and the extent to which the child is able to regulate the emotional disruption. Children who experience sadness, anger or fear and who regard the interparental conflict as both immediate and a potentially long-term threat to the quality of other family relationships (including the parent-child relationship) show heightened signs of emotional and behavioural distress which might be expressed as depressive and aggressive symptoms (Davies & Cummings, 1998).

Support for theories that propose direct detrimental influences of interparental conflict on children can be found in research which emphasises the importance of children's awareness of parental behaviour, specifically their awareness of interparental conflict (Cummings & Davies, 2002; 2011). If interparental conflict were to only impact children indirectly through mechanisms which will be described imminently (e.g. disrupted parent-child relationships), a child would not need to be directly exposed to, or witness this conflict for it to have a negative consequences on their well-being (Harold & Conger, 1997). However, studies have observed that exposure to overt interparental conflict has a greater negative impact on child distress than covert conflict, which the child would not necessarily have to have witnessed (Harold, Osborne, & Conger, 1997).

Disruptions in the interparental relationship might impact children indirectly through the deteriorations of other relationships within the family, specifically the parent-child relationship (Katz & Gottman, 1994). The spill-over hypothesis (Erel & Burman, 1995) proposes that behaviour or emotion transfers from one dyad to another within the family system, so that negativity within the spousal relationship effectively 'spills over' in to the parent-child relationship. Disruptions in the parent-child dyad are seen to be critical in understanding how interparental conflict impacts child adjustment (Fauber & Long, 1991). Before discussing the detrimental impact that interparental conflict might have on children's development (specifically the development of depressive and aggressive symptoms) through parent-child relationships, the importance of the parent-child relationship will be reviewed.

Parent-child relationship

The parent-child relationship has long been viewed as a central feature for the healthy emotional and behavioural development of children and adolescents (Kagan & Snidman, 1999; Serot & Teevan, 1961). Two theories that attempt to describe the importance of the parent-child relationship and parenting for healthy development in children, and that are used to describe maladaptive developmental outcomes (including child depressive and aggressive symptoms) are attachment theory (Bowlby, 1969) and social learning based coercion theory (Patterson et al., 1992; Patterson, 1982; Reid, Patterson, & Snyder, 2002).

Prior to Bowlby and Spitz's early work on the influence of parent-child relations, socioemotional bonds between infants and caregivers had largely been neglected as sources of impact on child development (Bowlby, 1953; Siegler, DeLoache, & Eisenberg, 2003; Spitz, 1945). Early studies on institutionalised children who were being provided for physically, in terms of nutrition and educational needs,

highlighted the risk factors associated with the absence of caregiver attachment. These children displayed a lack of the emotional development which is key to fostering future relationships (Bowlby, 1953) and often appeared sickly and physically stunted (Spitz, 1945, 1949). These studies began to challenge previously held beliefs about what a child required to develop normally; that is, even if adequate nourishment and health care is provided, an absence of emotional caregiving puts children at greater risk for negative developmental outcomes.

A child's early relationships have since been identified as instrumental in their development (Shonkoff, Levitt, Boyce, & Cameron, 2004). Bowlby's attachment theory (1953, 1969) revolutionised our understanding of how influential a child's early relationships are for their emotional and physical development. The quality of the parent-child relationship and its effects on child development continues to be studied, leading to a deeper understanding of the way these early relationships and emotional bonds likely influence children's interaction with others.

Mary Ainsworth, a student and protégée of Bowlby, researched mother-child interactions during infancy. Her observations highlighted the importance of two specific aspects of the relationship that might measure the quality of an infant's attachment to their primary caregiver; the extent to which an infant uses their caregiver as a secure base and the reaction of an infant to separations and reunions with the caregiver (Ainsworth, 1979). Researchers began attempts to empirically test these measures using the 'Strange Situation' and based on patterns of results associated with infants' reactions they identified several attachment categories: (1) secure attachment, (2) insecure/resistant attachment, (3) insecure/avoidant attachment, (4) disorganised/disoriented attachment.

Secure relationship attachments are the strongest form of attachment and are characterised by a child feeling they are able to depend on their parent or primary caregiver and feel confident that the parent will be able to meet their needs. Securely attached children use their primary attachments as a safe base to explore their environment but will seek their attachment figure when distressed, appearing confident that their parent will be able to soothe them (Main & Cassidy, 1988). Insecure resistant (or insecure ambivalent) attachments see a child behaving ambivalently towards their primary caregiver, exhibiting clingy and somewhat dependent behaviour, but will reject the caregiver when they try to engage in interactions. Children that demonstrate insecure avoidant attachment style appear independent from their parent or primary caregiver, emotionally and physically (Behrens, Hesse, & Main, 2007). Avoidant children do not seek comfort from their parent when they are distressed. Within disorganised attachments, a child appears to not be able to predict the reaction they will receive from their parent, uncertain as to whether a positive or negative response will be given to specific behaviours. The interplay between a parent's sensitivity to their child's emotional response governs attachment behaviour and is a prominent assumption that underlies attachment theory, as to which a parent's behaviour towards their child varies by their child's attachment style (Ainsworth, Blehar, Waters, & Wall, 2015). Parents of secure children are characterised as being responsive to their child's needs whilst aiding them to learn to control their emotions; parents of insecure-avoidant children on the other hand are less responsive to their child's needs and parents whose children are insecure-ambivalent are likely to be inconsistent in their responses to their child.

Empirical literature supports the association between positive and sensitive parenting and children's attachment; supportive, responsive and affectionate parenting is linked to attachment security (Gartstein & Iverson, 2014) whereas parents who are

not engaged, do not show support towards their child or who expose their child to maltreatment are linked to insecurely attached children (Goldberg, 2014). A wide range of negative outcomes are associated with children who are insecurely attached with their parent or primary caregiver. Insecurely attached children tend to exhibit more depressive and aggressive symptoms than children with more secure attachments (Burnette, Davis, Green, Worthington, & Bradfield, 2009; Fearon, Bakermans-Kranenburg, van Ijzendoorn, Lapsley, & Roisman, 2010).

Social learning based coercion theory provides an additional framework to explain how the parent-child relationship and parenting might impact on child development (Patterson, 1982). It was specifically designed to explain the development of externalising behaviours in children and adolescents (Patterson, 1982; Reid et al., 2002). Within coercion theory, child and adolescent psychological maladjustment, including the development of depressive and aggressive symptoms, are primarily explained through disrupted parental discipline skills and hostile relationships between children and their parents (Harold & Leve, 2011). Negative parenting practices including permissive and authoritarian parenting styles have been associated with antisocial behaviour in children and adolescents (Reid, Webster-Stratton, & Baydar, 2004). More specifically, hostile, critical, punitive and coercive parenting has been associated with externalising problems (Rutter, Giller, & Hagell, 1998; Scott, Doolan, Beckett, Harry, & Cartwright, 2012) with more modest associations between these negative parenting styles and child internalising problems (McLeod, Wood, & Weisz, 2007). High levels of parental warmth have been associated with lower levels of externalising behaviour problems in children (Garber, Robinson, & Valentiner, 1997).

A well-established aspect of the parent-child relationship that has been associated with negative child outcomes across a range of child developmental stages is

hostility expressed from parents towards their children (Harold et al., 2011; Lewis et al., 2014) which will be explored in depth throughout the current thesis. Parent-to-child hostility has been associated with disruptive behaviour with peers (Elam et al., 2014a), Attention Deficit Hyperactivity Disorder symptoms in children (Lifford, Harold, & Thapar, 2009) along with aggressive and depressive symptoms in children and adolescents (Elam et al., 2014; Lewis et al., 2014).

As previously mentioned, it is important to acknowledge that the father-child relationship, although considered far less frequently than the mother-child relationship, likely impacts on the risk for the development of negative outcomes in children.

Researchers have recently begun to examine processes within the father-child relationship that might negatively (Jaffee, Moffitt, Caspi, & Taylor, 2003) or positively impact a child's development (Cabrera, Fitzgerald, Bradley, & Roggman, 2007; McHale, 2007). This is especially important within the context of parental depression (Jarvis & Creasey, 1991).

Recognising the distinctions between mother-child and father-child subsystems is pivotal in understanding the mechanisms involved in the development of psychopathology in children as these relationships might provide a unique understanding as to the pathways that could increase, or decrease the risk for negative outcomes in a child (Collins & Russell, 1991; Lamb, 2004). Previous research has highlighted that interactions between parents and their children are likely to differ for fathers and mothers. Fathers are more likely to interact with their child through play, where mothers are more likely to interact with their child through direct caregiving (Russell & Russell, 1987). When fathers are more involved with routine child care, there are positive associations for children, such as, an improvement with academic achievement (Hoffman & Youngblade, 1999). More secure father-child attachments

might reduce the risk for children to develop anxious and withdrawn behaviours than secure mother-child attachments (Verschueren & Marcoen, 1999). Harsh paternal discipline is associated with higher instances of child behaviour problems compared to harsh maternal discipline (Lewis, Newson, & Newson, 1982). A limitation of this research is that it has typically examined these parent-child relationships with young infants, so it is unclear how these interactions might differ during middle childhood. The current thesis shall examine these associations across a broad range of developmental periods (early childhood, middle childhood and early adolescence) to expand on our understanding of this relationship.

Interparental conflict and parent-child relationships

The conflict expressed within a couple's relationship has previously been thought of as a starting point or catalyst for a sequence of negative changes in family interactions (Katz & Gottman, 1994). Theories that explain the indirect impact of interparental conflict on children though disrupted parent-child relationships will now be reviewed.

The spill-over hypothesis (Erel & Burman, 1995) explains negative outcomes for children living in households with interparental conflict being negatively impacted by the marital disputes through disruptions in the parent-child relationship. Parents in hostile relationships with their spouses tend to exhibit elevated levels of hostility and aggression toward their children and are observed as being less sensitive and emotionally responsive to their children's needs. Conflict between parents might indirectly affect children through a 'spill over' of negative emotions from the couple relationship to the parent-child relationship.

A key component of models that use a family systems theory or approach in an attempt to explain the influence of interparental conflict on children's outcomes is the

recognition of both the interdependence and reciprocal influences that exist between the individuals of a family, be that a parent or child (Reynolds et al., 2014). The familywide model of interparental conflict and its effects on children integrates both direct and indirect perspectives of how conflict impacts on child development using a processorientated perspective of relations between interparental conflict and child adjustment (David, Steele, Forehand, & Armistead, 1996; Davies et al., 2002; Wilhelm, Brownhill, & Boyce, 2000). Emphasis is placed within this model on assessing the family as a whole, not as separate units but integrated pieces that explain relationships between all members. The family-wide model outlines how children's expectations and representations of interparental conflict might impact on their long-term psychological development. The model highlights the importance of considering children's individual perspectives in questioning how exposure to conflict between parents adversely affects their psychological well-being as children's understanding of interparental conflict is an important factor in determining its impact on their psychological development (Harold et al., 1997). Children's expectations on how a parent will behave towards them derives from how they perceive them to behave with one and other. Under this premise, if a child observes hostility or conflict between their parents, they will expect hostility in their own relationship with their parent.

Nature versus Nurture or Nature via Nurture

A key issue when trying to determine influences on child development is whether individual differences arise as a result of genetic makeup (nature) or environmental influences (nurture). Biological and environmental risk factors associated with the development of depressive and aggressive symptoms in children and adolescents have previously been outlined within this chapter. It has become increasingly recognised that the over-simplistic divide between genetic and

environmental risk, including intrauterine risk, fails to capture the dynamic interplay between these complementary sources of influences on children's health and behaviour. Interest has, as a result, turned to exploring the nature of the relationship between genetic and environmental sources of risk in explaining the origin of disorder in childhood. We no longer conceptualise developmental outcomes as being purely a result of nature (genetic influences) or nurture (environmental influences), but rather examine the interplay between the two (nature via nurture). The recognition that multiple factors, both genetic and environmental, might contribute to the development of psychopathology in children (Natsuaki et al., 2014) has driven attempts to disentangle genetic and environmental influences to better understand the unique contributions they might play on a child's development. Within the context of the current thesis, the question is whether the presence of mental health problems in children and adolescents are a result of a genetic predisposition to psychopathology, a result of environmental influences that they are exposed to or a combination of these risks.

Previous research into the influence of family socialisation and parental depression on child development has often utilised families where parents are genetically related to the child that they are rearing (Barber, Olsen, & Shagle, 1994; Downey & Coyne, 1990b; Grych et al., 2003; Hill, Bush, & Roosa, 2003; Shelton & Harold, 2008). These studies have been informative to assess the different mechanisms involved in the development of psychopathology and contributed to our understanding of the presence of these associations. However, it is difficult to determine from this research whether parent-level, family-level and child outcomes arise as a result of shared genetic influences or are a by-product of the environmental effects the family are exposed to in studies that use biologically related families (Plomin, DeFries, & Loehlin,

1977). That is, these studies are unable to assess the separate contributions of genetic and environmental influences on the development of depressive and aggressive symptoms in children and adolescents as parents and children have both genes and environment in common. Similarities observed in characteristics between parents and children when they are biologically related might only be reflecting the presence of underlying shared genetic influences that simultaneously impact the characteristic in the parent and characteristics in the child (Harold et al., 2013). When determining whether the development of mental health problems in children and adolescents are predominantly a result of nature or nurture, it is important to be aware that genes might not only be impacting on the specific symptoms of psychopathology in question (depressive and aggressive symptoms), but they might also be influencing the environment that the child is exposed to (e.g. parental depressive symptoms, interparental conflict and parent-child hostility). The term used to describe this overlap of influence is genotype-environment correlation (rGE) (Plomin et al., 1977).

Genotype-Environment correlation

Genotype-Environment correlations are used to explain how genetic and environmental effects influence one another (Table 1). There are three configurations of *r*GE: passive, evocative, and active (Plomin et al., 1977). The passive gene-environment correlation is the association between an inherited genotype and the environment in which the child is raised. It specifically relates to the overlap of genetic and environmental influences on a child living with a parent whom the child is biologically related to as associations observed between parent and child characteristics (hostile parenting and child depressive and aggressive symptoms) could be a product of underlying shared or common genetic factors that might simultaneously influence the parent and child trait (Harold et al., 2011a; Harold et al., 2013; Jaffee & Price, 2007;

Price & Jaffee, 2008). A child might actively seek out experiences or circumstances that are correlated with the behaviour in question (e.g. depressive and aggressive symptoms) which is referred to as an active gene-environment correlation. The evocative gene-environment correlation refers to an individual's heritable behaviour evoking an environmental response. A child with depressive or aggressive symptoms might evoke negative reactions from their parents (Avinun & Knafo, 2013; Ge & Conger, 1996). The current thesis focuses specifically on passive *r*GE and evocative *r*GE (though these will not be examined within the first study).

Table 1: Configurations of Genotype-Environment Correlations

Туре	Description	Source of Environmental Influences
Passive	Children receive genotypes	Parents and siblings
	correlated with their family	
	environment	
Evocative	Individuals are reacted to on	Anybody with close
	the basis of their genetic	proximity (e.g. peers, friends,
	propensities	teachers)
Active	Individuals seek or create	Any person or environment
	environments correlated with	with close proximity
	their genetic proclivities	

SOURCE: Plomin, DeFries & Loehlin (1997)

Research designs that allow for the separation of passive rGE from indices of family relationships (hostile interparental and parent-child relationships) from associations with child outcomes (depressive and aggressive symptoms) aim to disentangle the influence of genetic and environmental contributions. This can ultimately aid in the development of interventions aimed at targeting modifiable environmental risk factors to reduce negative outcomes (Harold et al., 2013). Examining associations between parental depression, interparental conflict and hostile parenting practices with the development of child psychopathology in families where children are

genetically unrelated from the parents rearing them, the confound of passive rGE can be controlled for. If an association between a measure of the family environment (e.g. parent-child hostility) and a child outcome (e.g. depressive or aggressive symptoms) is present in genetically unrelated parent-child dyads, the association cannot be a result of common or shared genes, and we can more accurately determine that the association might be a result of an environmental influence.

The current thesis will be utilising two naturally occurring experimental research designs that allow for the separation of passive *r*GE from indices of the family environment (parental depression, interparental conflict, parent-child hostility) from child mental health problems (depressive and aggressive symptoms): an adoption at birth design and a sample of families where children have been conceived through in vitro fertilization (IVF). An overview of research designs used to assess the impact of family socialisation on children and adolescents will now be provided, including a review of adoption and IVF research.

Research designs & methodological issues

The research findings that have been reviewed suggest the existence/presence of associations between parental depression, interparental conflict, parent-child hostility and the development of negative outcomes (specifically depressive and aggressive symptoms) in children and adolescents. The methodology used to explore the relationships between parental depression, family socialisation and psychopathology in children and adolescents will now be reviewed.

A range of study designs can be used to assess the impact of parental depression and family socialisation on child outcomes. Advancements in methodology have allowed the examination of the association between parental depression, family relationships and child outcomes to be studied across a wide spectrum of research

designs including twin studies (Chen, Li, Natsuaki, Leve, & Harold, 2014;
Schermerhorn et al., 2011), adoption studies (Bornovalova et al., 2014; Harold et al.,
2013; Mannering et al., 2011), clinical studies (Grych, Fincham, Jouriles, & McDonald,
2000), longitudinal studies (Grych et al., 2003; Hanington, Heron, Stein, &
Ramchandani, 2012; Koss et al., 2013; Shelton & Harold, 2008), novel research designs
such as in-vitro fertilisation studies (Harold et al., 2013) and meta-analyses of research
(Amato & Keith, 1991; Amato, 2001; Emery, 1982; Krishnakumar & Buehler, 2000).

Twin and adoption studies have historically been used to demonstrate the contribution of genetic and environmental influences to the development of childhood negative emotional and behavioural outcomes (Rutter, Moffitt, & Caspi, 2006). The introduction of novel, genetically informative research designs, specifically In-Vitro Fertilisation studies (Haroldet al., 2008) has also contributed to research. These research designs will now be discussed with references to and consideration of the strengths and limitations of each design and a specific focus on how these designs have contributed to our understanding of how parental depression and family socialisation might influence the development of psychopathologies in children and adolescents.

Twin studies

Francis Galton's seminal paper (1875) on the history of twins outlined opportunities offered to explore the influences of genes and environment through two naturally occurring types of siblings: genetically identical (monozygotic) and non-identical (dizygotic) twins. Interpretations of results from twin studies are grounded in the theory that similarities in twins are the result of their genes and their shared environment.

Several assumptions are tied to twin research. Firstly, that monozygotic twins (MZ) are genetically identical to each other. Therefore if MZ twins are compared to first

degree relatives, who typically only share 50% genetic resemblance, these differences are hypothesised to be a result of shared genes. If greater similarities can be found in MZ twins than first degree relatives, this might indicate a greater genetic prevalence for a specific targeted trait. Dizygotic twins (DZ) are siblings who are a result of multiple zygosity (two fertilised zygotes who mature simultaneously in the womb). Although DZ twins might develop pre and postnatally alongside each other, they are only as genetically similar to each other as any typical first degree relatives (such as siblings). This assumption therefore postulates that comparisons can be made between MZ and DZ twins. If MZ twins are more similar for a specific trait than DZ twins, it is predicted that that trait is likely to be a result of greater genetic similarity between MZ twins, resulting in a greater contribution of genes to that trait.

The second assumption is that shared environmental influences contribute equally to MZ and DZ twin pairs. Shared environmental influences (or common environment influences) are environmental influences that have the effect of making siblings (or other family members) more similar to one another (Dick, 2005). Shared family influences might include the socioeconomic status of the family (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), being exposed to parental psychopathology (Burt, 2009), parental education and parenting behaviours (Plomin, DeFries, Knopik, & Neiderheiser, 2013) or other shared family experiences (Bokhorst et al., 2003).

Although family influences are the focus of the current study, it should be noted that additional shared environmental influences that might explain similarities between family members, especially siblings, can be a result of shared communities, shared peer groups, and attending the same school (Dick, 2005). To reiterate the assumption; twin studies are based on the hypothesis that any of these shared influences (be it parenting,

socioeconomic status, or shared social experiences) have a comparatively equal impact on MZ and DZ twins, regardless of genetic similarity. All twins will be exposed to these specific environmental influences in the same way as their twin.

When twin studies assess and compare MZ and DZ twins who are reared in households with genetically related parents, the final assumption made is that twins are also affected by non-shared environmental influences. These are influences that exist independently for different family members (Plomin & Knopik, 2012). This describes different environmental influences that twins' experience, including differential parental treatment or having different peer-groups and friends. If twin research is based on the assumptions that MZ twins are genetically identical and experience an equal, shared-environment influence, any differences found between MZ would be a result of individual experiences.

Studies that utilise traditional twin models therefore attempt to measure the contribution of genetic and environmental influences to phenotypic variations in twins, taking into account genetic, shared environment and non-shared or unique environment contributions to twins typically raised with their biological families. The methodology involved in twin studies however is continuously adapting and evolving to better fit the knowledge that has been acquired from the research area. The relationships between twins and their children or other family members for instance, are now explored in extended twin designs (Boomsma et al., 2006). The expansion of original twin studies provides the opportunity to study a variety of genetic and environmental relationships that are unable to be examined when studying twins alone, such as the influence of shared environment and non-additive gene effects simultaneously, which can be assessed if studying the children or siblings of twins.

The limitations of twin studies have been discussed in detail by Bundey (1991). Criticisms against twin designs include disputes over heritability estimates, issues relating to the equal environment assumption, misclassification of MZ twins and the notion that MZ twins are 100% genetically related. In terms of investigating the impact of family socialisation on children, the main limitation in traditional twin studies is its inability to control for passive gene-environment correlation (see gene-environment correlations section for an in-depth overview). A child is typically reared by a parent who they are genetically related to, thus they have both genes and environmental experiences in common. It is not possible therefore to determine if associations between parent and child (such as parental depression and negative child outcomes) are a result of genes or environment.

Traditional twin studies have been expanded as an attempt to remediate the limitations outlined. For example, as parents of biologically related children provide the environmental context and transmit genetic makeup to their children, environmental and genetic processes that might influence child development are confounded (D'Onofrio et al., 2005). To remediate this, researchers began exploring the children of twins in Children of Twins (CoT) research designs. The premises of these studies is that first cousins of identical twins will share 50% of their genes but will be raised in different environments, thus the CoT design is able to disentangle genetic and environmental contributions.

Findings from twin studies seem to support environmental risk factors for the development of psychopathology in children and adolescents. Rice et al. (2006) investigated the influence of family conflict in predicting depressive symptoms in a longitudinal twin design. Significant gene-environment interactions were observed, with effects of family conflict on depressive symptoms being greater in children and

adolescents at genetic risk of depression. This supports the importance of studying the impact of environmental influences in the development of depression, especially when children are at an increased risk (such as when living with a clinically depressed mother).

Adoption studies

The separation of genetic and environmental effects within a prospective adoption study operates differently from the previously described twin study designs (Leve et al., 2007). Genetic contributions and intrauterine environmental influences are assessed using biological birth parent information, if information on the birth parent is available (e.g. The Early Growth and Development Study; Leve et al., 2007). Post-natal environmental impacts are measured by assessing factors which might influence a child from their rearing environment (e.g. indices of family environment, adoptive parental psychopathologies, and school or community environments). Adoptive parents share post-natal environment but no genes with the adopted child whereas biological birth mothers share genes but typically only the intrauterine environment with the child. There are therefore a wide range of gene-environment processes that can be examined using longitudinal adoption studies. The influence of passive gene-environment correlation is removed as parents rear children they are biologically unrelated to. Evocative gene-environmental correlations can also be assessed, whereby a genetically inherited child trait may evoke a specific behavioural response from their adopted parent (Rutter et al., 2006).

Findings from research exploring the influence of family socialisation and parental psychopathology on child negative outcomes using adoption designs have contributed a wealth of knowledge to our understanding of how environmental and genetic influences might operate to impact development. Collectively, the results tackle

a range of questions that were previously unanswered within the field, such as how parenting might offset genetic risks to prevent the emergence of child problems in toddlerhood (Leve et al., 2009). They have also presented studies which compare and contrast the contribution of birth parent psychopathology with environmental risk factors, such as adoptive parent psychopathology or adoptive parenting on child outcomes (Marceau, Laurent, & Neiderhiser, 2015) highlighting, once again, how multifaceted the influences on development of psychopathology in children and adolescents appears to be.

Genetic contributions to negative child outcomes appear to be related to birth mother characteristics, including a slower cognitive processing speed (Roos et al., 2016), increased prenatal pregnancy risks and prenatal maternal drug usage (Marceau et al., 2015; Pemberton et al., 2010), internalising symptoms in birth mothers (Kerr et al., 2013; Marceau et al., 2015), and antisocial behaviour in birth mothers (Kerr et al., 2013). Postnatal environmental risks that have been highlighted within adoption studies include adoptive mother's uninvolved parenting (Roos et al., 2016), adoptive mother and adoptive father hostile parenting (Elam et al., 2014; Stover et al., 2012), and adoptive mother depression, with weaker environmental contributions of adoptive father depression (Elam et al., 2014; Natsuaki et al., 2014; Pemberton et al., 2010). Conflict within the adoptive parents' relationship has also been associated with negative outcomes (Harold et al., 2013; Stover et al., 2012).

A commonality in many of these studies however was the interaction observed between genetic and environmental risks for the development of negative outcomes in young children. Leve et al. (2009) observed how adopted children may be at differential risks for specific outcomes associated with forms of adoptive parenting, depending on a child's genetic risk for psychopathology. Toddlers who were deemed to

be at high genetic risk, based on birth mother psychopathology (anxiety, depression, antisocial and drug use) benefited from structured parenting, whereas toddlers who were deemed to be at low genetic risk (based on birth mother psychopathology: anxiety, depression, antisocial and drug use) were at increased risk for negative outcomes for the same aspect of parenting (structured parenting). Differential outcomes for children at low and high genetic risks have also been demonstrated. A further study found that infants at a genetic risk for externalising problems and affect dysregulation demonstrated heightened attention to frustrating events only in families where the adoptive mother had higher levels of anxious and depressive symptoms (Leve et al., 2010). This is an example of an environmental effect (GxE). Children might inherit an emotional liability which makes them more sensitive to the effects of marital hostility, as Rhoades et al. (2011) found associations between marital hostility and toddler anger was not moderated by a low genetic risk (low levels of birth mother anger and frustration), however for those children who had been considered to be at high genetic risk (high levels of birth mother anger and frustration), adoptive parents' inter-parental conflict predicted later levels of toddler anger and frustration levels. Adopted children who demonstrated greater attention control in infancy appeared to mitigate genetically based risks for internalising problems when raised by adoptive parents who were rated as having low levels of anxiety (Brooker et al., 2014). The authors concluded that the findings might suggest that, for children who are genetically susceptible to developing internalising problems and are raised in a low-risk environment, attentional control might serve as a protective factor from the development of internalising problems.

Finally, evidence of evocative effects of adopted child genetic influences on birth parents have also been identified; birth mother low behavioural motivation predicted toddler low social motivation which predicted both adoptive mother-child and

father-child hostility (Elam et al., 2014), which in turn has been shown to impact negative outcomes in children.

In-vitro fertilisation studies

The introduction and development of in-vitro fertilisation (IVF) has provided individuals who have faced problems with infertility and are unable to conceive children naturally with the opportunity to become parents through assisted reproductive technologies (Hammarberg, 2001). These assisted reproductive technologies might use egg and/or sperm donation, embryo donation, homologous IVF and gestational surrogacy.

IVF families provide a unique and novel opportunity to study genetic and environmental influences on the development of children, as children are reared by either genetically unrelated parents (embryo donation), genetically related parents (homologous IVF and gestational surrogacy), genetically unrelated fathers but genetically related mothers (sperm donation), or genetically unrelated mothers but genetically related fathers (egg donation) (Harold, Shelton, & Rice, 2008). This provides researchers with the chance to study the interaction of genetically related parents versus parent-child dyads that are not genetically related. It offers the opportunity to examine whether genetic, intrauterine and/or environmental influences uniquely, jointly or collectively contribute to variations in children's developmental outcomes. The ability to unambiguously differentiate between these effects cannot be accommodated through the application of any other genetic epidemiological design (Harold et al., 2013). The proportion of variance (σ^2) underlying any index of health and behaviour as contributed to by genetic (σ^2), intrauterine (σ^2) and environmental (σ^2) factors can be identified across the groups, as described in Table 2.

The premise of interpreting research from an IVF study therefore is that if parents are genetically unrelated to the child they are rearing, influences from parent-to-child will most likely be the result of environmental influences, or children may evoke specific behaviours from their parents that might have genetic attributes. This design remedies problems found in adoption designs where there might be a time delay between the birth of a child and adoption, or the inability to control for intrauterine environmental influences. It can therefore remediate the confounding influence of intrauterine influences of genetic effects on child outcomes in specific groups.

 Table 2: Variance components across IVF and surrogacy groups

Conception Group	Mother-child	Father-child
Homologous IVF	$0.5\sigma_{g}^{2}+\sigma_{u}^{2}+\sigma_{e}^{2}$	$0.5\sigma_{g}^{2} + \sigma_{e}^{2}$
Sperm donation	$0.5\sigma_{g}^{2}+\sigma_{u}^{2}+\sigma_{e}^{2}$	σ_{e}^{2}
Egg donation	$\sigma_{u}^{2} + \sigma_{e}^{2}$	$0.5\sigma_{g}^{2} + \sigma_{e}^{2}$
Embryo donation	$\sigma_{u}^{2} + \sigma_{e}^{2}$	σ_{e}^{2}
Surrogacy	$0.5\sigma_{g}^{2} + \sigma_{e}^{2}$	$0.5\sigma_{g}^{2} + \sigma_{e}^{2}$

SOURCE: Rice & Thapar (2010)

The Cardiff-IVF study (Harold et al., 2008) provides an example of how genetically sensitive research designs have evolved to better understanding the nature of nurture (Elam et al., 2014). It is however described as a genetically informative research design (as opposed to a genetically sensitive research design) as donor information is unavailable. Future extensions of this research design however aim to include this additional information. These studies highlight the importance of utilising such genetically sensitive designs to understand family processes that may impact on risk for child outcomes, removing passive rGE. The IVF design is therefore informative in identifying whether specific family processes have likely causal effects on child outcomes, or are better accounted for by gene-environment correlations.

Rationale of thesis

Overall, this introductory chapter presents evidence that suggests that parental depression, interparental conflict and parent-child hostility are risk factors for the development of depressive and aggressive symptoms in children and adolescents. In particular, it emphasises the importance of understanding how parental depression, interparental conflict and hostile parent-child relationships are related to one another, and how they contribute to the development of mental health problems in children and adolescents. It highlights the associations between parental depression on relationships within the family (both interparental and parent-child) and how interparental conflict can have a negative impact on parenting and the parent-child relationship and associations between hostility in family relationships and the development of depressive and aggressive symptoms in children and adolescents. It also indicates the need to further explore the role of father depression, fathers' perception of interparental conflict and father-child hostile relationships in the development of mental health problems in children. Furthermore, evidence has been presented that demonstrates advances in how we can examine these associations across a broad range of research designs, including novel, genetically informative research designs that allow us to disentangle genetic and environmental influences for the associations between parental depression, interparental conflict and parent-child hostility on the development of psychopathology in children and adolescents.

The current programme of research will expand on the existing evidence presented in this chapter. What remains unexplored is how parental depression, (specifically the inclusion of paternal depression) interparental conflict and parent-child hostility interact to impact the development of depressive and aggressive symptoms in a range of child developmental stages across genetically related and genetically unrelated

parent-child dyads. This thesis therefore utilises four data sets to examine associations between parental depression ,hostile family relationships and psychopathology in children and adolescents: (1) a community sample of low risk adolescents; (2) a sample of adolescents living with a clinically depressed mother; (3) a sample of children conceived through IVF; and (4) a sample of adopted children, their biological mothers and their adoptive parents. This thesis is comprised of three primary empirical chapters.

Overview of empirical chapters

Chapter 2: Study 1. The first study of this thesis utilises two longitudinal samples of adolescents living in the same geographical area of the UK; a community sample of low risk adolescents (The Welsh Family Study; WFS) and a sample of children living with a parent who has experienced recurrent depression (Early Prediction of Adolescent Depression; EPAD). It focuses specifically on the associations between maternal depression with interparental conflict and mother-child hostility in early adolescence. It will build on the existing literature by analysing and comparing associations between study variables (parental depression, interparental conflict, parent-child hostility and symptoms of psychopathology in children) across the two samples outlined. The purpose of this study is to examine these associations in two samples of early adolescents to determine if the risks associated with maternal depression, interparental conflict and mother-child hostility for the development of depressive and aggressive symptoms in adolescents differ for children living with or without a mother who has experienced clinical depression.

Chapter 3: Study 2. The second study of this thesis examines how parental depression, interparental conflict and parent-child hostility are associated with the development of depressive and aggressive symptoms in middle childhood. It builds on the previous study (chapter 2 study 1) in several important ways. Firstly, it will consider

the influence of fathers within these contexts; how fathers' depression and father-child hostility might be associated with the development of depressive and aggressive symptoms in their children. Secondly, how fathers may be influenced by interparental conflict and impact on processes associated with child depression and aggression symptoms. Thirdly, it will use a genetically informative research design (in-vitro fertilisation sample; IVF) to disentangle genetic and environmental influences (of both mother and father) for child developmental psychopathology (Rutter, 2007; Rutter, Pickles, Murray, & Eaves, 2001). Thirdly, it will examine these processes during middle childhood so that we might see how these mechanisms operate during different developmental periods. This could provide an indication of where to target early interventions to reduce negative outcomes in children exposed to parental depression and hostile relationships within the family.

Parental factors associated with development in children have historically focused on the influence of mothers. There are several reasons that might offer explanations as to why mothers have been the primary focus of these investigations. Mothers have typically been the primary caregivers of infants, as such it has been suggested that she has more interactions with the child, therefore these influences have been viewed as more important on child developmental outcomes than father-child interactions (Cabrera et al., 2000).

Initial explorations as to a fathers' capabilities to influence their child appeared to be primarily concerned with their presence-vs-absence, as opposed to the individual characteristics that might impact a child's development. Researchers have more recently sought to recognise and understand the importance of fathers' role within the family.

Lamb & colleagues work has been prominent in promoting early and continued empirical evidence into the importance of the father for child development. In 'The Role

of the Father' (Lamb, 2004) he re-articulates the decades of work that has demonstrated that fathers not only play a role in child development, but they are also present and involved in their children's lives, having both positive and negative impacts on their development.

The second study of this thesis will not only examine the associations between maternal depression and mother-child hostility, but will also include examination of the association between father depression, father-child hostility and child psychopathology. As outlined in the previous chapter, multiple researchers have shown that maternal depression can have detrimental effects on their child including increased risks for developing both internalising, emotional problems and external, behavioural issues (for a review see Goodman et al., 2011). Far fewer researchers have directed their attention to paternal depression on the development of psychopathology in children (Reeb et al., 2015).

Chapter 4: Study 3. The first study of this thesis outlined associations of maternal depression, interparental conflict and mother-child hostility with the development of depressive and aggressive symptoms for high-risk and low-risk adolescents. Findings here suggested the importance of processes from maternal depression to interparental conflict and interparental conflict to mother-child hostility on the development of depressive and aggressive symptoms in adolescents. The second study extended these analyses by including an examination of father depression, father's perception of interparental conflict and father-child hostility on the development of depressive and aggressive symptoms in children within a genetically informative research design. Here it was found that environmental mechanisms helped explain the associations between parental and child psychopathologies, perhaps more strongly for child aggression than child depressive symptoms. The study was limited as it was a

cross-sectional model that could not simultaneously examine the associations between mother depression, father depression and mother-child and father-child hostility. The third study brings together findings from study 1 and study 2, and contributes further to the examination of parental depression and family socialisation processes on the development of depressive and aggressive symptoms by examining these processes in a longitudinal model, which can examine these processes across time thus allowing more confidence in the direction of associations between these measures due to the temporal ordering of study variables. It also includes mothers and fathers in the same model, which enables us to examine whether there are independent effects of mother depression, father depression and parent-child relationship hostility, with the additional benefit of being able to assess a marker of background genetic risk (birth mother depressive symptoms). The study also utilises a younger sample of children (aged 18 months during first assessment, 7 years at final assessment) which will enable us to examine very early processes to identify whether there are earlier intervention points to reduce risks of negative mental health problems in children, and to see whether these processes (parental depression, interparental conflict, parent-child hostility) are similar at different phases of child development.

Chapter 5: Summary, conclusions & putting findings into practice. The final chapter serves to provide a summary of the primary findings of the thesis, highlighting the strengths of the individual studies and the thesis as a whole. Limitations will be examined, whilst directions for future research will be considered. The results are discussed with respect to the benefits of using a process-orientated approach to understanding the development of psychopathology in children and adolescents. Specific attention will be given to environmental influences that have been identified within the studies that might be potentially modifiable to improve outcomes in children

exposed to parental depression and disrupted family relationships. The implications of the findings for practice and policy will be explored, with an overview provided of the current interventions and preventions that aim to reduce the negative influences of parental depression and hostile marital relations exerted on children and a discussion of processes these interventions target that the current study adds.

Chapter 2: Examining associations of maternal depression, interparental conflict and mother-child relationships with the development of depressive and aggressive symptoms in early adolescence using a community and a clinical sample

Part of this chapter has been presented at the following presentations;

- Oral Presentation on "Examining links between maternal and paternal mental health & child psychopathology: How parents may (or may not) help children", at Postgraduate Conference, Leicester, Dec 2012.
- Oral Presentation: "The influence of maternal depression and family socialisation on offspring psychopathology" at 2nd year Psychology PhD Conference, Sussex, June 2014
- Oral Presentation: "Parental depression and family socialisation on the development of depressive and aggressive symptoms in children and adolescents", presentation at CDRiP, Sussex, June 2015
- Oral Presentation: "The influence of maternal depression and family socialisation on the development of depression in adolescents", presentation at International Congress of Clinical and Health Psychology with Children and Adolescents, Madrid, November 2015

The introductory chapter of this thesis reviewed literature and theories outlining how parental depression and family relationships (interparental and parent-child) impact on the development of negative outcomes in children and adolescents. The thesis primarily focuses on understanding how these influences impact the development of depressive and aggressive symptoms in children and adolescents. It aims to improve understanding of these associations by using a range of research designs to assess how, why and when these influences might impact children.

The first study of this thesis aims to examine the influence of maternal depression, interparental conflict and hostile mother-child relationships on the development of depressive and aggressive symptoms in early adolescence. It will assess how these associations might differ for high-risk adolescents living with a mother who has been identified as experiencing clinical depression compared to low-risk adolescents from a community sample. Evidence presented in the first chapter highlighted that children reared by a parent with depression are more likely to experience symptoms of psychopathology (Goodman & Gotlib, 2002).

Psychopathology in adolescence

Early adolescence is viewed as being a critical developmental transition, characterised by rapid growth and change (World Health Organization, 2015) and is considered to be a predominantly difficult period in development (Arnett, 1999; Duffett, Johnson, & Farkas, 1999). Adolescents are at a high risk for developing psychopathologies and have been identified as an age group that is particularly vulnerable to experience problems with mental health given the simultaneous influence of physical and social change (American Psychological Association, 2002; Gluckman, 2011). The majority of adolescents however, transition through these changes and

challenges without developing significant social, emotional or behavioural difficulties (Steinberg & Morris, 2001).

Although prevalence rates for psychopathologies in adolescents varies across studies, what consistently appears is that symptoms of psychopathology within this age group are increasing (Collishaw et al., 2004; Rubin & Pepler, 2013). Conservative estimates of the prevalence rates for psychopathologies are around 10% for adolescents aged between 11 to 12 years which increases to 12% for adolescents aged between 13 to 15 years (Ford et al., 2003). Costello and colleagues (2003) assessed children and adolescents at age 9, 11 and 13 years. At the beginning of the study, around 23% of the samples were reported to have mental health problems, which increased to almost 38% at the end of the three-month study. Similarly, Kessler et al. (2005) followed 13 to 17 year olds for a period of 12 months and observed rates of mental health problems increasing from 23% to 40%.

Psychopathologies experienced in early adolescence are typically grouped into internalising and externalising problems. Internalising problems are viewed as emotional difficulties and include symptoms of depression, anxiety and somatization (Reynolds et al., 2014). Externalising problems are more typically associated with harmful, disruptive and disinhibited behaviours and include conduct disorders and aggressive behaviours (Ford et al., 2003; Kovacs & Devlin, 1998).

Although not specifically examined within this study, gender differences for internalising and externalising problems begin to emerge during adolescence. Prior to adolescence, gender differences in internalising problems such as depression and anxiety, are rarely found. During adolescence these problems become more common in females than their male counterparts (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998; Nolen-Hoeksema & Girgus, 1994). Additionally, externalising problems appear

to be more common in males than females after adolescence (Leadbeater & Kuperminc, 1999).

The current study will be specifically focussing on associations of maternal depression, interparental conflict and hostile mother-child relations with the development of psychopathologies (depressive and aggressive symptoms) in adolescents.

Depressive symptoms in adolescence

Adolescents who suffer from depression and display depressive symptomology, are characterised as having low moods (Lau & Eley, 2010), lack of enthusiasm (Huberty, 2012) and frequently withdraw socially from family members and peers (Witvliet et al., 2010). Depression in adolescents is also associated with a number of harmful and destructive behaviours including increased substance abuse, cigarette smoking, high risk sexual behaviour, physical health problems, impaired social relations, and increase in suicidal ideation with a thirty-fold increased risk of suicide completion (Birmaher et al., 1996; Brent et al., 1998; Stolberg et al., 2002; Thapar et al., 2012). Interest in the development of and presence of depression in adolescents has grown dramatically in the last decade (Nolen-Hoeksema & Hilt, 2013). The prevalence of depression in adolescents aged between 13 to 18 years is 11.2% and as such, it is viewed as one of the most prevalent mental health disorders experienced by this age group (Merikangas et al., 2010).

Depressive symptoms in adolescence are associated with significant impairment to interpersonal relationships, social competence, academic performance, and increased levels of substance abuse and behavioural problems (Birmaher et al., 1996; Hankin, 2006). Studies that have examined trajectories for depression and depressive symptoms using longitudinal data have identified that depression in adolescence predicts increased

risk of suicide which is the third leading cause of adolescent fatality (Gould, Greenberg, Velting, & Shaffer, 2003). There is also evidence to suggest depressive symptoms experienced during adolescents are not only associated with current problems such as higher levels of substance abuse (Hankin, 2006), but are also consistently associated with depression in adult populations (Costello et al., 2003).

The current study focuses on symptoms of depression in adolescents, rather than a clinical diagnosis. Depressive symptoms show a strong continuity to clinical depressive disorders (Rice et al., 2002) and it is widely accepted that a continuum exists on which both symptoms and clinical diagnosis of depression show both concurrent and future impairment (Costello et al., 2003).

Aggressive symptoms in adolescence

A global problem has been identified regarding an increase in antisocial and aggressive behaviour incidences within adolescent populations (Collishaw et al., 2004; Piko, Keresztes, & Pluhar, 2006). The individual, societal, clinical and political implications of increasing rates of behavioural problems in adolescents is deserving of intensive research in order to better understand the mechanisms involved in the development of aggression and antisocial behaviour (Rubin & Pepler, 2013).

Aggression in adolescents can manifest in an array of behaviours including problems with emotional regulations, manipulative behaviours, argumentative reaction to situations, and verbally and physically threatening behaviour or bullying tendencies (Karriker-Jaffe, Foshee, Ennett, & Suchindran, 2008). Studies have emphasised the stability of aggressive behaviours within this age group, however a substantial amount of youth desist in aggressive behaviours over time (Loeber & Hay, 1997).

Identifying risk factors for the development of aggressive behaviours and targeting environmental mechanisms involved in the aetiology of these disorders is a

necessary step in alleviating the risk of potential negative outcomes. Risk factors associated with the development of aggressive symptoms in adolescents include overly harsh parental discipline, maltreatment, maternal consumption of nicotine during pregnancy, parental separation, teen pregnancy, association with deviant peers, social and economic disadvantages and parental psychopathology (Jaffee, Strait, & Odgers, 2012). Aggressive behaviours are more common amongst adolescents who have previously experienced symptoms of psychopathology, including depression and anxiety (Forero, McLellan, Rissel, & Bauman, 1999; Piko & Pinczés, 2014).

Risks associated with the development of psychopathology in adolescence

Identifying factors that contribute to the development of depressive and aggressive symptoms in adolescents might help to inform intervention and prevention strategies that could alleviate the negative impact of psychopathology from an individual, societal and clinical perspective (Harold et al., 2011; Silberg, Maes, & Eaves, 2010). This is especially salient for those adolescents who have been identified as being at a high risk of developing behavioural and emotional problems.

Three major risk factors which have previously been identified as putting adolescents at an increased danger of developing psychopathology are; maternal psychopathology (Downey & Coyne, 1990), negative mother-child relationships (Erel & Burman, 1995; Kochanska, Brock, Chen, Aksan, & Anderson, 2015) and interparental conflict (Grych & Fincham, 1990; Harold, Elam, Lewis, Rice, & Thapar, 2012; Reynolds et al., 2014). What has not been examined is how these risk factors that are associated with the development of depressive and aggressive symptoms in adolescents living with a clinically depressed mother differ for adolescents who have not been exposed to maternal clinical depression.

Maternal Depression

Parental psychopathology has repeatedly been highlighted as a major risk factor for negative developmental outcomes in children (Mars et al., 2013; Sellers, 2012). More specifically, children who live with a depressed mother are at a greater risk than those residing with a non-depressed mother to develop a multitude of negative outcomes including depression, anxiety, antisocial behaviour, impaired inter-personal relationships with family members and peers, and a marked reduction in academic achievement (Weissman et al., 2006). Maternal depression has been identified as major risk factors for negative developmental outcomes in children (Beardselee et al., 1998; Mars et al., 2013; Sellers, 2012; Weissman, et al., 2006) and it has also been associated with disruptions in the parent-child relationship, exposure to stressful family life events and child depressive symptoms (Rice, Lewis, Harold, & Thapar, 2013).

Associations between maternal depression and child outcomes are well replicated across a variety of study designs including longitudinal (Sellers et al., 2013), novel methodology such as IVF studies for genetically related and unrelated families (Rice et al., 2013) and adoptive studies (Kerr et al., 2013). Children who live with a depressed mother are up to four times more likely to develop depression themselves (Beardselee et al., 1998). Attempts have been made in recent years to understand the processes underlying the transmission of elevated depressive symptoms from mothers to their children. Although it appears that shared genes can act as a predisposition for depression (Harold et al., 2011; Rice et al., 2002), genetically sensitive research designs (whereby genetic and environmental contributions can be studied relatively separately) have also supported the importance of environmental risks in the transmission of depression from mother-to-child. This evidence provides sufficient justification for the examination of environmental mechanisms involved in the transmission of depression,

as although being genetically related to a mother with depression might predispose a child to developing depressive symptomology, studies have demonstrated that depression can be transmitted, even in the absence of shared genes (Harold et al., 2011; Lewis et al., 2013; Rice et al., 2002).

Research has shown associations not only between depression in mothers and internalising problems (such as depression) in adolescents, but also with externalising disorders (such as aggression) in children (Kovacs & Devlin, 1998). Elevated rates of maternal depression were found to be predictive of conduct problems in children (Gravener et al., 2012). Environmental factors previously associated with the transmission of maternal psychopathology to aggressive symptoms in their children, include harsh or hostile parenting in infancy, though a more complex interaction between genes and biological process have been put forward to help understand why children might develop externalising as oppose to internalising problems (Rutter & Silberg, 2002).

As evidence has been consistently found for the effects that maternal depression has on children, research now aims to establish the mechanisms and processes through which maternal depression might explain these negative outcomes. That is, how and why are these children put at additional risks compared to those with non-depressed mothers (Goodman & Gotlib, 1999). Pathways that might explain associations between maternal depression and negative outcomes include: i) genetic heritage ii) impairment of neuroregulatory systems iii) the experience and effects of living with a depressed mother and (iv) exposure to environmental stress (Goodman & Gotlib, 1999). The present study will be focussing on the experience and effects that children of depressed mothers are exposed to, specifically the associations between maternal depression and increased marital hostility and maternal depression with mother-child relations.

Parent-child relationships

Early relationships that a child has with their primary caregiver have long been viewed as instrumental in their development (Bowlby, 1969; Shonkoff et al., 2004). Parent-child relationships have also been consistently recognised as key for the development of children's well-being (Kagan & Snidman, 1999; Serot & Teevan, 1961). The parent-child relationship during adolescent development has receive also received much attention (Laursen & Collins, 2009). Although the parent-child relationship is likely to alter during this developmental period, research indicates that it remains important as a social and emotional resource for adolescents (Collins & Steinberg, 2006). Impairments in parenting that impact the parent-child relationship are associated both with maternal depression and the increased risk for child psychopathology. The mother-child relationship has been identified as a central mechanism for the transmission of psychopathology across generations of a family. Maternal parenting behaviours and maternal hostility have been associated with both externalising problems, such as aggression (Murray, Dwyer, Rubin, Knighton-Wisor, & Booth-LaForce, 2014) and internalising problems, such as depression in adolescents (Lewis et al., 2013; McLeod et al., 2007).

Parenting disruptions in mothers who experience depression are regularly reported; mothers who are depressed express more hostility and lower levels of warmth towards their children than parents without depression, they are also more likely to be less emotionally available to, and less supportive of their children (Cummings & Davies, 1994; Suveg, Shaffer, Morelen, & Thomassin, 2011). These disruptions in parenting have been examined as one mechanism through which maternal depression may exert negative effects on children (Downey & Coyne, 1990; Goodman & Gotlib, 1999; Marmorstein et al., 2004), with findings from Davies & Windle (1997) suggesting

that the parent-child relationship mediates the association between parental depression and adolescent depression.

The association between parenting and children's internalising symptoms appears to be inconclusive with evidence from empirical studies both supporting and discrediting the relationship. Findings from both community and clinical samples have suggested that child internalising problems are associated with disruptions in parenting (Psychogiou, Daley, Thompson, & Sonuga-Barke, 2007). It is predicted that children might feel distressed when they have experienced hostile interactions with their parents and that this stress might manifest in part in their depressive symptoms (Lewis et al., 2013). Elevated levels of maternal criticism appears to be more closely associated with childhood experiences of depression than other child psychopathologies (Asarnow, Tompson, Woo, & Cantwell, 2001). Disruptions in parenting might be a mediating mechanisms explaining associations between maternal depression and child internalising problems (Davies & Windle, 1997) or, parenting and maternal depression might act as two separate risk factors for the development of internalising problems. However, some evidence suggests that maternal parenting factors are unrelated to children's internalising problems (Foster et al., 2008; Frye & Garber, 2005).

Parenting, especially high levels of maternal hostility and criticism, have been associated with the development of externalising problems in children including conduct problems and antisocial behaviours (Burt et al., 2005; Vostanis, Nicholls, & Harrington, 1994). Parenting disruptions have also been identified as mediating the association between maternal depression and child externalising symptoms (McCarty & McMahon, 2003). As with internalising problems, some studies suggest maternal depression and parenting might have independent associations with externalising problems in children (Nelson, Hammen, Brennan, & Ullman, 2003).

Interparental conflict

Adolescents who live in households that are exposed to high levels of frequent and poorly resolved interparental conflict, are at a higher risk for a range of negative outcomes (Grych & Fincham, 2001). These include problems with academic achievement and impaired cognitive functioning (Ghazarian & Buehler, 2010; Harold et al., 2007; Hinnant et al., 2013). They are also more likely to report negative experiences in social situations such as issues with social competence and withdrawal than children who have not been exposed to increased levels of interparental conflict (Grych & Fincham, 1990; Paley et al., 2000). Interparental conflict has also been implicated as a risk factor for the development of psychopathology in adolescents, with interparental conflict being associated with internalising and externalising problems in children, including the presence of depressive and aggressive symptoms (Cummings, George, McCoy, & Davies, 2012; Grych et al., 2000; Grych & Fincham, 1990; Hanington et al., 2012).

Research into the association between interparental conflict and marital discord with adolescent's behavioural and psychological problems has emerged as a central feature for the impact of family socialisation on the development of psychopathology in children and adolescents during the past 30 years. The 1970's produced a diverse spectrum of research hypothesising the effects (or lack thereof) of negative parental interactions on children (Emery, 1982). Some psychologists postulated that adolescents with any form of behavioural problems would have parents with marital problems (Framo, 1975). Others did not believe there was enough evidence to demonstrate a clear or direct association between the two (Herzog & Sudia, 1973). Emery concluded that the discrepancies of findings and conclusions drawn from studies within this period were a result of unsophisticated research techniques. Chapter 1 of this thesis outlined

improvements in research techniques and they will be discussed in greater depth at a later point.

Cowan and Cowan (2002) highlighted the importance of understanding and identifying the links between destructive couple relations and negative outcomes for children. As with other indices of family socialisation, it is critical to understand *how* interparental conflict affects negative child development. Emery (1982) concluded that marital turmoil produces childhood disorders through the disruption of attachment bonds and altered parental discipline practices. The spill-over hypothesis offers further explanations of how these disrupted spousal relations might impact on children (Erel & Burman, 1995). Negative emotions and effects of conflict are believed by the authors to indirectly impact the child by disrupting the parent-child relationship. Animosity and hostility effectively 'spills over' in to the parent-child relationship from the couple relationship. The association between high levels of conflict in the parental relationship and high levels of hostility within the parent-child relationship seems to support this assumption.

Previous studies have demonstrated the impact of marital conflict on a host of family indices including parent-child relationships (Reynolds et al., 2014) and domains of children's emotional and social development (El-Sheikh, Buckhalt, Mize, & Acebo, 2006). Whilst several studies have examined them within the context of parental depression (Kouros, Papp, Goeke-Morey, & Cummings, 2014) these aspects of family socialisation and intergenerational transmission of depression have rarely been looked at in groups of at-risk versus not at-risk adolescents. This study aims to provide a better understanding of how these mechanisms operate for different groups of adolescents.

Comparing clinical and community samples

Maternal depression might increase risk for adolescent psychopathology indirectly through family processes, such as interparental conflict and parent-child hostility. Maternal depressive disorder and symptoms of maternal depression have both been identified as major risk factors for negative developmental outcomes in children (Beardselee et al., 1998; Mars et al., 2013; Sellers et al., 2013; Weissman, Myrna, Wickramaratne et al., 2006). Children who live with a depressed mother are at greater risk than those residing with a non-depressed mother to develop a multitude of negative consequences, including depressive symptoms and aggressive behaviours (Beardselee et al., 1998; Weissman, Wickramaratne et al., 2006). Furthermore, maternal depression also negatively impacts the interparental (Cummings & Davies, 1994; Downey & Coyne, 1990) and parent-child relationship (Foster et al., 2008; Weissmanet al., 2006).

Consequently, it is important to examine the effect of maternal depression symptoms on risk for child psychopathology via these family processes to help elucidate the mechanisms through which children and adolescents develop mental health problems. In addition, it is important to examine and compare these processes in children and adolescents exposed to differential levels of risk (e.g. at-risk adolescents who live with a mother who has experienced clinical depression compared with children and adolescents from a community sample). As the previously outlined, risk mechanisms (interparental conflict and hostile mother-child relationships) might operate differently for these groups of children and adolescents. Improving our understanding of how these processes operate within these different individuals might aid the targeting of specific interventions where children are at differential levels of risk, for example, due to exposure to maternal depression.

Current study

The aim of the current study was to examine the association between maternal depression, interparental conflict, mother-to-child hostility and measures of negative adolescent outcomes (depressive and aggressive symptoms), after adjusting for earlier levels of child problems with normative adolescents, and adolescents identified as being at high-risk as a result of residing with a mother who has experienced clinical depression. Few studies have examined these associations in a high risk/clinical sample compared with a community sample of adolescents.

It was hypothesised that there would be associations between levels of maternal depression and the indices of family socialisation (interparental conflict and mother-child-hostility) but these might be different for adolescents within the clinical and community samples, with previous research suggesting that adolescents living with a mother who has experienced clinical depression would be at greater risk of developing depressive and aggressive symptoms, which may be via increased vulnerability to the impact of negative relationships.

The present study employed two longitudinal samples; first, a high-risk group of adolescents from a UK study of parents with clinical major depressive disorder and their adolescent children (The Early Prediction of Adolescent Depression Study, EPAD, Mars et al., 2012). Second, a community sample of not at-risk adolescents was obtained from a longitudinal community sample of families and their children from the UK (Welsh Family Study, WFS; Grych et al., 2003; Harold et al., 2004; Shelton & Harold, 2008).

Method

Sample 1: The Welsh Family Study

The Welsh Family Study (WFS) was a three-wave longitudinal community-based study which recruited families (parents and one participating child) from the South Wales area of the UK. The study was primarily designed to assess experiences of family life and well-being and psychosocial development. Twelve secondary schools in South Wales were selected on the basis of their economic and social characteristics to obtain a demographically representative range and families were recruited from these schools. The sample is representative of families living in Wales and England with regards to family composition and ethnicity (ONS, 2002). See procedure for further details on recruitment. The three waves of data collection were conducted one year apart: Time 1 (1999); Time 2 (2000) and Time 3 (2001). The study comprises of 543 children living in the South Wales area, UK. Children aged 11to 13 years and 387 parents initially took part in the study.

The current study uses data from Time 1 (1999) and Time 2 (2000) of the study, where children were aged 11-12 years at Time 1 (M = 11.7 years) and 12 to 13 years at Time 2 (M = 12.7 years). Given the interest in the current study on interparental relationships, only data from two-parent families were used. This included 478 children, of whom 86.2% lived with their mother and father, 11.9% lived with their mother and step-father and 1.9% lived with their father and step-mother. Of this sample (N = 478), 37 did not participate at follow up (Time 2), therefore the maximum valid sample was 441.

Data from the children and their parents were only included if observations across all of the chosen measures were available, thus the final sample comprised of 264 families (60% of valid sample), 51.5% of this sample were boys. Child and parent

questionnaires were completed to assess overall well-being of the children and their families whilst the child was transitioning from primary (year 6) to high school (year 7) (UK school system) including assessments of the quality of family relations and economic conditions.

Procedure

Approval was sought by the researchers through contacting schools in the South Wales area. Initial contact was made with secondary schools in the South Wales area, following this parents of pupils were sent a letter to invite them and their child to participate in a research project that would be focused on the link between everyday family life and children's development. Researchers provided a presentation for parents at a parent-teacher evening and gave additional information and a letter about the project which included a consent form outlining goals for each stage of the project. No financial initiative was provided for participating families but parents were informed that a summary booklet outlining key research findings would be distributed to all participating families once the project had been completed.

Parent questionnaires were sent to the families by post as well as instructions for completing measures and stamped addressed envelopes for each parent to complete. The current study only used maternal ratings for the questionnaire to ensure comparisons with the Early Prediction of Adolescent Depression sample. Parents were advised to complete their questionnaires independently. A contact number provided if they had any concerns or queries. A variety of measures were included on parental questionnaires, parents were asked to report on marital and family interactions, parenting, child psychological health, along with family demographics and economic conditions.

Children completed their questionnaires during school hours. Children's questionnaires included measures which examined their perceptions on the quality of

their family environment and child psychological health. An overall debriefing was provided where researchers and children discussed conflict resolution tactics and children were encouraged to be open and speak how they felt after completing the questionnaires. No concerns were raised by children participating in this study during the debriefing session (Harold, Aitken, Shelton, 2007). The data utilised in the current study used mother and child-completed questionnaires from two time points (1999 and 2000).

Sample 2: The Early Prediction of Adolescent Depression Study

The EPAD study is a longitudinal study of the adolescent children of parents with recurrent depression (N = 337). Parents were primarily recruited from general practise after receiving treatment for at least two episodes of depression (78%), a previous study of adults with recurrent unipolar depression (19%) and from additional sources (3%). For the current study, families were selected from two-parent households due to the nature of the study (assessment of mother-child relationship and fathermother relationships) (N = 217). Two cases were not included due to ambiguity of whom the adolescent was living with at Time 1 of the study. Of the remaining sample, 80.2% resided with their mother and father, 19.8% with their mother and step-father. The study focuses predominantly on depressed mothers and their adolescent children, though there are 22 cases in which the index parent is a depressed father. These cases were not included in the current study. Children and adolescents were tested at two time points and had an age range from 9-17 years at Time 1 (M = 12.23 years, SD = 1.96) and 10-18 years at Time 2 (M = 13.67 years, SD = 2.00) approximately a year later. The final sample included 195 participants with females accounting for 58.3% of the sample.

Procedure

To be included in the study, parents were required to have a history of recurrent unipolar depression (at least two lifetime episodes, later confirmed at interview) with no previous history of a psychotic disorders, schizoaffective disorder or mania. Parents had to be living with a child that they were biologically related to and who was aged between 9 to 17 years. In families with multiple children, the youngest child was selected for two reasons. Firstly researchers wanted to avoid parents selecting children with the most problems. Secondly, the focus of the study was to examine the development of psychopathology across the course of adolescence. Children with moderate to severe intellectual disability that would impair their ability to complete questionnaires (IQ<50) were excluded. Within the duration of the study, two families were excluded as a result of the depressed parent reporting bipolar diagnosis.

Sixty-two GP surgeries from the South Wales area of the UK assisted with recruitment. Patients with recurrent depression were identified through electronic records using depression read-codes and/or more than one repeat prescription of anti-depressant medication for at least two episodes of depression. After an initial telephone screening, patients identified were contacted via a letter from the surgery asking if they would like to hear information regarding the research project. After 2 weeks, non-responders were sent a reminder letter, after this no further attempts to contact the patients were made. Patients who were willing to participate returned a reply card with their contact details to the research team. Patients were contacted by telephone to assess eligibility for participation in the study but before this point no contact had been made between the research team and patients. Due to patient confidentiality, the researchers were unable to see if there were any differences between responders and non-responders.

Participants from two pre-existing studies of adults with recurrent unipolar depression from the community (the Depression case control study, DECC; Depression Network, DeNT, Farmer et al., 2004; Korszun et al., 2004) were also used in the study. Recruitment for these studies had been through Community Mental Health teams and advertisements in local media and care centres. Participants who had previously consented to future contact were contacted with a letter which gave information relating to the current research study.

Lastly, community volunteers were recruited via posters that were situated in health centres, the University Hospital of Wales, and advertisements in a 'Depression Alliance' newsletter. Volunteers contacted the research team and were then provided with information about the study. They were then contacted and screened for eligibility and to confirm participation.

During the assessments, parents and children were sent questionnaires two weeks prior to interview. Interview assessments were primarily conducted in the family home. Researchers where possible, interviewed parents and children in different room using different interviewers. Parents and children were provided with a description of the study and provided written informed consent (or assent if under 16 years) at each assessment. These assessments lasted around 3 hours, families were compensated for their time (£20 voucher per family). Interviewers were aware of parental depressive symptoms but were not informed of additional clinical problems in parent or child psychopathology (Sellers et al., 2013).

Summary of Datasets

The current study uses data from the two data sets to examine the impact of family socialisation (interparental conflict and mother-child hostility) on the development of psychopathology (depression and aggression) in children and

adolescents, within the contexts of maternal depression. Where WFS is a community sample, EPAD is a sample of parents with clinical depression, all of whom had a history of recurrent unipolar depression, though they did not necessarily all have clinical depression during the assessment period. Almost one quarter of the mothers in the EPAD sample (23.2%) met criteria for major depressive disorder at the time of the baseline interview. This allowed for a comparison of the impact of family socialisation within an at-risk and not at-risk groups of children and adolescents.

Measures

Adult depressive symptoms (Time 1). In both samples, maternal depressive symptoms were assessed using the Beck Depression Inventory (BDI), a 21-item measure of depressive symptoms (Beck & Beamesderfer, 1974). The questionnaire asks adults to describe the way they have been feeling in the past week on a scale of 0-3, such as 'I do not feel sad' to 'I am so sad or unhappy that I can't stand it'. Maternal reports on the BDI had very good internal consistency for WFS (α = .83) and EPAD (α = .93). Mothers from the EPAD sample reported significantly higher depressive symptoms than the WFS sample (t (538) = 14.69, p<.001).

Child psychological adjustment (Time 1 & Time 2). The current study utilised a combined rater approach for children's depressive symptoms and aggressive behaviours (mothers and children reporting) to avoid reliance on a single reporter and provide a more robust measure of child and adolescent psychopathology (Harold & Conger, 1997).

For the WFS, *child depression* was measured using a mother-rated version of the Child Behaviour Checklist (CBCL; Achenbach, 1991). The Anxious-Depressed subscale of the CBCL was used to measure depression. Mothers were asked to indicate how well specific statements described their child within the past six months. Mothers

indicated that the statements were 'Not True', 'Sometimes True' or 'Very True' (on a scale of 0-2). Statements included child 'Complains of loneliness' and child 'Cries a lot'. Internal consistency across Time 1 and Time 2 were very good (α = .83 and α =.83 respectively). Children's self-report of depression was completed using the Child Depression Inventory (CDI; Kovacs, 1981). Children were given 26 groups of statements and were asked to pick one sentence that best described how they felt and thought for the past two weeks from each group. Groups included statements such as 'I am sad once in a while', 'I am sad many times' or 'I am sad all the time', and 'I do not feel alone', 'I feel alone many times', or 'I feel alone all the time'. Child-reports on the CDI had very good internal consistency for Time 1 (α = .84) and Time 2 (α = .86). Maternal and child ratings were summed at each time point to create the depression outcome measure.

Child aggression in the Welsh Family Study included the mother-rated aggression subscale of the Child Behaviour Checklist (Achenbach, 1991). Mothers were asked to indicate how well specific statements described their child within the past six months. Mother's indicated that the statements were 'Not True', 'Sometimes True' or 'Very True'. Statements included child is 'Mean to others' and child 'Gets into fights'. Internal consistency was very good across Time 1 and Time 2 (α = .86 and α = .86 respectively). Children rated their own levels of aggression in the WFS using the Buss and Durke 1957) hostility subscale. Children were given nine statements and asked to indicate how much each statement was like them. The available answers were 'Not at all', 'A little bit', 'Somewhat', 'A lot' and 'Exactly'. Statements included 'When I get angry I say nasty things' and 'If I have to use physical violence to defend myself, I will'. Internal consistency was very good at Time 1 (α =.82) and Time 2 (α = .84). Maternal

and child ratings were summed to create the depression outcome measure at each time point.

For the EPAD study, mothers and their child completed the Strength and Difficulties Questionnaire (SDQ; Goodman, 1997). The questionnaire is a brief, behavioural screening tool of 25 items. Subscales for the questionnaire are emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems and pro-social behaviour. The current study used the emotional symptoms subscale to assess depression symptoms (five items) which included statements such as 'I get a lot of headaches, stomach-aches or sickness', and 'I am often unhappy, down-hearted or tearful'. As with the CBCL, informants are asked to indicate if the statements are 'Not True', 'Somewhat True', or 'Certainly True'. Reliability estimates for mother's ratings of child emotional symptoms were good for Time 1 ($\alpha = .75$) and Time 2 ($\alpha = .81$). Child ratings of emotional symptoms had acceptable internal consistency (Time 1, $\alpha =$.66; Time 2, $\alpha = .68$). The conduct problem subscale was used to assess child aggression and included statements such as 'I get very angry and often lose my temper' and 'I fight a lot'. Reliability estimates for the conduct problem subscale for mothers were Time 1 $(\alpha = .70)$ and Time 2 $(\alpha = .69)$. Internal consistency for child reports of conduct problems were acceptable both at Time $1(\alpha = .60)$ and Time $2(\alpha = .66)$. Maternal and child ratings were summed to create the depression outcome measure at each time point.

Although the samples did not allow for a direct comparison of a single measurement of child psychological adjustment, the maternal rating for child depression (CBCL) for the WFS sample and SDQ (as used in EPAD) have been shown to be highly correlated with each other and both measures are able to discriminate between a psychiatric and community sample in detection of internalising and externalising problems in children and adolescents (Goodman & Scott, 1999). Furthermore, the child

rating for depressive symptoms (CDI) used in WFS has been shown to also be highly correlated with the SDQ (Koskelainen, Sourander, & Kaljonen, 2000).

Interparental conflict (Time 1). Interparental conflict (IPC) was measured in both samples using the hostility subscale of the Iowa Youth and Families Project (IYFP) Rating Scales (Melby, Ge, Conger, & Warner, 1995). IPC was assessed using mothers' ratings of hostility from their partners. The subscale of the questionnaire asked mothers to consider a period during the past month when they had spent time with their partners and asked them to rate on a scale of 1 to 7 (1 being 'Always' through to 7 being 'Never') that their partners got angry at them, criticised them or their ideas, shouted when upset and argued with them if there was a disagreement. Responses were reverse scored so higher scores indicated higher levels of hostility from partner to mother. Internal consistency was very good for WFS ($\alpha = .89$) and EPAD ($\alpha = .92$).

Parental appraisals of parent-child relations (Time 1). Hostile mother-child relationships were measured using the mother-rated hostility subscale of the IYFP Rating Scales (Melby et al., 1995). Mothers were asked to rate how often they had responded in a hostile manner towards their child in the past month. Internal consistencies were very good for WFS (α = .83) and EPAD (α = .89).

Analysis

To examine the associations between maternal depression, IPC, mother-child hostility and negative child outcomes in a high-risk and not at-risk group of adolescents, Structural Equation Modelling (SEM) was conducted using Mplus (Muthen & Muthen,1998-2001) to test the empirical validity of the proposed theoretical models (Figure 1 and 2). Child outcomes (child depression and child aggression) were tested in separate models examining the association with maternal depression, interparental

conflict and mother-to-child hostility as risk processes for child psychopathology in the WFS and EPAD samples.

Associations between maternal depression, family socialisation and negative child outcomes were further examined using path analysis for high-risk (EPAD) and adolescents from the community sample (WFS). The models were conducted using Mplus 7 (Muthen & Muthen, 2007). As the samples were obtained from two separate data sets, we were unable to use stacked modelling to compare pathways within the models. Differences between pathways in the models were examined using the Satorra-Bentler Scaled Chi-Square Difference Test (Satorra, 2000). Pathways were examined and compared separately e.g. pathways between mother-child hostility and child depressive symptoms within the EPAD and WFS models were compared.

Missing data

In the at-risk study (EPAD), there were no differences between those who had complete data and those who were missing for adolescent aggression symptoms (M = 3.87 for completed, M = 5.14 for missing, p = .051)_or adolescent depression symptoms (M = 6.49 for completed, M = 5.79 for missing, p = .43). In contrast, those families that did not participate at follow up had significantly higher maternal depression scores at baseline (M = 15.32 for completed, M = 21.81 for missing, p = .003).

There was a similar pattern of findings for missingness in the not at-risk (WFS) study; there were no difference between those who had complete data and those who were missing for adolescent aggression symptoms (M = 28.89 for completed, M = 29.78 for missing, p = .482) or adolescent depression symptoms (M = 14.47 for completed, M = 15.75 for missing, p = .226). In contrast, those families that did not participate at follow up had significantly higher maternal depression scores at baseline (M = 5.56 for completed, M = 6.96, p = .039).

The Little's test of missing data (Little, 1988) indicated that data were not missing completely at random for WFS child depression (χ^2 (38) = 63.76, p=.006), WFS child aggression (χ^2 (39) = 66.93, p = .004), EPAD child depression (χ^2 (26) = 48.52, p = .005), and EPAD child aggression (χ^2 (26) = 41.06, p = .031) models. List-wise deletion was used when running the models to ensure that models reflected the same families, so that the children and mothers used in the child depression models were the same that were used within the child aggression models (WFS child depression N=219; WFS child aggression N = 231; EPAD child depression N = 195; and EPAD child aggression N = 195).

Model Fit Statistics

Model fit statistics can be used to evaluate how accurately a statistical model reflects the raw data. The model fit statistics used within this study will be outlined. The conventional test of significance, the chi-square test (χ^2), examines the magnitude of difference between the sample and model fitted covariance matrices (Gierl & Mulvenon, 1995). The Bentler Comparative Fit Index (CFI) and the Tucker Lewis Index (TLI) are incremental fit indices which measure the improvement of the proposed model to the baseline (or independence model) and the author's suggest a value of 0.9 indicates an acceptable and 0.95 a good fit. The Root-Mean-Square Error of Approximation (RMSEA) is a measure of model misfit or poor fit per degree of freedom and a value of .08 implies acceptable model fit and .05 good model fit (Browne & Cudeck, 1993). It is important to avoid a reliance on one particular test or cut-off criteria as these can both be impacted by sample size and distribution bias, so fits should never be considered as absolute (Hu & Bentler, 1999). The extent to which the data fits the models will thus be assessed with multiple fit statistics reported based on the criterion described.

Results

Correlational analysis

Intercorrelations, means and standard deviations are presented for the associations between maternal depression and family socialisation for adolescent depressive and aggressive symptoms (Table 1) for the community (WFS) and clinical (EPAD) samples of adolescents.

For the clinical sample (EPAD), maternal depression was associated with interparental conflict (r = .22, p < .01), mother-to-child hostility (r = .31, p < .01), adolescent depressive symptoms at time 1 (r = .31, p < .01) and time 2 (r = .29, p < .01) and adolescent aggressive behaviours at time 1 (r = .29, p < .01) and time 2 (r = .28, p < .01). A different pattern of results was observed for the community sample (WFS) with maternal depression only being associated with interparental conflict (r = .36, p < .01), adolescent depressive symptoms at time 1 (r = .14, p < .05) and adolescent aggressive symptoms at time 2 (r = .17, p < .05).

Interparental conflict was associated with mother-to-child hostility for both WFS (r=.26, p<.01) and EPAD (r=.30, p<.01). Interparental conflict was also associated with child aggressive symptoms in WFS at time 1 (r=.16, p<.05) and time 2 (r=.16, p<.05).

In EPAD, interparental conflict was associated with adolescent depressive symptoms at time 2 (r = .17, p < .05) and adolescent aggressive symptoms at time 2 (r = .19, p < .05).

In WFS and EPAD samples, mother-child hostility was associated with all adolescent negative outcomes; adolescent depressive symptoms at time 1 (WFS, r = .49, p<.01; EPAD, r = .20, p<.01), adolescent depressive symptoms at time 2 (WFS, r = .29, p<.01; EPAD, r = .27, p<.01); adolescent aggressive symptoms at time 1 (WFS, r =

= .49, p<.01; EPAD, r = .46, p<.01) and aggressive symptoms at time 2 (WFS, r = .39, p<.01; EPAD, r = .47, p<.01).

For WFS, adolescent depressive symptoms at time 1 were associated with depressive symptoms at time 2 (r = .67, p < .01), adolescent aggressive symptoms at time 1 (r = .40, p < .01) and adolescent aggressive symptoms at time 2 (r = .39, p < .01). Similarly, adolescent depressive symptoms at time 1 in the EPAD sample were associated with adolescent depressive symptoms at time 2 (r = .72, p < .34) and adolescent aggressive symptoms at time 2 (r = .72, p < .34) and adolescent aggressive symptoms at time (r = .34, p < .01) and time 2 (r = .26, p < .01).

Table 1. Intercorrelations, Means, and Standard Deviations (S.D.) Among Constructs for Welsh Family Study (Lower Diagonal, N = 264) and Early Prediction of Adolescent Depression (Upper Diagonal, N = 195) Mothers and Children for Child Depression and Child Aggression.

Variable	(1)	(2)	(3)	(4)	(5)	(6)	(7)	Mean	S.D.
(1) Mother Depression	-	.22**	.31**	.31**	.29**	.29**	.28**	15.23	10.43
(2) Interparental Conflict	.36**	-	.30**	.09	.17*	.09	.19*	12.42	5.71
(3) Mother-to-child hostility	.09	.26**	-	.20**	.27**	.46**	.47**	12.71	4.48
(4) Child depressive symptoms T1	.14*	.12	.49**	-	.72**	.34**	.26**	6.63	4.31
(5) Child depressive symptoms T2	.08	.01	.29**	.67**	-	.30**	.39**	5.66	4.15
(6) Child aggressive symptoms T1	.13	.16*	.49**	.40**	.24**	-	.69**	3.89	3.08
(7) Child aggressive symptoms T2	.17*	.16*	.39**	.39**	.39**	.66**	-	3.22	3.08
Mean	5.33	10.27	27.43	14.52	14.93	28.33	29.13		
S.D.	4.45	4.16	7.57	7.99	8.90	8.97	8.99		

^{*}p <.05, ** p<.01

Path analysis

Associations between maternal depression, family socialisation and negative child outcomes were further examined using path analysis for at-risk (EPAD) and not at-risk (WFS) adolescents. The models were conducted using Mplus 7 (Muthen & Muthen, 2007). At-risk and not at-risk models were then compared to assess differences between these groups using the Satorra-Bentler Scaled Chi-Square Difference Test (Satorra, 2000).

Maternal depression, family socialisation and depressive symptoms in adolescents in the community sample

Figure 1a illustrates results for community sample of adolescents (WFS) with adolescent depression as an outcome measure for assessing the associations between maternal depression, IPC, and mother-child hostility. Maternal depression symptoms at Time 1 were significantly associated with adolescent depression symptoms at Time 1 (β = .23, p < .01). Maternal depression predicted an increase in interparental conflict (β = .35, p < .01) which in turn was associated with mother-child hostility (β = .31, p < .01). The model also showed that although adolescent depressive symptoms at Time 1 were associated with mother-to-child hostility at Time 1 (β = .35, p < .01), mother hostility at Time 1 did not predict adolescent depressive symptoms at Time 2 (β = .03, p=.59).Model fit indices were moderate (χ^2 (2) = 13.35, SD = 7.68; RMSEA = .10; CFI = .97; TLI = 0.88).

T1 T2

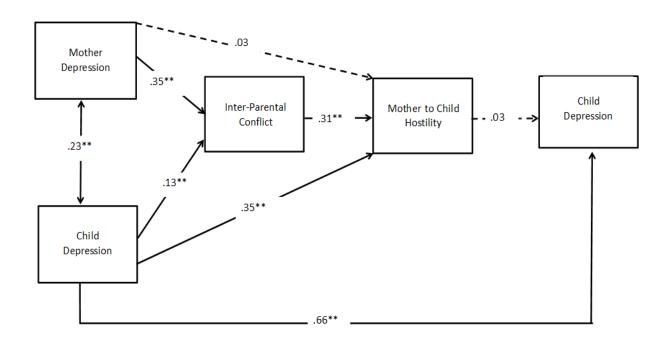


Figure 1a.. Model Results for Welsh Family Study for Child Depression. *p <.05, ** p < .01

Maternal depression, family socialisation and depressive symptoms in adolescents in the clinical sample

Figure 1b illustrates results for adolescents from the clinical sample (EPAD) with adolescent depressive symptoms as an outcome measure for assessing the associations between maternal depression, IPC, and mother-child hostility. Maternal depression at Time 1 was associated with adolescent depressive symptoms at Time 1 (β = .22, p < .01). Maternal depression also predicted mother-child hostility (β = .22, p < .01). Furthermore, maternal depression predicted interparental conflict (β = .20, p < .01) which in turn showed associations with mother-child hostility (β = .24, p < .01). Mother-child hostility (Time 1) was significantly associated with adolescent depressive symptoms at time 2 (β = .13, p < .05), this was a unique finding for the adolescents in the clinical sample as this pattern of associations was not observed for the adolescents

from the community sample (WFS). Unlike the adolescents in the community sample however, adolescent depressive symptoms at time 1 were not associated with interparental conflict (β = .06, p =.47) or mother-child hostility (β = .10, p =.19). Model fit indices were good ($\chi^2(2)$ = 2.16, SD = 1.63; RMSEA = .02; CFI = 1.00; TLI = 1.00).

The primary observational differences between the models for adolescent depressive symptoms for the clinical and community samples was the significant association between mother-child hostility and adolescent depressive symptoms which was only observed for the at-risk group, the direct association between maternal depression and mother-child hostility was also only observable in the clinical sample. Adolescent depressive symptoms at time 1 were associated with interparental conflict and mother-child hostility in the community sample, but not the clinical sample.

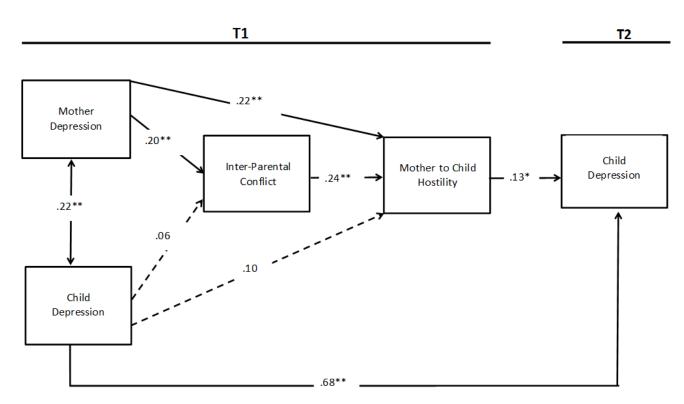


Figure 1b. Model Results for Early Prediction of Adolescent Depression for Child Depression. *p < .05, ** p < .01

Maternal depression, family socialisation and aggression symptoms in adolescents in the community sample

Figure 2a illustrates results for adolescents from the community sample (WFS) with adolescent aggressive symptoms as an outcome measure for assessing the associations between maternal depression, IPC, and mother-child hostility. Significant associations were exhibited between adolescent aggressive symptoms at time 1 with interparental conflict (β = .12, p < .05) and mother-child hostility (β = .37, p < .01). Maternal depression predicted increases in interparental conflict (β = .36, p < .01) which in turn was associated with mother-child hostility (β = .31, p < .01). The results also demonstrated a significant pathway from mother-child hostility (Time 1) to adolescent aggressive symptoms at time 2 (β = .14, p < .01) which was not observed for adolescents from the community sample for the depressive symptoms outcome. Model fit indices were good (χ^2 (2) = 1.87, SD = 1.2; RMSEA = .01; CFI = 1.00; TLI = 1.00).

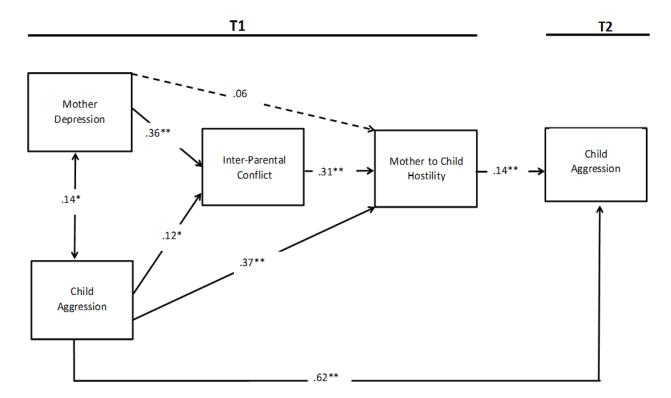


Figure 2a. Model Results for Welsh Family Study for Child Aggression. *p < .05, ** p < .01

Maternal depression, family socialisation and aggression symptoms in adolescents in the clinical sample

Figure 2b illustrates results for adolescents from the clinical sample (EPAD) with adolescent aggressive symptoms as an outcome measure for assessing the associations between maternal depression, IPC, and mother-child hostility. Associations between adolescent aggressive symptoms at time 1 with mother-child hostility were observed (β = .42, p < .01). Mother depression was associated with adolescent aggressive symptoms at time 1 (β = .25, p < .01). Maternal depressive symptoms predicted increases in interparental conflict (β = .20, p < .01). Associations between maternal depression with mother-child hostility were observed (β = .14, p < .05) which was not observed adolescents from the community sample. As with all models for adolescents from the clinical and community samples, interparental conflict was significantly associated with mother-child hostility (β = .22, p < .01) and in turn,

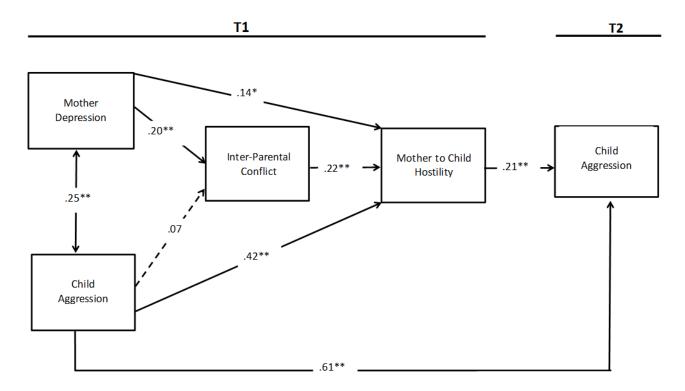


Figure 2b. Model Results for Early Prediction of Adolescent Depression for Child Aggression. *p < .05, ** p < .01

mother-child hostility was associated with adolescent aggressive symptoms at time 2 (β = .21, p < .01). Model fit indices were adequate ($\chi^2(2)$ = 1.79, SD = 1.02; RMSEA = .02; CFI = 1.00; TLI = 1.00).

Comparisons of models

Clinical (EPAD) and community (WFS) models for adolescent depressive and aggressive symptomologies were compared using the Satorra-Bentler Scaled Chi-Square difference test (Satorra, 2000). The pathway from maternal depression to interparental conflict differed between adolescents from the clinical and community sample ($\Delta \chi^2$ (1) = -5.35, p <.05) and paths from mother-child hostility and initial ratings of adolescent aggressive symptoms (Time 1) differed between the groups ($\Delta \chi^2$ (1) = -16.88, p<.01). No other significant differences were observed between measures in the models.

Discussion

The current study utilised two complementary, longitudinal research designs to examine the impact of maternal depression, interparental conflict and hostile mother-child relationships on the development of depressive and aggressive symptoms in two groups of adolescents from (i) a clinical and (ii) community sample. Although previous research has identified these processes as impacting the development of psychopathologies in adolescents, findings from the current study can help to improve our understanding of how these mechanisms might operate for adolescents living with a mother who has experienced clinical depression, compared to a community sample of adolescents who have not been exposed to the same risk.

The associations of maternal depression, interparental conflict and hostile mother-child relationships with adolescent depressive symptoms

Firstly, associations between the previously identified risk factors (mother depression, IPC and negative mother-child relationships) on the development of depressive symptoms in adolescents in clinical and community samples were examined. A repeated finding across both groups was the relationship between mother depression and interparental conflict and interparental conflict with mother-child hostility, highlighting the importance and strengths of these relationships for adolescents who have either been identified as at-risk or not at-risk. The findings of how these indices of family socialisation and parental psychopathology impact negatively on adolescents is supported by a range of previous studies (Downey & Coyne, 1990a; Harold et al., 2012; Kochanska et al., 2015). The current study enhances an understanding through the ability to assess these mechanisms across adolescents deemed to be at different risks for developing depressive symptomologies as a result of living with a mother who has experienced clinical depression.

The association between maternal depression, interparental conflict and hostile mother-child relationships on adolescent depressive symptoms in both groups should reinforce the importance of focussing on modifiable aspects of adolescents' home environment to reduce negative outcomes. However, there were two key differences in the group of adolescents from the clinical and community samples for the development of depressive symptoms in this study; first, hostile mother-child relationships were associated with a greater level of adolescent depressive symptoms in the adolescents from the clinical sample, but not in the community sample. Second, earlier levels of child depressive symptoms in adolescents from the community sample were associated with mother-to-child hostility. These findings might enable us to make inferences about

the development of depression in adolescents living with or without a mother who has experienced clinical symptoms of depression.

Given that the mother-child relationship is a consistent factor in predicting the development of psychopathology for adolescents in the clinical sample, but not to such a high degree for the community sample, it highlights the importance of maternal mechanisms in high-risk environments, where mothers have experienced clinical depression. Furthermore, adolescents in the community sample impacted on the mother-child relationships, but this was not a replicated finding in adolescents from the clinical sample. This might indicate that in low-risk families (with respect to the absence of a mother with clinical depression) other aspects of the family environment might be associated with the parent-child relationship which can then affect symptoms of psychopathology in adolescence (Lewis et al., 2013).

Overall it seems possible that adolescents living with a mother who has experienced clinical depression might be more vulnerable to being negatively affected by hostile mother-child relationships than those adolescents who have not. It is possible that they are exposed to multiple risks associated with their mother's depression such as increases in hostility within the interparental and parent-child relationship. Perhaps children within the high-risk sample are more vulnerable to hostility within these relationships which negatively impacts them in multiple ways. Children of depressed mothers might be more distressed than children of non-depressed mothers following expressions of hostility and this stress might manifest in depressive symptoms. This should be taken into account when assessing areas for interventions for different family types as if a depressed mother resides within the households, processes which increase risks for children developing symptoms of depression might need to be additionally addressed.

The associations of maternal depression, interparental conflict and hostile mother-child relationships with adolescent aggressive symptoms

Secondly associations between the previously identified risk factors (mother depression, IPC and negative mother-child relationships) on the development of aggressive symptoms in adolescents from the clinical (EPAD) and community sample (WFS) were examined. Once again, associations were found for adolescents from the clinical and community sample for maternal depression, interparental conflict and hostile mother-child relationships. More similarities were found between adolescents from the clinical and community samples for the adolescent aggressive symptoms than for adolescent depressive symptoms. Primarily, there were associations in both groups between hostile mother-child relationships and later levels of adolescent aggressive symptoms. This implies that mother-child hostility can act as a risk factor for the development of aggressive behaviours in adolescents considered to be at either high or low risk for developing psychopathology (by residing with or without a mother who experiences clinical depression).

The adolescents from the clinical sample revealed associations between all the measures, except earlier levels of adolescent aggressive symptoms with interparental conflict. There was a significant relationship between mother depression and adolescent aggressive symptoms. This relationship was not found in the community study which suggests adolescents living with clinically depressed mothers might be at a greater risk of developing aggressive symptoms than adolescents living with non-clinically depressed mothers. It has previously been demonstrated that children of depressed parents are at increased risk of developing psychopathology (Natsuaki et al., 2014), the current study demonstrates that the process may be different for high risk adolescents (living with a mother who has clinical depression). It also indicates the potential

multifinality of clinical maternal depression, as it appeared to be associated with adolescent depressive and aggressive symptoms.

Clinical implications

A potential target of intervention to reduce negative child outcomes, based on the association between mother depression and adolescent emotional and behavioural problems in the current study, would be to target maternal depression. Indeed, previous studies have indicated that risks to children might be reduced with successful treatment of mother's depression and associated maladaptive family environment factors (e.g. family stress and parent-adolescent conflict). Parenting interventions aimed at reducing maternal hostility in mothers with depression may be especially beneficial for preventing or reducing symptoms of aggression in adolescents and might also be beneficial for reducing maternal depression symptoms (Barlow, Coren, & Stewart-Brown, 2002; Barlow & Coren, 2001; Barlow, Smailagic, Huband, Roloff, & Bennett, 2014; Coren, Barlow, & Stewart-Brown, 2003; Gross, Fogg, & Tucker, 1995). It should also be noted that maternal depression may moderate the effectiveness of parenting interventions, parents with lower depression score have been observed as having more beneficial responses to parenting training and interventions (Beauchaine, Webster-Stratton, & Reid, 2005). It is clear that multiple risk factors should be considered to ensure that the most appropriate intervention is used is based on specific risk factors.

The findings of the current study demonstrated the indirect and direct effects of mother-to-child hostility via interparental conflict. If interparental conflict is targeted for intervention, families may benefit from improved mother-child relationships, with reduced hostility expressed from mother-to-child. This might then decrease the risk associated with the development of adolescent depressive and aggressive symptoms. Previous intervention studies which have included programs to improve couple

relationships with parenting programs seem to demonstrate additional benefits than parenting programs alone, with greater longevity for improving interparental relationships, parenting skills and parent-child relationships (Cowan & Cowan, 2002; Dadds, Schwartz, & Sanders, 1987; Griest, Forehand, & Rogers, 1982; Webster-Stratton, 1994).

Limitations and future work

Although the current study offers unique findings for the associations between maternal depression with indices of family socialisation in adolescents, the conclusions should be considered in light of some limitations. Firstly, different measures of adolescent psychopathology were used for the clinical and community samples.

Although the different measures for child psychopathology have been previously associated (Goodman & Scott, 1999; Koskelainen et al., 2000; Lynch, Mills, Daly, & Fitzpatrick, 2004) we cannot compare them directly, we can only compare the magnitude of effects across different pathways for the adolescents from the clinical and community samples, though we know that rates of psychopathology for the adolescents living with a depressed mother are higher than general populations.

A further limitation is that we were unable to examine whether pathways were similar or different for father depression or father-child hostility. The relationship between parental depression and psychopathology in children appears to be a more robust and consistent for maternal depression compared to paternal depression (Natsuaki et al., 2014). The relationship between paternal depression and child outcomes appears to be more ambiguous (Lewis et al., 2013; Tully, Iacono, & McGue, 2008), which suggests that different pathways might explain the intergenerational transmission of depression and psychopathologies for fathers and mothers. Some previous research however, has identified links between paternal depression and

psychopathology in children (Davis, Davis, Freed, & Clark, 2011; Laurent et al., 2013; Wilson & Durbin, 2010). Kane and Garber's meta-analysis (2004) demonstrated that father depression was significantly related to father-child hostility, plus emotional disorders and behavioural problems in adolescents. Research is now beginning to highlight the importance of fathers and the father-child relationship for adolescent development. Limitations outlined in this study included the absence of exploration of the influence of fathers; however, it is important that when methodologically possible, father-child relationships are also examined within a similar framework. It is also crucial to understand how different relationships within the family function, not only with the presence of parental psychopathology, but also how family members might react to conflict. For instance, males appear to react differently to spousal conflict than females; some research suggests that females might be more likely to internalise problems associated with partner conflicts, whereas males might be more prone to react with externalising behaviours, such as hostility (Newland, Freeman, & Coyl, 2014). The focus of the current study however, was the examination of the processes in different study designs to have a conceptual understanding of the mechanisms which explain the development of depressive and aggressive symptoms in adolescents. This is why gender differences were not specifically examined within the sample.

Research into family socialisation has historically been conducted in families where children are genetically related to the parents who rear them. The current study uses data from two samples of genetically related families. Parents in the studies reared children whom they were biologically related to. Limited inferences can therefore be made from the results in determining the contribution of genetic and environmental influences on the development of depression and aggression in children as parents and children share both genes and environment. However, previous research that has used

genetically sensitive research designs (where parents are genetically unrelated to their children and allow a partial separation of genetic and environmental influences) have indicated the importance of environmental factors to the aetiology of depression and aggression in children and adolescents. Although genetic predispositions in the relationship between maternal depression and child psychopathologies have been researched, associations between maternal depression and negative child outcomes have been found in genetically unrelated families (Harold et al., 2011; Leve et al., 2007; Tully et al., 2008) suggesting that environmental factors might significantly contribute to the development of depression and aggression in children (Silberg et al., 2010).

Research in the remaining chapters of this thesis addresses some of these issues by examining family socialisation processes in both mothers and fathers in genetically informative research designs (e.g. utilising IVF; Chapter 3, and Adoption studies; Chapter 4).

Chapter 3: Examining the associations of parental depression and family socialisation with the development of depressive and aggressive symptoms in middle childhood using a genetically informative research design

Part of this chapter has been presented at the following presentations;

- Oral Presentation: "The influence of maternal depression and family socialisation on offspring psychopathology" at 2nd year Psychology PhD Conference, Sussex, June 2014
- Oral Presentation: "Parental depression and family socialisation on the development of depressive and aggressive symptoms in children and adolescents", presentation at CDRiP, Sussex, June 2015
- Oral Presentation: "The influence of maternal depression and family socialisation on on the development of depression in adolescents", presentation at International Congress of Clinical and Health Psychology with Children and Adolescents, Madrid, November 2015

Introduction

The first study of this thesis outlined associations of maternal depression, interparental conflict and mother-child hostility with the development of depression and aggression for high-risk and low-risk adolescents. The current study extends the understanding of how these mechanisms are associated with the development of psychopathology in several ways. Firstly, it will consider the impact of fathers. As outlined in the previous chapter, multiple researchers have shown that maternal depression can have detrimental effects on their child including increased risks for developing both internalising, emotional problems and external, behavioural issues (for a review see Goodman et al., 2011). Far fewer researchers have directed their attention to paternal depression on the development of psychopathology in children (Reeb et al., 2015). The current study will examine how fathers' depression and father-child hostility might affect the aetiology of depression and aggression in their children. Secondly, it will use a genetically informative research design (in-vitro fertilisation sample; IVF) to disentangle genetic and environmental influences (of both mother and father) for child developmental psychopathology (Rutter, 2007; Rutter et al., 2001). Thirdly, it will examine these processes during middle childhood so that we might see how these mechanisms operate during different developmental periods. This could provide an indication of where to target early interventions to reduce negative outcomes in children exposed to parental depression and hostile relationships within the family.

Psychopathology in children

Symptoms of psychopathology in childhood primarily consist of internalising (e.g. depression) and externalising (e.g. aggression) problems (Bayer et al., 2012). Internalising problems are often viewed as emotional difficulties in which feelings of fearfulness, anxiety, shyness and sadness and experienced (Ollendick & King, 1994;

Reynolds et al., 2014; Zahn-Waxler et al., 2000). Externalising problems are characterised by harmful, disruptive and disinhibited behaviours that can include aggression, rule-breaking, conduct disorders and oppositional behaviours (Ford et al., 2003; Kovacs & Devlin, 1998).

Middle childhood is viewed as a unique but under-researched developmental period for children (Schonert-Reichl, 2011), this is especially true when contrasted with research on early childhood and adolescence. There appears to be a large amount of research and literature dedicated to early childhood and adolescence compared with middle childhood which presents a window of opportunity to better examine development within this age group. During this developmental period, children experience physical, cognitive and social changes; between the ages of 5 and 7 years, children enter formal education, learn to read and are given more independence to regulate their own behaviour (Huston & Ripke, 2006). Middle childhood is part of a continuum which is dependent on influences from infancy and early childhood and will in itself, influence behaviours and outcomes of adolescents and adults. Research on psychopathology within in this developmental period would thus complement progress on our understanding of general health during early childhood and psychopathology in adolescence (Biehl, Park, Brindis, Pantell, & Irwin, 2002).

Estimates of prevalence rates for mental health problems for children during middle childhood are limited (Biehl et al., 2002) though some data exists which estimates that around 6% of children aged between 6 to 11 years' experience emotional and behavioural problems including depressive and aggressive symptoms (Moore, Hatcher, Vandivere, & Brown, 2000). Mental health problems experienced in this age group can include symptoms of anxiety, aggression and disruptive disorders, Attention Deficit Hyperactivity Disorder and depressive symptoms, with the presence of

depressive symptoms being recognised at increasingly earlier ages (Son & Kirchner, 2000).

The importance in studying depressive and aggressive symptoms in middle childhood can be attributed to findings that it is often within this age group that one might first observe depressive symptoms (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015) and manifestations of aggression (Huesmann, Eron, Lefkowitz, & Walder, 1984). Before this age it can be difficult to disentangle internalising, emotional problems (such as depression) and externalising, behavioural problems (such as aggression) (Leve et al., 2009). Children within middle childhood have greater cognitive capabilities and self-awareness than in earlier developmental periods but do not face the additional stressors associated with adolescence. Given this it might be the most effective period to consider the influence of family factors and how psychopathology develops, and it might be the most effective stage of which to intervene to maximise the potential for positive growth (Huston & Ripke, 2006).

Depression and aggression in middle childhood

Depression refers to an overall lowering of normative functioning, often relating to feelings of sadness and dejection. Researchers and clinicians recognise that although depressive disorders are rare in preadolescence samples with prevalence rates of 1%-2% (Angold & Costello, 2001), many depressive symptoms, including sadness, loss of interest in activities and decreased energy are observed in this age group (Wilkinson, Trzaskowski, Haworth, & Eley, 2013). Parent reports indicate that 10 to 20% of children aged below 11 years are rated as feeling sad, miserable or depressed (Achenbach, 1991; Rutter, Tizard, & Whitmore, 1970).

Children identified as having early onset depression are at a higher risk of later depressive episodes in adolescence and adulthood (Emslie et al., 1997; Harrington et al.,

1990; Kovacs & Devlin, 1998; Lewinsohn et al., 1999; Rao et al., 1999) with evidence that these early signs and symptoms of depression show strong continuities with clinical depression in adulthood (Fombonne et al., 2001; Pine et al., 1999).

Aggression is typically used to describe behaviours which are verbally or physically threatening (Nagin & Tremblay, 1999). In middle childhood however, the intent to hurt individuals might not be a component of the behaviour (Wakschlag et al., 2010). Aggressive behaviours in middle childhood might be a result of fear or anger in specific situations, or a result of underdeveloped self-regulation skills or frustration at unmet needs (Cole et al., 1996; Wakschlag et al., 2010). Aggressive behaviours are very common in early childhood, in fact it is rarer for children to not exhibit physical aggression before the age of 3 years (Buchmann et al., 2014). However, persistent aggression has been associated with several negative outcomes for children in middle childhood as they transition into adolescents and later adulthood including unemployment, criminal behaviours and negative consequences on socioeconomic status (Buchmann et al., 2014).

Parental depression

Maternal depression has long been associated with negative developmental outcomes in children and adolescents (Claessens, Engel, & Curran, 2015; Cummings & Davies, 1994; Johnson & Flake, 2007). Prior research has largely focused on maternal depression, with few studies considering the potential impact of paternal depression in relation to child mental health (Phares, Fields, Kamboukos, & Lopez, 2005; Phares, Lopez, Fields, Kamboukos, & Duhig, 2005). The focus on maternal depression within this area might be due to the increased prevalence of depression in women (Albert, 2015) with estimates that 1 in 4 women will require treatment for depression, compared to 1 in 10 men (National Collaborating Centre for Mental Health, 2010). Other reasons

might include previously highlighted expectancies for fathers to not be as involved in child rearing as mothers (Russell & Russell, 1987) and the difficulties often faced by researchers in recruiting fathers for research on child development (Cassano et al., 2006; Mitchell et al., 2007). However, research has recently begun to consider the potential impact of father depression on their children (Bradley & Slade, 2011).

Researchers who have examined the influence of paternal depression have demonstrated that depression in fathers is associated with a greater level of father-to-child hostility (Harold et al., 2011a), negative effects on children's general psychological functioning (LeFrançois, 2012) as well as increased risk for child depressive and anxiety symptoms when maternal depressive symptoms have been controlled for (Reeb et al., 2015). Paternal depression also appears to impact negatively on depressive symptoms in mothers (Laurent et al., 2013).

Research debating the relative impacts of maternal and paternal depression on children appears to be inconsistent; some researchers state that compared with depression in fathers, depression in mothers is associated with greater risk for negative outcomes in children (Connell & Goodman, 2002) and more severe episodes of child depression (Klein et al., 2005). Other studies show more comparatively similar impacts of maternal and paternal depression on negative child outcomes (Jacob & Johnson, 1997; Marchand & Hock, 1998). There does appear to be a gap in the literature for studies which assess both maternal and paternal depression and child outcomes (Reeb et al., 2015). It is therefore important that research examines the processes through which both maternal and paternal depression may influence risk for the development of child psychopathology, specifically depressive symptoms and aggressive behaviours.

Parent-child relationship

The parent-child relationship has historically been viewed as having substantial influence on the development of children, as such, a child's relationship with their parent is seen within developmental psychology as a key determinant of emotional and behavioural development (Kagan & Snidman, 1999; Serot & Teevan, 1961). Parental factors that influence a child's development have primarily focused on mothers (Phares & Compas, 1992; Phares, Fields, et al., 2005). There are several reasons that might offer explanations as to why mothers have been the primary focus of these investigations; mothers have typically been the primary caregivers of infants, as such it has been suggested that she has more interactions with the child, therefore these influences have been viewed as more important on child developmental outcomes than father-child interactions (Cabrera et al., 2000).

Initial explorations as to a fathers influence on their child appeared to be primarily concerned with their presence-vs-absence, as opposed to the individual characteristics that might be associated with child's development (Phares & Compas, 1992). It is important to acknowledge that the father-child relationship, although considered far less frequently than the mother-child relationship, likely impacts on the risk for the development of negative outcomes in children. Researchers have recently begun to examine processes within the father-child relationship that might negatively (Jaffee et al., 2003) or positively impact on child development (Cabrera et al., 2007; McHale, 2007). This is especially important within the context of parental depression (Jarvis & Creasey, 1991).

Researchers have previously identified the importance of exploring the differences between mother-child and father-child relationships involved in the development of psychopathology. An enriched understanding of the uniqueness of these family

subsystems helps in identifying modifiable areas of the child environment that might reduce the risk for negative outcomes in children (Lamb, 2004). Early research exploring differences between mother-child and father-child relationships were often concerned with comparing how, when and why parents would spend time with their child. Russell & Russell (1987) described how fathers were more likely to interact with their child through play, with mothers primarily interacting with the child through direct caregiving. More recent research attempts to examine the influence of wider family subsystems such as interparental conflict alongside parent-child relationships on the development of psychopathology in children.

Interparental conflict

The influence of the marital relationship on parenting and parent-child relationships has become well established (Cox, Paley, & Harter, 2001), however it is only more recently that researchers have begun to examine the association between father depression and the marital relationship and subsequent child outcomes (Stevenson & Fabricius, 2014). Although some previous research has suggested that there were no differences between the negative impact of interparental conflict on mothers and fathers (Erel & Burman, 1995) more recent studies have indicated that fathers might be more negatively impacted by hostility in the spousal relationship (Cummings et al., 2004; Cummings, Merrilees, & George, 2010; Krishnakumar & Buehler, 2000).

The spill-over hypothesis predicts that animosity experienced in the interparental relationship will effectively 'spill-over' into other relationships within the home, including that of the parent-child (Erel & Burman, 1995; Krishnakumar & Buehler, 2000). Evidence suggests that interparental relationships may affect fathers more than mothers (Cummings et al., 2004; Cummings et al., 2010; Krishnakumar & Buehler,

2000; Shelton & Harold, 2008). If this is the case, the father-child relationship might be at a greater risk of being negatively affected as a result of interparental conflict than the mother-child relationship. Cummings and colleagues (2004) 'fathering vulnerability hypothesis' outlines how the spill-over might be more likely to impair father-child relationships due to mothers having a clearer idea of their parental role, and finding it easier to compartmentalise their role as a spouse and as a parent. Evidence suggests that a decrease in interparental relationship security (associated with interparental conflict) predicted harsher parenting for fathers but not mothers (Amato, 2000).

In-vitro fertilisation families

The current study will utilise a data set of families who have conceived a child through in-vitro fertilisation (IVF). Approximately 1-4% of all children are conceived through IVF (Andersen, Gianaroli, Felberbaum, De Mouzon, & Nygren, 2006). Examining children who have been conceived through IVF, where children may or may not be genetically related to their parents, offers the opportunity to assess the importance of genetic relatedness for children who have not had additional risk factors typically associated with step-families and adoption (Golombok, 2015).

Family stress

Parents who conceive a child through IVF may have often experienced higher levels of stress compared to those who conceive naturally. This stress might be associated with dealing with infertility problems prior to using artificial reproductive technologies. Infertility has been shown to have negative impacts on the individuals affected by it and on the relationship of the couples experiencing it (Connolly & Edelmann, 1992; Luk & Loke, 2015). Stresses that might be experienced within IVF families could be a result of increased expectations that parents have for themselves (Gameiro, Nazaré, Fonseca, Moura-Ramos, & Canavarro, 2011) and for their children

(Pottinger & Palmer, 2013) or perhaps related to the emotions that surrounded the previous infertility challenges that have not been successfully resolved (Onat & Beji, 2012). However, Hammarberg and colleagues (2008) found that parents general stress levels related to parenting a first-born were comparable for IVF and families who have naturally conceived.

As the current study involves measuring negative indices of the relationship between parents, it is worth specifically evaluating how couple relationships in IVF families compare to those who have conceived a child naturally. Findings appear to be somewhat inconclusive, with some researchers concluding that relationships amongst IVF couples are more positive (Golombok, Cook, Bish, & Murray, 1995), whilst others have concluded that family relationships, amongst both interparental and parent-child relationships are lower (Gibson, Ungerer, Tennant, & Saunders, 2000). These inconsistencies might be related to different family structures within IVF families (e.g. whether mother/father/both are genetically related to the child). Some research exploring IVF family structure has found that where there is no genetic link between mother and child (egg donation), psychological well-being might be higher for both mothers and fathers (Golombok, Murray, Brinsden, & Abdalla, 1999). Less favourable associations have been observed in families where fathers are genetically unrelated to their child (sperm donation) such as higher levels of family disruptions (Harold et al., 2008).

Parenting in IVF families

Parents who conceive children through IVF have often struggled with infertility and treatment for several years prior to a successful insemination and birth and although, as previously mentioned this might result in higher levels of stress for couples and families (Luk & Loke, 2015) it also appears that individuals who overcome the

challenges associated with the experience become very committed parents when a child arrives (Golombok, 2015). This is perhaps due to their motivation to have a child and the planning that is irrevocably involved in having a child though IVF (Carson, Redshaw, & Sacker, 2013). It seems that contrary to previous beliefs that family stress associated with the situations parents might have experienced prior to the birth of their child might have a negative impact on their parenting, that these parents appear to be very committed and involved with their children and empirical evidence suggests that there are more positive outcomes for these families (Hayatbakhsh et al., 2011). Differences in IVF parents compared to parents who have naturally conceived might be attributed to prenatal attachment of IVF fathers; fathers of IVF children who reported stronger attachment prior to their child's birth demonstrated lower levels of anxiety and irritability postnatally (Hjelmstedt & Collins, 2008). Studies which have compared parental adjustment in IVF and families who have naturally conceived however, appear to find no differences in parental adjustment (Colpin & Bossaert, 2008; Wagenaar, van Weissenbruch, Knol, Cohen-Kettenis, Delemarre-van de Waal, & Huisman, 2009).

Children conceived through IVF

The risk of preterm delivery, low birth weight and obstetric complications seem to be increased significantly for children born through IVF (Halliday, 2007), however this might be related to the higher instances of multiple births (e.g. twins, triplets) through IVF than natural conceptions (Lédée-Bataille et al., 2001). Other than these problems associated with potential perinatal and birth complications, children conceived through IVF appear indistinguishable from those conceived in traditional families in terms of their development and psychological well-being (Golombok, 2015) and any differences that are observed appear to point favourably towards IVF children (Hahn & DiPietro, 2001; Repokari et al., 2007).

Lack of difference in outcomes between children born through IVF and children born through natural conceptions could be considered to be an unusual finding based on the indication that parents of IVF children might even be more committed and involved with their children (Hayatbakhsh et al., 2011). One might expect therefore, that these children have the capacity to show even higher levels of psychological adjustment than those from more traditional families (Golombok, 2015). Some researchers have stated that there appears to be a minimum amount of 'good' parenting that a child needs to be able to develop in a positive manner, but after that point, the quality of parenting might have negligible differences to a child's well-being (Maccoby, 2007). This might provide an explanation as to why researchers more commonly focus on negative aspects of parenting in studies looking at developmental outcomes in children. Alternatively, much research has expected to find negative outcomes in children, thus they applied assessments to rate on the negative side of problems so the tools might not have the ability to measure the other end of the spectrum for positive outcomes (Golombok, 2015).

Using IVF families to separate genetic and environmental influences

It is well recognised that genetic and environmental factors both contribute to the development of child psychopathology (Plomin et al., 1977; Plomin, DeFries, Knopik, & Neiderhiser, 2016). The interplay between genetic and environmental influences on negative child outcomes has more recently been explored (Harold et al., 2008; Natsuaki et al., 2014) with the recognition that previously held beliefs about the divide between genes and environment was overly simplistic and failed to capture the dynamic interactions that might consequently help to describe the aetiology of psychopathology in children (Thapar & Harold, 2014).

Previous research examining associations between parental depression, indices of family socialisation and child psychopathology has primarily been conducted with parents and children who are biologically related (Combs-Ronto, Olson, Lunkenheimer, & Sameroff, 2009; Psychogiou et al., 2007; Sellers et al., 2013). It is therefore difficult to disentangle whether associations that have been observed are a result of genetic or environmental contributions, or both (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). This is a result of shared or common genes between parents and children who are biologically related (Rutter et al., 2006) and has been defined as gene-environment correlation (*r*GE; Plomin et al., 1977).

Several rGE processes have been outlined; evocative, active and passive (Ge et al., 1996; Jaffee & Price, 2007, 2012; Rutter et al., 1997). Passive rGE occurs when parents and children's genes (which are shared when children are biologically related to the rearing parents) confound the association between family and child level variables genes (Jaffee & Price, 2007, 2012). For example, shared genetic attributes between the parent and child can influence the parenting that is provided (Ge, et al., 1996; Rutter, Moffitt, & Caspi, 2006). The current study focuses specifically on passive rGE, employing a genetically informative design to remove this as a possible confound of the association between parent and child psychopathology. This research design is based on families where children have been conceived through assisted reproductive technologies. Children conceived via these methods, specifically, IVF may be genetically related to both parents (homologous IVF), the mother only (sperm donation), the father only (egg donation), or to neither parent (embryo donation). By comparing the similarity of parents and their children across each of these groups, we can disentangle the effects of genetic and environmental influences on child psychopathology.

Studies that have used IVF families to assess parental depression and family socialisation on child outcomes have made a unique contribution to our knowledge of how these mechanisms might impact child development. In recent years, studies have identified that parental depression operates as an environmental risk factor for the development of emotional and behavioural problems in children (Natsuaki et al., 2014) with both maternal and paternal depression being directly associated with child depressive symptoms (ages 4 to 10 years) in genetically unrelated families (Harold et al., 2011). Although family socialisation processes have been highlighted as one mechanism through which parental depression might influence child outcomes, parental depression and family socialisation processes have not been *simultaneously* examined in the context of a genetically informative design to disentangle genetic and environmental contributions to the development of child depressive symptoms and child aggression via these processes.

Previous studies have begun to address questions in relation to family processes and child outcomes. Lewis and colleagues (2013) separately examined the impact of maternal and paternal depression on parent-child hostility and child depression. They found direct associations between maternal depression and child depression for genetically unrelated mother-child dyads. For genetically related dyads associations were observed through mother-to child hostility and warmth. However this study did not take into account the contribution of interparental conflict or consider the effect of parental depression on child aggressive behaviours, both of which have been identified as important risk factors for child psychopathology. The current study will extend these findings, firstly by examining the impact of interparental conflict on parent-child relationships (i.e. directly testing the spill-over hypothesis), secondly by examining how

these mechanisms operate within the context of parental depression for both child depressive symptoms and aggressive behaviours.

Associations have also been observed between interparental conflict and child externalising behaviour through mother-to-child and father-to-child hostility, regardless of genetic relatedness, although these associations were significantly stronger for fathers compared to mothers (Harold et al., 2013). Although this study did consider the impact of interparental conflict on parent-child hostility and child externalising problems, it did not examine these processes within the context of parental depression, nor did it examine the effects of these hostile family relationships on child depressive symptoms.

Overall these studies demonstrate that paternal depression, as well as maternal depression, likely have negative impacts on children (Claessens et al., 2015; Reeb et al., 2015). Secondly; that mother depression and father depression can each influence negative indices of the couple relationship, such as interparental conflict (Hanington et al., 2012). Thirdly, interparental conflict has been previously associated with increases in negative parent-child relations, including parent-child hostility (Harold et al., 2013). Fourthly, that mother-to-child and father-to-child hostility have previously been associated with the development of psychopathology in children (Lewis et al., 2011). Finally it has demonstrated that these mechanisms might operate to promote negative developmental outcomes in children who may be genetically related, or genetically unrelated to their parents (Harold et al., 2013). What remains unexplored is how these processes (maternal depression, paternal depression, interparental conflict, mother-child hostility, father-child hostility) operate on the development of depressive symptoms and aggressive behaviours during middle childhood in the context of a genetically sensitive research design. The current study aims to look at the processes on the development of depressive and aggressive symptoms, thus remediating the lack of research on fathers in general. More specifically the effect of paternal depression and the role of fathers in family socialisation processes on child outcomes will be examined, as well as providing a genetically informative framework to examine genetic and environmental contributions to such processes. To address this aim, the present study employed a cross-sectional, genetically informative research design to investigate the impact of parental depression, interparental conflict and parent-to-child hostility on symptoms of depression and aggression in middle childhood (children aged 4 to 11 years) in genetically related and genetically unrelated mother-child and father-child dyads.

Method

Participants

The Cardiff IVF study includes participants from families who have conceived a child through one of the assisted reproductive methods and were recruited though several different fertility clinics who had agreed to participate. Twenty-two fertility clinics were initially contacted with 19 agreeing to participate. Of the participating fertility clinics, 18 were based in the UK and 1 in the US. Families who had conceived a child through IVF between 1994 and 2002 (child age of 4 to 10 years) using any of the conception subgroups were considered eligible to participate within this study. The fertility clinic contacted the families on behalf of the research team via mail. The gamete donors and surrogates needed to be unrelated to either of the rearing parent to aid comparisons. Questionnaires were mailed to participating families through the fertility clinics. The questionnaire included measures of parental health, parental mental health, couple relationship quality, life events a range of child outcomes measures including psychological well-being as well as sociodemographic information.

The Cardiff IVF (C-IVF) sample includes parents who had information on the specific study variables being explored (mother genetically related, *N*=433; mother

genetically unrelated, N=129, father genetically related, N=405; father genetically unrelated, N=159). Forty-nine percent of the children were girls. Children were aged between 4-11 years (M=6.8 years, SD=1.24). The number of families in each conception group for genetically related fathers was 444 homologous IVF, 175 IVF with egg donation, 23 IVF with gestational surrogacy. The sample has been compared with UK norms, with minimal differences in mean levels of child behaviour symptoms (ONS, 2002).

Only children living with two parents were included in the current study (92% of sample) due to the focus on how the interparental relationship is associated with parental depression and parent-child relationship. The homologous IVF group contributed to around 50% of this sample, sperm donation 24%, egg donation 20%, embryo donation 3% and gestational surrogacy 3%. The current study requires comparing genetically unrelated parent-child dyads; 77% of the families had genetically related mother-child dyads and 73% had genetically related father-child dyads. Only families with complete data for the measures of interest were included. Four groups were used within the analyses;

- (i) Mothers who were genetically related to their child with genetically related and genetically unrelated fathers (N = 427)
- (ii) Mothers who were genetically unrelated to their child with genetically related and genetically unrelated fathers (N = 127)
- (iii) Fathers who were genetically related to their child with genetically related and genetically unrelated mothers (N = 400)
- (iv) Fathers who were genetically unrelated to their child with genetically related and genetically unrelated mothers (N = 154)

Measures

Parent depression. Parental depression was assessed using the depression subscale of the Hospital Anxiety Scale (HADS-D; Zigmond & Snaith, 1983). The seven items of the depression subscale request individuals to read statements including 'I feel I have lost interest in things' and 'I feel life is not worth living' and rate whether they relate to that statement on a scale of 0-3 (0= No, not at all; 3= Yes, definitely). Two of the items were reverse coded ('I have a good appetite' and 'I still enjoy the things I used to') to maintain a consistency within the scale, with a higher score indicating higher levels of depression. Internal consistency estimates were good for both mothers (α = 0.75) and fathers (α = 0.76)

Interparental conflict. Mothers and fathers reported on hostility they experienced from their partner using the spousal hostility subscale of the Iowa Youth and Family Project (IYFP) Rating Scales (Melby et al., 1998; item scale range = 1-7). Individuals were asked to report on levels of hostility they experienced from their partner during the past three months. Statements required parents to think how often their partners exhibited a specific behaviour, including how often does your spouse 'Get angry at you' and 'Criticise you or your ideas'. Internal consistency was very good for mothers ($\alpha = 0.90$) and fathers ($\alpha = 0.89$).

Parent-to-child hostility. The hostility subscale of IYFP (Melby et al., 1998) was used to assess expressed hostility towards children. Mothers and fathers reported on their own behaviours towards their child. As with the spousal IYFP, parents were asked to think about their behaviour toward their child in the past three months. Items included "*How often do you get angry at him/her*?" with parents responses ranging from 1-7 (1=Always; 7= Never). Internal consistency was good for both mothers ($\alpha = 0.80$) and fathers ($\alpha = 0.82$).

Child depression. Child outcomes were assessed through opposite parent report, e.g. mothers' reported for child depression and aggression for father models, and fathers' reported on child depression and aggression for mother models. Child depression was assessed using parents' reports of the Short Mood and Feelings Questionnaire (Angold & Stephen, 1995). Parents were asked to consider their child's behaviour over the past 3 months and report on a 3-point scale, how true 13 item statements were (0 = not true; 3 = very true). Items included the child 'Complains of loneliness', 'Cries a lot' and 'Fears he/she might think or do something bad'. Internal consistency was good for both mothers' ($\alpha = 0.78$) and fathers' ($\alpha = 0.79$) reports of child depression. The Mood and Feelings Questionnaire has been shown to highly correlate with more detailed evaluations of children's depression, including the Children's Depression Inventory (r = .67, Kovacs, 1983) demonstrating that it is a reliable and a valid measure.

Child aggression. Parents reported on their children's aggression using the 5 items of the conduct subscale of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997); item scale range = 0-2). Statements about that child included that the child 'Often has temper tantrums' and 'Often fights with other children or bullies them'. Internal consistency was acceptable for mothers ($\alpha = 0.52$) and fathers ($\alpha = 0.54$). Scores from the SDQ have been shown to distinguish between community and clinic samples equally well as more in-depth assessments such as the Child Behaviour Checklist (CBCL; Achenbach, 1991).

Results

Correlational analysis

Intercorrelations, means and standard deviations are presented for genetically related and genetically unrelated mother-child (Table 1) and father-child (Table 2)

dyads. For genetically related mother-child dyads (N=427), significant associations were found between symptoms of maternal depression and interparental conflict (r=.24, p<.01) mother-to-child hostility (r=.30, p<.01), father rated child depressive symptoms (r=.18, p<.01) and father rated child aggressive symptoms (r=.29, p<.01). Interparental conflict was significantly associated with mother-to-child hostility (r=.32, p<.01), father rated child depression (r=.13, p<.01) but not father rated child aggression (r=.09, p=.077). Significant associations were observed between mother-to-child hostility with father rated child depressive symptoms (r=.23, p<.01) and father rated child aggressive behaviours (r=.31, p<.01). Father rated levels of child depression were significantly associated with father rated child aggressive behaviours (r=.35, p<.01).

For genetically unrelated mother-child dyads (N=127), significant associations were found between symptoms of maternal depression and interparental conflict (r=.31, p<.01), mother-to-child hostility (r=.33, p<.01), father rated child depressive symptoms (r=.19, p<.05) and father rated child aggressive symptoms (r=.33, p<.01). Interparental conflict was significantly associated with mother-to-child hostility (r=.35, p<.01), father rated child aggression (r=.22, p<.01) but not father rated child depression (r=.14, p=.13). Significant associations were observed between mother-to-child hostility with father rated child aggressive symptoms (r=.23, p<.01) and father rated child aggressive behaviours (r=.31, p<.01). Father rated levels of child depression were significantly associated with father rated child aggressive behaviours (r=.27, p<.01).

For genetically related father-child dyads (N = 400), significant associations were found between symptoms of paternal depression and father rated IPC (r = .27, p < .01) father-to-child hostility (r = .25, p < .01), mother rated child depressive symptoms

(r=.18, p<.01) and mother rated child aggressive symptoms (r=.14, p<.01). Father rated IPC was significantly associated with father-to-child hostility (r=.31, p<.01), mother rated child depression (r=.22, p<.01) and mother rated child aggression (r=.14, p=.077). Significant associations were observed between father-to-child hostility with mother rated child depressive symptoms (r=.19, p<.01) and mother rated child aggressive behaviours (r=.33, p<.01). Mother rated levels of child depression were significantly associated with mother rated child aggressive behaviours (r=.30, p<.01).

For genetically unrelated father-child dyads (N=154), significant associations were found between symptoms of paternal depression and father rated IPC (r=.23, p < .01) father-to-child hostility (r=.33, p < .01), and mother rated child depression symptoms (r=.19, p < .05) but there was not a significant association between father depression and mother rated child aggression symptoms (r=.14, p=.08). Father rated IPC was significantly associated with father-to-child hostility (r=.45, p < .01) and mother rated child aggression symptoms (r=.17, p < .05) with weak evidence of an association between mother rated child depression symptoms, which just approached significance (r=.16, p=.50). Significant associations were observed between father-to-child hostility with mother rated child aggression symptoms (r=.35, p < .01) but not with mother rated child depression symptoms (r=.16, p=.07). Mother rated levels of child depression were significantly associated with mother rated child aggressive behaviours (r=.50, p < .01).

Table 1. Intercorrelations, means and standard deviations among constructs for genetically related (lower diagonal, N = 427) and genetically unrelated (upper diagonal, N = 127) mothers and children

Variable	(1)	(2)	(3)	(4)	(5)	Mean	S.D.
(1) Father depression	-	.23**	.33**	.19*	.14	3.82	2.74
(2) Inter-parental Conflict (father rated)	.27**	-	.45**	.16	.17*	13.68	4.59
(3) Father-to-child hostility (father rated)	.25**	.31**	-	.15	.35**	11.47	3.80
(4) Child Depression (mother rated)	.18**	.22**	.19**	-	.50**	2.93	3.41
(5) Child Aggression (mother rated)	.15**	.14**	.33**	.30**	-	1.39	1.48
Mean	3.75	13.17	11.77	2.66	1.47		
S.D	3.06	4.63	3.73	2.79	1.42		

^{*}p <.05, ** p<.01

Table 2. Intercorrelations, means and standard deviations among constructs for genetically related (lower diagonal, N = 400) and genetically unrelated (upper diagonal, N = 154) fathers and children

Variable	(1)	(2)	(3)	(4)	(5)	Mean	S.D
(1) Mother depression	-	.31**	.33**	.19*	.33**	4.37	2.80
(2) Inter-parental Conflict (mother rated)	.24**	-	.35**	.14	.22*	11.82	4.47
(3) Mother-to-child hostility (mother rated)	.30**	.32**	-	.05	.38**	12.10	3.68
(4) Child Depression (father rated)	.18**	.13**	.23**	-	.27**	2.24	2.55
(5) Child Aggression (father rated)	.29**	.09	.31**	.35**	_	1.88	1.58
Mean	4.23	11.81	11.86	2.70	1.41		
S.D	3.17	4.48	3.55	2.94	1.37		

^{*}p <.05, ** p<.01

Path Analysis

Associations between parental depression, family socialisation and negative child outcomes were further examined using path analysis. Path models were conducted separately for genetically related and genetically unrelated parent-child dyads. The models were conducted using Mplus 7 (Muthen & Muthen, 2007). Genetically related and genetically unrelated models were then compared using a stacked modelling procedure in which paths were fixed to be equal across groups and subsequently compared using the Satorra-Bentler Scaled Chi-Square Difference Test (Satorra, 2000).

Model fit statistics

Model fit was examined using several fit statistics, as outlined in the previous chapter (Chapter 2). Briefly, a good model fit was indicated by a non-significant chi-square test (χ^2) test. As the chi-square test is sensitive to sample size, additional indicators were used. These included a CFI and TLI of greater than 0.95 and an RMSEA of less than 0.5.

Maternal depression, interparental conflict, mother-child hostility and child depression

Figure 1 illustrates results for genetically related and genetically unrelated mother-child dyads for child depressive symptoms. For genetically related mother-child pairs, there were significant pathways between maternal depression symptoms with interparental conflict ($\beta=.24, p<.01$), mother-to-child hostility ($\beta=.24, p<.01$) and child depression ($\beta=.10, p<.05$). Interparental conflict predicted mother-to-child hostility ($\beta=.26, p<.01$) but there was no significant pathway between interparental conflict and father rated child depressive symptoms ($\beta=.04, p=.43$). There was a significant pathway between mother-to-child hostility and child depression symptoms ($\beta=.19, p<.01$).

There were some similar associations observed for the genetically unrelated mother and child dyads. There were significant pathways between maternal depression symptoms with interparental conflict ($\beta = .28$, p < .01) and mother-to-child hostility ($\beta = .25$, p < .01). Interparental conflict predicted mother-to-child hostility ($\beta = .27$, p < .01) and there was no significant pathway between interparental conflict and father rated child depressive symptoms ($\beta = .10$, p = .30). However, in contrast to the genetically related mother-child pairs, in the genetically unrelated mother-child pairs, there was no significant pathway from genetically unrelated maternal depressive symptoms to child depressive symptoms ($\beta = .04$, p = .70) or from genetically unrelated mother-child hostility to child depression symptoms ($\beta = .18$, p = .054).

The genetically related and genetically unrelated mother-child groups were compared using Chi-square difference testing using the Satorra-Bentler Scaled Chi-Square (Satorra, 2000). The pathway from mother hostility towards her child to child depression differed between these groups ($\Delta \chi^2 = 6.944$, $\Delta df = 1$, p < .01). No other significant differences were observed between pathways in the models.

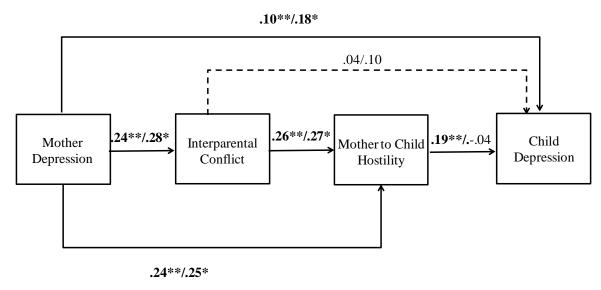


Figure 1. Genetically related/genetically unrelated mother-child dyads for child depressive symptoms *p < .05, **p < .01

Paternal depression, interparental conflict, father-child hostility and child depression

Figure 2 illustrates results for genetically related and genetically unrelated father-child dyads for child depressive symptoms. There were significant pathways between paternal depression symptoms with interparental conflict (β = .29, p <.01), father-to-child hostility (β = .18, p <.01) and child depression (β = .11, p <.05). Interparental conflict predicted father-to-child hostility (β = .23, p <.01) and mother rated child depressive symptoms (β = .15, p <.01). Father-to-child hostility went on to predict child depressive symptoms (β = .12, p <.05).

There were some similar associations observed for the genetically unrelated father and child dyads. There were significant pathways between paternal depression symptoms with interparental conflict (β = .24, p <.01) and father-to-child hostility (β = .23, p <.01). Interparental conflict predicted father-to-child hostility (β = .37, p <.01). However, some differences were observed between the genetically related and genetically unrelated father-child dyads. There was not a significant pathway between paternal depressive symptoms and mother rated child depression symptoms (β = .12, p = .14) or between interparental conflict and child depressive symptoms (β = .10, p = .32) and no pathway between father-to-child hostility and child depressive symptoms (β = .07, p = .44). The genetically related and genetically unrelated father-child groups were compared using Chi-square difference testing using the Satorra-Bentler Scaled Chi-Square (Satorra, 2000). No significant differences were observed between any pathways in the model.

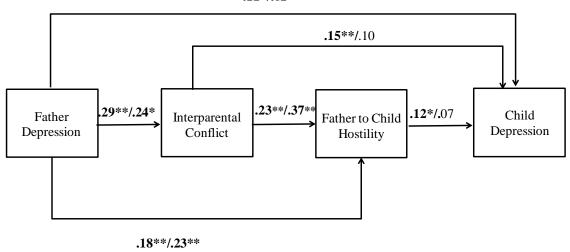


Figure 2. Genetically related /genetically unrelated father-child dyads for child depressive symptoms *p < .05, ** p < .01

Maternal depression, interparental conflict, mother-child hostility and child aggression

Figure 3 illustrates results for genetically related and genetically unrelated mother-child dyads for child aggressive symptoms. There were significant pathways between maternal depression symptoms with interparental conflict (β = .24, p < .01), mother-to-child hostility (β = .24, p < .01) and father rated child aggressive behaviours (β = .21, p < .01). Interparental conflict predicted mother-to-child hostility (β = .25, p < .01) but there was no significant pathway between interparental conflict and child aggressive symptoms (β = -.05, p = .28). There was a significant pathway between mother-to-child hostility and child aggressive symptoms (β = .26, p < .01).

These findings were replicated for the genetically unrelated mother and child dyads. There were significant pathways between maternal depression symptoms with interparental conflict (β =.30, p <.01), mother-to-child hostility (β = .26, p <.01) and father rated child aggressive behaviours (β = .19, p <.05). Interparental conflict predicted mother-to-child hostility (β = .25, p <.01) but there was no significant

pathway between interparental conflict and child aggressive symptoms (β = .05, p = .56). There was a significant pathway between mother-to-child hostility and child aggressive symptoms (β = .30, p <.01).

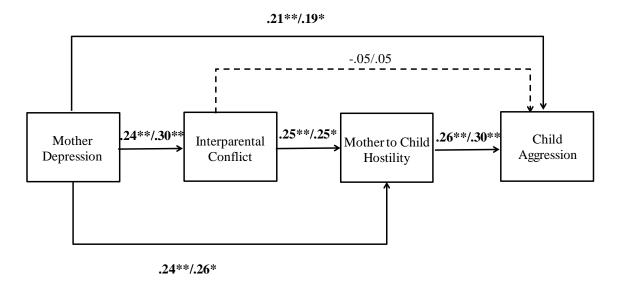


Figure 3. Genetically related/genetically unrelated mother-child dyads for child aggression *p < .05, ** p < .01

The genetically related and genetically unrelated mother-child groups were compared using Chi-square difference testing using the Satorra-Bentler Scaled Chi-Square (Satorra, 2000). No significant differences were observed between any pathways in the models.

Paternal depression, interparental conflict, father-child hostility and child aggression

Figure 4 illustrates results for genetically related and genetically unrelated father-child dyads for child aggressive symptoms. There were significant pathways between paternal depression symptoms with interparental conflict (β = .29, p <.01), father-to-child hostility (β = .18, p <.01) but not for child aggressive symptoms (β = .08, p = .12). Interparental conflict predicted father-to-child hostility (β = .24, p <.01) but not

father rated child aggression symptoms ($\beta = .03$, p = .55). Father-to-child hostility went on to predict child aggressive symptoms ($\beta = .30$, p < .01).

These findings were replicated for the genetically unrelated father and child dyads. There were significant pathways between paternal depression symptoms with interparental conflict (β = .24, p <.01), father-to-child hostility (β = .23, p <.01) but not for child aggressive symptoms (β = .02, p = .78). Interparental conflict predicted father-to-child hostility (β = .37, p <.01) but not father rated child aggression symptoms (β = .002, p = .98). Father-to-child hostility went on to predict child aggressive symptoms (β = .35, p <.01).

The genetically related and genetically unrelated father-child groups were compared using Chi-square difference testing using the Satorra-Bentler Scaled Chi-Square (Satorra, 2000). No significant differences were observed between any pathways in the models.

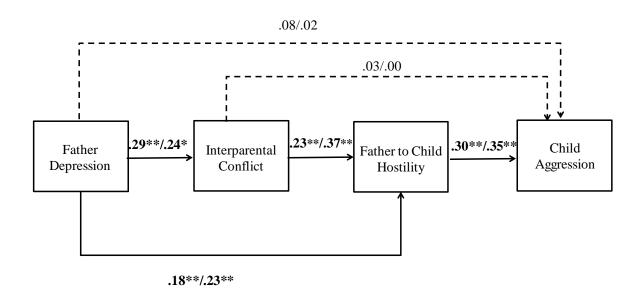


Figure 4. Genetically related/genetically unrelated father-child dyads for child aggression p < .05, **p < .01

Discussion

The current study used a genetically informative research design to examine the impact of parental depression, interparental conflict and hostile parent-child relationships on the presence of depressive and aggressive symptoms in children. The sample of children used for this study were conceived via different types of IVF. As a consequence, this 'adoption-at-conception' study included children who varied in regard to their genetic relatedness to their parents. IVF studies are particularly good at disentangling two sources of environmental influences: prenatal and postnatal environments which is important when we are interested in parental depression because maternal distress serves as a prenatal adversity to developmental outcomes in children through alterations in epigenetic and environmental pathways (Monk, Spicer, & Champagne, 2012).

The sample was genetically informative as children were either genetically related or genetically unrelated to their parents. This extends previous findings by examining how these processes (maternal depression, paternal depression, interparental conflict, mother-child hostility, father-child hostility) operate on the development of depressive symptoms and aggressive behaviours during middle childhood, thus remediating the lack of research on paternal depression and the role of fathers in family socialisation processes on child outcomes. Furthermore, by employing a genetically informative research design we were able to examine the unique contribution of genetic and environmental influences on the development of child depression and aggression symptoms.

Associations between maternal depression, interparental conflict and hostile mother-child relationships with child depressive and aggressive symptoms

The observations of associations for maternal depression and family socialisation with child psychopathology symptoms were similar for genetically related and genetically unrelated mother-child dyads. The presence of a direct association between maternal depression with child depressive and aggressive symptoms for genetically related and genetically unrelated mother-child dyads demonstrates that this relationship cannot purely be attributed to shared genes, a finding supported by studies exploring environmental mechanisms for the transmission of maternal depression to childhood psychopathology (Harold et al., 2011b; Lewis et al., 2013; Natsuaki et al., 2014). For all groupings, associations were found between maternal depression and interparental conflict, and between interparental conflict and mother-to-child hostility. Although direct observations between mother-child hostility and child depressive symptoms were observed for genetically related dyads, this was not a repeated finding for genetically unrelated mother-child pairings. Explanations for this finding could encompasses several elements of the mother-child relationship. Firstly, it might be a result of shared or common genes for genetically related dyads, perhaps the additive effects of genetic and environmental risks within these pairings have a greater impact, so that these children are more susceptible to negative consequences from hostility in the maternal relationship. Secondly it is possible that the finding might be associated with differential parenting for children whom mothers are genetically related to, that is children's differential susceptibility to the environmental aspects of their mothers' depression might be impacting on the development of psychopathology (Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2007; Belsky & Pluess, 2009; Boyce & Ellis, 2005).

Direct associations between maternal depression and child aggressive behaviours were observed for both genetically related and unrelated mother-child dyads. Furthermore, they were also present for mother-to-child hostility and child aggression, but not through interparental conflict for both genetically related and unrelated pairings. These findings are supported by studies which have demonstrated environmental mechanisms for the transmission of maternal depression to a range of child outcomes which might increase the risk for externalising, as well as internalising problems in children (Downey & Coyne, 1990b).

Associations between paternal depression, interparental conflict and hostile father-child relationships with child depressive and aggressive symptoms

A greater number of differences were observed for father-child dyads than mother-child dyads when assessing the associations between paternal depression, interparental conflict and father-to-child hostility on child depressive symptoms for genetically related and genetically unrelated dyads. Paternal depressive symptoms were directly associated with child depressive symptoms for genetically related, but not genetically unrelated father-child dyads. Both measures of family socialisation (interparental conflict and father-child hostility) were associated with child depressive symptoms for genetically related father-child dyads only. This finding contradicts previous studies that found more problematic relationships in households where fathers were genetically unrelated to the child whom they were raising (Golombok et al., 1999). As with the mother-child dyads, associations between genetically related pairings for father-child hostility with child depression might be a result of common, or shared genes, or differential susceptibility.

The associations between paternal depression and child aggressive behaviours appeared to share more similar associations between genetically related and unrelated

dyads than child depressive symptoms. There were no direct associations between father depression or interparental conflict with child aggression for either group. However, indirect associations were found for genetically related and unrelated father-child pairings, in that paternal depression was associated with interparental conflict, which was associated with father-to-child hostility which in turn was associated with child aggressive behaviour. Given that this finding was consistent for genetically relate and genetically unrelated father-child dyads, this association cannot be accounted for passive *r*GE which suggests that this is an environmental effect.

Implications of findings

Researchers who have previously explored the associations between parental depression and indices of family socialisation with child depressive symptoms and aggressive behaviours have been eager to identify environmental mechanisms that might help explain the transmission of psychopathologies from parent-to-child (Harold et al., 2011a; Natsuaki et al., 2014). Identifying environmental mechanisms is beneficial in finding potential modifiable aspects of a child's environment to decrease the risk of negative outcomes, to ultimately improve children's well-being. The current study highlights both direct and indirect pathways from these risk factors to child depressive symptoms and aggressive behaviours. A consistent association that has been replicated thus far has been that of parental depression with interparental conflict and interparental conflict with parent-to-child hostility which are areas that could be targeted with interventions to reduce negative outcomes in children. Furthermore, parent-to-child hostility appears to be a risk factor for the development of depressive symptoms when children are genetically related to their parents, and to aggressive behaviours in all the evaluated situations, for mother-child and father-child dyads in both genetically related and unrelated groupings. This finding suggests that the association observed between

parent-to-child hostility and child depressive symptoms may be better accounted for by passive rGE and therefore may not be a suitable target for intervention. In contrast the association between parent-to-child hostility was observed in both genetically related and genetically unrelated groups. Where parent-child pairings are genetically unrelated, associations cannot be accounted for by passive rGE. This suggests that it might be a modifiable aspect of the child's environment that could be targeted to the remediation of aggressive behaviours in children. It also highlights that different pathways and mechanisms are likely to explain the development of depressive symptoms and aggressive behaviours in children. Therefore, different intervention strategies are likely to be needed to target child depressive symptoms and aggressive behaviours. Parent-to-child hostility appeared to show a more consistent relationship with child aggressive behaviours than depressive symptoms which might indicate that although parent-to-child hostility would be an effective target for intervention to reduce aggressive behaviours, different indices of the parent-child relationship might be more important for the development of depressive symptoms in children.

Limitations

Some caution should be taken when assessing findings from the current study with regards to direction of effects as all measures were taken at the same time point. Although the current study used a cross-sectional data set (all measures taken at the same time point) previous studies have demonstrated some reciprocal relationships between child psychopathology and parenting, it appears that parenting more consistently predicts child outcomes (Lewis et al., 2013). A further limitation of the cross-sectional nature of the design is that although the study can disentangle passive rGE from associations between family relationship and child outcomes, evocative rGE is unable to be examined as a possible explanation for the associations. Evocative rGE

is when genetically inherited attributes of the child (e.g. temperament and personality) can influence the parenting the child receives (Ge & Conger, 1996).

A further limitation relates to the reporting of psychopathology and indices of family socialisation within the study. Mothers and fathers reported on their own depressive symptoms, the hostility experienced in family relationships and child outcomes. However, to reduce the impact of shared rater-bias, cross-raters were used where possible (i.e. mothers reported on child psychopathology for father models and vice versa). Future studies should aim to use composites from multiple raters for the parent-child relationship and child psychopathology, either through observational measures of these constructs, or child self-report (Bögels & van Melick, 2004).

A limitation of the current study was that due to the construction of the groups for the models, we were unable to control for the influence of opposite parent depressive symptoms (e.g. in the genetically related and genetically unrelated father-child dyads, mother depressive symptoms were not measured or controlled for). This could be a potential limitation based on our knowledge about assortative mating and psychopathology in partners. Assortative mating describes the tendency for individuals of similar phenotypes to mate more frequently than expected by chance (Mathews & Reus, 2001; Vandenberg, 1972). Couples have been found to show close similarities with each other for a variety of traits, including symptoms of psychopathology. In a meta-analysis of assortative mating and psychopathology, Mathews & Reus (2001) concluded that assortative mating occurs for depression in couples (however assortative mating for other measures of psychopathology, such as bipolar disorder was more common than assortative mating with depression). For the current study, the evidence that couples can have comparatively similar levels of depression could mean that unmeasured parental depressive symptoms might be impacting on associations within

the model. Additionally we know that children who are exposed to two parents with depression might be at greater risk for the development of psychopathology (Weissman et al., 2006). Therefore future studies should attempt to control for assortative mating for depression and the additional risks posed to children living with multiple depressed parents. This could be done by simultaneously measuring depressive symptoms in mothers and fathers so that they their associations can be compared and controlled for when examining the influence of parental depressive symptoms on family relationships and the development of child psychopathology.

Although the current study utilised a genetically informative research design, information on donors was not available. The next study in this thesis will employ a research design that overcomes some of these limitations by assessing the simultaneous impact of parental depression, interparental conflict and parent-child hostility on the development of child depressive symptoms and aggressive behaviours in a longitudinal adoption study where birth mother information can also be assessed (Harold et al., 2013).

A final limitation of the current study is that the analyses did not allow genetically related / unrelated mothers and fathers to be examined within the same model. This means that we could not directly compare the associations between mothers and fathers and genetically related-genetically unrelated parent dyads. The next study will allow for the examination of genetically unrelated parents in an adoption study at the same time.

Chapter 4: Examining associations of birth mother and adoptive parent depression, interparental conflict and parent-child hostility with the development of depressive symptoms and aggressive behaviours in young children using a genetically sensitive research design

Part of this chapter has been presented at the following presentations;

- Oral Presentation: "Parental depression and family socialisation on the development of depressive and aggressive symptoms in children and adolescents", presentation at CDRiP, Sussex, June 2015
- Oral Presentation: "The influence of maternal depression and family socialisation on on the development of depression in adolescents", presentation at International Congress of Clinical and Health Psychology with Children and Adolescents, Madrid, November 2015

Introduction

The first study of this thesis outlined associations of maternal depression, interparental conflict and mother-child hostility with the development of depression and aggression for adolescents living either with or without a mother who had experienced clinical depression. The findings from Study 1 indicated that maternal depression was associated with interparental conflict and interparental conflict was in turn associated with mother-child hostility for families within the clinical and community sample. Mother-child hostility was associated with increased levels of aggressive symptoms for both samples, but adolescent depressive symptoms were only associated with motherchild hostility in the clinical sample. The limitations of the first study included the reliance on mother-child dyads that were genetically related and the absence of fathers in the study. The second study extended these analyses by including an examination of father depression and father-child hostility on the development of child outcomes in a genetically informative research design (IVF). Parents were either genetically related or genetically unrelated to the child that they were rearing. Here it was found that environmental mechanisms helped explain the associations between parental and child psychopathology (specifically internalising and externalising symptoms), more strongly for child aggression than child depressive symptoms.

The second study used a cross sectional design so was able to examine the associations between parental depression, interparental conflict, parent-child hostility and child outcomes during the same time point. However it is difficult to determine cause and affect relationships when all the variables are measured during the same time point. The current study contributes further to the examination of parental depression and family socialisation processes on the development of depressive symptoms and aggressive behaviours by examining these processes in a longitudinal model, which can

examine these processes across time, allowing more confidence in the direction of associations between these measures. It also includes mothers and fathers in the same model, which enables us to examine whether there are independent effects of mother depression, father depression and parent-child relationship hostility, with the additional benefit of being able to assess a marker of background genetic risk (birth mother depressive symptoms). The study also utilises a younger sample of children (aged 18 months during first assessment, 7 years at final assessment) which will enable us to examine processes earlier to identify whether there are earlier intervention points to reduce risks of negative mental health problems in children, and to see whether these processes (parental depression, interparental conflict, parent-child hostility) are similar at different phases of child development.

Mental health problems in childhood are categorised as internalising (e.g. depression and anxiety) and externalising (e.g. aggression, conduct and oppositional defiance) problems (Bayer et al., 2012). As mental health problems in children have been found to often continue into adolescence and adulthood (Bosquet & Egeland, 2006) understanding the development of psychological problems in this age group has become increasingly prominent within empirical literature.

Psychopathology in early childhood

There is some debate amongst researchers and clinicians about how early children demonstrate symptoms that are associated with psychological or mental health problems (DelCarmen-Wiggins & Carter, 2004). Half of all lifetimes cases of mental health problems will have started by the age of 14 years (Kessler et al., 2005). Children and adolescents between the ages of 6 years and 17 years are believed to experience emotional and behavioural problems, with younger children not verbally reporting on their own internalising and externalising symptoms until the age of 11 years (American

Psychological Association, 2013). However, some studies have identified children as young as 4 years verbally reporting to their parents or teachers that they are experiencing emotional and behavioural difficulties (Simpson, Cohen, Pastor & Reuben, 2008) with signs of problematic behaviours associated with child psychopathology present in even younger age groups (Gardner & Shaw, 2008). There currently appears to be a shift in recognising emotional and behavioural problems in increasingly younger age groups (Bayer et al., 2012) though with the exception of autism and Attentional Deficit-Hyperactivity Disorder (ADHD), understanding of child psychopathology in younger children (under 4 years) is far behind understanding of similar problems in older children (Angold & Egger, 2004). Early instances of internalising and externalising problems have been associated with problematic interactions for the child with parents and peers (Fanti & Henrich, 2010). Differentiating between internalising and externalising problems in such early age groups appears to be problematic (Leve et al., 2009). This could be due to several factors such as the recognised co-occurrence of emotional and behavioural problems (Achenbach & Conners, 1989; Weiss & Catron, 1994). Co-occurrence of internalising and externalising problems might be a result of both shared risk factors and temperamental factors which might put children at greater risk of developing these problems. Risk factors that have been associated with both internalising and externalising problems include hostile and negative home environments (e.g. interparental conflict), parental depression and shared genetic liability for the development of emotional and behavioural problems (Silberg et al., 2010; Tully et al., 2008).

There is also evidence that the presence of either an internalising or externalising disorder might increase the risk for the development of the other, that is, one condition

might predispose individuals to another condition (Gjone & Stevenson, 1997).

Externalising problems might predispose children to have an elevated risk for developing future internalising problems, including symptoms of depression (Fergusson et al., 1996). This might be a result of disruptive behaviours having a negative impact on adolescent development, generating stressful life events such as to conflict with peers and parents (Capaldi & Patterson, 1991; Capaldi & Stoolmiller, 1999; Champion, Goodall, & Rutter, 1995). These environmental stressors might increase a child's vulnerability to develop depressive symptoms (Pine, Cohen, Johnson, & Brook, 2002). Depressive symptoms might also lead to the development of externalising behavioural problems (Capaldi, 1992). Depressive symptoms have been associated with a range of disorders including anxiety and aggressive behaviours and it predicts a range of externalising problems in adolescents such as substance misuse (Costello et al., 2011, 2003). Appreciating that these problems co-occur in childhood is vital in understanding the development and aetiology of internalising and externalising problems in early childhood (Fanti & Henrich, 2010).

Further problems in differentiating between internalising and externalising problems occur when looking at trajectories of these problems into adolescents; a high percentage of children that display internalising symptoms such as anxiety and depression in earlier life, go on to display externalising behaviours later in their childhood (Perle et al., 2013). Gilliom & Shaw (2004) assessed trajectories of internalising and externalising problems in a group of males who were aged 2 to 6 years at the first assessment. Associations between higher levels of internalising problems with higher levels of externalising problems were observed, with higher levels of externalising problems predicting rapid increases in the risk of children developing internalising problems (Gilliom & Shaw, 2004).

Continuities between child and adult mental health problems appear to be strong, even for children who have been identified with symptoms at an earlier age; in a study examining children aged between 4-16 years at the first assessment, high levels of childhood symptoms predicted up to a 6 fold increase for adult diagnoses of psychopathology (Hofstra, Ende, & Verhulst, 2002) and children with externalising behaviour problems are more likely to become delinquent as adolescents and portray higher levels of criminality and violence as adults (Liu, 2004).

Despite the regular co-occurrence of internalising and externalising problems in young children, some differences have been observed, for instance, children experiencing internalising problems appear to be more prone to sadness, exhibit less impulsivity and higher levels of effortful control than children of the same age who exhibit externalising problems (Eisenberg et al., 2001). The current study aims to expand current understanding on the development of depressive and aggressive symptoms in early childhood but considering how early levels of these problems (combined internalising and externalising symptoms) at a young age are associated with either internalising or externalising as the child approaches middle childhood.

Internalising problems in early childhood

There is evidence that internalising problems in early childhood has been a neglected area of research in relation to the assessment of psychopathology and mental health problems in this age group (Tandon, Cardeli, & Luby, 2009). Reasons for this gap in empirical literature might be due to the difficulty in detecting the presence of internalising problems and symptoms in children who have limited verbal skills and might lack the ability to effectively communicate their imbalanced internal states or a result of internalising problems being viewed as less problematic by parents and teachers (Tandon, Cardeli & Luby, 2009). Advances in methodology have given

researchers the ability to capture, quantify and interpret the presence and development of internalising problems in earlier age groups (Tandon, Cardeli & Luby, 2009).

Stability has been evidenced for internalising problems in young children; one study demonstrated that almost 40% of toddlers between the ages of 1 to 3 years exhibiting internalising problems continued to exhibit these problems one year later (Briggs-Gowan & Carter, 2006) and reports of children's internalising symptoms at age 3 significantly predicted similar symptoms at age 5 (Kerr, Lunkenheimer, & Olson, 2007). Further evidence shows that early levels of internalising problems can prevail for extended periods of time. Children between the ages of 2 and 3 who exhibit symptoms of depression have three times the risk of exhibiting similar symptoms 8 years later than infants who did not have internalising problems at that early age (Mesman & Koot, 2001). This highlights the importance of early intervention to reduce the development of internalising problems through childhood.

Externalising problems in early childhood

Externalising behaviours are very common in early childhood, with some research indicating that it is more likely for children under the age of three to exhibit externalising behaviours such as aggression than for children to not show these behaviours (Buchmann et al., 2014; Tremblay, 2004). However, persistent aggression has been associated with several negative outcomes for young people as they transition into adolescences, which in turn has been associated with negative consequences in adulthood including unemployment, criminal behaviours and negative consequences on socioeconomic status (Buchmann et al., 2014; Tremblay, 1994). Higher levels of externalising problems in early childhood is often seen as the best predictor for later instances of conduct disorders and antisocial behaviour problems (Campbell, et al., 2000).

A wealth of knowledge has been accumulated on externalising problems in children, research on the presence and development of these problems in younger children have been more vigorously examined than internalising problems (Fergusson & Horwood, 2014; Harold et al., 2013; Loeber & Hay, 1997; Tandon et al., 2009).

Accurately identifying young children with higher levels of externalising problems is necessary to improve our understanding of the development of and stability of externalising problems, which might aid the development of preventions and interventions to reduce negative outcomes in children (Offord, Kraemer, Kazdin, Jensen, & Harrington, 1998). This is particularly salient when considering the stability of externalising behaviour problems in children, that is it appear that it can persist for many years having negative implications on a child's health and education (Fergusson, Lynskey & Horwood, 1996).

Evidence has been provided that shows internalising and externalising problems can be exhibited in young children. This highlights the importance of understanding early mechanisms and processes through which children may develop mental health problems to aid the targeting of timely intervention and prevention strategies, as well as informing such strategies. The current study aims to examine the relative associations of birth mother depression, adoptive parental depression, adoptive parent interparental conflict and adoptive parent parent-child relationship with the development of depressive and aggressive symptoms in young children.

Parental depression

Children of depressed parents are at a higher risk of developing both internalising and externalising problems than children of non-depressed parents (Beardslee, Bemporad, Keller, & Klerman, 1983). The association between parental depression and child psychopathology could be related to multiple factors including

child exposure to parental depressive symptoms, or that child psychopathology and parental depressive symptoms might share common aetiology (McAdams et al., 2015); common or shared genes might explain the higher instances of internalising and externalising problems in children of depressed parents. Negative child outcomes could also be a result of multiple risks including the influence of genes, parental relations (eg. interparental conflict) and parenting (Pemberton et al., 2010).

Exposure to parental depressive symptoms might increase the risk for the development of psychopathology problems in children by disrupted parenting practices that have been associated with parental depression. Parents who experience depression are more likely to be preoccupied and respond to their children with higher instances of negatively (England & Sim, 2009). Negative parenting practices that have been observed in depressed parents include lack of engagement and warmth and increases in hostile parent-child relationships (Lovejoy et al., 2000). These parenting practices have been linked to the presence, development and maintenance of internalising and externalising problems in children and adolescents (Galambos, Barker, & Almeida, 2003; Liu, 2004). Disrupted parenting practices as a result of parental depression remain a concern even when parents are no longer experiencing depressive episodes; poor or disrupted parenting practices can endure even after a depressive episode (England & Sim, 2009; Seifer, Dickstein, Sameroff, Magee, & Hayden, 2001).

Children exposed to parental depressive symptoms are likely to also observe disruptions in their parents' relationship, due to associations found between parental depressive symptoms and interparental conflict (Shelton & Harold, 2008). Interparental conflict has also been associated with the development of internalising and externalising symptoms in children and adolescents (Reynolds et al., 2014).

It is improbable that just one of these genetic or environmental risks is responsible for the development of internalising and externalising problems in children and adolescents, an examination of the interplay between the recognised risks would provide a more informative understanding of how, why and when these problems develop and persist (Masten et al., 2005). The current study aims to investigate the extent to which parental depression might increase negative outcomes in children through environmental mechanisms (adoptive parent depression, interparental conflict and parent-child relationship) by utilising a sample of children who have been adopted at birth and are biologically unrelated to the parents who are rearing them. The study also simultaneously considers the potential genetic risks associated with these negative outcomes by assessing the influence of birth mother depression.

Some studies have previously investigated the impact of adoptive parental depression on child and adolescent outcome. Tully, Iacono & McGue (2008) focused on the development of internalising and externalising disorders in adolescents (including depression and conduct disorder) exposed to adoptive parental depression. They concluded that maternal depression had an environmental impact on the development of depression and disruptive disorders in adolescence and that paternal depression did not appear to have an environmental liability on increased risks for the development of psychopathology in adolescents. Adoptive mother depression has also been associated with toddlers externalising problems (Pemberton et al., 2010). Environmental risk factors measured in a study of adopted children, their birth and adoptive parents suggested that adoptive mother's uninvolved parenting might be involved in the development of children's co-occurring symptoms of both internalising and externalising problems (Roos et al., 2016). Birth mother prenatal drug use appeared to be a mechanism for the development of genetic and environmental influences on the

development of internalising problems in young children, but only parental influences contributed independently to externalising problems (Marceau et al., 2015). These studies demonstrate that the intergenerational transmission of psychopathology might not just be a result of common or shared genes and that the environmental impact of children living with depressed parents might affect development of internalising and externalising problems in children.

The environmental impact of parental depression has been explored in studies that are able to disentangle environmental and genetic influences; specifically, the IVF design (see Chapter 3) and the adoption design that will be utilised in the current study. Harold et al. (2013) examined the familial transmission of depression and antisocial behaviour symptoms using the Cardiff IVF sample. Direct associations between maternal depression and child depression were observed in genetically unrelated dyads supporting the premise that there are environmental factors that influence the transmission of depression from parent-to-child. An interesting observation was that a differential pattern of genetic and environmental mediation underlying the intergenerational transmission of depressive symptoms was found among genetically related and generically unrelated father-child and mother-child dyads.

Interparental conflict

The negative consequences associated with children exposed to marital discord have been extensively examined within empirical research, with two of the most well established outcomes being the presence of internalising and externalising problems in children (Cummings & Davies, 1994; Grych & Fincham, 2001; Rhoades et al., 2011). Children appear to be particularly sensitive to parental discord even from a young age, with distress as a result of parental conflict being evidenced in children as young as 6 months (Harold et al., 2004).

Exposure to interparental conflict might negatively affect children through several mechanisms. Directly observing interparental conflict might act as a direct stressor for children as children appear to react with fear and anger when there are exposed to their parents arguing (Amato & Cheadle, 2008). This finding might be particular salient for younger children exposed to interparental conflict, as this age group are likely to be more egocentric thus more likely to blame themselves for disrupted parental relationships which could result in feelings of guilt and lowered self-esteem (Grych & Fincham, 1990). Additional explanations as to how interparental conflict impacts on a child's psychological development focus on how disrupted marital relationships affect the parent-child relationship (Erel & Burman, 1995). These models suggest that negative emotions experienced within the parental relationship spill-over into the parent-child relationship.

The association between child internalising and externalising problems and interparental conflict might also be attributed to common or shared genes between parents and children simultaneously impacting on parental and child characteristics.

Behavioural genetic studies have demonstrated that personality traits including depression and aggression have a strong genetic component (Plomin et al., 2013). As children share approximately 50 % of their genes with each of their biological parents, genetic predispositions might mean that conflict within the spousal relationship could be positively associated with children's emotional and behavioural problems, but that shared genes might partially inform this relationship (Amato & Cheadle, 2008). To try and disentangle the influence of genes and environment on the association between interparental conflict and children's negative outcomes, researchers might utilise an adoption design, where children are reared by parents whom they are biologically related to. If an association is observed between interparental conflict and child

outcomes in these families, we can be more confident in determining the environmental impact of interparental conflict as the confound of passive gene environment correlations have been removed, thus common or shared genes cannot explain the relationship between these variables. Studies that have examined the relationship between interparental conflict and child outcomes using such designs have found that associations between marital conflict and negative outcomes for both genetically related and adopted children (Amato & Cheadle, 2008), providing evidence for an environmental effect that is not confounded by passive *r*GE.

Parent-child relationship

The influence of the parent-child relationship on children has long been viewed as a fundamental aspect of child development, particularly during early childhood (Kagan & Snidman, 1999; Serot & Teevan, 1961; Shonkoff et al., 2004). The parentchild relationship is likely to be linked with parenting styles; different parenting styles have previously been linked with various outcomes in children. Negative parenting practices including permissive and authoritarian parenting styles being associated with a range of negative outcomes in children including higher levels of anxiety, depressive and aggressive symptoms, lower rates of self-esteem and problems with academic attainment (Smokowski, Evans, Cotter, & Webber, 2014). A well-established aspect of the parent-child relationship that has been associated with negative child outcomes through a range of child developmental stages, is hostility expressed from parents towards their children (Harold et al., 2011; Lewis et al., 2013). Parent-to-child hostility has been associated with disruptive behaviour with peers (Elam et al., 2014), Attention Deficit Hyperactivity Disorders symptoms in children (Lifford et al., 2009) along with aggressive and depressive symptoms in children and adolescents (Elam et al., 2014; Lewis et al., 2013).

As previously discussed, associations observed between specific aspects of the parent-child relationship (e.g. hostile parent-child relationships) and negative child outcomes might be a result of shared genes when children are biologically related to the parents who are rearing them. That is genes might be simultaneously impacting on different traits and behaviours in the parents and children (such as hostile parenting styles and negative child outcomes). The current study will be use a sample of children who are genetically unrelated from their rearing parents, thus it is important to consider research which has been able to disentangle genetic and environmental influences to assess how the parent-child relationship impacts children who are genetically unrelated from their rearing parents. Hostile parent-child relationships have been associated with child externalising problems in both genetically related and genetically unrelated parent-child dyads, though this association appears to be less prominent for internalising problems (Harold et al., 2011; Harold et al., 2013; Rhoades et al., 2011).

Adoption study design

It is well recognised that genetic and environmental factors both contribute to the development of child mental health (Plomin et al., 1977; Scarr & McCartney, 1983). The interplay between genetic and environmental influences on negative child outcomes has more recently been explored (Harold et al., 2008; Natsuaki et al., 2014) with the recognition that previously held beliefs about the divide between genes and environment was overly simplistic and failed to capture the dynamic interactions that might consequently help to describe the aetiology of psychopathology in children (Thapar & Harold, 2014).

A problem with much previous research is the difficulty in interpreting associations between indices of family socialisation (e.g., interparental conflict and hostile parenting) and child outcomes as it is unclear whether associations operate as

environmental or genetic risk factors. Where these associations are investigated within biologically related families (parents genetically related to the child whom they are rearing), relationships between a parent's and child's trait might be due to an underlying shared genetic characteristic that co-influences the characteristic. These shared genes might also be impacting on the family environment in which the child is living, e.g., interparental conflict and hostile parenting. This phenomenon is referred to as genotype-environment correlation (rGE). Adoption studies attempt to disentangle this overlap as a natural experiment occurs where children are reared by parents whom they are genetically unrelated to (because there are no underlying shared genes, we can control for genetic contributions to a child trait).

Adoption studies examine the resemblance between biologically related and unrelated parent-child dyads. Similarities between adopted children and their biological parents are assumed to be due to shared genes whereas similarities between adopted children and their non-biological parents are assumed to be due to shared environment. Genetic contributions and intrauterine environmental influences are assessed using biological birth parent information, if information on birth parent is available (e.g. The Early Growth and Development Study Leve et al., 2013). Post-natal environmental impacts are measured by assessing factors which might influence a child from their rearing environment (e.g. indices of family environment, adoptive parental psychopathologies, and school or community environments). Adoptive parents share post-natal environment but no genes with the adopted child whereas biological birth parents share genes but typically only inter-uterine environment with the child. There are therefore a wide range of gene-environment interactions that can be examined using adoption studies. The influence of passive gene-environment correlation (passive rGE) is removed as parents rear children they are biologically unrelated to. Where adoption

studies are longitudinal, evocative rGE can also be assessed, whereby a genetically inherited child trait may evoke a specific behavioural response from their adopted parent (Rutter et al., 2006).

Findings from research exploring the influence of family socialisation and parental psychopathology on child negative outcomes using adoption designs have contributed a wealth of knowledge to improve our understanding of how environmental and genetic influences might operate to impact development. Collectively, the results tackle a range of questions that were previously unanswered within the field, such as how parenting might offset genetic risks to prevent the emergence of child problems in toddlerhood (Leve et al., 2009). They have also presented studies which compare and contrast the contribution of birth parent psychopathology with environmental risk factors, such as adoptive parent psychopathology or adoptive parenting on child outcomes (Marceau et al., 2015) highlighting once again, how multifaceted influences on the development of psychopathology in children and adolescents appears to be.

Genetic contributions to negative child outcomes appear to be related to birth mother characteristics, including slower maternal processing speed (Roos et al., 2016), increased prenatal pregnancy risks and prenatal maternal drug usage (Marceau et al., 2015; Pemberton et al., 2010). Associations have also been found between internalising symptoms and antisocial behaviour in birth mothers with negative outcomes in children (Kerr et al., 2013; Marceau et al., 2015).

Postnatal environmental risks that have been highlighted within adoption studies include adoptive mother's uninvolved parenting (Roos et al., 2016), adoptive mother and adoptive father hostile parenting (Elam et al., 2014b; Stover et al., 2012), adoptive mother depression, and adoptive father depression (Natsuaki et al., 2014; Pemberton et

al., 2010), and conflict within the adoptive parents relationship (Harold et al., 2013; Stover et al., 2012).

Finally, evidence of evocative effects of adopted child genetic influences on adoptive parents have also been identified; birth mother low behavioural motivation predicted toddler low social motivation which predicted both adoptive mother-child and father-child hostility (Elam et al., 2014) which in turn has been shown to impact negatively on outcomes in children. One of the benefits of having a research design which is both genetically sensitive and longitudinal is the ability to further investigate direction of effects in associations between child depressive symptoms and aggressive behaviours on family socialisation (interparental and parent-child relationships).

Exploring the interactional nature of the parent-child relationship in a situation where effects of shared genes have been eliminated might prove important in contributing to our understanding of how these mechanisms operate.

Previous studies have examined the influence of heritable characteristic of child evoking parental responses. Inherited externalising behaviour in children (including aggression) has been shown to elicit responses in rearing parents whom they are genetically unrelated to; children whose birth parents reported higher levels of aggressive behaviour evoked more negative parenting responses, including harsh discipline and hostility (Ge & Conger, 1996). The Nonshared Environment in Adolescent Development Study (NEAD; Neiderhiser, Reiss, & Hetherington, 2012) supported these findings by demonstrating that a child based genetic influence on hostile parenting preceded the later presence of antisocial behaviour. A recent meta-analysis of children of twins on parenting as a reaction evoked by children's genotype (Avinun & Knafo, 2013) concluded that developmental research must view children as active agents in their home environment and consider not only the impact that parents

have on children, but the impact that children have on their parents. Evoked parental responses to heritable child characteristics appear to be present even with children as young as one (Reiss, 2005). A clearer understanding about this relationship between child characteristics and parental responses would enable practitioners to provide interventions/preventions and support to parents whose children might display early signs of psychopathology which aim to reduce hostile parent-child relationships, thus reducing the risk of the child developing future externalising problems.

A gap in the literature that this study addresses is a further understanding of how child depressive and aggressive characteristics may impact on the marital relationship and parent-child relationship. Hajal and colleagues (2015) recently examined the associations between positive child-parent and family level characteristics in the EGDS study in families with 9-month olds. Evocative rGE was tested by examining the effect of birth parent temperament on harsh-parenting from adoptive parents. Findings highlighted the influence of evoked effects on adoptive fathers'. Harsh parenting for adoptive fathers was inversely related to marital quality and to birth mother positive temperament. Marceau and colleagues (2015) study examined prenatal genetic risk (as measured by birth mother substance abuse, internalising problems and externalising problems), children's cortisol and parenting on the aetiology of internalizing and externalizing problems on children in the EGDS sample. Different pathways were identified between internalising and externalising problems in children which further supports the importance of studying these two dimensions separately. There were direct effects of parenting influences on externalising problems in children and. For internalising problems, prenatal risk and child cortisol transmitted effects of genetic and parenting influences via indirect pathways. However, the current study aims to expand on these findings by considering how birth mother depressive symptoms is associated

with early levels of child psychopathology which might evoke parent-child hostility from adoptive parents.

Current study

The current study will focus on associations of birth mother and adoptive parental depressive symptoms, adoptive parents' marital hostility and hostile adoptive parent-child relationships with the presence and development of depressive and aggressive symptoms in young children. The current study builds on Harold and colleagues (2013) study that focuses on the associations between interparental conflict, hostile parenting, and children's externalising behaviour problems. The study found indirect associations between parental conflict and child externalising problems through mother-to-child and father-to-child hostility in genetically unrelated groups. These associations were stronger for genetically unrelated fathers than genetically unrelated mothers. The current study will extend the previous analyses by taking into consideration the impact of the role of the father, assessing both adoptive father and mother depressive symptoms and adoptive mother-to-child and father-to-child hostility. Birth mother levels of depression will serve as an estimate of risk for the development of psychopathologies in children.

Method

Sample: The Early Growth & Development Study

The sample consisted of linked adopted children, adoptive parents, and birth parents participating in the first cohort of the Early Growth and Development Study (EGDS), a longitudinal, presently on-going multisite study of adoptive families and birth parents (for a detailed overview see Leve et al., 2013). The eligibility criteria for participation within the study were: (a) the adoption placement was domestic, (b) the infant was placed within 3 months postpartum, (c) the infant was placed with a

nonrelative adoptive family (d) the infant had no known major medical conditions, including extreme prematurity or extensive medical surgeries and (e) the parents were able to comprehend English at eighth-grade level.

The first cohort of the EGDS sample includes 350 triads; 350 adopted children, 350 sets of adoptive parents, 350 birth mothers and 115 birth fathers.

The EGDS participants were representative of adoptive populations that completed adoption plans at the participating agencies during the same time period (Leve et al., 2013). Differences in sociodemographic characteristics between birth and adoptive parents were also representative of typical adoptive populations, with adoptive parents having more advantaged socioeconomic background than birth parents (DeFries, Plomin, & Fulker, 1994).

Data for the participants in the first cohort of the EGDS study was taken at several different time points or waves (Wave 1 adopted children 9 months old, Wave 2 adopted children 18 months old, Wave 3 adopted children 27 months, Wave 4 adopted children 4 ½ years, Wave 5 adopted children 6 years, Wave 6 adopted children 7 years old). Child outcomes for the current study were measured when the adopted children were 18 months old (M = 18.1 months, SD = 1.3 months) during Wave 2 and 84 months old (7 years) during Wave 6. Forty-two percent of the children were female (N = 151); 63% of the children were the adoptive parents' first child. The mean infant age at adoption placement was 7 days (SD = 13 days, Mdn = 2 days, mode = 2 days). The adoptive families were typically college educated, middle-class families. The mean ages of adoptive mothers (AM) and adoptive fathers (AF) at Wave 1 were 38 years (SD = 5.4 years) and 39 years (SD = 5.9 years) respectively, and the couples have been married an average of 11.9 years (SD = 5.1 years). The infants and adoptive parents were predominantly White (64% infants, 91% mothers, 88% fathers). There were also a

significant number of multi-ethnic participants (21% infants, 4% mothers, 3% fathers) and African American participants (11% infants, 4% mothers, 6% fathers). The overall number of families used who had completed the relevant questionnaires and measures was 114. The final sample used was reduced due to several selection criterion; same sex families were removed (to remain consistent across other studies within the thesis), only two parent households were used. As this is a longitudinal study and multiple variables were considered across the time points, only families who had complete data across all points for all variables were included.

Sample recruitment

Recruitment for the first Cohort of EGDS was initiated in March 2003 with adoption agency liaisons helping to identify families who had completed through their agency in the Northwest, Mid-Atlantic and Southwest regions of the United States. Five key aims for recruitment were considered during this process (1) to reduce the likelihood of recruiting only one member of the adoption triad (child, adoptive parents and birth parents); (2) to minimise potential ethical concerns by not initiating contact until after the period of revocation; (3) to minimise the probability that participation in the study would cause information to be transferred across participants, including adoption agencies; (4) to recruit a sample that would contain ethnic diversity and varying levels of adoption openness (contact and knowledge between birth and adoptive families); and (5) to recruit a large subsample of birth fathers (Leve et al., 2013)

Once the participants were deemed to be eligible for participation (see inclusion criteria in Sample Description), the adoption agency liaised with researchers and sent families a letter describing the study to all eligible adoptive families 2 to 4 weeks after placement of the child. Adoptive families were at this point given the opportunity to opt out of the study and/or contact from the researchers by returning a self-addressed,

stamped postcard. Two weeks after mailing the letters, liaisons contacted the birth mothers linked to the adoptive families that had not opted out of the study (80% of adoptive families). During this call, the liaison provided a description of the study and asked for permission to have a recruiter from the study contact her directly.

Measures

Parent depression. Birth mothers (BM), adoptive mothers (AM) and adoptive fathers (AF) completed the Beck Depression Inventory (BDI) a 21-item measure of depressive symptoms (Beck & Beamesderfer, 1974). The questionnaire asks adults to describe the way they have been feeling in the past week on a scale of 0-3 such as 'I *do not feel sad*' to 'I am so sad or unhappy that I can't stand it'. BM completed the same questionnaire at Wave 1 (M = 11.31, SD = 9.18), Wave 2 (Mean = 11.03, SD = 9.49) and Wave 3 (M = 7.11, SD = 7.38) Internal consistencies were very good for birth mother BDI at Wave 1 ($\alpha = .92$), Wave 2 ($\alpha = .91$) and Wave 3 ($\alpha = .90$). Birth mother responses to the BDI were summed to create a composite measure of birth mother depressive symptoms (M = 28.55, SD = 23.32). Adoptive parents questionnaires responses to the BDI were used from Wave 2 (AM, Mean = 3.84, SD = 3.59; AF M = 2.70, SD = 3.32) internal consistencies were good for AM ($\alpha = .77$) and AF ($\alpha = .80$).

Interparental conflict. Interparental conflict between adoptive parents was measured using two questionnaires and an observational measure of relationship quality during Wave 5 of EGDS.

The Behaviour Affect Rating Scale (Melby et al., 1995) requires each spouse to report on their own and their partner's behaviour, answering questions using a 7-point scale ranging from never to always. Questions on the respondent's view of their partner's behaviour included 'How often did your partner get angry at you?' and 'How often did your partner criticize you?' The current study used the hostility factor of

responders reporting the hostility that had been evidenced towards them by their partners. Adoptive mothers reported a mean score of 10.90 (SD = 2.81) and adoptive fathers had a mean report of 11 (SD = 3.03). Internal consistencies were very good for AM ($\alpha = .91$) and AF ($\alpha = .90$).

The second questionnaire used to assess the interparental relationship was the reduced Marital Instability Index (Booth, Johnson, & Edwards, 1983) which is an abbreviated form of the Marital Instability Index using five extracted items that best predict instability within the spousal relationship. For each item, AMs and AFs reported independently on the frequency of their own behaviour using a 4-point scale: 1(not in the last year) to 4 (within the last 3 months). Sample items include: "Have you or your partner seriously suggested the idea of divorce" and "Has the thought of separating or getting a divorce crossed your mind?". Items were summed to compute a score for each parent; higher scores indicated greater marital instability. AMs had a mean marital instability index score of 6.26 (SD = 2.64) and AFs had a mean score of 6.32 (SD = 4.28). Internal consistencies were very good for AM ($\alpha = .83$) and AF ($\alpha = .90$).

The marital interaction task is an observational measure of interparental relationships. Adoptive parents were asked to discuss 19 topics about their marital relationship topics for 20 minutes. Discussion topics were purposely designed to elicit positive and negative emotions such as 'How long have you been living together? Where did you meet? What are some of the things you first liked about each other and what are the things you enjoyed doing together? and 'What frustrates you about your relationship' Trained observational coders who were blind to all study hypotheses coded this task by using the Iowa Family Interaction Rating Scales- Marital Interaction Code (Dogan, Lei, Milne-Kahn, & Pong, 2005). Mood, communication, antisocial behaviour, coercion, hostility, relationship quality, responsiveness and warmth/support

are coded for. Due to the novelty of this measure with the current data set we began by focussing on the codes for 'hostility', 'reciprocal hostility' and 'relationship quality'. As the specific hostile subscales of the marital interaction task did not correlate with the questionnaire measures for interparental conflict, we decided to instead purely focus on 'relationship quality' from the marital interaction task. This scale assesses the observer's evaluation of the quality of the dyad's relationship with a low score indicating unhappy, emotionally unsatisfying or brittle relationship. Higher scores are indicative of the observer's impression that the relationship is warm, open and emotionally satisfying. This particular scale offers an overview of what the observer's impression of the overall relationship was. Lower scores indicated low warmth/support, low positive mood, low listener responsiveness, high angry coercion, and high hostility and reciprocate hostility so the scale was recoded before analysis (higher score indicates a more negative relationship). Coder inter-rater reliability for relationship quality was good(K = .60).

The Behaviour Affect Rating Scale, Marital Instability Index and relationship quality subscale of the marital interaction task were highly correlated and therefore standardised using z-score transformations and summed to create a measure of combined mother, father and observational reports of interparental conflict.

Parent-to-child hostility. Hostile parent-child relationships were measured using the mother and father rated hostility subscale of the IYFP Rating Scales (Melby et al., 1998) with each parent reporting on their own relationship with their child at Wave 6 (child 7 years old). Parents were asked to rate how often they had responded in a hostile manner towards their child on a scale of 1 to 7 (always to never) in the past month. Examples of the statements the parents rated for frequency were 'Get angry at

him/her' and 'Criticise his/her ideas'. Internal consistencies were good for adoptive mothers ($\alpha = .77$) and fathers ($\alpha = .77$).

Child outcome. Child depression and child aggression were measured by adoptive mothers and adoptive fathers using a parent-rated version of the Child Behaviour Checklist (CBCL; Achenbach, 1991) during Wave 2 (adopted children 18 months old and Wave 6 (adopted children 7 years). The Anxious-Depressed subscale was used to measure depression and the aggression subscale was used to measure child aggressive behaviours. Adoptive parents were asked to indicate how well specific statements described their child within the past six months. Mother's indicated that the statements were 'Not True', 'Sometimes True' or 'Very True' (on a scale of 0 to 2). Statements included child 'Complains of loneliness' and child 'Cries a lot' for the anxious-depressed subscale. For the aggression subscale, statements included 'Destroys his/her own things' and 'Argues a lot'. AMs and AFs independently completed assessment items were aggregated and computed into one measure of child depression, and another measure of child aggression. Internal consistencies were poor for adoptive mother and father depression at Wave 2 (AM $\alpha = .50$; AF $\alpha = .54$), perhaps related to difficulties in assessing psychopathologies in children at such a young age, but good for both parents reports of child depressive behaviour at Wave 6 (AM α = .62; AF α = .62). For child aggression, internal consistencies were very good for both mother and father at Wave 2 (AM α = .84; AF α = .86) and Wave 6 (AM α = .63; AF α = .60). During Wave 2, adopted children were 18 months old, Leve et al (2009)have previously identified the difficulties in differentiating between internalising and externalising behaviour problems in this age group so AM and AF ratings for child depression and aggression symptoms were summed to create a measure of child psychopathology for Wave 2.

Model fit statistics

Model fit was examined using several fit statistics, as outlined in the previous chapter (Chapter 2). Briefly, a good model fit was indicated by a non-significant chi-square test (χ^2) test. As the chi-square test is sensitive to sample size, additional indicators were used. These included a CFI and TLI of greater than 0.95 and an RMSEA of less than 0.5.

Results

Correlational analysis

Intercorrelations, means and standard deviations are presented for the associations between parental depression (birth mother depression, adoptive mother depression, and adoptive father depression), adoptive family socialisation (interparental conflict and parent-child hostility) and child outcomes (child depression and child aggression) in Table 1.

Significant associations were observed between birth mother depression and child aggression at Wave 6 (r=.19, p<.05), adoptive mother depression was significantly associated with interparental conflict (r=.21, p<.05), father-child hostility (r=.24, p<.01) and child emotional and behavioural problems at Wave 2 (r=.29, p<.01). Adoptive father depression was significantly associated with father-child hostility (r=.23, p<.05) and child emotional and behavioural problems at Wave 2 (r=.29, p<.01). Interparental conflict was significantly associated with father-child hostility (r=.31, p<.01). Mother-child hostility had significant correlations with father-child hostility (r=.35, p<.01), child emotional and behavioural problems at Wave 2 (r=.27, p<.01) and child aggressive behaviours at Wave 6 (r=.46, p<.01). Father-child hostility was significantly associated with child emotional and behavioural problems at Wave 2 (r=.21, p<.05) and child aggression at Wave 6 (r=.38, p<.01). Child

emotional and behavioural problems at Wave 2 were significantly associated with child aggression at Wave 6 (r = .32, p < .01) with a significant association also observed between child depression at aggression at Wave 6 (r = .46, p < .01).

Table 1. Intercorrelations Among Constructs for Early Growth and Development Study (N = 114)

Va	riable	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
1)	Birth Mother	-								
	Depression									
2)	Adoptive		_							
	Mother	.27**								
	Depression									
3)	Adoptive Father	04	.11	_						
	Depression									
4)	Interparental	18	.21*	.12	_					
	Conflict ^a									
5)	Mother-Child	.05	.17	.03	.09	-				
	Hostility									
6)	Father-Child	01	.24**	.23*	.31**	.35**	-			
	Hostility									
7)	Child									
	Psychopathology	.07	.29**	.19*	.15	.27**	.21*	-		
	(Wa 2)									
8)	Child	.07	13	00	.09	.10	.16	.16	_	
	Depression									
	(Wa6)									
9)	Child	.19*	01	.11	01	.46**	.38**	.32**	.46**	-
	Aggression									
	(Wa6)									
	Means	28.55	3.51	2.69	63	10.90	11.00	19.14	3.53	10.41
	SD	23.32	3.36	3.19	3.22	2.81	3.02	10.21	3.23	8.40

^{*}*p* <.05, ** *p* < .01.

Path analysis

The Little's test of missing data (Little, 1988) indicated that data was missing at random (χ^2 (313) = 346.95, p = .091). Proportion of missing data ranged from 11% to 55% across all study variables. Given that one of the aims of this study is to examine processes specifically for child depressive and aggressive symptoms, listwise deletion was used across all study variables to ensure samples were identical across each model

Wa = wave of the study

^a Standardized z-scores of interparental relationship

(N = 114). This ensured that any differences in processes/pathways are not due to sample size differences across the models, and likely reflects theoretical differences.

Associations between birth mother depression, adoptive parent depression, interparental conflict, mother-child and father-child hostility with child depressive symptoms and aggressive behaviours were further examined using path analysis. The models were conducted using Mplus7 (Muthen & Muthen, 2007) using maximum likelihood estimation procedures. Model fit was examined using chi-square statistic (χ^2), Root Mean Square Error of Approximation (RMSEA), Confirmatory Fit Index (CFI) and the Tucker-Lewis Fit Index (TLI). Good model fit was indicated by non-significant χ^2 , RMSEA close to zero (\leq .06), and a CFI and TLI \geq .95 (Kline, 2005).

Birth mother depression, adoptive parent depression, family socialisation and child depressive symptoms

Figure 1 illustrates results for child depressive symptoms as an outcome measure for assessing the associations between parental depression, interparental conflict and parent-child hostility. No association was observed between birth mother depression and child psychopathology at Wave 2 (β = .16, p = .07). Children's general measure of psychopathology at Wave 2 was associated with adoptive mother depression (β = .25, p < .05) but not adoptive father depression (β = .10, p = .26).

Child psychopathology at Wave 2 was associated with mother-to-child hostility at Wave 6 (β = .25, p < .05) and adoptive father depression was associated with father-to-child hostility at Wave 6 (β = .17, p < .05). A significant pathway was observed between adoptive mother depression at Wave 2 and interparental conflict at Wave 3 (β = .17, p < .05) which was associated with father-to-child hostility at Wave 6 (β = .25, p < .01) but not mother-to-child hostility (β = .17, p=.07). Mother-to-child hostility and father-to-child hostility were strongly associated at Wave 6 (β = .31, p < .01). The

Model fit indices were poor (N=114, $\chi^2(8)$ = 19.64, p = .01; RMSEA = .11; CFI = .78; TLI = 0.30).

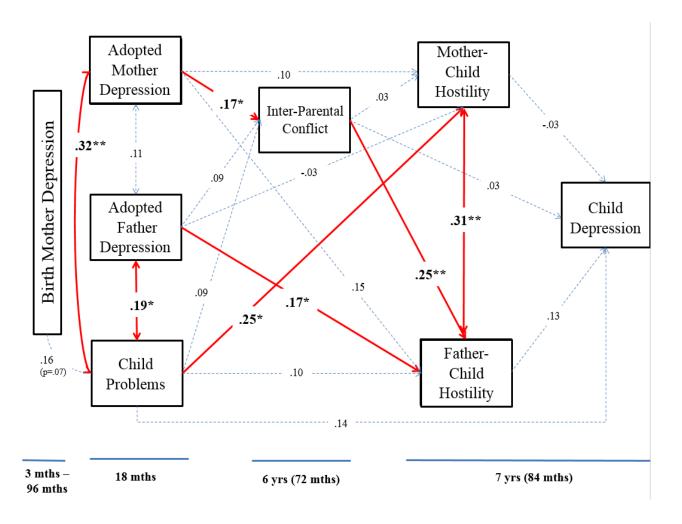


Figure 1. Parental depressive symptoms, interparental conflict, adopted parent-child hostility and child depressive symptoms *p < .05, **p = < .01 (N = 114, $\chi^2(8) = 19.64$, p = .01; RMSEA = .11; CFI = .78; TLI = 0.30)

Birth mother depression, adoptive parent depression, family socialisation and child aggression

Figure 2 illustrates results for child aggressive symptoms as an outcome measure for assessing the associations between parental depression, interparental conflict and parent-child hostility. No association was observed between birth mother depression with child psychopathology at Wave 2 (β = .16, p = .07). Children's general measure of

psychopathology at Wave 2 was associated with adoptive mother depression (β = .32, p < .01) but not adoptive father depression (β = .10, p =.26).

Child psychopathology at Wave 2 was associated with mother-to-child hostility at Wave 6 (β = .25, p < .05) and adoptive father depression was associated with father-to-child hostility at Wave 6 (β = .17, p < .05). A significant pathway was observed between adoptive mother depression at Wave 2 and interparental conflict at Wave 3 (β = .17, p < .05) which was associated with father-to-child hostility at Wave 6 (β = .25, p < .01). Mother-to-child hostility and father-to-child hostility were strongly associated at Wave 6 (β = .31, p < .01). Mother-to-child hostility was associated with child aggressive behaviours at Wave 6 (β = .32, p < .01) and father-to-child hostility was also associated with child aggressive behaviours at Wave 6 (β = .28, p < .01). Early levels of child psychopathology were associated with child aggressive behaviour at Wave 6 (β = .21, p < .05). The Model fit indices were poor (N =114, χ ²(8) = 20.62, p = .008; RMSEA = .12; CFI = .86; TLI = 0.56).

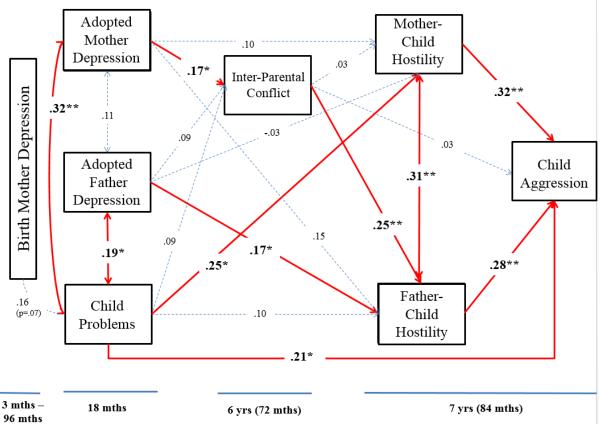


Figure 2. Parental depressive symptoms, interparental conflict, adopted parent-child hostility and child aggressive symptoms *p < .05, **p = < .01 (N = 114, $\chi^2(8) = 20.62$, p = .008; RMSEA = .12; CFI = .86; TLI = 0.56)

Discussion

The final study of this thesis built on the first two studies whilst considering their limitations. It aimed to piece together the primary components that have been examined within the thesis; parental depression (adoptive mother and father), interparental conflict, parent-child hostility (adoptive mother and father) and the development of child psychopathology (depression and aggression) in children and examine these within a longitudinal, genetically sensitive research design. Associations of parental depression (birth mother, adoptive parents), interparental conflict, and hostile adoptive parent-child relationships with the presence of depressive and aggressive symptoms in young children were assessed. The findings of this study will now be discussed in detail and an overview of the limitations and how these might inform future research will also be considered. The primary findings of the study which will be discussed include the presence of an early association between adoptive parental depression and child symptoms of psychopathology, the absence of an association between birth mother depressive symptoms and early levels of child psychopathology, the separate associations observed between maternal and paternal relationships within the family and child psychopathology, the significant association between early levels of child psychopathology with mother-child hostility (but not with interparental conflict or father-child hostility) and the relationship between elevated levels of parental hostility with later symptoms of aggression, but not on depressive symptoms in children.

The first notable finding that will be discussed is the relationship between early symptoms of child psychopathology (18 months) and parental depressive symptoms (birth mother, adoptive mother, and adoptive father). There was not a significant association observed between birth mother depression and adopted child

psychopathology at 18 months, although this was just below conventional levels of significance. Birth mother depression was not correlated with depressive symptoms in children at age 7 years, it was however associated with child aggressive symptoms.

Both adoptive parent depressive symptoms were associated with the early measure of child psychopathology. These findings could be explained in several ways. Firstly, they add support to theories that environmental mechanisms have a strong influence on the aetiology of psychopathology in young children (Natsuaki et al., 2014), this would explain why an association at such a young age exists between genetically unrelated parent-child dyads but not genetically related mother-child dyads. Secondly, the results support previous findings that a genetic predisposition for the development of externalising problems exists (Liu, 2004). The findings could also signal that birth mother depression is not a significant risk in terms of genetic predisposition for the development of psychopathology in children, other measures of birth mother (and birth father) psychopathology should be considered and this association should be examined when the child is older as the genetic risk might not manifest until later in development.

Results from the current analysis support the spill-over hypothesis (Erel & Burman, 1995); that hostility within the marital relationship spills over to hostile parent-child relationships. However, this was only found for the interparental conflict and father-child hostility which is supported by previous research highlighting that fathers might be more vulnerable to the effects of marital conflict in terms of how it impacts on their relationship with their child (Goldberg & Easterbrooks, 1984; Katz & Gottman, 1994). Even though the interparental conflict did not predict elevated levels of mother-child hostility, the finding still has implications for intervention efforts. The findings support results from early prevention and treatment studies targeting parenting (Dishion, Shaw, & Connell, 2008; Shaw, Dishion, Supplee, Gardner, & Arnds, 2006; Van Zeijl et

al., 2006) or specifically in the case of the current study, paternal parenting, and/or marital relationships (Cowan, Cowan, & Ablow, 2005) in interventions to reduce externalising problems in young children. Considering the results indicated father-child relationships are at a higher risk of negativity as a result of the interparental conflict, this should be further examined in future research, especially given how few interventions specifically include fathers.

There was a significant direct pathway between 18 month child symptoms of psychopathology and adoptive mother hostile parenting which is consistent with other reports of reciprocal relationship between child behaviour and parenting (Colder, Lochman, & Wells, 1997; Scaramella & Conger, 2003) and is suggestive of evocative *r*GE (Plomin et al., 1977). However, this relationship was not observed between early symptoms of child psychopathology and adoptive father-child relationship at 84 months. This might be because mothers typically spend more time with their young infants, even if both parents are in full time employment (Craig, 2007; Pleck & Masciadrelli, 2004) so might be more susceptible to a child evoking a negative response. However, adoptive fathers have been previously identified as interacting with their infants and spending more time with their infants than biologically related fathers (Holditch-davis, Sandelowski, & Harris, 2007) which may impact on other aspects of AF parenting not examined in current model.

The development of depressive symptoms was not associated with any of the measures examined in the model, including early measures of psychopathology.

Previous studies focussing on the associations between parent-to-child hostility and child depression have been inconclusive (Lewis et al., 2013) which suggests that this specific measure of the parent-child relationship might not be the right mechanism to be examining. It might be the case that 18 months is too early an age to be looking for

behaviours which might later develop, or provide trajectories for child depressive symptoms, or that these early symptoms of psychopathology are more closely associated with child aggression (Gilliom & Shaw, 2004). It is also important to remember that children were aged 7 during the assessment for depression, where the typical age for manifestation of depression in children and adolescents is 14-15 years (Perle et al., 2013). Therefore, future research should examine these processes once the children have entered the peak period of risk for depression. It is also possible that other aspects of parenting (e.g. parental rejection, lack of warmth) might be more relevant for the development of depressive symptoms in early childhood.

Leve et al. (2009) observed how adopted children may be at differential risks for specific forms of adoptive parenting, related to a child's genetic risk for psychopathology. Toddlers who were deemed to be at high genetic risk, based on birth mother psychopathology (anxiety, depression, antisocial and drug use) benefited from structured parenting, whereas negative outcomes for the same aspect of parenting was associated with increased behavioural problems for adopted children who had been rated as having low genetic risk. Differential outcomes for children at low and high genetic risks have also been demonstrated in a study which found that infants at a genetic risk for externalising problems and affect dysregulation demonstrated heightened attention to frustrating events only in families where the adoptive mother had higher levels of anxious and depressive symptoms (Leve et al., 2009). Children might inherit an emotional liability which makes them more sensitive to the effects of marital hostility, as Rhoades et al. (2011) found associations between marital hostility and toddler anger was not moderated by a low genetic risk (birth mother anger and frustration), however for those children who had been considered to be at high genetic risk, adoptive parents inter-parental conflict predicted later levels of toddler anger and

frustration levels. Adopted children who demonstrated greater attention control in infancy appeared to mitigate genetically based risks for internalising problems when raised by adoptive parents who were rated for low levels of anxiety (Brooker et al., 2014). This might suggest that, for children who are genetically susceptible for developing internalising problems and raised in a low-risk environment, high attentional control might serve as a protective factor from the development of internalising problems.

Evidence from the current study suggests that environmental mechanisms influence development of psychopathology during early childhood, furthermore, environmental processes associated with parental depression might increase the risk of the development of externalising problems within this age group. Previous studies that have used biologically related families to assess associations between family socialisation with the development of psychopathology in children have not been able to rule out that the associations observed between marital hostility, parenting and child outcomes might be a result of genetic influences. In the present study, the potentially confounding presence of shared genes was removed as children were genetically unrelated to their rearing parents. The results can therefore confirm that there are significant environmental effects of parental depression and marital hostility on children's symptoms of aggression via harsh parenting in early childhood.

The environmental processes identified within this study associated with the development of child externalising problems might be able to contribute to prevention programs which identify and target modifiable environmental factors that are associated with child development. The family factors that were identified within the current study included maternal depression, marital hostility and parent-child hostility, and also the way in which early child characteristics relate to negative maternal responses to their

child. Previous research has identified the influence of marital conflict on child outcome via hostile parenting (Rhoades et al., 2011) but the current study expands on these findings by demonstrating that levels of parent-child hostility may be important for the development of child aggression. Attempting to reduce the levels of parent-child hostility present in households might require different areas to target for interventions for mothers and fathers. It is important to consider how parent-child hostility was affected by hostility from the marital relationship. Interparental conflict was associated with father-child hostility so for fathers, the parent relationship might be a more effective place for intervention. For mothers, interventions which target their depressive symptoms influencing hostility in their marital relationship and providing them with tools to approach difficult early child characteristics might reduce the hostility expressed towards their infants. For fathers, interventions which target the influence of paternal depression on parental hostility and their responses to marital conflict might reduce father-child hostility. The areas targeted for interventions are supported by previous research which has identified that there may be more risks associated for father-child than mother-child dyads in this environment (Cabrera & Mitchell, 2009).

This final study of the thesis aimed to build on the first two studies of this thesis. Within the first study associations between maternal depression, interparental conflict and mother-child hostility were found. This supported previous findings of the impact that maternal depression has on hostile spousal relationships which in turn were associated with disrupted parent-child relationships (Burke, 2003; Krishnakumar & Buehler, 2000). The first study was specifically designed to examine how these variables were associated with the development of depressive and aggressive symptoms in adolescent children living either with or without a mother who had experienced clinical depression. Differences were observed in that mother-child hostility was

associated only with depressive symptoms in adolescents who lived with mothers who had experienced clinical depression. Mother-child hostility was associated with adolescent aggressive symptoms for both groups of adolescents. This study, as noted in chapter 2, although had its strengths in that it was longitudinal, using a clinical and community sample, did not include measures of paternal depression or father-child relationships. It was also a biologically related sample, therefore we could not disentangle genetic and environmental influences on child outcomes. The second study built on this by including fathers and examining these associations in genetically related and genetically unrelated parent-child dyads to determine if common or shared genes contributed to the associations observed within the first study. Environmental mechanisms that might explain the transmission of parental depressive symptoms and hostile parental relationships on aggressive symptoms on children through disrupted parent-child relationships were identified. The second study (Chapter 3) did however have a few limitations in that it was cross-sectional, it was genetically informative but not genetically sensitive and we could not examine mothers and fathers in the same model. The final study extended these findings further by utilising a unique genetically sensitive research design which has specific attributes that overcome these limitations. It measured birth mother depressive symptoms and using a longitudinal design to examine how parental depression, interparental conflict and hostile parent-child relationships would be associated with depressive and aggressive symptoms in young children.

The key findings were that adoptive mother depression was associated with interparental conflict, which was in turn predicted increased levels of father-child hostility. Early levels of child psychopathology symptoms predicted increases in mother-child hostility. Parent-child hostility was associated with child aggressive

symptoms at age 7 years, but not for depressive symptoms in the same age group. The findings of the study highlighted the importance of environmental mechanisms in the transmission of psychopathology from parents to their children. The study demonstrated early associations between adoptive parent depression and child psychopathology when the adopted child was only 18 months old, an association that was not present for children's psychopathology with their birth mother depression, illustrating the importance of environmental mechanisms on the aetiology of psychopathology in young children (Natsuaki et al., 2014).

The inclusion of fathers in the current study should also be further explored. There were differences in the associations between parental depression, marital hostility and hostile parenting for mothers and fathers. Although maternal depression was directly related to marital hostility, the same was not observed for paternal depression. This supports findings that the marital relationship might be placed under greater strain from the presence of maternal depression than paternal depression (Fincham et al., 1997). Marital hostility was directly associated with increases in father-child hostility, but not mother-child hostility which replicates previous findings that fathers' parenting might be more effected by disruptions in the marital relationships than mother's parenting and that fathers show greater evidence for the spill-over of negativity from the marital relationship to their relationship with their children than do mothers (Kerig, Cowan, & Cowan, 1993; Schacht, 2009). Explanations for this could relate to a mothers clearer, more salient role within the family so she might be able to compartmentalise relationships within the family, so that her role as a spouse is distinct from that of a mother (Cummings et al., 2004). Thus although both mother-child and father-child hostility resulted in increased levels of externalising problems, the processes and mechanisms appear to be differential for mothers and fathers.

Limitations

Inferences made from the findings of this study should be balanced by the limitations associated with it. The first limitation that will be discussed is the poor model fit statistics for the models within this study. It is possible that the poor fit reflects the fact that there are other measures, not included in the model, contributing to the associations. Future research should consider what these additional measures might be.

Parent-child hostility and child depressive symptoms and aggressive behaviours at aged 7 years were collected during the same assessment time. It is therefore difficult to draw inferences about the directions and causal relationship between these measures (Hajal et al., 2015). However, previous research suggests that there might be a bidirectional relationship of effects between parent-child hostility and symptoms of psychopathology (Lewis et al., 2013). Mediation through the parent-child relationship was only found in genetically related parent-child pairs (parent-child warmth or hostility did not mediate association in genetically unrelated dyads) which suggests the presence of passive rGE in this group (Harold et al., 2011). Parent-child hostility did not appear to represent an environmental mediated link between parent and child depression. The findings from the current study supports this, given that parent-child dyads were genetically unrelated and no association was observed between parent-child hostility and later symptoms of depression in children. For the development of aggression however, parent-child hostility was associated with aggressive symptoms in this sample of genetically unrelated dyads, indicating that this association could be environmental.

Future research should examine the relationship between parent-child hostility and child outcomes over different time points in the study, as and when later measures

of child psychopathology are available from the EGDS study, to improve the understanding how these measures might impact each other (Rutter, 2007).

Several of the constructs were measured via adoptive parent self-report which risks the influence of shared method variance, however, an observational measure of interparental conflict was used which eliminates some elements of reporter bias for this specific construct. Future studies should aim to use composites from multiple raters for the parent-child relationship and child psychopathology, either through observational measures of these constructs, or child self-report at later age ranges (Bögels & van Melick, 2004).

Finally, in the current study, none of the family processes that were examined (interparental conflict, parent-child hostility) were associated with depressive symptoms in children at age 7 years. Previous studies have highlighted that the intergenerational transmission of depression is largely environmental (Lewis et al., 2013; Natsuaki et al., 2014). This highlights the need for future research to examine alternative environmental mechanisms that might help to explain the development of depressive symptoms in young children.

The current study aimed to expand understanding of the associations of parental depression, interparental conflict and parent-child hostility with the development of psychopathology in young children. The environmental measures examined (interparental conflict and parent-child hostility) did not appear to be associated with the development of depressive symptoms in early childhood, there also did not appear to be an association between early measures of child psychopathology with later measures of child depressive symptoms. For the development of aggressive symptoms in early childhood, multiple processes within the adoptive environment were associated with higher levels of aggression when children were 7 years old with both mother-child and

father-child hostility leading to child aggression. Higher levels of adoptive mother depression were associated with increases in interparental conflict, with both adoptive father depression and interparental conflict being associated with father-child hostility. Early levels of child psychopathology were associated with increased mother-child hostility which provides evidence to support evoked adoptive maternal responses to early child emotional and behavioural problems. In summary, the findings highlight that multiple family-based environmental mechanisms might be associated with the development of externalising problems in children.

Chapter 5: Summary, Conclusions & Putting Findings into Practice

This thesis set out a number of aims in order to examine the role of genetic and specific family relationship factors on the development of psychopathology in children and adolescents. To address these aims, four samples of families were examined; a longitudinal community sample; a longitudinal clinical sample of adolescents living with mothers' who had experienced depression; a cross-sectional genetically informative design of children conceived through IVF and a longitudinal sample of children who had been adopted which assessed the associations of birth mother and adoptive parental influence with development in young children. The central aim of this thesis was to examine the extent to which parental depression, interparental conflict and hostile parent-child relationships were associated with the development of depressive and aggressive symptoms in children and adolescents. In particular, it aimed to consider these mechanisms across a variety of different developmental stages and to isolate modifiable environmental influences through assessing these relationships in families where children were either genetically related or genetically unrelated to the parents who were rearing them.

Overview of primary findings

The first chapter of this thesis aimed to provide a broad overview the aetiology of psychopathologies in children and adolescents with a specific focus on internalising and externalising problems. It explored theories that are currently used to explain the development of depression and aggression in childhood. These include biological risk factors, imbalances in neurotransmitters, genetic factors associated with child psychopathology, parental psychopathology and environmental risk factors. Three

primary risk factors for understanding the development of depression and aggression in children were identified as parental depression, interparental conflict and parent-child hostility. Evidence of the association between these risk factors and child psychopathology was provided. These specific risk factors were explored in detail, taking into consideration the importance of parental mental health, the interparental relationship and the parent-child relationship on child development. The interactions between these risk factors was also examined, for example, the impact of parental depression on interparental conflict and associations between couple relationships and parent-child relationships. The chapter then considered the nature-versus-nurture debate in understanding the presence of negative outcomes in children. Within this chapter, it was found that early research into the aetiology of child psychopathology was primarily concerned in determining whether biological or environmental influences played a more important role. Current understanding of the topic was outlined which represents a more holistic approach as to how these influences are associated with one another (Nature via Nurture). Research designs that examine the influence of parental depression and family relationships on child development were outlined. Widely used designs, including family and twin studies were critiqued and the benefits of using genetically informative IVF and genetically sensitive research designs were explored. Finally, a rationale for the current thesis was delivered and empirical studies briefly outlined. There were two primary goals of the chapter. The first was to present the case that parental depression, interparental conflict and parent-child relationships are associated with depression and aggression in children. The second was to identify what remains unexplored and to outline the importance of improving our understanding of the mechanisms involved in the development of psychopathologies so that we might more effectively reduce mental health problems in childhood.

The second chapter of the thesis and first empirical study (Study 1) identified associations of maternal depression and family relationships with the development of depressive and aggressive symptoms in adolescents. It used two longitudinal samples, a community sample of adolescents and a clinical sample where adolescent lived with a mother who had experienced clinical depression. It intended to explore if the mechanisms involved in the development of psychopathology were similar for adolescents from a community sample and adolescents identified as being at a high-risk for the development of negative outcomes due to the presence of a clinically depressed parent. Maternal depressive symptoms were associated with heightened levels of conflict within the marital relationship, which in turn was associated with increased instances of mother-child hostility for both the community and clinical sample. Differences were found whilst examining the associations between mother-child hostility with the development of depressive and aggressive symptoms on early adolescents; mother-child hostility was associated with the development of aggressive symptoms in both samples. However, mother-child hostility was only associated with heightened levels of depressive symptoms in the adolescent children within the clinical sample.

There were several key findings from this study in relation to the aims of the thesis. First, significant pathways were repeatedly found between maternal depression, interparental conflict and mother-child hostility, which supports extensive previous findings examining the connection between these measures of family influence. Second, that mother-child hostility appears to be closely related to the development of aggressive symptoms in early adolescents. Third, children who are living with a mother who has experienced clinical depression might be at an increased risk of developing depressive symptoms, perhaps as a result of mother-child hostility. The findings of this

study were interpreted with the understanding that the associations between interparental conflict, mother-child hostility and symptoms of depression and aggression might not reflect environmental influences. Children and mothers were genetically related in both of these samples so shared/common genes might be associated with the presence of psychopathologies in children and hostile behaviours in the mother. The first study was also not able to examine associations of paternal depression and parent-child hostility with the development of psychopathologies in adolescents, given the limited number of fathers' available within the clinical sample.

The third chapter and second empirical study (Study 2) aimed to tackle some of the problems identified within the first study such as its inability to examine environmental influences on the development of depressive and aggressive symptoms in children and the absence of fathers in exploring the associations of parental depression and parent-child hostility with child outcomes. This chapter reviewed evidence for the presence of psychopathologies in middle childhood and focused on the importance of utilising genetically informative research designs that are able to disentangle genetic and environmental influences on child development. It also considered the importance of fathers' in evaluating the aetiology of psychopathology in children.

Study 2 utilised data from a cross-sectional study of families who had conceived a child through assisted reproductive technologies, specifically IVF. The second study examined the same risk factors as Study 1 (maternal depression, interparental conflict, mother-child hostility) to examine their associations with the development of depressive and aggressive symptoms in middle childhood. Additional relationships examined within this study were the associations between paternal depressive symptoms, father perceptions of interparental conflict and father-child hostility on negative outcomes in

children. These associations were examined for children who were either genetically related or genetically unrelated to their mother or father who was rearing them.

The findings in study 2 revealed that maternal and paternal depression were associated with interparental conflict and interparental conflict was associated with mother-to-child and father-to-child hostility. Interparental conflict was directly associated with child symptoms of depression when children were genetically related to their fathers but this finding was not exhibited in any of the other models. Maternal depression was directly associated with depressive and aggressive symptoms for both genetically related and genetically unrelated mother-child dyads. The relationship between parent depression and child outcomes was not as consistent across father-child dyads; paternal depression was only directly associated with child symptoms of depression when fathers' were genetically related to their child. Both mother-child and father-child hostility were associated with child aggressive symptoms for genetically related and unrelated parent-child dyads. Parent-child hostility was associated with child depressive symptoms when mother-child and father-child dyads were genetically related but this was not present for parent-child hostility and child depression when dyads were genetically unrelated.

One of the benefits of using the IVF sample for Study 2 is the ability of the studies to disentangle genetic and environmental influences on these associations. When associations were observed for genetically unrelated dyads, the confounding influence of common or shared genes has been removed so we might more confidently determine that an association is due to an environmental influence which might then be targeted for interventions aimed at reducing negative outcomes in children. Thus the observations of the associations in study 2 of note are firstly that parent-child hostility is associated with child aggressive symptoms which cannot be completely a result of

shared genes as this relationship was also found in genetically unrelated dyads.

Secondly, parent-to-child hostility did not seem to be as strong a predictor for the development of child depressive symptoms where parents and children were genetically unrelated. Thirdly that paternal depression is associated with interparental conflict and interparental conflict is associated with father-to-child hostility which provides a broader perspective of the mechanisms involved in the development of psychopathology than Study 1.

There are some factors that should be taken into consideration when interpreting the results from Study 2. Firstly, the sample used a cross-sectional design so all the measures were taken from the same time point so we are unable to determine if one measure might predict increased levels in following measures, we may only state that those measures are associated. However, the direction of effects seems to be closely related to studies that have utilised longitudinal data sets. The second issue is that although the sample is genetically informative, data was not available for genetically related donors where parent-child dyads were genetically unrelated.

The fourth chapter of the thesis comprises of the final empirical study (Study 3). This chapter first provided an overview of the presence and development of psychopathologies in early childhood. It importantly examined existing literature on how early internalising and externalising problems might be detectable in childhood. The associations of parental depression, interparental conflict and parent-child hostility with development during early childhood were specifically outlined. The chapter then focused on findings from adoption studies that have examined how parental depression and family relationships relate to child development. The problems experienced in interpreting findings from Study 2 were taken into consideration and Study 3 aimed to expand on the findings from the previous studies in several ways. Using a longitudinal

sample of families from an adoption study provided additional ways of examining associations of family relationship indices with child psychopathology. A younger sample of children was examined than the previous two studies; children were aged 18 months during the first time point and 7 years of age in the final time point which provided the study with the ability to examine how very early symptoms of psychopathologies might be associated with later levels of depressive and aggressive symptoms. The study included measures from birth mother (depressive symptoms) and adoptive parents (adoptive parent depressive symptoms, interparental conflict, and parent-child hostility) providing the opportunity to examine genetic and environmental influences separately as children were genetically unrelated to their rearing parents so mechanisms can be more confidently determined as having an environmental influence. The associations between adoptive parent depressive symptoms with parent-child hostility were able to be examined simultaneously. Study 1 was unable to examine associations of father depression and family relationships with child psychopathology, Study 2 provided separate models for the mother and father parent-child dyads. This benefits the thesis as it offers the opportunity to consider if there are differential features between maternal and paternal depression and parent-child hostility on the development of psychopathologies in young children.

The final study demonstrated the complexity that different relationships within the family might add to the development of depressive and aggressive symptoms in young children. A combined measure of symptoms of psychopathology was used for child outcome when the adopted child was 18 months old, due to issues with being able to disentangle internalising and externalising problems in young children. Birth mother depression was not associated with early levels of child psychopathology. Adoptive mother depression and adoptive father depression were both associated with early levels

of child psychopathology. Adoptive mother depression when the child was 18 months was associated with increases in interparental conflict when adopted child was 6 years old. Although adoptive father depression was not directly associated with interparental conflict, it was associated with increases in father-child hostility. Interparental conflict however, was associated with father-child hostility. Early measures of child psychopathology were significantly associated with mother-to-child hostility. Neither mother-child nor father-child hostility was associated with child depressive symptoms when adopted child was 7 years old. Adopted parent-child hostility was however associated with child aggressive symptoms. This supports findings from previous studies which suggested that associations between parent-child hostility and symptoms of depression in children might be a result of shared genes (Harold et al., 2011) and strong associations between parent-child hostility and aggression in children (Burt et al., 2005; Vostanis, Nicholls, & Harrington, 1994).

The results from the three studies when considered together provide an over view of how psychopathology in children and adolescents develops across different stages of childhood and how risk factors associated with negative outcomes might partially explain the development of depressive and aggressive symptoms in children. Perhaps the most consistent finding across the three studies was the relationship between parental depression and interparental conflict and interparental conflict and parent-child hostility. This was reported for mothers in the community sample of low-risk adolescents and clinical sample of high-risk adolescents in Study 1, and for mothers and fathers who were genetically related and genetically unrelated from their child in Study 2, which supports previous studies that have examined the relationship between these measures (Shelton & Harold, 2008). However, the final study challenged these associations. It appeared that adoptive mother depression was associated with

interparental conflict and interparental conflict was associated with father-child but not mother-child hostility. This suggests that early interparental and parent-child relationships might be different for younger age groups, or be a reflection of the adoption sample. It is also consistent with previous findings that the father-child relationship might be more susceptible to disruptions as a result of negatively experienced within the interparental relationship (Cummings et al., 2004).

A result that was consistently found across all the studies and data sets was the association between mother-child hostility (Studies 1, 2, and 3) and father-child hostility (Studies 2 and 3) with elevated levels of aggressive symptoms in children and adolescents. This finding is in accord with previous studies that have demonstrated elevated risks for aggressive symptoms when children are exposed to hostility in their relationships with their family (Elam et al., 2014). This highlights the robustness of this finding as it has been identified across different developmental periods, using a range of samples and measures. The findings for parent-child hostility and child depressive symptoms appear to be more complex. When adolescents were living in with a mother who had experience clinical depression, mother-child hostility was related to elevated depressive symptoms in adolescents. This finding was not observed for the community sample. The second study utilised a genetically informative research design and identified associations between mother-child hostility and father-child hostility and depressive symptoms in middle childhood when the dyads were genetically related. Given that this association was observed only in genetically related parent-child dyads (the clinical sample of high-risk adolescents in Study 1 and genetically related parentchild dyads in Study 2) we might conclude that this association is only a result of shared or common genes. The tenuous association between parent-child hostility and child depressive symptoms has previously been examined (Lewis et al., 2013). We cannot

conclude from these findings however that the parent-child relationship is not important in the development of depressive symptoms in children and adolescents as parent-child hostility is just one measure of the relationship. However targeting parent-child hostility would not be the most effective place of intervention to reduce negative depressive symptoms in children within this age group.

It should also be noted that paternal depression has been examined far less frequently than maternal depression for its impact on child psychopathology (Phares, Fields, et al., 2005). The results from the second study found that paternal depression was associated with interparental conflict and father-child hostility. These are therefore interesting results and contribute to the growing body of knowledge on the impact of paternal depression on family relationships and child psychopathology. For example, fathers with depression are more likely to have increased hostility in their father-child relationship and decreased positive parenting behaviours than fathers without depression (Kane & Garber, 2004; Wilson & Durbin, 2010). The impact of paternal depression on child outcomes has also been recognised as independent of the effects of maternal depression symptoms (Kane & Garber, 2008) which might explain why in the final study (Study 3; EGDS) paternal depression was associated with increases in father-child hostility whereas maternal depressive symptoms was associated with interparental conflict.

In summary, three key findings have been identified across the three empirical studies are that parental depression is typically associated with increased levels of conflict within the spousal relationship which in turn is associated with higher levels of parent-child hostility. Mothers might me more impacted by early levels of child psychopathologies that fathers. Parent-child hostility is a good predictor of elevated levels of aggressive symptoms in children and adolescents. As these findings were also

exhibited where children were genetically unrelated from their parents, we can more confidently state that parental depression, interparental conflict and parent-child hostility are at least in part environmental influences.

Limitations of studies conducted

Some limitations should be considered in relation to the studies described in this thesis. Firstly, although some of the findings are interesting and potentially practically significant, conclusions must be tentatively drawn and conclusions about causality would be premature when comparing the findings across the studies owing to the observational nature of the research designs. Where possible, the same or similar measures were used but some more direct comparisons could not be fully realised due to this issue. The range of measures across the studies that have demonstrated complementary findings could also be viewed as a strength.

Perhaps a major limitation in the current thesis was the lack of diversity of family types, given the focus of the research on two parent families. All the studies utilised two parent, heterosexual families. There were practical reasons behind this decision as although some information on same sex couples was available in the EGDS sample, it was important that where ever possible the samples to be as similar as possible to allow for closer comparisons.

To the author's knowledge, there have been no studies that have directly examined the impact of parental depression, interparental conflict and parent-child relationship on the development of child psychopathology for children raised by same sex parents. However, given existing research, there is no reason to suggest that the relationship and associations found within this thesis and previous studies would present itself any differently in same sex parent families.

Studies that have compared parenting attitudes and parenting skills for same-sex and hetrosexual parents have found no systematic differences (Perrin, 2002). Similarly, there is a robust finding within the literature that there are no additional negative outcomes for children reared by same sex parents, if anything, these children seem to be more well-adjusted when considering health, psychosocial and emotional development (Lick, Schmidt, & Patterson, 2011; Perrin, 2002). Several studies have shown that children of heterosexual parents when compared to children of same sex parents identify as being more aggressive, and reports from parents and teachers describe them as more bossy and negative (Steckel, 1987; Tasker, 1999, 2005). Within these studies, children of lesbian parents were not only rated by adults as being more affectionate and responsible, but identified themselves as more loveable. It is also important to remember that the diversity of same sex parents has been underexplored (Perrin, 2002). It seems that regardless of parental sexual orientation, children are generally rated as more well-adjusted when parents report high relationship satisfaction and lower levels of interparental conflict and for those children whose parents do divorce, children seem to be better adjusted if their parents still have an amicable relationship (Emery, 1982; Perrin, 2002). Overall, children seem to be more influenced by family processes and relationships than by family structure.

The current thesis was primarily concerned with the way in which parental depressive symptoms and family relationships relate to the development of negative child outcomes. Parents in all of the studies completed questionnaires for the presence of depressive symptoms but it is important to take into consideration additional psychopathology regularly co-occurs with depression including substance misuse (alcohol and/or drugs) and externalising problems. Approximately 50% of adults with a mental health problem have at least one other concurrent disorder (Clark, Watson, &

Mineka, 1994). The presence of depression, in the absence of other mental health problems is now viewed to be far less common than depression which co-occurs with other disorders (Kessler, DuPont, Berglund, & Wittchen, 1999). The most common disorders that are comorbid with depression in adults are anxiety (Zimmerman, McDermut, & Mattia, 2000), antisocial behaviour (Kim-Cohen & Moffitt, 2005) and substance misuse (Chen et al., 2013). None of the studies within this thesis measured the presence of these additional problems in parents. The associations of parental psychopathologies and measures of family relationships with the development of depressive and aggressive symptoms in children and adolescents should be considered and developed in future research.

The studies used in the current thesis used parental reports of the parent-child relationship and the interparental conflict. Past research supports the importance of the child and adolescent perceptions of these relationships (Harold et al., 1997). Again, this was primarily a result of the difficulty in trying to replicate measures across all of the studies; not all the studies had available child reports of interparental conflict and parent-child hostility, so to remain consistent, parental reports of these measures were used. The final study included a combination of parent and child report of symptoms. The problem with parents reporting on their child's symptoms of psychopathologies are that if they have a hostile relationship with their child, this is unlikely to facilitate open communication and supportive bonds between the child which is important if the child is to spontaneously disclose information (Kerr & Stattin, 2000). This might be more of an issue for internalising problems (e.g. depression) which might be harder to detect without disclosure from the child about how they are feeling.

Finally the current thesis did not examine child gender within the models. Previous research has indicated that gender differences for depressive and aggressive symptoms

start to appear in adolescents, with females being more likely to be affected by internalising problems and males more likely to exhibit externalising behavioural problems (Leadbeater & Kuperminc, 1999; Lewinsohn et al., 1998; Nolen-Hoeksema & Girgus, 1994). There also appear to be gender differences in the effects of interparental conflict and parent-child relationships on the development of child psychopathology. Male children more typically respond with aggression to hostility within the interparental and parent-child relationship and female children are more likely to respond with distress (Carolyn Zahn-Waxler, Cummings, & Iannotti, 1991). Female children might also be more sensitive to the negative effects of parental depression symptoms than boys (Lewis et al., 2013).

In a recent examination of the impact of interparental conflict on children in a longitudinal study, Brock & Kochanska (2015) examined parent-child relationships as a mechanisms that might account for the impact of interparental conflict on children's long-term risk of internalising problems. Increased levels of interparental conflict increased levels of girls internalising problems, whereas consequences of maladaptive marital conflict (e.g. unresolved conflict and lingering tensions) increased internalising problems for boys and girls.

Contextualising findings: implications for practice and policy

The aim of research of this nature is to improve our understanding of the processes that underpin the associations between parental depression and family socialisation with negative child adjustment outcomes. Having a clearer understanding of how these mechanisms operate enables the development of theoretical foundations on which effective interventions that aim to improve children's well-being may be built. The findings of this thesis therefore have potentially significant implications for research and practice. The identification of environmental pathways to the development

of depressive and aggressive symptoms in children and adolescents highlights potential areas that can be targeted for interventions to reduce the development of negative outcomes in children. The studies in this thesis have highlighted three key areas that might impact the development of psychopathology in children; parental depression, interparental conflict and parent-child hostility.

The findings from the studies in this thesis demonstrated that parental depression and interparental conflict can have an indirect effect on child well-being by affecting the parent-child relationship which is supported by findings from previous studies (Schoppe-Sullivan, Schermerhorn, & Cummings, 2007). As parental depression appears to be associated with interparental conflict, and given the robustness of the relationship that has been repeatedly found between parent-child hostility and interparental conflict (Harold et al., 2012), interventions that are targeted at the couple relationship may limit the potential spillover effect by promoting effective communication and parenting skills (Reynolds et al., 2014). Although not a focus on the current thesis, previous research has identified that children might not only be indirectly influenced by interparental conflict (through disruptions in the parent-child relationship) but that their interpretations and comprehension of parental conflict might directly impact on them (Harold et al., 2007).

The findings of the current thesis highlight three primary areas as potential targets for interventions: parental depression, interparental conflict and parent-child relationships. Given the evidence from the current thesis of the relationship between parental depression with interparental conflict, and hostile parent-child relationships with interparental conflict. This overview will primarily focus on examining current interventions targeting the couple relationship to reduce negative outcomes in children

and adolescents. It is hoped that targeting the couple relationship might benefit parents' depressive symptoms and disrupted parent-child relationships.

The current chapter aims to provide an overview of existing literature of interventions where improving parental relationships are a key component. It will be specifically examining programmes which target intact families as these families have been the focus of the current thesis. The approaches examined aim to reflect the key findings of the thesis; addressing hostile parenting practices/parent-child relationships while enhancing couple relationships. As such, many of these programmes focus on the Family Systems Model of understanding developmental outcomes in children which proposes that development is predicted by risks in the quality of the parent-child relationship and the quality of the interparental relationship. The chapter will not include direct discussion of clinical or therapeutic practice or programmes which aim to support families affected by domestic violence as these issues are beyond the scope of this thesis.

Focus on parental depression

Parental depression was associated with increases in interparental conflict for mothers within the community and clinical sample (Study 1), mothers and fathers in both biologically related and biologically unrelated parent-child dyads (Study 2) and for adopted mothers (Study 3). The findings from the study within this thesis therefore support previous evidence that parental depressive symptoms are likely to disrupt marital relations, which then impact parent-child relationships. It seems that an effective area to target for interventions to reduce negative outcomes in children and adolescents would therefore be parental depression. This section will review evidence from interventions that have aimed to target parental depression to reduce negative impact on family relationships.

Given the detrimental impact that parental depression can have on the parent-child relationship (Downey & Coyne, 1990; Martoccio, Brophy-Herb, Maupin, & Robinson, 2016) some interventions have focused on treating and preventing depressive symptoms in children of depressed parents. A review of eight interventions of parental depression which aimed to impact the functioning and well-being of 18 month to 18 year old children of depressed parents highlighted several points for future research and interventions; increasing psychoeducation about parental depression, addressing parenting whilst treating parents for adult depression, focussing on promoting positive parent-child interactions and teaching skills to help children cope with parental depression that they have been exposed to (Boyd & Gillham, 2009). However several limitations were discussed including problems with small sample sizes, lack of replication, and problems with implementing the interventions within the current mental healthcare system.

As marital discord has also been associated with depression in adults (Hops, Perry, & Davis, 1997), interventions which aim to target couple relationship in treating adult depression will now be discussed. It seems that a bidirectional relationship might exist between depressive symptoms and marital discord/interparental conflict. This is supported by longitudinal research that has found relationship discord can predict an increase in depressive symptoms (Beach & Katz, 2003). Furthermore, relationship discord and depressive symptoms often covary (Davila, Karney, Hall, & Bradbury, 2003). Evidence for this relationship promotes a strong case for marital interventions for depressed patients and several interventions have been designed in the form of marital therapy models that have been developed specifically for treating couples with at least one depressed partner (Jacobson, Dobson, Fruzzetti, Schmaling, & Salusky, 1991; Kung, 2000). The marital discord model of depression (Beach, Sandeen, & O'Leary,

1990) proposes that for some individuals who are embroiled in conflict with their partners, this conflict has a role in the development of and or maintenance of their depression. It aims to increase marital support by enhancing couple intimacy, emotional expression, perception of spousal dependability, and direct self-esteem support between partners. Strategic marital therapy (Coyne & DeLongis, 1986) aims to alter the negative interactional cycle between depressed patients and their partners.

Focus on parenting and the couple relationship

The findings from the empirical studies within this thesis support previous research that indicate that when parents are embroiled in conflict with their spouse, they are more likely to be hostile towards their child (Erel & Burman, 1995). Disruptions to parenting skills and parent-child relationships are therefore common when households are marked by marital hostility (Cox et al., 2001). Therefore, parenting intervention programmes that target parenting skills and the parent-child relationship could protect children from the negative effects of interparental conflict. Parenting interventions aimed at reducing parent-child hostility may be especially beneficial for preventing or reducing externalising behaviours such as aggression in children and adolescents, whereas parental hostility within the studies thesis did not appear to be as associated with child and adolescent depressive symptoms. Evidence of the effectiveness of parenting programmes for reducing externalising behaviours in young children has been found (Posthumus, Raaijmakers, Maassen, van Engeland, & Matthys, 2012) which demonstrated sustained benefits for both parenting skills and child behaviour.

Bidirectional effects between child psychopathology and mother-child hostility were observed within three of the four samples used within this thesis; within Study 1, aggression in middle childhood was associated with mother-child hostility in both community and clinical samples. These measures were taken during the same time point

so causality is difficult to determine. Another limitation about this finding was that the mother-child dyads were biologically related in both these samples, the issues for the implications for this have been discussed at length within this thesis. The finding however was also found within a longitudinal measure of this association in a sample of biologically unrelated mother-child dyads (Study 3). Early levels of adopted child psychopathology (18 months) were associated with mother-child hostility (84 months). Child training might therefore be an important component of intervention programmes to attempt to reduce disruptive behaviours in children. Interventions that have combined parent training and child training have shown positive results (Webster-Stratton, Reid, & Hammond, 2004; Webster-Stratton & Hammond, 1997).

In programmes that are designed to improve parenting practices, neglecting to address couple conflict appears to provide less long term benefits than those which take the importance of promoting positive relations between parents into consideration (Webster-Stratton & Hammond, 1999). Cowan et al. (2005)have repeatedly demonstrated this through their assessment of programmes which are purely aimed at improving parenting skills compared with ones which incorporate interventions aimed at reducing conflict and improving communication skills in the interparental relationship.

A meta-analysis of prevention programmes with a focus on parenting yielded no statistically significant results for improving child outcomes (Durlak & Wells, 1997).

Parenting intervention programmes which have included components aimed at supporting and improving the interparental relationship appear to offer more promising findings (Cowan & Cowan, 2002; Reynolds et al., 2014). An example of this can also be viewed with the 'Incredible Years' parenting course; couples who completed the ADVANCE version of the course, which included a focus on improving communication

and problem solving skills, were reported as not only exhibiting improvements in their parenting skills but communication effectiveness within the marital relationship was also improved (Webster-Stratton, 1994). Couples who received the BASIC training did not demonstrate these favourable results. The 'School Children & their Families' programme randomly allocated 100 couples to either a control condition, a couple discussion group with the couple relationship issues as a focus or a couple discussion group with parenting issues as a focus. Parent-child relationships were improved in both of the discussion groups for couples discussing either parenting or couple issues. That is, couples who were exposed to the condition that did not specifically aim to target parenting issues had improved parent-child relationships as a by-product of improved interparental relationships and reduced levels of interparental conflict (Cowan et al., 2005). Improvements in family relationships appeared to persist 6 and 10 years after the initial intervention (Cowan et al., 2011). The findings from the 'Incredible Years' and 'School Children & their Families' programmes highlight that programmes which target interparental relationships can benefit the whole family with long lasting effects (Webster-Stratton & Reid, 2003).

A range of programmes that cultivate enhanced parent and family relationships might improve couple relationships by improving communication and reducing conflict, whereas other programmes might be more specifically focused on reducing interparental conflict (Reynolds et al., 2014). These interventions provide training for conflict and anger management whilst educating parents on the impact that interparental conflict can have on children (Cummings & Davies, 2002). The association between maternal depression, interparental relationships and parent-child relationship should be considered when reviewing the efficacy of interventions. Research suggests that parent

interventions which are specifically aimed at reducing maternal hostility may have an additional benefit of reducing maternal depression symptoms (Barlow et al., 2002).

Future directions of research

The current thesis outlined specific processes and mechanisms by which parental depressive symptoms and hostile family relationships are associated with the development of internalising and externalising in children and adolescents. Future research could formally examine indirect pathways by which parental depressive symptoms might predict increases in child psychopathology. Another issue with the current set of studies was that test of mean differences between specific measures and samples were not always possible (e.g. different measures used for different variables across the studies). Future research could examine and compare mean differences across different groups. For example, the current thesis did not examine if levels of interparental conflict were higher for community or high-risk samples, or if interparental conflict levels were any different for IVF and adoptive families. The spread of variance across some of the samples has potential limitations for the interpretation of the findings. For Study 1 for example, there is a large amount of variance in the levels of maternal depression within the clinical sample. Future examinations of the same data sets could control for this by separating individuals within the same samples that have high or low levels of current depressive symptoms.

There are covariates that were not measured within the current thesis that could have influenced the development of depressive and aggressive symptoms which should be considered in future research. Socioeconomic status of families has been previously identified as a risk factor in the development of negative outcomes in children. In a study examining the extent to which mother well-being and family economic circumstances in infancy were associated with children's behavioural problems at age 3,

researchers established that economic deprivation and maternal depression separately and collectively diminished the emotional well-being of children (Kiernan & Huerta, 2008). Peer influences have also been associated with the development of psychopathology in children and adolescents; peer rejection has been associated with both the development of internalising and externalising problems (Laird, Jordan, Dodge, Pettit & Bates, 2001; Split, Lear, Leflot, Onghena & Colpin, 2014).

The family environment

Parent-child hostility was associated with child aggressive symptoms across all developmental stages (Study 3- early childhood; Study 2- middle childhood; Study 3- early adolescence) which is why interventions have been reviewed to reduce the presence of behavioural problems in children by targeting parenting and the parent-child relationship. However, parent-child hostility within the studies for the thesis was rarely associated with child depressive symptoms. Previous interventions have highlighted the effectiveness of parenting interventions in reducing externalising problems (e.g. conduct disorders) in children and adolescents (Woolfenden, Williams, & Peat, 2002) but seem to not be as effective when trying to reduce the presence of depressive symptoms within the same age group. Therefore future research should consider exploring aspects of the family environment which might be more relevant to the development of depressive symptoms given the associations found within the present studies (Gilman, Kawachi, Fitzmaurice, & Buka, 2002).

Other at-risk groups

The studies included in the current thesis used parental depression as a measure of risk for the development of psychopathology and examined how these risk factors might also be associated with family processes (specifically interparental conflict and parent-child hostility). Future research could expand on this by examining children and

adolescents exposed to different or additional risks. This could include other measures of parental psychopathology and perhaps a deeper exploration of paternal psychopathology. For example, Study 1 used a sample of adolescents living with a mother who had clinical depression. A natural extension to this could be to consider the associations between family relationships and child outcomes for adolescents exposed to paternal depression. Additionally, Study 3 used birth mother depression as a risk factor for the development of negative child outcomes, birth father depression should also be examined within this theoretical framework.

Impact on child outcomes

The current study found associations between parent-child hostility and aggressive symptoms in children and adolescents (and depressive symptoms for high-risk adolescents living with a mother who experienced clinical depression). Other child outcomes that are associated with the pathways for negative outcomes identified within the present thesis should also be examined. For example, the impact of parental depressive symptoms, interparental conflict and parent-child hostility on childrens' social skills and academic ability could be examined.

Genetically informative and genetically sensitive research design

The current thesis utilised a genetically informative (IVF) and a genetically sensitive (EGDS, adoption) research design. It was able to highlight that parent-child hostility is an environmental risk factor for the development of externalising problems, such as aggressive behaviours in children and adolescents. These studies could be developed in a few ways to further explore some of the findings from the current study. The genetically sensitive research designs used were not conducted with high-risk groups of children and adolescents as these designs are typically community based. Future studies might expand on the IVF design to obtain longitudinal information and

information of the donor when parents who rear their children are genetically unrelated (Harold et al., 2013). As EGDS continues to collect data from the families who have participated in the study, the development of psychopathology can be examined as the children transition from middle childhood to early adolescence to see if the pattern of findings are similar as the child goes through different developmental stages.

The premise of identifying environmental mechanisms is to identify potentially modifiable areas that could be targeted with interventions to reduce negative outcomes. It should be noted however, that just because a genetic association has been identified, this does not necessary preclude it from being changeable via interventions. For complex traits, which can include symptoms and behaviours associated with psychopathology, Haworth & Davies (2014) acknowledge that heritability does not necessarily mean that a trait is unchangeable. Robust empirical evidence has demonstrated that genetic influences can change with age (Deary et al., 2012), context (Blakemore & Mills, 2014) and in response to behavioural and drug interventions (Bakermans-Kranenburg & Van Ijzendoom, 2015). Being able to identify and separate genetic and environmental causes, and establishing how they might interact can provide researchers with the opportunity to develop effective environmental interventions (Haworth & Davies, 2014).

Conclusion

The presence of depressive and aggressive symptoms in children and adolescents can be associated with multiple risk factors within the family environment. Parental depressive symptoms, and more consistently maternal depressive symptoms, are associated with marital discord which is associated with disruptions within the parent-child relationship and parenting. These disruptions might manifest as increased incidences of hostility expressed from parents towards their children. Parent-child

hostility was an important risk factor for the development of aggressive symptoms in children and adolescents across a range of developmental stages. For children living with clinically depressed mothers, mother-child hostility was also associated with elevated depressive symptoms. The association between parent-child hostility and child aggressive symptoms was identified as an environmental pathway as these associations were present in families where children were genetically unrelated from the parents who were rearing them. An additional finding was that early levels of psychopathology (depressive and aggressive symptoms) appear to have a bidirectional association with parent-child hostility as within some of the studies depressive and aggressive symptoms predicted increased levels of mother-child hostility. The current thesis therefore demonstrates the importance of considering the associations between multiple environmental pathways within the family with the development of psychopathology in children and adolescents.

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