

**Paternal psychological distress and child emotional, behavioural,  
and cognitive outcomes: Findings from the Millennium Cohort  
Study**

Thesis submitted for the degree of Doctor of Philosophy

UCL Institute of Education

Maria Sifaki

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## **Declaration**

I, Maria Sifaki, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

## **Abstract**

**Background:** Recent research studies indicate that fathers' psychological distress affects child emotional, behavioural, and cognitive development. Parenting practices are suggested to account for these pathways. Despite the increasing understanding of the role of paternal psychological distress, important questions remain, especially in relation to adolescent outcomes. Therefore, the aim of this thesis is to explore further the relationship between paternal psychological distress and child development, from early childhood to adolescence.

**Methods:** The thesis utilizes secondary data from the Millennium Cohort Study. It involves three research aims: i) Using cross-lagged models, to investigate the bi-directional associations between fathers' psychological distress and child emotional and behavioural difficulties, across child ages 3-14; ii) Through group-based trajectory modelling, to identify trajectories of paternal psychological distress across ages 3-11 and subsequently, using regression modelling, to examine how these trajectories are linked to offspring cognitive functioning at age 11; iii) Finally, through path analysis, to assess if fathers' psychological distress at child ages 3, 7, and 11, predicts offspring engagement in health risk behaviours (smoking, alcohol use, binge drinking, and sexual activity) at age 14.

**Results:** Findings show that paternal psychological distress predicts child emotional and behavioural difficulties, and that child difficulties also predict paternal psychological distress, though to a less extent. Moreover, reciprocal interactions occur between fathers' distress and offspring peer difficulties, across ages 11 and 14. Additionally, when it comes to cognitive functioning, paternal psychological distress is associated with less impulsive decision-making. Last, some evidence

emerged that, for intact families, fathers' distress is linked to a decreased likelihood of adolescent alcohol and binge drinking.

**Conclusions:** This thesis shows that paternal psychological distress influences child outcomes. Clinical and policy implications arise, as results suggest that promoting the mental well-being of fathers can be beneficial for child development.

## **Impact Statement**

This thesis explored the relationship between fathers' psychological distress and child emotional, behavioural, and cognitive outcomes. The thesis also examined the links between fathers' psychological distress and adolescent engagement in health risk behaviours, including smoking, alcohol use, and sexual activity. Data from a large and nationally representative cohort were used, looking at children from the age of 3 to the age of 14. Findings have practical implications while also highlighting some directions for future research.

Specifically, results showed that paternal distress does negatively influence child development. At the same time child difficulties can deteriorate paternal well-being. For some of the paths identified, parenting practices and marital conflict were found to play a causal role. These conclusions indicate that, when working with either children or adults, clinicians should consider the well-being of all family members, and try to improve the interactions that take place between them, to hinder adverse effects. For instance, it may be the case that by preventing distressed fathers from being hostile towards their children, the development of emotional and behavioural difficulties is to some extent restricted. Moreover, conclusions underline that policies promoting the healthy development of children should consider the mental health of their caregivers, including their fathers.

In terms of future research, one issue that arises and could be addressed is the causal mechanisms operating between paternal distress and adolescent outcomes. This thesis does look into causal mechanisms, though only for young children. It is unclear if the same pathways are in place as children grow older. In addition, findings showed that paternal distress is associated with less adolescent risk

engagement. This was an unexpected result; it would be useful if future studies evaluated the reasons why this might be the case. Last, this thesis has looked at the general population. However, it is not known how the paths that emerged may differ for vulnerable or disadvantaged groups (i.e., ethnic minorities); this is another research direction that could be investigated by scholars.

Part of this thesis' findings have already been disseminated in the academic journal *European Child & Adolescent Psychiatry* (Sifaki et al., 2021). These findings have also been presented in an academic poster, at the UCL Institute of Education's doctoral conference, in 2020. In the future, we plan to disseminate more of this thesis' outputs in the form of academic papers, as this will help inform both the research community and the public on the research questions that were explored, and the conclusions reached.

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# Table of Contents

Declaration.....	2
Abstract.....	3-4
Impact statement.....	5-6
Acknowledgements.....	7
Table of contents.....	8-12
List of Tables.....	13-14
List of Figures.....	14
List of abbreviations.....	15-16
<b>Chapter 1: Introduction.....</b>	<b>17-20</b>
1.1. The role of fathers and thesis aims.....	17-19
1.2. Thesis outline.....	19-20
<b>Chapter 2: The relationship between paternal psychological distress and child outcomes.....</b>	<b>21-41</b>
2.1. Paternal psychological distress and its prevalence.....	21
2.2 The link between paternal psychological distress and child outcomes.....	21-32
2.2.1. Child internalising difficulties.....	21-23
2.2.2. Child externalising difficulties.....	23-25
2.2.3. Child cognitive abilities.....	25-26
2.2.4. Child executive functions.....	26-27
2.2.5. Child decision-making.....	27-29
2.2.6. Child health risk behaviours.....	29-32
2.3. Reasons why paternal distress and child outcomes are linked.....	32-42
2.3.1. Bronfenbrenner’s bio-ecological model.....	33
2.3.2. Transactional family dynamics theory.....	33-34
2.3.3. Bandura’s social-learning theory.....	34-35



2.3.4. The mediating role of marital quality.....	35-37
2.3.5. The mediating role of paternal warmth.....	37-39
2.3.6. The mediating role of paternal hostility.....	40
2.3.7. The mediating role of paternal involvement.....	40-42
<b>Chapter 3: Data and measures.....</b>	<b>43-49</b>
3.1. Data.....	43
3.2. Measures.....	43-49
3.2.1. Paternal measures.....	44-46
3.2.2. Child measures.....	46-49
<b>Chapter 4: Reciprocal links between paternal distress and child internalising and externalising difficulties (research aim 1).....</b>	<b>50-88</b>
4.1. The effects of child difficulties on paternal distress.....	51-52
4.2. Mediating pathways from child difficulties to paternal distress.....	52-53
4.3. Bi-directional links between father distress and child difficulties.....	53-56
4.4. The role of child's gender.....	56-57
4.5. Conclusions and the present study.....	57-59
4.6. Methods.....	59-63
4.6.1. Participants.....	59
4.6.2. Measures.....	59-60
4.6.3. Analytic strategy.....	61-63
4.7. Results.....	63-76
4.7.1. Descriptive statistics.....	63-65
4.7.2. Correlations.....	65
4.7.3 SEM results: Full analytic sample.....	65-68
4.7.4. Moderation.....	69
4.7.5 First Sensitivity analysis (5412 cases).....	69-71
4.7.6. Second sensitivity analysis (5258 cases).....	72-73
4.7.7. Mediation analysis.....	74-76

4.8. Discussion.....	76-88
4.8.1. Father effects.....	77-78
4.8.2. Child effects.....	78
4.8.3. Reciprocal links.....	78-80
4.8.4. Moderation results.....	80-81
4.8.5. Mediation results.....	81-83
4.8.6. Sensitivity analysis.....	83-85
4.8.7. Limitations and directions for future research.....	85-87
4.8.8. Conclusions and implications.....	87-88
<b>Chapter 5: Trajectories of paternal psychological distress and cognitive functioning in early adolescence (research aim 2).....</b>	<b>89-123</b>
5.1. Cognitive functioning in early adolescence.....	90-91
5.2. The role of paternal distress.....	91-92
5.3. The role of maternal distress.....	92
5.4. Trajectory modelling.....	93
5.5. Trajectories of paternal distress.....	93-94
5.6. Trajectories of maternal distress.....	94-95
5.7. Gender differences in cognitive functioning.....	95
5.8. Individual and family factors related to cognitive functioning.....	96
5.9. Conclusions and the present study.....	96-97
5.10. Methods.....	97-102
5.10.1. Participants.....	97-98
5.10.2. Measures.....	98-100
5.10.3. Analytic strategy.....	100-102
5.11. Results.....	102-112
5.11.1. Descriptive statistics.....	102-104
5.11.2. Correlations.....	105
5.11.3. Modelling parallel trajectories for mothers and fathers using GBTM.....	105-107

5.11.4. Multiple regression results.....	107-109
5.11.5. Moderation by gender.....	110
5.11.6. Sensitivity analysis.....	110-112
5.12. Discussion.....	114-122
5.12.1. Trajectories of paternal and maternal distress.....	114
5.12.2. Verbal ability.....	114-115
5.12.3. Visuospatial working memory.....	115-116
5.12.4. Decision-making.....	116-119
5.12.5. Sensitivity analysis.....	119
5.12.6. Gender differences.....	119-120
5.12.7. Limitations.....	120-121
5.12.8. Conclusions and implications.....	121-122

**Chapter 6: Paths from paternal psychological distress to adolescent health risk**

<b>engagement (research aim 3).....</b>	<b>123-155</b>
6.1. Maternal distress and adolescent health risk engagement.....	124-126
6.2. Reasons why paternal distress and adolescent health risk engagement are linked.....	126-127
6.3. Gender differences.....	127-128
6.4. Conclusions and the present study.....	128-129
6.5. Methods.....	129-133
6.5.1. Participants.....	129-130
6.5.2. Measures.....	130-131
6.5.3. Analytic strategy.....	131-133
6.6. Results.....	133-146
6.6.1. Descriptive statistics.....	134-135
6.6.2. Path analysis results.....	136-143
6.6.2.1. Smoking results.....	136-138

6.6.2.2. Alcohol drinking results.....	138-139
6.6.2.3. Binge drinking results.....	139-141
6.6.2.4. Sexual activity results.....	141-143
6.7. Sensitivity analysis.....	143-146
6.7.1. Smoking results.....	143
6.7.2. Alcohol drinking results.....	143-144
6.7.3. Binge drinking results.....	145
6.7.4. Sexual activity results.....	145-146
6.8. Trajectory modelling results.....	146
6.9. Discussion.....	147-155
6.9.1. Smoking.....	148
6.9.2. Alcohol drinking.....	148-150
6.9.3. Binge drinking.....	150-151
6.9.4. Sexual activity.....	151
6.9.5. The links between covariates and adolescent health risk behaviours.....	151-153
6.9.6. Links with paternal and maternal trajectories.....	153
6.9.7. Limitations.....	154
6.9.8. Conclusions and implications.....	154-155
<b>Chapter 7: Discussion.....</b>	<b>156-160</b>
7.1. Strengths and implications.....	156-158
7.2. Limitations and directions for future research.....	158-160
7.3. Conclusions.....	160
<b>References.....</b>	<b>161-229</b>
<b>Appendix 1.....</b>	<b>230-232</b>
<b>Appendix 2.....</b>	<b>233-235</b>

## List of Tables

<b>Table 1:</b> Description of the MCS sweeps.....	43
<b>Table 2:</b> Description of the SDQ difficulties scales.....	46
<b>Table 3:</b> Descriptives of the analytic and non-analytic samples (Research aim 1).....	64-66
<b>Table 4:</b> Cross-lagged model results.....	68
<b>Table 5:</b> Sensitivity analysis results (5412 cases).....	71
<b>Table 6:</b> Sensitivity analysis results (5258 cases).....	73
<b>Table 7:</b> Direct, indirect and total effects between child difficulties and paternal distress....	76
<b>Table 8:</b> Descriptives of the analytic and non-analytic samples (Research aim 2).....	103-104
<b>Table 9:</b> Multiple regression results (coefficients and standard errors) for the unadjusted models.....	109
<b>Table 10:</b> Multiple regression results (coefficients and standard errors) for the adjusted models.....	109
<b>Table 11:</b> Multiple regression results (coefficients and standard errors) for the unadjusted models (sensitivity analysis).....	112
<b>Table 12:</b> Multiple regression results (coefficients and standard errors) for the adjusted models (sensitivity analysis).....	112
<b>Table 13:</b> Descriptives of the analytic and non-analytic samples (Research aim 3)....	134-135
<b>Table 14:</b> Gender differences for each of the risky behaviour outcomes at age 14.....	135
<b>Table 15:</b> Smoking results for the unadjusted models.....	137
<b>Table 16:</b> Smoking results for the adjusted models.....	137-138
<b>Table 17:</b> Alcohol drinking results for the unadjusted models.....	138-139
<b>Table 18:</b> Alcohol drinking results for the adjusted models.....	139
<b>Table 19:</b> Binge drinking results for the unadjusted models.....	140-141
<b>Table 20:</b> Binge drinking results for the unadjusted models.....	141
<b>Table 21:</b> Sexual activity results for the unadjusted models.....	142
<b>Table 22:</b> Sexual activity results for the unadjusted models.....	142-143

<b>Table 23:</b> Smoking results for the adjusted models, sensitivity analysis.....	143-144
<b>Table 24:</b> Alcohol results for the adjusted models, sensitivity analysis.....	144
<b>Table 25:</b> Binge drinking results for the adjusted models, sensitivity analysis.....	145
<b>Table 26:</b> Sexual activity results for the adjusted models, sensitivity analysis.....	146

## List of Figures

<b>Figure 1:</b> Cross-lagged model for paternal distress and child problem behaviour.....	63
<b>Figure 2:</b> Paternal distress trajectories, child ages 3-11.....	106
<b>Figure 3:</b> Maternal distress trajectories, child ages 3-11.....	107
<b>Figure 4:</b> Path analysis model for paternal and maternal psychological distress.....	132

## List of abbreviations

**ADHD:** Attention-Deficit Hyperactivity Disorder

**AIC:** Akaike Information Criterion

**AvePP:** Average Posterior Probabilities

**BAS:** British Abilities Scales

**BIC:** Bayesian Information Criterion

**CANTAB:** Cambridge Neuropsychological Test Automated Battery

**CFI:** Comparative Fit Index

**CGT:** Cambridge Gambling Task

**CPRS:** Pianta's Child-Parent Relationship Scale

**FIML:** Full Information Maximum Likelihood

**GBTM:** Group-Based Trajectory Modelling

**GRIM:** Golombok Rust Inventory of Mental State

**GSEM:** Generalized Structural Equation Modelling

**IOE:** Institute of Education

**K-6:** Kessler Psychological Distress Scale

**MCS:** Millennium Cohort Study

**MICE:** Multiple Imputation by Chained Equations

**NCS-A:** National Comorbidity Survey Replication Adolescent Supplement

**NHS:** National Health System

**NSDUH:** National Surveys on Drug Use and Health

**NVQ:** National Vocational Qualifications

**ODD:** Oppositional Defiant Disorder

**OECD:** Organization for Economic Co-operation and Development

**RMSEA:** Root Mean Square of Approximation

**SD:** Standard Deviation

**SDQ:** Strength Difficulties Questionnaire

**SE:** Standard Error

**SEM:** Structural Equation Modelling

**SES:** Socio-Economic Status

**STRS:** Student-Teacher Relationship Scale

**TLI:** Tucker-Lewis Index

**UK:** United Kingdom

**VIF:** Variance Inflation Factor



## **Chapter 1: Introduction**

This chapter will discuss the role of fathers in today's family environment and how their role has changed over time. It will also present the evidence regarding paternal mental health and psychological distress. Finally, it will present the outline of the current thesis.

### **1.1. The role of fathers and thesis aims**

In the traditional family model, mothers are the main child caregivers. Fathers, on the other hand, are the 'breadwinners', responsible for financially supporting the family. Their involvement in child upbringing is limited, and concerns mainly disciplining their children (Cabrera et al., 2000; Lamb, 2004).

Nowadays, due to societal changes and women's increased participation in the workforce, family roles are not the same. Fathers are expected to have more parenting responsibilities and provide day-to-day care for their children (Lamb, 2004). Indeed, from 1970 to 2010, it is estimated that the amount of daily time UK fathers spend with their children has doubled (Altintas & Sullivan, 2017). Greater paternal involvement means that fathers' impact on their offspring is likely to be stronger than it used to be (Opondo et al., 2016). As a result, research interest regarding their role and how they shape their children's development has significantly risen (Cassano et al., 2006).

One important parameter to consider is the fathers' mental health. Research on mothers has documented that maternal mental health difficulties have severe, adverse effects on child development, including emotional, behavioural, social, cognitive, academic, and health outcomes (Dahlen, 2016; Goodman et al., 2011;

Mughal et al., 2019; Oh et al., 2020; van der Waerden et al., 2017; Wickham et al., 2015). Effects are also shown to be long-term; for instance, maternal perinatal depression has been associated with offspring depression in adolescence and adulthood (Tirumalaraju et al., 2020).

Taking into account the importance of maternal mental health, and the greater role fathers today have on their children's lives, it becomes crucial to explore what the influence of paternal mental health might be. Such findings could contribute to informing policies and practices promoting the wellbeing of families, by helping identify vulnerable youth and understand family processes.

To measure paternal mental health difficulties, different studies utilized different approaches. Specifically, while some examined paternal clinical depression or depressive symptoms (Paulson, Keefe, & Leiferman, 2009), others measured psychological distress. Psychological distress is a useful construct in assessing the general wellbeing of the population. It can involve symptoms of depression or anxiety, that fluctuate from mild to severe (Drapeau et al., 2010). High levels of psychological distress could mean a psychiatric diagnosis; however, even lower levels can imply that the individual is experiencing negative emotions that interfere with their daily lives (Drapeau et al., 2010). The present thesis will focus on fathers' psychological distress and will use the terms depression or depressive symptoms when referring to past studies that assessed those.

This thesis will aim to expand the current knowledge regarding the role of fathers' psychological distress, by further investigating links with child outcomes and addressing existing research gaps. Specifically, the thesis aims are:

1. To investigate the reciprocal relationships over time between paternal psychological distress and child emotional and behavioural difficulties. In particular, the thesis will examine, from offspring age 3 to 14, whether transactional associations occur between fathers' psychological distress and children's difficulties, or whether only one-way influences take place (either from fathers to children or vice versa). Moreover, the moderating effects of gender will be explored, as well as the role of potential mediators, to test theory about why and how these relationships exist.
2. To investigate how trajectories of paternal psychological distress across childhood (ages 3 to 11) affect cognitive functioning in early adolescence (age 11), including verbal abilities, visuospatial working memory, and decision-making. It will also be explored if these relationships are moderated by the child's gender.
3. To investigate how paternal psychological distress at child ages 3, 7, and 11 relates to adolescent (age 14) health risk behaviours, including smoking, alcohol drinking, binge drinking, and sexual activity. Due to the different rates of engagement in risky behaviours for boys and girls, paths for each gender will be run separately.

To address these research aims, secondary longitudinal data from the UK's Millennium Cohort Study (MCS) will be analyzed. Data from sweeps 2-6 will be used, when the children were aged 3, 5, 7, 11, and 14 years respectively.

## **1.2. Thesis outline**

The following chapter of this thesis will present a literature review on the relationship between paternal psychological distress and child outcomes. This will involve an

examination of the current research evidence and the potential reasons the two might be linked. The next chapter will describe the Millennium Cohort Study (MCS) data and the main measures that were used for this thesis. Subsequently, the following three chapters will each present an empirical piece of work, corresponding to the three thesis aims, respectively. These chapters will provide an overview of the relevant research literature, the methods used for each analysis, the results, and a discussion. The final chapter will involve an overall evaluation, including a summary of the findings, limitations, implications, and directions for future research.

## **Chapter 2: The relationship between paternal psychological distress and child outcomes**

### **2.1. Paternal psychological distress and its prevalence**

Nowadays, an increasing amount of research is exploring the role of fathers in their children's lives and how their well-being can shape child development. Compared to maternal depression, the rates of paternal depression are shown to be lower (Escribà-Agüir & Artazcoz, 2011; Kiviruusu et al., 2020). Nevertheless, it is estimated that approximately 8%-24% of fathers will experience depression at some point, while the average for the male adult population is 4% (Cameron et al., 2016; Cui et al., 2021; Paulson & Bazemore, 2010). These findings suggest that men who become parents face increased risks of developing mental health difficulties. The periods of pregnancy and postpartum are those of the highest vulnerability (Paulson & Bazemore, 2010; Philpott et al., 2017; Rao et al., 2020), and fathers who develop depressive symptoms during that time are likely to continue to present difficulties during later years (Nath et al., 2016a). Prior history of psychiatric disorders, maternal mental health difficulties, unemployment, low household income, marital tension, and parenting stress are all shown to be important risk factors (Chhabra, McDermott, & Li, 2020; Escribà-Agüir & Artazcoz, 2011; Kiviruusu et al., 2020; Nath et al., 2016a; Nishigori et al., 2020; Recto & Champion, 2020; Tambelli et al., 2019).

### **2.2. The link between paternal psychological distress and child outcomes**

#### **2.2.1. Child internalising difficulties**

Internalising difficulties refer to emotional difficulties developed by children, including depression and anxiety symptoms, as well as peer-related difficulties, such as withdrawal and loneliness (Achenbach & Edelbrock, 1979). Youth experiencing

internalising difficulties is at higher risk for delinquency, substance abuse, academic underachievement, as well as mental health problems in adulthood (Isaksson et al., 2020; McLeod et al., 2016; Pedersen et al., 2019). Internalising difficulties are present in both boys and girls; however, during adolescence they become more prevalent in females (Gutman & McMaster, 2020). Maternal psychopathology, low family socio-economic status, and stressful life events are demonstrated to be important risk factors (Côté et al., 2018; Hosokawa & Katsura, 2017; March-Llanes et al., 2017).

Recently, research studies have shown that paternal psychological distress can also be a risk factor for child internalising difficulties (Dietz et al., 2009; Kahn, Brand & Whitaker, 2004; Psychogiou, Russell, & Owens, 2020; Ramchandani & Psychogiou, 2009; Weitzman, Rosenthal & Liu, 2011), including emotional (Aunola et al., 2015; Ayano et al., 2021; Dette-Hagenmeyer & Reichle, 2014; Fletcher et al., 2011; Flouri, Sarmadi, & Francesconi, 2019; Giallo et al., 2014; Gulenc et al., 2018; Gutierrez-Galve et al., 2019; Kvalevaag, et al., 2013; Letourneau et al., 2019; Lewis et al., 2017; Malmberg & Flouri, 2011; Reeb & Conger, 2009; Reeb, Conger & Wu, 2010; Reeb et al., 2015; Smith et al., 2013) and social difficulties (Dette-Hagenmeyer & Reichle, 2014; Fletcher et al., 2011; Flouri et al., 2019; Giallo et al., 2014; Gulenc et al., 2018; Kvalevaag, et al., 2013; Letourneau et al., 2019; Smith et al., 2013). Paternal effects appear to be present during preschool years (Aunola et al., 2015; Fletcher et al., 2011; Flouri et al., 2019; Giallo et al., 2014; Gulenc et al., 2018; Kvalevaag, et al., 2013; Letourneau et al., 2019; Malmberg & Flouri, 2011; Smith et al., 2013), middle childhood (Dette-Hagenmeyer & Reichle, 2014), adolescence (Ayano et al., 2021; Flouri et al., 2019; Lewis et al., 2017; Reeb & Conger, 2009; Reeb et al., 2015) and even young adulthood (Gutierrez-Galve et al., 2019; Reeb et

al., 2015). Notably, paternal effects occurred even when controlling for family and socio-demographic factors, such as maternal distress and poverty. What is more, most research has used longitudinal, community-sample designs, demonstrating the long-term influence of paternal distress in the general population (Fletcher et al., 2011; Flouri et al., 2019; Giallo et al., 2014; Gutierrez-Galve et al., 2019; Kvalevaag, et al., 2013; Lewis et al., 2017; Smith et al., 2013).

Meta-analytic studies have also yielded supportive conclusions about the role of fathers' distress on children's emotional and social well-being. Specifically, Connell & Goodman (2002) argued that paternal mental health problems (including alcohol abuse and schizophrenia) predict offspring internalising difficulties from early years to adolescence, with paths being the strongest in adolescence. Kane & Garber (2004) also reported that paternal depression is linked to offspring internalising difficulties, particularly for community samples. More recently, Cui et al., (2020) claimed that paternal perinatal depression affects emotional and social development in the early years.

### **2.2.2. Child externalising difficulties**

Youth externalising difficulties include delinquency, aggression, and hyperactivity (Achenbach & Edelbrock, 1979), and are more frequent in males than females (Chi & Cui, 2020), particularly during childhood (Bongers et al., 2004). As with internalising difficulties, externalising difficulties are associated with a range of adverse and long-term outcomes (Cox et al., 2021; Kremer et al., 2016; Reef et al., 2011). Family and environmental factors, such as maternal depression (Carlone & Milan, 2021) are shown to predict their emergence (Kjeldsen et al., 2021).

Several studies have investigated the relationship between paternal distress and offspring externalising difficulties (Dietz et al., 2009; Kahn et al., 2004; Kvalevaag, et al., 2013; Psychogiou et al., 2020; Ramchandani & Psychogiou, 2009; Weijers et al., 2018; Weitzman, Rosenthal & Liu, 2011). Their findings suggest that fathers' distress is linked to conduct (Dette-Hagenmeyer & Reichle, 2014; Fletcher et al., 2011; Flouri et al., 2019; Giallo et al., 2014; Gulenc et al., 2018; Gutierrez-Galve et al., 2015; Ramchandani et al., 2005, 2008) as well hyperactivity difficulties (Chen et al., 2020; Dette-Hagenmeyer & Reichley, 2014; Fletcher et al., 2011; Flouri et al., 2019; Giallo et al., 2014; Gulenc et al., 2018; Gutierrez-Galve et al., 2015; Ramchandani et al., 2005). Meta-analytic studies have also showed significant effects (Cheung & Theule 2019a; Cui et al., 2020; Kane & Garber, 2004).

Alike the research looking into internalising difficulties, most of the afore-mentioned studies have applied longitudinal research designs to general population samples. Furthermore, the majority concern the early years or elementary school children; while the studies exploring these paths in adolescence are considerably more limited (Flouri et al., 2019; Weijers et al., 2018; Weitzman et al., 2011).

Additionally, there has been a narrow amount of research examining clinically referred children (Herbert et al., 2013; Weijers et al., 2018). Particularly, Herbert et al. (2013) conducted a 3-year longitudinal study, in which they investigated the impact of paternal depressive symptoms on preschoolers with severe externalising problems, including ADHD and ODD diagnosis. Results indicate that fathers' difficulties were related to different aspects of those children's development, such as emotional and behavioural adjustment, as well as social functioning. Similar conclusions were reached by Weijers et al. (2018), who argued that paternal distress



was strongly associated with externalising symptoms in offspring aged 6-20 years old, diagnosed with various psychiatric disorders.

### **2.2.3. Child cognitive abilities**

Cognitive abilities are defined as ‘the mental actions or processes of acquiring knowledge and understanding through thought, experience, and the senses’ (Davis, Pitchford, & Limback, 2011). Higher cognitive abilities in childhood are linked to many positive outcomes in later life, such as better mental health and higher academic attainment (Deary et al., 2007; Gale et al., 2010). The family environment, including socio-economic status, parenting, marital discord, and maternal mental health, is shown to play an important role in the development of offspring cognition (Kiernan & Huerta, 2008; van Der Waerden et al., 2017). Nonetheless, only a limited number of studies so far have evaluated the role of fathers’ psychological distress.

To begin with, Cheng et al., (2016) in a community sample of 5350 families, investigated the influence of maternal and paternal depression, measured when the child was 9 months, on child cognitive outcomes at 24 months. The types of outcomes examined were memory, problem-solving, comprehension, expressive and receptive language skills. The researchers accounted for several covariates, such as premature birth, which was the case for approximately 10% of the sample. Results revealed that paternal depression was related to lower cognitive ability, irrespective of confounding factors. Similarly, Paulson et al., (2009) using the same source of data, concluded that paternal depression at 9 months predicted poorer child expressive language skills at age 24 months. Lastly, Fredriksen et al., (2019) in a Norwegian community sample of approximately 1000 families, argued that fathers’

elevated depressive symptoms, measured at 6 weeks, 6 months, and 12 months, led to receptive and expressive language problems at 18 months.

A number of cross-sectional studies have also been conducted, with the results being ambiguous. In particular, Mensah & Kiernan (2010) using data from the Millennium Cohort Study, explored the impact of maternal and paternal psychological distress on child outcomes at age 5. Three types of outcomes were assessed: communication/language, math ability, and socio-emotional skills. After adjusting for covariates, only the paths for maternal distress remained significant. Conversely, Dave et al., (2009) offer some support for the association between paternal depression and off-spring cognition. Based on data from 248 families in England, the researchers concluded that children whose fathers had major depression were 9 times more likely to be referred to a professional for speech and language problems, compared to children who had mentally healthy fathers. Nuttall et al., (2019) also found that paternal depressive symptoms were linked to preschoolers' low literacy skills.

Overall, the existing studies looking into the relationship between paternal distress and offspring cognition are limited and focused on the early years. Further research is needed to explore whether paternal effects occur during middle childhood or adolescence.

#### **2.2.4. Child executive functions**

Executive functions refer to a set of cognitive processes, such as working memory, inhibitory control, interference control and cognitive flexibility, that enable individuals to undertake goal-oriented behaviour (Diamond, 2013; Best & Miller, 2010; Miyake & Friedman, 2012). They are considered to emerge during the first year of life and

continue to develop until early adulthood (Best & Miller, 2010; Diamond, 2013; Isbell et al., 2015). Executive functions play a significant part in child development, as they relate to mental health, self-regulated behaviour, school readiness, academic performance and social competence (Best, Miller, & Naglieri, 2011; Bierman et al., 2008; Dias et al., 2017; Pearson et al., 2016; Razza & Blair, 2009; Riggs, Blair & Greenberg, 2004; Roman, Ensor & Hughes, 2016).

There is a limited number of studies that have evaluated the impact of paternal mental health on children's executive functions (Belleau et al., 2013; Halse et al., 2019; Micco et al., 2009). Specifically, Belleau et al., (2013) and Micco et al., (2009) assessed whether offspring of parents (both fathers and mothers) with psychiatric disorders have lower executive function skills, compared to their peers. Both studies were cross-sectional and looked at children from early childhood to late adolescence. Another study, Halse et al., (2019), investigated the longitudinal influence of paternal depression and anxiety symptoms on primary school children, within the general population. Supportive evidence for the role of parental mental health was found by Belleau et al., (2013); however, Halse et al., (2019), as well as Micco et al., (2009), failed to identify any effects.

It should though be noted that in all afore-mentioned studies the proportion of fathers included was small. Conducting studies with a larger sample of fathers would enable to further examine the path from paternal distress to offspring's executive functions.

#### **2.2.5. Child decision-making**

Decision-making is also an important aspect of cognitive functioning, particularly in adolescence, as it is linked to a range of different behaviours and outcomes. From a neurobiological perspective, adolescents are more prone to risky decision-making

than other age groups, as areas of the brain corresponding to cognitive control mature slowly (Somerville, Jones, & Casey, 2010). Nonetheless, it should be noted that individual variation is present, and some adolescents may display much more conservative decision-making than their peers.

Both ends of the spectrum, high riskiness, and high risk-avoidance can be problematic, for different reasons. On one hand, high riskiness is associated with an increased likelihood of engagement in delinquency, substance abuse, problematic eating, and self-harm (Adjorlolo, Asamoah, & Adu-Poku, 2018; De Bellis et al., 2013; Francesconi et al., 2020; Oldershaw et al., 2009; Xiao et al., 2009). On the other hand, risk-avoidance has been linked to depression and anxiety symptoms (Galván & Peris, 2014; Lewis et al., 2021), and is considered to be a risk factor for their emergence (Mannie et al., 2014).

There is very limited research exploring if and how paternal distress may be associated with decision-making. Specifically, in the study by Mannie et al., (2015), researchers, using a computerized task, compared decision-making in youth with familial risk for depression (who had never had depression themselves) to controls. Differences were small; however, vulnerable youth presented less risk-taking behaviours. Overall, adult depression is associated with a lack of sensitivity towards rewards, and hence with more conservative decision-making (Halahakoon et al., 2020). Therefore, children of depressed individuals may adopt this cognitive style either through environmental reinforcement or genetic susceptibility (Richards et al., 2016).

One limitation of the research by Mannie et al., (2015) is that it concerned only adolescents who had been exposed to clinical levels of depression. Moreover, the

study involved adolescents who had a depressed parent; it did not differentiate between fathers and mothers. Hence, it is unclear how fathers' distress specifically, may affect decision-making in the general population.

#### **2.2.6. Child health risk behaviours**

To some extent, exploring and risk-taking could be considered a part of typical adolescent development (Steinberg, 2008). However, it is not uncommon for some youth to engage in behaviours that are dangerous for their health, safety, and well-being. Examples of such behaviours include smoking, alcohol drinking, binge drinking (heavy episodic alcohol drinking), and sexual activity. All of them are major public concerns and have been linked to a number of adverse consequences, such as mental health difficulties, substance abuse disorders, poor academic performance, delinquency, aggression, neurocognitive alterations, and impaired decision-making (Ali et al., 2015; Boden, Blair & Newton-Howes, 2020; Degenhardt et al., 2013; Edwards et al., 2014; Enstad et al., 2019; Heradstveit et al., 2017; Lawler et al., 2021; Levola et al., 2020; Malone et al., 2014; Melotti et al., 2011; Najman et al., 2019).

Smoking is one of the leading causes of poor physical health and preventable deaths both in the UK and across the world (Department of Health, 2017; Forouzanfar et al., 2016). Early initiation of smoking is associated with an increased risk for nicotine dependence (Kendler et al., 2013) and out of the people who smoke, a significant amount report that they initiated it in adolescence (East et al., 2018). Despite the laws prohibiting it, a considerable number of adolescents in the UK smoke, most of them disclosing that they buy cigarettes from shops (Department of Health, 2017). Specifically, it is estimated that approximately 14% of 14-year-olds in the UK have

smoked at least once, while 2% smoke recurrently (Lavery et al., 2019). The use of cigarettes by youth, particularly those under 15, has declined in the last years, both in the UK and across Europe (Department of Health, 2017; Hallingberg et al., 2020; Kraus et al., 2018). However, there have been some reports that vaping has increased among adolescents, possibly because of the perceived reduced risks (Bauld et al., 2017).

When it comes to alcohol, by the age of 15, approximately 73% of the UK's adolescents report that they have tried it (Public Health England, 2018). Although underage drinking in the UK has lowered during the last few years, the percentage still remains one of the highest in Europe (Danielsson et al., 2012). Moreover, youth in the UK are shown to have more favourable attitudes towards alcohol compared to other countries (Healey et al., 2014).

Binge or heavy drinking refers to the consumption of excessive amounts of alcohol in one take (typically more than 5 drinks). A high number of adolescents in the UK, compared to other countries, engage in binge drinking (Danielsson et al., 2012), with the proportion being higher for females than males (Healey et al., 2014).

Last, even though sexual development is part of adolescence, sexual debut at a very early age (broadly defined as 15 or younger) is associated with unsafe practices and health risks (Tolman & McClelland, 2011), such as inconsistent use of contraception (Magnusson et al., 2012), unprotected sexual activity (Prendergast et al., 2019), increased likelihood for sexually transmitted infections (Epstein et al., 2014), and unintended pregnancies (Prendergast et al., 2019). Some evidence indicates that the average age of sexual debut in the UK has declined in the recent years;

nonetheless, recent studies estimate that about 3% of 14-year-olds have already had a sexual intercourse (Kelly et al., 2019; Mercer et al., 2013).

Only a narrow number of studies have so far investigated the relationship between fathers' psychopathology and adolescent risky behaviours (Ali et al., 2016; Essau & de la Torre-Luque, 2021; Herman-Stahl et al., 2008). Analysing data from the National Surveys on Drug Use and Health (NSDUH), Herman-Stahl et al., (2008) explored the impact of severe paternal and maternal psychological distress on adolescent binge drinking and illicit drug use. Having a mother with severe psychological distress was associated with higher risks for adolescent engagement in both these behaviours. When it comes to fathers, severe psychological distress predicted lower risks for binge drinking, but only for black families. There was no relationship between paternal distress and illicit drug use. Using the same source of data, Ali et al., (2016) examined how parental comorbid mental health and substance abuse disorder related to adolescent substance abuse disorder, including alcohol or drugs. Findings showed that, after adjusting for confounders, this link exists only for mothers, not fathers. Finally, Essau & de la Torre-Luque (2021), using data from the National Comorbidity Survey Replication Adolescent Supplement (NCS-A), identified psychopathology and substance abuse trajectories for fathers and mothers. Two trajectories emerged for fathers: 'low' (low psychopathology symptoms and substance and use) and 'high' (high psychopathology symptoms and drug use). Adolescents whose fathers belonged in the 'high' trajectory faced increased risks for illicit drug abuse. Off-spring of mothers with similar characteristics were at heightened risk for nicotine dependence, drug, and alcohol abuse.

Overall, these outcomes indicate that there might be an association between fathers' distress and adolescent engagement in risky behaviours. However, the existing research has some important limitations. For instance, in two of the afore-mentioned studies (Ali et al., 2016; Essau & de la Torre-Luque, 2021) parental mental health and substance abuse were examined simultaneously. Therefore, it is not clear what effects parental mental health on its own might have had. Moreover, no longitudinal studies have been conducted so far.

### **2.3. Reasons why paternal distress and child outcomes are linked**

Many reasons have been proposed as to why paternal distress and child outcomes are related to each other. For instance, genetic links are shown to exist between them (Heinrich et al., 2016; Silberg, Maes, & Eaves, 2010). It is also possible that common risk factors, such as poverty, predict both higher levels of distress and worse developmental outcomes for fathers and children, respectively (Adjei et al., 2022; Chzhen & Bruckauf, 2019; Nath et al., 2016a).

Theories such as Bronfenbrenner's bio-ecological model (Bronfenbrenner, 1977), transactional family dynamics theory (Schermerhorn & Cummings, 2008), and Bandura's social-learning theory (Bandura, 1978) provide a lens through which these associations can be interpreted. All the above theories support that the family environment, and particularly parental behaviour, play a fundamental role in child development. Indeed, a plethora of research evidence confirms their importance. The following paragraphs will first provide an overview of those theories and subsequently an evaluation of the research evidence.



### **2.3.1. Bronfenbrenner's Bio-ecological Model**

Bronfenbrenner's bio-ecological model argues that children's development occurs from the interactions between the children's unique characteristics and their immediate and more distal environments (Bronfenbrenner & Ceci, 1994). The environment nearest to the child is referred to as the microsystem and includes his/her immediate context, such as family, peers, and school. The microsystem influences child development, but at the same time, the child also impacts the microsystem. The bio-ecological model considers that the child's unique genetic make-up, along with characteristics such as age and gender, shape the child's response to the microsystem's effects. Depending on the "response", the microsystem could then also be altered. These reciprocal interactions between them are referred to as "proximal processes" (Bronfenbrenner & Ceci, 1994).

In the present context, paternal distress can be viewed as part of the microsystem, affecting child development. It may do so directly, or indirectly, through parenting and family practices. Children, depending on individual characteristics, may be more or less vulnerable to these effects. At the same time, children's reactions to these effects can cause changes to paternal behaviours or paternal distress.

### **2.3.2. Transactional family dynamics theory**

Similar to the bio-ecological model, the transactional family dynamics model also supports the reciprocal nature between the family environment and the child. Specifically, it argues that mutual influences occur between all family members (Schermerhorn & Cummings, 2008). It also argues that these influences are modified based on the individuals' characteristics, such as temperament (Schermerhorn & Cummings, 2008). Both theories come in contrast to child development models,

which view the child as a passive recipient and consider that family processes are unidirectional.

According to the transactional family dynamics model, individuals within families are nested in dyads (e.g., father-mother) or triads (e.g., father-mother-child). Interactions that occur in one setting (e.g., play) may affect interactions in other settings. The transactional theory also supports that the pathways between family members do not remain stable over time but could be altered across different time-periods (Schermerhorn & Cummings, 2008).

In line with the afore-described models, a part of the present thesis will focus on exploring the reciprocal links between paternal distress and child outcomes (internalising and externalising difficulties). Such findings can contribute to providing a more holistic understanding of the father-child relationship.

### **2.3.3. Bandura's Social-Learning Theory**

Bandura's social learning theory could also be used to explain the association between paternal distress and child outcomes (Bandura, 1978). According to the social learning theory, children learn how to behave by observing and imitating those around them (Bandura, 1978). Thereafter, as the socially desirable behaviours get reinforced, and the undesirable ones get punished, children maintain those that result in beneficial outcomes (Bandura, 1978).

Negative cognitive styles, such as viewing oneself as unworthy, are common in adults with mental health difficulties (Sutton et al., 2011). Distressed fathers could be reinforcing those negative cognitive processes in their children, increasing their susceptibility to psychopathology (Lewis et al., 2018). Paternal distress has also

been linked with hostile types of behaviour (Davis et al., 2011; Dette-Hagenmeyer & Reichle, 2014); consequently, children of distressed fathers are more likely to be exposed to aggression and to imitate it. In case these aggressive behaviours remain unchallenged by their family and their school environment, they could potentially to long-term conduct problems. Similarly, children could imitate their fathers if they engage in substance abuse (Vermeulen-Smit et al., 2012), which is also common among distressed individuals (Åhlin et al., 2015; Boden et al., 2010; Lev-Ran et al., 2014).

#### **2.3.4. The mediating role of marital quality**

High levels of paternal psychological distress have been linked to low marital quality (Bronte-Tinkew et al., 2007; Cheung et al., 2019), including dissatisfaction (Bower et al., 2013), discord (Smith et al., 2013) and lack of affection (Ramchandani et al., 2011). Distressed partners tend to engage in more negative verbal and non-verbal interactions (Johnson & Jacob, 2000) and are more likely to make accusations (Keller et al., 2009), forming relationships of poor quality, with frequent arguments and disagreement (Wee et al., 2011). Being in a disharmonious marriage can in turn result in an even higher risk for depression (Franck & Buehler, 2007; Nath et al., 2016a).

Low marital quality is shown to predict youth internalising (Low & Stocker, 2005; van der Valk et al., 2007; Weymouth et al., 2019) and externalising difficulties (Franck & Buehler, 2007), as well as substance abuse (Fosco & Feinberg, 2018; Hair et al., 2009; Yap et al., 2017). Particularly girls and older adolescents might be more vulnerable to the effects of parental conflict, as they tend to be more involved in their parents' relationship (van der Valk et al., 2007). Children living in families with high

levels of conflict may perceive their environment to be unstable and threatening, resulting in distress and maladaptive ways of coping (Du Rocher Schudlich & Cummings, 2007; Cummings et al., 2005). Furthermore, observing aggressive interactions could reinforce similar, disruptive behaviour (Bandura, 1978). Parents might also use their children as “messengers” or as “allies” against the other parent, causing them feelings of guilt and emotional insecurity (Franck & Buehler, 2007). Lastly, the impact on children can be indirect, since marital hostility often provokes father-child hostility, which is demonstrated to have very adverse effects on their well-being (Low & Stocker, 2005).

Some studies have supported that marital quality mediates the relationship between father distress and child internalising and externalising difficulties (Du Rocher Schudlich & Cummings, 2007; Cummings, Keller, & Davies, 2005; Gutierrez-Galve et al., 2015; Hanington et al., 2012; Keller et al., 2009; Smith et al., 2013). Nevertheless, the strength of this mediating pathway is found to vary, depending on the length of the timespan examined. For instance, some evidence suggests that marital quality during infancy does not mediate the long-term effect of paternal postpartum depression on young adult depressive symptoms (Gutierrez-Galve et al., 2019). However, for shorter periods of time, its role was significant (Du Rocher Schudlich & Cummings, 2007; Gutierrez-Galve et al., 2015; Hanington et al., 2012).

Moreover, marital quality might fully mediate the link with some types of child difficulties, but not others. For instance, Cummings et al. (2005) reported that marital quality fully accounted for the relationship between paternal dysphoria and peer problems as well as prosocial behaviour. Nonetheless, for emotional and conduct difficulties, the mediation was only partial. Similarly, Hanington et al. (2012) found that parental conflict partially explained the association with conduct symptoms;

however, they did not find any links with emotional. This discrepancy could perhaps be attributed to the fact that Cummings et al. (2005), conducted a cross-sectional study while Hanington et al. (2012), a longitudinal, suggesting that marital quality can explain the presence of current but not long-term emotional problems. Future research could expand the existing knowledge by exploring further the role of marital quality in different types of child outcomes, including substance abuse.

### **2.3.5. The mediating role of paternal warmth**

Parental warmth is a parenting behaviour that involves high levels of affection, comfort, sensitivity, support, and encouragement towards the child (Lee et al., 2018). Parental warmth refers to the quality of parental involvement, rather than the quantity of it (Lee et al., 2018).

Lack of paternal warmth has been associated with a range of adverse offspring outcomes, including mental health problems (Elgar et al., 2007; Malmberg & Flouri, 2011). Lee et al., (2018) argue that children whose caregivers do not exhibit affection might feel unloved, which can negatively influence their well-being. Additionally, those children are more likely to engage in delinquency and alcohol misuse (Calafat et al., 2014; Yap et al., 2017). Having a poor-quality relationship with their fathers could perhaps prevent them from sharing their whereabouts, resulting in a lack of monitoring, and consequently increased vulnerability for engagement in risky and maladaptive behaviours (Fletcher et al., 2004). In contrast, high levels of warmth are shown to have a protective effect, as those children are more likely to communicate with their caregivers and act in line with their instructions (Danielsson et al., 2011; Ryan et al., 2010; Windman et al., 2016; Yap et al., 2017).

Lack of paternal warmth is also linked to poor cognitive skills (Coley, Lewin-Bizan & Carrano, 2011; Madigan et al., 2019; Malmberg & Flouri, 2011; Malmberg et al., 2016; Martin, Ryan & Brooks-Gunn, 2007; Sethna et al., 2017) and impaired executive functions (Hertz et al., 2019; Towe-Goodman et al., 2014), particularly for families of lower socio-economic status and ethnic minority backgrounds (Madigan et al., 2019). According to Bowlby's attachment theory (Bowlby, 1982), infants who share a warm and secure relationship with their caregivers do not need to worry about their needs being covered or being abandoned. Therefore, they have more cognitive resources available to invest in exploring and learning. In other words, those offspring feel safe to delve into new and unfamiliar situations, through which they can advance their skills, acquire new information and engage in stimulating and enriching activities (Bernier et al., 2012; Madigan et al., 2019; Towe-Goodman et al., 2014). Furthermore, children raised in a supportive environment are likely to build high self-esteem, so they persist and perform to the best of their ability while undertaking cognitive tasks (Kaur & Gera, 2016; Valcan et al., 2018).

Fathers experiencing psychological distress tend to be less warm with their children (Cheung & Theule, 2019b; Wilson & Durbin, 2010). Symptoms associated with high levels of distress, such as low energy and fatigue, as well as the lack of mental resources, may prevent them from responding to their children's needs in a sensitive and engaging way (Dix & Meunier, 2009; Sethna et al., 2015). Moreover, distressed caregivers are likely to appraise their children's behaviour in a more negative way than non-distressed caregivers (Ordway, 2011), a cognitive bias that could influence their parenting responses (Dix & Meunier, 2009).

Studies have investigated the mediating role of paternal warmth in the relationship between paternal distress and child outcomes. Dubois-Comtois et al., (2021) based on an observational study of low-income families, argued that the quality of father-toddler interaction mediates the relationship between paternal distress to internalising and externalising difficulties. Studying the longitudinal associations in approximately 4000 families, Elgar et al., (2007) showed that paternal warmth mediates the link from paternal depression to offspring externalising difficulties in late childhood and adolescence. Dette-Hagenmeyer & Reichle (2014) also supported its mediating effect, regarding children's socio-emotional competence and hyperactivity.

There have been no studies so far assessing the role that paternal warmth might have in the relationship between paternal distress and child cognitive or health-related outcomes. Additionally, when it comes to internalising and externalising difficulties, not all studies offer support for the mediating role of paternal warmth (Franck & Buehler, 2007; Giallo et al., 2014; Herbert et al., 2013). In particular, Herbert et al. (2013), using a sample of children with attention-deficit hyperactivity disorder (ADHD), reached the same conclusions. Similarly, no evidence was found by Giallo et al. (2014) as well as Franck and Buehler (2007), who examined community samples of preschoolers and adolescents respectively.

This discrepancy could possibly be explained by differences in the samples' characteristics; the studies that did not identify warmth as a mediator researched children with higher levels of difficulties (Franck and Buehler, 2007; Herbert et al., 2013). For instance, Herbert et al. (2013) focused specifically on children with severe behaviour problems, while Franck and Buehler (2007) included a higher percentage of children with severe difficulties compared to other studies. In other words,

decreased paternal warmth could be a mediator for community samples but not clinical ones.

### **2.3.6. The mediating role of paternal hostility**

Paternal hostility is characterized by negative behaviours towards the child, including high levels of aggressiveness, irritability, criticism as well as lack of patience, through which frequent arguments and conflicts emerge (Kane & Garber, 2004; Low & Stocker, 2005). Paternal hostility is a strong predictor of adverse child outcomes (Halse et al., 2018; Lam et al., 2018; Lucassen et al., 2015; Reeb et al., 2010; Valcan et al., 2018). In contrast to warmth, hostility undermines children's feelings of security (Halse et al., 2019), and burdens their development and well-being (Low & Stocker, 2005; Valcan et al., 2018). Paternal hostility is commonly observed among fathers experiencing psychological distress (Cheung & Theule, 2019b).

In a cross-sectional study involving families with children around 10 years old, Low & Stocker (2005) argued that hostility mediates the association between paternal depression and child internalising and externalising difficulties. Similarly, using longitudinal data from the UK, Nath et al., (2016b) suggested that it mediates the link from postnatal depressive symptoms to child emotional regulation at age 7, with the link being stronger in boys. These findings highlight the need to further explore the ways in which hostility affects the father-child relationship.

### **2.3.7. The mediating role of paternal involvement**

Paternal involvement refers to the 'quantity'; the amount of time fathers spend engaging with their children (Bronte-Tinkew et al., 2007). For instance, that might include involvement with academic activities, such as practicing math or support with



literacy (Baker, 2013; Huntsinger et al., 2016; Manolitsis et al., 2013; Skwarchuk et al., 2014). It may also include ‘fun’ activities, such as shared book reading, storytelling, playing or visiting the library, all of which are beneficial for children’s well-being and development (Baker, 2013; Fagan et al., 2016). By engaging in enriched and stimulating activities, children are provided with the opportunity to enhance their abilities (Fay-Stammbach et al., 2014). Additionally, through interactions with their fathers, they can practice their social and self-regulation skills (Cabrera et al., 2007). Indeed, many studies suggest that lack of paternal involvement is linked to child internalising and externalising difficulties (Allen-Meares et al., 2010; Flouri et al., 2016; Kroll et al., 2016; McMunn et al., 2016), lower cognitive abilities (Fagan et al., 2016; Huntsinger et al., 2016; Manolitsis et al., 2013), risky sexual behaviours (Coley et al., 2009), and substance misuse (Ryan et al., 2010; Yap et al., 2017), as children of uninvolved fathers may miss on those benefits.

Lower levels of involvement are observed in distressed fathers, who tend to be withdrawn and not engaged with their children (Bronte-Tinkew et al., 2007; Chu & Lee, 2019; Shorey et al., 2019). Nevertheless, only two studies so far have evaluated whether paternal involvement may play a mediating role in the relationship between fathers’ distress and child outcomes (Guilierrez-Galve et al., 2015; Paulson et al., 2009). Specifically, using large-scale, longitudinal, UK data, Guilierrez-Galve et al., (2015) supported that paternal involvement at child age 18 months, partially mediated the relationship between paternal postnatal depressive symptoms and child internalising and externalising difficulties at 42 and 81 months. Furthermore, Paulson et al., (2009) argued that paternal reading frequency partially mediated the link from postnatal symptoms to child language abilities at 2 years.

Overall, though these findings highlight the importance of paternal involvement, research has so far only examined its effects in the early years. To gain a fuller understanding, it would be beneficial to examine its role during later developmental stages.

## Chapter 3: Data and Measures

The aim of this chapter is to describe the data used in the thesis. It will also describe the main paternal and child measures.

### 3.1. Data

To address the research aims, this thesis analyzed data from the UK's Millennium Cohort Study (MCS) (<https://www.cls.ioe.ac.uk/mcs>). The MCS is an ongoing birth cohort survey, including information on 19,243 UK families (19,517 children) who had a child born in 2000–2002. Participating families were disproportionately selected, to ensure that all UK minority groups, and disadvantaged wards are represented (Plewis, 2007). So far, data have been collected for 7 MCS sweeps, details for which can be found in Table 1. For the current project, data were used from sweeps 2-6 (ages 3 to 14). Ethical approval for the MCS has been obtained from NHS Multi-Centre Ethics Committees. Parents gave informed consent, and children (at the ages of 11, 14 and 17) informed assent. This thesis has received additional ethics approval from the IOE Research Ethics Committee.

**Table 1** Description of the MCS sweeps

Sweep	Sweep 1	Sweep 2	Sweep 3	Sweep 4	Sweep 5	Sweep 6
Child's age	9 months	3 years	5 years	7 years	11 years	14 years
Number of Families	18,552	15,590	15,246	13,857	13,287	11,726

## **3.2. Measures**

### **3.2.1. Paternal measures**

This project used paternal measures which assess psychological distress, marital satisfaction, father-child relationship quality (warmth and hostility), and involvement. Detailed information on how these variables were utilized to address each of the research aims will be provided in the corresponding chapters.

*Paternal psychological distress* was assessed with the 6-item Kessler Psychological Distress scale (K-6) across child ages 3-14 (sweeps 2-6). K-6 is a self-administered measure of emotional state and overall distress, with good psychometric properties (Kessler et al., 2002). It evaluates depressive and anxiety symptoms experienced within the last month with the following 6 questions: “During the last 30 days, about how often did you feel so depressed that nothing could cheer you up/ hopeless/ restless or fidgety/ everything was an effort/worthless/nervous?”. These items were rated on a five-point scale, ranging from “none of the time” to “all the time”. Responses were added to create a final score, varying from 0 to 24, with higher values indicating more difficulties. A score equal to or higher than 14 indicates clinical levels of difficulties (Staples et al., 2019).

*Paternal marital satisfaction* was measured at child ages 3 and 5 (sweeps 2 and 3) with 4 items derived from the Golombok Rust Inventory of Marital State (GRIMS), a self-completed measure with good validity and reliability (Rust et al., 1990). These items were: “my partner is usually sensitive and aware of my needs”, “partner doesn’t listen to me”, “I sometimes feel lonely even when I’m with my partner” and “I suspect we may be on the brink of separation”. They answered on a 1-5 scale, with 1 corresponding to “strongly agree” and 5 to “strongly disagree”. After being

appropriately reversed, items were added to create a total score, varying from 4 to 20, with higher values suggesting higher marital satisfaction.

*Father-child closeness* (or warmth) and *father-child conflict* (or hostility) were measured with the two subscales of Pianta's Child-Parent Relationship Scale (CPRS) Short Form, adapted from the Student-Teacher Relationship Scale (STRS, Pianta & Steinberg, 1992). This scale assesses parental perceptions regarding the parent-child relationship quality and was completed by the father at child age 3 (sweep 2). Questions were rated on a 1-5 scale, ranging from "definitely does not apply" to "definitely applies". The closeness sub-scale includes 7 items, such as "I share an affectionate, warm relationship with my child" and "if upset, my child will seek comfort from me". Responses are added, to generate a final score, which varies from 5 to 35, with higher values reflecting higher levels of closeness. The conflict sub-scale consists of 8 items, examples including "my child and I always seem to be struggling with each other" and "my child is uncomfortable with physical affection or touch from me". The final score may range from 5 to 40; higher values show increased conflict.

*Paternal involvement* was measured at child age 5 (sweep 3), with 7 questions, answered by the father. These questions include: "how often do you read to the child?", "how often do you tell stories to the child?", "how often do you do musical activities with the child?", "how often do you draw with the child?", "how often do you play physical active games with the child?", "how often do you play games indoors with the child?" and lastly "how often do you take the child to the park/playground?". They were rated on a 1-6 scale, with 1 corresponding to "everyday" and 6 to "not at all". Scores were reversed and added, creating a total score, ranging from 7 to 42, with higher scores suggesting greater involvement.

The maternal variables which have been used involve *psychological distress* and *mother-child relationship quality*. These have been measured at the same time-points and in the same way as paternal variables.

### 3.2.2. Child measures

The child outcomes that were assessed include emotional and peer (internalising) difficulties, conduct and hyperactivity (externalising difficulties), verbal ability, spatial working memory, decision-making, and health risk behaviours.

**Table 2** Description of the SDQ difficulties scales

Emotional difficulties	Conduct problems	Hyperactivity/Inattention	Peer difficulties
Often complains of headaches, stomach-aches or sickness	Often has temper tantrums or hot tempers	Restless, overactive, cannot stay still for long	Rather solitary, tends to play alone
Many worries	Generally obedient	Constantly fidgeting or squirming	Has at least one good friend
Often unhappy, downhearted	Often fights with other children	Easily distracted, concentration wanders	Generally liked by other children
Nervous or clingy in new situations	Often lies or cheats	Thinks things out before acting	Picked on or bullied
Many fears, easily scared	Steals from home, school or elsewhere	Sees tasks through to the end	Gets on better with adults than with other children

*Child internalising and externalising difficulties*, across child ages 3-14 (sweeps 2-6), were measured with the Strengths and Difficulties Questionnaire (SDQ), completed by the main caregiver, which was in most cases the mother (Goodman, 2001). The SDQ, a measure of good psychometric properties (Goodman, 2001), consists of five sub-scales: emotional symptoms, conduct problems, hyperactivity/inattention, peer relations (the 'difficulties' subscales), and prosocial behaviour (the 'strengths' subscale). Only the 4 difficulties subscales have been used in this project. Each of

the sub-scales includes 5 items. These were answered on a 0–2 scale, with 0 corresponding to “not true”, 1 to “somewhat true” and 2 to “certainly true”. The items for each subscale are presented in table 2. Items were added, generating a final score for each subscale. Summing all the subscales creates a total difficulties score.

*Verbal ability* was assessed at child age 11 (sweep 5), using the British Ability Scales (BAS–II) Verbal Similarities subscale. BAS-II was administered to children in their homes, as part of the main interview process. This subscale measures verbal knowledge and verbal reasoning, by asking children how 3 words are related to each other (Hansen, 2014). In the current project, the scores used were standardized to have a mean of 50 and a standard deviation of 10. They have also been adjusted for both item difficulty and child age.

*Spatial working memory* was also assessed at child age 11 (sweep 5), by the Cambridge Neuropsychological Test Automated Battery (CANTAB) Spatial Working Memory test (Robins et al., 1994). The task was administered to children at their homes, during the main interview. The test included 3 blocks of 4 trials each. Children were presented with a computer screen and asked to look for the blue tokens which were hidden under coloured boxes. Once a token was found, children had to move it to the right-hand column of the screen, and then start a new search. When all tokens were found, the trial was completed. Tokens would never appear under the same box twice. In case children searched again a box in which they had previously found a token, it was marked as an error (between errors). It was also marked as an error if, within the same search, they returned to the same box which they had previously found to be empty (within errors). Gradually, the number of tokens increased from 4 to 6 to 8, raising the difficulty of the task. The colour and location of the boxes varied between trials, to discourage stereotyped search

strategies. Two spatial working memory variables were produced: *total errors* and *strategy*. Total errors consist of the sum of between and within errors, with higher scores suggesting poorer performance. The optimal way to complete this task was to begin a search from the same box that the previous search began with. Strategy scores were measured by counting the number of times a search began from a different box. Hence, a high strategy score indicates that the child did not search for tokens effectively.

*Decision-making* was measured at child age 11 (sweep 5), using the Cambridge Gambling Task (CGT; Rogers et al., 1999). Children completed the CGT at their homes. Children were presented with a row of ten boxes across the top of a computer screen. These boxes were either red or blue, with the ratio between these 2 colours varying. One of those boxes always contained a yellow token. Children were asked to choose the box colour in which they believed the token was hidden. The task involved 5 stages, each of which included a block of trials. The last 4 stages were the gambling stages. During those, children were given 100 points, which appeared in either ascending or descending order. They were asked to select a percentage of these points to bet on their colour decision. Points were added or subtracted, depending on the actual location of the token. The aim was to gain as many points as possible. Six outcomes were produced from this task: (1) *Deliberation time*, which refers to the average amount of time (in milliseconds) it took to make a box colour response; (2) *Quality of decision-making*, which describes the mean number of trials children chose the correct colour; (3) *Delay aversion*, which shows the difference in percentage bet between the ascending and the descending conditions; (4) *Overall proportion bet*, which refers to the mean proportion of points bet across all trials; (5) *Risk adjustment*, which measures the extent at which



children's betting behaviour was affected by the ratio of boxes (in other words, children's tendency to bet more points when most boxes were of their chosen colour); and (6) *Risk-taking*, which describes the mean proportion of points bet on trials at which the most possible outcome was selected.

Finally, *adolescent health risk behaviours* were assessed at age 14 (sweep 6), through self-report. *Smoking* was measured with a binary variable showing if the child had ever smoked (including electronic cigarettes) or not. *Alcohol consumption* was assessed with a 3-level categorical variable, reflecting: whether the child had never had an alcoholic drink, whether the child had had an alcoholic drink but had never engaged in binge drinking, and lastly, whether the child had engaged in binge drinking at least once. Binge drinking was defined as consuming 5 or more alcoholic drinks in one take. Last, *sexual activity* was measured with a dichotomous variable, demonstrating whether the child had ever engaged in any type of sexual intercourse or not (Gage & Palatay, 2021).

## **Chapter 4: Reciprocal links between paternal psychological distress and child internalising and externalising difficulties (research aim 1)**

The aim of this chapter is to examine the reciprocal associations over time between paternal psychological distress and child internalising and externalising difficulties, from early years to adolescence. The chapter will also investigate the effects of the child's gender, as well as the role of potential mediators, for both father and child paths.

Exploring transactional relationships in the family context can be beneficial for policies and clinical practice, which aim to promote the well-being of families. By identifying whether it is fathers, children, or both, who most influence each other, it is possible to design practices that will effectively support all family members. What is more, it is important to understand whether these links vary across different child developmental stages. In this way, it can be determined whether there are phases during which assisting vulnerable families is most critical.

This chapter will begin with an overview of the research literature concerning the one-way effects of child internalising and externalising difficulties on paternal distress. It will also discuss the reasons why child difficulties may be related to paternal distress. Subsequently, it will examine the research literature focusing on the reciprocal relationships between paternal distress and child difficulties and will investigate the role of the child's gender in these relationships. Next, the chapter will present the methods and results of the analysis conducted to address the first research aim. Finally, findings, limitations, and implications will be discussed.

#### **4.1. The effects of child difficulties on paternal distress**

As demonstrated in Chapter 2, paternal psychological distress is a strong predictor of child internalising and externalising difficulties. At the same time, though limited, some studies have also assessed the role of the child (Goldstein et al., 2013; Podolski & Nigg, 2001; Settapani et al., 2013; Silverman et al., 2009). Research evidence suggests that child difficulties can also affect fathers and are perhaps associated with higher levels of psychological distress. This evidence is in line with theoretical models, such as Bronfenbrenner's bio-ecological model and family systems theory, which argue that the child is not simply a passive recipient of environmental effects. Instead, the child plays an active role, by interacting with and influencing the surrounding environment.

There is a small number of studies exploring whether children's emotional symptoms might be negatively impacting the caregivers' well-being; however, these studies have so far focused exclusively on mothers (Settapani et al., 2013; Silverman et al., 2009). Specifically, using samples of clinically referred children, researchers compared maternal distress before and after the children's effective psychological treatment (Settapani et al., 2013; Silverman et al., 2009). Results indicate that children's recovery was accompanied by a significant improvement in their mothers' psychological well-being, unaccounted by other factors. It is possible that the same effects would be observed in fathers, though this has yet to be evaluated by research studies.

Regarding externalising difficulties, although there have been no research studies so far assessing fathers' psychological distress per se, a few have examined the one-way child pathways to fathers' parenting stress (Goldstein et al., 2007; Podolski &

Nigg, 2001; Settapani et al., 2013; Silverman et al., 2009). Parenting stress is used to describe how anxious a caregiver feels regarding his/her role as a parent (Graziano et al., 2011), and is closely linked to psychological distress (Bamishigbin et al., 2020). It has been shown that fathers of children with hyperactivity and conduct problems report higher parenting stress than fathers of children with no behavioural problems, suggesting that those child difficulties could possibly have an adverse impact (Goldstein et al., 2013; Podolski & Nigg, 2001).

#### **4.2. Mediating pathways from child difficulties to paternal distress**

So far, the variables mediating the pathways from child difficulties to paternal distress have not been investigated. Nevertheless, it can be hypothesized that family factors, such as marital conflict and parenting practices, may have a role to play.

As highlighted in previous chapters, marital conflict contributes to explaining father effects. At the same time, reciprocal associations have been found between marital conflict and adolescent internalising and externalising behaviours, with one escalating the other (Cui et al., 2007; Van der Valk et al., 2007). A child experiencing difficulties can provoke a disharmonious family environment, preventing positive interactions between all members. For instance, parents may engage in disagreement over the reasons for the child's difficulties or for the actions needed to manage the situation (Cui et al., 2007; Van der Valk et al., 2007). While child difficulties are shown to predict marital conflict, there has been no evaluation so far, as to whether marital conflict may mediate the relationship from child difficulties to paternal distress. However, independent links between marital conflict and individuals' psychological distress have been found (Franck & Buehler, 2007).

Therefore, it is possible that child difficulties could influence paternal psychological distress through marital conflict.

Other potential pathways from child difficulties to paternal distress include parenting practices, such as paternal warmth, hostility, and involvement. Parenting a child with difficulties can impose many challenges on caregivers, leading them to become emotionally disengaged, distant, and harsh (Pearl et al., 2016). As a result of these behaviours, caregivers may experience feelings of guilt or inadequacy, which could in turn deteriorate their mental well-being. Research findings do indeed indicate child internalising and externalising difficulties can trigger a decline in positive parenting practices (Burke et al., 2008; Hipwell et al., 2008; Hou et al., 2021), and that these associations can sometimes be bidirectional (Pearl et al., 2016; Verhoeven et al., 2010; Zvara et al., 2018). What is more, Hou et al., (2021) demonstrated that adolescent depressive symptoms might be associated with paternal depressive symptoms through a reduction in paternal warmth. Nevertheless, further research on these relationships is required, across different ages and for different types of child difficulties.

#### **4.3. Bi-directional links between father distress and child difficulties**

To further understand the father-child relationship, existing research has not only examined child paths, but also the bidirectional paths between fathers and offspring. In other words, whether child difficulties and paternal distress might influence each other simultaneously.

Two studies have explored these bidirectional links in clinical samples (Antúnez et al., 2018; Tichovolsky et al., 2018). In particular, Antúnez et al., (2018) studied 331 children with oppositional defiant disorder (ODD) and their families. Researchers

concluded that child externalising difficulties at the age of 3 predict paternal anxiety and depression symptoms at the age of 8, though only for boys. There were no father effects. Furthermore, Tichovolsky et al., (2018) investigated the reciprocal relationships between father and child depressive symptoms, in a sample of 153 families with preschoolers who had severe behavioural problems. Children were 3 years old at baseline and data was collected annually until they reached 6. Cross-lagged models revealed that paternal difficulties consistently predicted child difficulties, but not vice-versa. It should be noted that in both studies maternal depressive symptoms were accounted for.

A few studies have evaluated the bidirectional links between fathers' distress and child difficulties in the early years and middle childhood (Cioffi et al., 2018; Gross et al., 2008; Villarreal & Nelson, 2018). First, Gross et al., (2008) analyzed longitudinal data from the Early Steps Multisite Study, a cohort in which approximately half of the families have taken part in a parenting intervention, while the other half was the control group. Researchers assessed the impact of child non-compliance at age 2 years on the progress of paternal depressive symptoms across child ages 2-4 years; however, they did not find a significant association. On the contrary, the intercept of paternal depressive symptoms across child ages 2-4 years predicted child internalising difficulties at age 4. The conclusions were similar for families who received the intervention and those who did not. In addition, Cioffi et al., (2018) explored the relationships between parental depressive symptoms and child internalising difficulties, from 18 months to 6 years of age, in a sample of families with adopted children. Though paternal symptoms were linked to child difficulties, neither child nor cross-lagged effects were identified. Last, Villarreal & Nelson (2018), analyzing data from 633 families, examined the paths between father and

child internalising difficulties, across child ages 6, 8, and 10 years. There was one child effect, with child difficulties at age 8 resulting in more paternal difficulties at age 10, but no father effects.

Using cross-lagged models, Hastings et al., (2020) and Hou et al., (2021) investigated the bidirectional associations between fathers and their offspring in adolescence. Specifically, Hastings et al., (2020), following a sample of 220 families, explored the influences between youth and parent depression, and youth and parent anxiety, across 2 years. While transactional relationships were revealed between mother and youth symptoms, neither father nor youth symptoms predicted one another. On the contrary, Hou et al., (2021) demonstrated that child effects take place, with youth depressive symptoms predicting paternal depressive symptoms, for 2 consecutive years. Furthermore, findings support that paternal warmth partially mediated this path; adolescent depression provoked a less warm father-child relationship, which in turn deteriorated paternal symptoms.

Fanti et al., (2013) is the only study from the past research literature to have looked into the transactional links between offspring difficulties and paternal distress from the early years to adolescence. Specifically, researchers examined those associations across 6 time-points, from child ages 4.5 years to 15 years, for both externalising and internalising difficulties. Regarding externalising difficulties, outcomes showed that father effects occur in early childhood, and that both father effects and child effects occur in adolescence (though these never occurred simultaneously). For internalising difficulties, there were mainly father effects, while also reciprocal relationships were identified, between the ages of 11 to 12 years. Reciprocal relationships also took place between the ages of 12 to 15, though only for female adolescents.

The mixed conclusions derived from the existing research could be potentially attributed to methodological limitations. For instance, some studies have not adjusted for key confounders, such as maternal distress or family income (Hastings et al., 2020; Hou et al., 2021; Fanti et al., 2013). Moreover, with a few exceptions (Cioffi et al., 2018), most studies included only intact, biological families, limiting the generalisability of their conclusions (Fanti et al., 2013; Gross et al., 2008; Villarreal & Nelson, 2018). Studies including non-intact families did generally have a small sample size (Hastings et al., 2020). The present research will aim to address these limitations, by adjusting for a number of confounders, and examining a large and UK-representative sample.

#### **4.4. The role of child's gender**

Overall, it is shown that boys are more likely to develop internalising difficulties, while girls are more likely to develop externalising difficulties (Chaplin & Aldao, 2013). Research also suggests that the impact of paternal distress might differ for boys and girls. For instance, several studies found that paternal distress affects sons more strongly than daughters (Fletcher et al., 2011; Flouri et al., 2019; Franck & Buehler, 2007; Low & Stocker, 2005; Ramchandani et al., 2005, 2008), particularly when it comes to externalising difficulties (Fletcher et al., 2011; Flouri et al., 2019; Ramchandani et al., 2005, 2008). A possible interpretation is that fathers tend to spend more time with their sons (Flouri & Buchanan, 2003; Kroll et al., 2016) because they identify more with them and feel more capable of raising a same-sex child (Lamb, 2004). Consequently, the influence of paternal distress is likely to be more evident in boys.



Conversely, other studies reported significantly stronger effects for daughters. This includes internalising (Fletcher et al., 2011; Gutierrez-Galve et al., 2019; Lewis et al., 2017; Reeb & Conger, 2009; Reeb et al., 2010) as well as externalising difficulties (Herbert et al., 2013; Fanti et al., 2018). It is argued that, compared to boys, girls place greater value on their interpersonal relationships, including their relationship with their fathers (Herbert et al., 2013; Reeb & Conger, 2009; Reeb et al., 2010). Hence, having a low-quality relationship with them is likely to negatively affect their well-being (Queen et al., 2013).

What is more, some findings suggest that the child's gender moderates the strength of child paths (Antúnez et al., 2018; Fanti et al., 2013). To illustrate, Antúnez et al. (2018), found that only boys' conduct problems, at ages 3-8, predicted paternal depression and anxiety symptoms. On the contrary, Fanti et al. (2013) claimed that only girls' internalising difficulties at age 12 predicted paternal depressive symptoms, 3 years later.

Overall, the role of the child's gender in the paths between paternal distress and child difficulties is ambiguous. Further research is needed to determine if these relationships manifest differently between boys and girls.

#### **4.5. Conclusions and the Present Study**

To conclude, existing research suggests that bidirectional influences between paternal distress and child difficulties may take place. Nevertheless, there is a lack of consistency in the findings. The present research will aim to expand current knowledge on the father-child relationship by studying the transactional associations between paternal distress and child difficulties from child age 3 to age 14, in a large and representative sample, while also adjusting for socioeconomic and family

factors. The study will expand on the conclusions of Flouri et al., (2019), who, using data from MCS, explored the impact of paternal psychological distress on child emotional, conduct, hyperactivity/inattention, and peer difficulties. By fitting growth curve models, researchers showed that paternal psychological distress predicted all types of child difficulties, in biological and non-biological families, though to a lesser extent than maternal distress did. Findings also indicate that paternal effects are stronger for boys.

Based on these conclusions and the overall research literature, it is hypothesized that across all ages, paternal distress will predict both internalising and externalising difficulties; however, the link with internalising difficulties may be stronger, due to the genetic factors underlying this relationship (Kendler et al., 2003). Regarding child effects, these are expected to be found for both internalising and externalising difficulties; nonetheless, in line with the existing research, these are likely to be less common than paternal effects. Considering that those paths may differ between boys and girls, the moderating role of the child's gender will also be explored. Given the mixed findings past studies have yielded regarding the role of the child's gender, it is not possible to speculate what the results of the current research may be.

Last, identifying the reasons why these relationships take place will not only strengthen current knowledge but will also help provide pragmatic guidance on how negative influences within the family environment can be hindered. Existing research on potential mediators concerns mostly father effects, with one study so far examining their role in child effects (Hou et al., 2020). Using the measures available (father-child closeness, father-child conflict, paternal marital satisfaction, and paternal involvement), the current research will aim to provide further insight into the causal pathways between paternal distress and child difficulties. It is hypothesized

that all the above mediators will play a role in explaining both father-child and child-father pathways.

## **4.6. Methods**

### **4.6.1. Participants**

The analytic sample for this analysis includes children and their families from the MCS who met the following criteria across child ages 3-14 (sweeps 2-6): 1) Child was either a singleton or a first-born twin or triplet; 2) Child had a total difficulties score in at least one of sweeps 2-6; 3) There were valid psychological distress data available for at least one of sweeps 2-6 (therefore, a father or father-figure was living in the same household as the child for at least one of the assessed sweeps). This resulted in an analytic sample of 13,105 children (49.2% female children, 94.2% biological fathers at child age 7, and 89.4% biological fathers at child age 14).

### **4.6.2. Measures**

*Paternal psychological distress* was measured with the K-6. Cronbach's alpha was .81, .82, .83, .86 and .85 across child ages 3, 5, 7, 11, and 14, respectively.

*Children's internalising and externalising difficulties* were measured with the 4 SDQ domains: emotional, conduct, hyperactivity/inattention and peer difficulties. Across child ages 3, 5, 7, 11, and 14, respectively, Cronbach's alpha, for the emotional domain, was .57, .59, .64, .70 and .72; for the conduct domain, it was .69, .64, .59, .61 and .65; for the hyperactivity domain it was .73, .77, .79, .79 and .77; lastly, for the peer domain, it was .53, .51, .57, .64 and .62.

Potential mediators include *paternal marital satisfaction*, *paternal closeness* to the child, *paternal conflict* with the child, and *paternal involvement*. Paternal marital

satisfaction was assessed in child ages 3 and 5, with Cronbach's alpha equalling 0.73 and 0.75, respectively. Paternal closeness and paternal conflict were both measured in child age 3, with Cronbach's alpha being 0.65 and 0.73, respectively. Finally, paternal involvement was assessed only in child age 5, with Cronbach's alpha equalling 0.73.

All covariates were selected based on findings from past research. *Key child covariates* included *gender* (Chi & Cui, 2020; Gutman & McMaster, 2020) and *ethnicity* (Ahmad et al., 2021; Goodman et al., 2008). Child gender was assessed with a dichotomous variable (male or female), as did ethnicity (white or other). *Paternal covariates* include *education* (Bjelland et al., 2008; Torvik et al., 2020) and *biological status* (Perales et al., 2017). Paternal education was measured with a binary variable, showing if the father had a university degree or not. For each case, the information available from the latest sweep was used. Biological status was also measured with a binary variable, indicating whether the father living in the same household as the child was the biological father or not. Due to the high multicollinearity between sweeps ( $VIF > 4$ ), biological status was controlled for only in child ages 7 and 14 (sweeps 4 and 6, respectively). Other covariates include *maternal psychological distress* (Hope et al., 2019; Nath et al., 2016a) and *family poverty* (Adjei et al., 2022; Nath et al., 2016a; Pryor et al., 2019), both of which were assessed at each sweep (2-6). Maternal psychological distress was measured with the K-6, while family poverty was measured with a binary variable, demonstrating if the family lived above or below 60% of the UK's household median income.

### 4.6.3. Statistical Analysis

All analyses were carried out in STATA. First, to examine if there was any sample selection bias, all study variables were compared between the analytic and non-analytic samples. Subsequently, the correlations between child difficulties, paternal distress, and maternal distress were explored, only for the analytic sample. Next, for each of the SDQ domains (emotional, conduct, hyperactivity, and peer difficulties) cross-lagged structural equation models (SEM) were run across child ages 3 to 14 (model depicted in Figure 1). Initially, models were run unadjusted and then adjusted for all covariates as well as prior child difficulties and paternal distress. In case cross-lagged effects were found, the formula recommended by Clogg et al. (1995),  $z = (\beta_1 - \beta_2) / \sqrt{(SE\beta_1)^2 + (SE\beta_2)^2}$  was applied, to test which of the effects (child or father) was stronger. Moreover, for any significant paths identified, the moderating role of the child's gender was investigated, using sub-group analysis.

In the MCS, families from disadvantaged populations, disadvantaged electoral wards, and ethnic minorities (in England only) were over-sampled. There were 9 strata in total for the whole of the UK; England ethnic, England advantaged, England disadvantaged, Scotland advantaged, Scotland disadvantaged, Wales advantaged, Wales disadvantaged, Northern Ireland advantaged, and Northern Ireland disadvantaged. To account for this issue, as well as for the issues of non-response and systematic attrition, data were weighted using clustering, stratification, and weight variables.

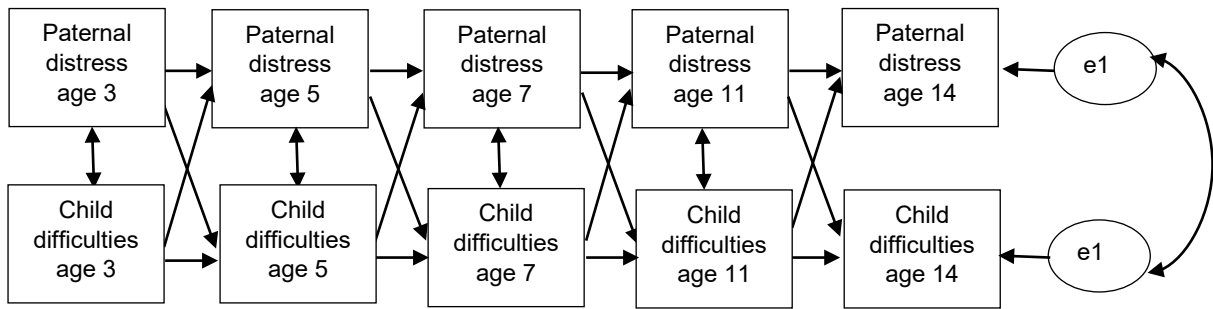
Missing data were dealt with by the maximum likelihood with missing values (mlmv) estimation method. In this way, it was possible to use the full sample of 13,105 cases in each model. Missing cases for paternal distress were 48.5%, 51.2%, 56.8%,

57.4% and 65.6%, across child ages 3, 5, 7, 11, and 14, respectively. For child total difficulties, missing cases were 25.2%, 31.1%, 36.4%, 39.4%, and 45.8%, across ages 3-14, respectively.

To be included in the analytic sample, a father/father-figure needed to reside in the same household as the child for at least one of the assessed sweeps, but not in every sweep. Hence, through mlmv, paternal distress scores were generated for sweeps in which there was no father in the household. To account for this issue, a sensitivity analysis was performed, involving only families which had a father-figure living consistently in the same household as the child, across child ages 3-14. This resulted in a sub-sample of 5412 families.

Additionally, another sensitivity analysis was conducted, involving only families in which there was a *biological* father in the household in all the assessed sweeps, leading to 5258 cases. In this way, it was possible to control for any potential changes in the father figure across sweeps (Pearce et al., 2014).

With the use of this sub-sample, a mediation analysis was conducted, to explore which family factors might help explain the significant relationships found. This sub-sample was selected for the mediation analysis, as the effects of change in family structure and change in the father-figure could be accounted for. Mediation was tested using Sobel's test, which is considered appropriate in research involving a large sample size (Özdil & Kutlu, 2019). Due to the measures available, it was possible to examine mediation paths only between child ages 3 to 5 and 5 to 7. Models were run for each type of child difficulties, adjusting for covariates, and including all mediators simultaneously. Missing data were again accounted for using the mlmv method.



**Figure 1** Cross-lagged model for paternal distress and child problem behaviour. Control variables included maternal distress (child ages 5-14), household income poverty (child ages 5-14), paternal education (latest available sweep), paternal biological status (ages 7 and 14), child ethnicity and child gender. The control variables predicted both paternal distress and child difficulties in every model.

## 4.7. Results

### 4.7.1. Descriptive Statistics

Families in the analytic sample were more advantaged than those in the non-analytic, suggesting some sample selection bias (table 3). To illustrate, across all sweeps, children presented fewer internalising and externalising difficulties, and maternal distress was lower. Paternal distress was lower as well; however, it was not possible to assess whether the difference was statistically significant, since most fathers with valid scores were included in the analysis, and only a few were in the non-analytic sample. In terms of socio-demographic characteristics, fathers in the analytic sample were more likely to be university-educated and to be biological fathers. Furthermore, families were less likely to live below the 60% poverty median and children were more likely to be female. There were no differences regarding ethnicity.

**Table 3** Descriptives of study variables in the analytic sample and in the non-analytic sample (unweighted data)

Categorical variables	Analytic Sample (N=13105)		Non-Analytic Sample (N=6138)		$\chi^2$ <sup>a</sup>
	N	%	n	%	
Girl	6446	49.2	2901	47.3	6.197*
White ethnicity (child)	9801	81.7	4123	82.0	ns
Father is university-educated	4909	38.4	233	18.4	198.863***
Age 5 years					
Household income poverty	2969	25.5	1623	67.1	1574.843***
Age 5 years					
Household income poverty	2470	23	1132	60.4	1096.915***
Biological father	9058	94.2	382	86.8	40.002***
Age 11 years					
Household income poverty	1894	18.3	900	51.7	939.700***
Age 14 years					
Household income poverty	2118	22.7	893	61.3	930.927***
Biological father	6448	89.4	216	85	4.881*
Continuous variables	N	Mean (SD)	n	Mean (SD)	t <sup>b</sup>
Age 3					
Child emotional symptoms	12,029	1.33 (1.48)	2707	1.62 (1.68)	8.221***
Child conduct problems	12,050	2.72 (2.02)	2713	3.33 (2.24)	12.985***
Child hyperactivity	11,949	3.82 (2.33)	2676	4.40 (2.47)	10.981***
Child peer difficulties	11,958	1.50 (1.58)	2681	1.83 (1.66)	9.316***
Paternal distress	9856	2.87 (3.13)	43	3.91 (4.55)	-
Maternal distress	11,140	3.10 (3.58)	2343	4.15 (4.44)	10.764***
Age 5					
Child emotional symptoms	11,317	1.33 (1.55)	2053	1.64 (1.76)	7.534***
Child conduct problems	11,328	1.42 (1.45)	2060	1.94 (1.72)	12.899***
Child hyperactivity	11,272	3.18 (2.33)	2037	3.88 (2.50)	11.721***
Child peer difficulties	11,299	1.09 (1.41)	2059	1.53 (1.62)	11.500***
Paternal distress	9384	2.95 (3.34)	13	3.31 (2.84)	-
Maternal distress	10,950	2.96 (3.63)	1927	4.08 (4.57)	10.215***
Age 7					
Child emotional symptoms	10,527	1.47 (1.71)	1784	1.86 (1.98)	7.759***
Child conduct problems	10,545	1.30 (1.48)	1793	1.81 (1.74)	11.599***
Child hyperactivity	10,520	3.25 (2.49)	1779	3.95 (2.63)	10.449***
Child peer difficulties	10,530	1.15 (1.50)	1789	1.62 (1.76)	10.721***



Paternal distress	8310	2.95 (3.41)	4	3.25 (2.75)	-
Maternal distress	10,210	2.93 (3.63)	1681	4.09 (4.72)	9.636***
Age 11					
Child emotional symptoms	10,055	1.80 (1.94)	1639	2.22 (2.24)	7.161***
Child conduct problems	10,053	1.29 (1.50)	1641	1.82 (1.79)	11.273***
Child hyperactivity	10,037	3.00 (2.42)	1634	3.70 (2.57)	10.281***
Child peer difficulties	10,061	1.29 (1.64)	1640	1.76 (1.87)	9.565***
Paternal distress	8203	3.85 (3.90)	4	5.00 (4.24)	-
Maternal distress	9904	3.79 (4.19)	1931	5.44 (5.31)	12.841***
Age 14					
Child emotional symptoms	9051	1.96 (2.09)	1389	2.48 (2.31)	7.981***
Child conduct problems	9054	1.32 (1.55)	1389	1.80 (1.82)	9.356***
Child hyperactivity	9050	2.88 (2.36)	1387	3.52 (2.51)	8.959***
Child peer difficulties	9055	1.65 (1.78)	1391	2.21 (1.94)	10.226***
Paternal distress	6612	3.63 (3.60)	2	3.50 (3.54)	-
Maternal distress	8526	4.15 (4.07)	1228	5.35 (4.86)	8.189***

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

#### 4.7.2. Correlations

For the analytic sample, bivariate correlations were used to explore the associations between paternal psychological distress, maternal psychological distress, and child difficulties (findings presented in tables A1-A5, in Appendix 1). All correlations were significant with effect sizes ranging from weak to moderate. Correlations between paternal and maternal psychological distress were weak (.11-.22), as did correlations between paternal psychological distress and child difficulties (.07-.19). Correlations between maternal distress and child difficulties ranged from weak to moderate, (.15-.39).

#### 4.7.3. SEM results: Full analytic sample

For each SDQ scale, models were initially run unadjusted. Model fit was adequate, with the CFI ranging from 0.90 to 0.93, TLI from 0.81 to 0.86, and the RMSEA from

0.088 to 0.0981 (the recommended values for a good model fit are  $CFI \geq .95$ ,  $TLI \geq .95$  and  $RMSEA < 0.07$ ; Hooper, Coughlan, & Mullen, 2008). Model fit improved after controlling for covariates, with the CFI ranging from 0.91 to 0.93, TLI from 0.84 to 0.88, and the RMSEA from 0.045 to 0.051 (results are displayed in table 4).

Each of the SDQ domains was examined separately. For emotional symptoms ( $CFI=0.92$ ,  $TLI=0.86$ ,  $RMSEA=0.045$ ), father paths were significant across all sweeps, with higher distress being associated with more child symptoms. Child symptoms were not shown to have any influence on paternal distress.

The same pattern of effects was observed for peer difficulties ( $CFI=0.91$ ,  $TLI=0.84$ ,  $RMSEA=0.047$ ). Nevertheless, cross-lagged effects occurred between peer difficulties and paternal distress, ages 11 to 14, with higher levels of difficulties predicting higher levels of distress, and vice versa. The formula by Clogg et al., (1992) was used, to determine which of the effects was larger. It was found that  $z=1.43$ , meaning the two effects did not differ significantly in size.

When it comes to the conduct domain ( $CFI=0.91$ ,  $TLI=0.84$ ,  $RMSEA=0.051$ ), paternal distress at ages 5 and 11 predicted more problems at ages 7 and 14, respectively. Moreover, conduct problems at the age of 3 were associated with elevated levels of paternal distress at the age of 5. Paternal distress at ages 3 and 5 predicted more hyperactivity at ages 11 and 14, respectively ( $CFI=0.93$ ,  $TLI=0.87$ ,  $RMSEA=0.050$ ), while child hyperactivity at the age of 7 predicted higher paternal distress at the age of 11.

Significant cross-sectional relationships were found. Specifically, for the conduct and peer domain, paternal and child difficulties were linked at the ages of 3, 7, 11, and

14. For emotional and hyperactivity difficulties, they were linked at the ages of 3, 11, and 14. At the age of 5, no there were no significant associations.

Regarding covariates, increased maternal distress and living in poverty predicted both higher paternal distress and more child difficulties. The same was the case for lack of paternal higher education, though for fathers, only at child age 14. Girls were more likely to display emotional difficulties at 11 and 14 years, while boys were more likely to display peer and hyperactivity difficulties across all ages. In addition, boys were more likely to display conduct problems at the ages of 5, 7, and 11. Finally, child ethnicity was not shown to have any impact.

**Table 4 Cross-lagged model results** (unstandardized coefficients, standard errors and standardized coefficients), adjusted for covariates

Regression paths	Emotional symptoms				Conduct problems				Hyperactivity				Peer relations			
	B	SE	$\beta$	95% CIs	B	SE	$\beta$	95% CIs	B	SE	$\beta$	95% CIs	B	SE	$\beta$	95% CIs
Stability in paternal psychological distress over time																
Age 3 → Age 5	0.59***	0.01	0.55	0.57, 0.61	0.59***	0.01	0.55	0.57, 0.61	0.59***	0.01	0.55	0.57, 0.61	0.59***	0.01	0.55	0.57, 0.61
Age 5 → Age 7	0.59***	0.01	0.58	0.58, 0.61	0.59***	0.01	0.58	0.58, 0.61	0.59***	0.01	0.58	0.58, 0.61	0.59***	0.01	0.58	0.58, 0.61
Age 7 → Age 11	0.64***	0.01	0.56	0.62, 0.66	0.64***	0.01	0.56	0.62, 0.66	0.64***	0.01	0.56	0.62, 0.66	0.64***	0.01	0.56	0.62, 0.66
Age 11 → Age 14	0.57***	0.01	0.61	0.55, 0.59	0.57***	0.01	0.61	0.55, 0.59	0.57***	0.01	0.61	0.55, 0.59	0.57***	0.01	0.61	0.55, 0.59
Stability in child difficulties over time																
Age 3 → Age 5	0.41***	0.009	0.39	0.40, 0.43	0.32***	0.006	0.44	0.30, 0.33	0.52***	0.008	0.63	0.51, 0.54	0.32***	0.008	0.36	0.31, 0.34
Age 5 → Age 7	0.51***	0.009	0.46	0.49, 0.53	0.56***	0.008	0.55	0.54, 0.57	0.68***	0.008	0.63	0.67, 0.70	0.53***	0.009	0.50	0.51, 0.55
Age 7 → Age 11	0.50***	0.01	0.45	0.48, 0.52	0.53***	0.009	0.52	0.51, 0.54	0.61***	0.007	0.64	0.59, 0.62	0.51***	0.01	0.47	0.49, 0.52
Age 11 → Age 14	0.52***	0.01	0.48	0.50, 0.54	0.59***	0.009	0.57	0.57, 0.61	0.62***	0.008	0.52	0.60, 0.63	0.57***	0.01	0.53	0.56, 0.59
Cross-sectional relationships (covariance) between paternal psychological distress and child difficulties																
Age 3	0.45***	0.05	0.10	0.36, 0.55	0.70***	0.06	0.11	0.58, 0.83	0.60***	0.07	0.08	0.45, 0.74	0.49***	0.05	0.10	0.39, 0.59
Age 5	-0.01	0.04	-0.04	-0.09, 0.07	0.05	0.04	0.01	-0.02, 0.12	0.09	0.06	0.02	-0.01, 0.21	0.05	0.04	0.01	-0.02, 0.13
Age 7	0.08	0.05	0.02	-0.004, 0.17	0.11**	0.04	0.03	0.04, 0.18	0.10	0.06	0.02	-0.009, 0.21	0.08*	0.04	0.02	0.006, 0.16
Age 11	0.24***	0.06	0.05	0.12, 0.37	0.18***	0.05	0.05	0.09, 0.27	0.21**	0.07	0.04	0.08, 0.34	0.17**	0.05	0.04	0.07, 0.28
Age 14	0.30***	0.06	0.06	0.17, 0.42	0.20***	0.05	0.06	0.11, 0.29	0.24***	0.06	0.05	0.12, 0.37	0.13*	0.05	0.03	0.03, 0.24
Cross-lagged relationships between paternal psychological distress and child difficulties																
PD <sub>age3</sub> → CD <sub>age5</sub>	0.02***	0.005	0.04	0.009, 0.03	0.007	0.004	0.01	-0.001, 0.02	0.02**	0.006	0.02	0.006, 0.03	0.01**	0.004	0.03	0.004, 0.02
CD <sub>age3</sub> → PD <sub>age5</sub>	0.008	0.02	0.003	-0.03, 0.05	0.05**	0.02	0.03	0.02, 0.08	0.02	0.01	0.01	-0.008, 0.04	0.0008	0.02	0.0003	-0.04, 0.04
PD <sub>age5</sub> → CD <sub>age7</sub>	0.01*	0.005	0.02	0.001, 0.02	0.01*	0.004	0.02	0.002, 0.02	0.007	0.006	0.003	-0.004, 0.02	0.01**	0.004	0.03	0.004, 0.02
CD <sub>age5</sub> → PD <sub>age7</sub>	0.03	0.02	0.01	-0.01, 0.07	0.006	0.02	0.002	-0.04, 0.05	0.02	0.01	0.01	-0.01, 0.04	0.03	0.02	0.01	-0.02, 0.07
PD <sub>age7</sub> → CD <sub>age11</sub>	0.02**	0.005	0.03	0.006, 0.03	-0.0006	0.004	-0.01	-0.008, 0.008	-0.002	0.006	0.01	-0.01, 0.01	0.01*	0.005	0.03	0.003, 0.02
CD <sub>age7</sub> → PD <sub>age11</sub>	0.008	0.02	0.004	-0.04, 0.05	0.02	0.03	0.008	-0.03, 0.08	0.04*	0.02	0.02	0.008, 0.07	0.05	0.03	0.02	0.0006, 0.11
PD <sub>age11</sub> → CD <sub>age14</sub>	0.02**	0.005	0.03	0.006, 0.03	0.02***	0.004	0.04	0.008, 0.02	0.01**	0.05	0.02	0.004, 0.02	0.02***	0.005	0.04	0.009, 0.03
CD <sub>age11</sub> → PD <sub>age14</sub>	0.02	0.02	0.01	-0.01, 0.06	0.04	0.03	0.02	-0.01, 0.10	-0.004	0.02	0.003	-0.04, 0.03	0.05*	0.02	0.02	0.001, 0.09

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .  $\beta$  = standardized beta coefficient, CIs=Confidence Intervals, PD=paternal psychological distress, CD = child difficulties

#### **4.7.4. Moderation**

Multi-group comparisons were used to examine whether the child's gender might moderate the significant effects observed. Using the Likelihood-Ratio test, the unconstrained models (in which all parameters were free to differ between groups) were compared to the constrained ones (in which all parameters were set to be equal across groups). For each SDQ domain, the difference between the models was significant;  $\chi^2(191)=665.23$ ,  $p<.001$ ;  $\chi^2(191)=749.91$ ,  $p<.001$ ;  $\chi^2(191)=1189.19$ ,  $p<.001$ ; and  $\chi^2(191)=547.47$ ,  $p<.001$ ; for emotional, conduct, hyperactivity and peer difficulties, respectively. The significant differences indicate that the paths should be allowed to vary, meaning that there were differences between boys and girls.

Subsequently, to pinpoint which specific paths differed between the 2 genders, step-by-step multi-group comparisons were performed. The Likelihood-Ratio test was used to compare the unconstrained model to the constrained one (in which, though, only 1 path was constrained each time).

Outcomes revealed one moderation effect, regarding peer difficulties,  $\chi^2(1)=4.53$ ,  $p=.03$ . Paternal distress at age 11 predicted boys' difficulties at age 14, but not girls' difficulties. No other moderation effects were identified.

#### **4.7.5. First Sensitivity Analysis (5412 cases)**

Results for the sensitivity analysis including only households with a father-figure continuously across child ages 3-14 are presented in Table 5. There were a few differences, compared to the full analytic sample. Specifically, for emotional difficulties, only father paths at the ages of 7 and 11 remained significant, while for peer difficulties, only father paths at the ages of 3, 5, and 11 remained significant. The cross-lagged effects between the ages of 11 and 14 persisted with the two paths

not differing significantly in strength ( $z = 1.17$ ). Furthermore, another set of cross-lagged effects was found between paternal distress and child conduct problems, across the ages of 3 and 5, with none of them being stronger than the other ( $z = 0.56$ ). Additionally, paternal distress at the age of 5 ceased to predict child conduct problems at 7, and hyperactivity difficulties at the age of 7 ceased to predict paternal distress at 11.

Following the same procedure as with the full analytic sample, multi-group comparisons were performed, to examine the moderating role of gender. The models were shown to differ significantly for emotional, conduct, hyperactivity, and peer difficulties;  $\chi^2(191)=578.15$ ,  $p<.001$ ;  $\chi^2(191)=607.98$ ,  $p<.001$ ;  $\chi^2(191)=859.07$ ,  $p<.001$ ; and  $\chi^2(191)=553.03$ ,  $p<.001$ ; respectively. There were no differences between boys and girls, with paternal distress at 11 predicting both genders' peer difficulties.

**Table 5 Sensitivity analysis 5412 cases** (unstandardized coefficients, standard errors and standardized coefficients), adjusted

Regression paths	Emotional symptoms				Conduct problems				Hyperactivity				Peer relations			
	B	SE	β	95% CIs	B	SE	β	95% CIs	B	SE	β	95% CIs	B	SE	β	95% CIs
<b>Stability in paternal psychological distress over time</b>																
Age 3 → Age 5	0.60***	0.01	0.54	0.57, 0.63	0.60***	0.01	0.54	0.57, 0.63	0.60***	0.01	0.54	0.57, 0.63	0.60***	0.01	0.54	0.57, 0.63
Age 5 → Age 7	0.59***	0.01	0.57	0.57, 0.61	0.59***	0.01	0.57	0.57, 0.61	0.59***	0.01	0.57	0.57, 0.61	0.59***	0.01	0.57	0.57, 0.61
Age 7 → Age 11	0.63***	0.01	0.55	0.60, 0.66	0.63***	0.01	0.55	0.60, 0.66	0.63***	0.01	0.55	0.60, 0.66	0.63***	0.01	0.55	0.60, 0.66
Age 11 → Age 14	0.58***	0.01	0.61	0.56, 0.60	0.58***	0.01	0.61	0.56, 0.60	0.58***	0.01	0.61	0.56, 0.60	0.58***	0.01	0.61	0.56, 0.60
<b>Stability in child difficulties over time</b>																
Age 3 → Age 5	0.42***	0.01	0.39	0.39, 0.44	0.30***	0.01	0.53	0.28, 0.32	0.54***	0.01	0.54	0.52, 0.56	0.33***	0.01	0.37	0.30, 0.35
Age 5 → Age 7	0.52***	0.01	0.47	0.50, 0.55	0.53***	0.01	0.50	0.51, 0.56	0.68***	0.01	0.64	0.66, 0.71	0.53***	0.01	0.50	0.51, 0.56
Age 7 → Age 11	0.50***	0.01	0.45	0.48, 0.53	0.50***	0.01	0.54	0.47, 0.52	0.60***	0.01	0.62	0.58, 0.62	0.49***	0.01	0.45	0.46, 0.51
Age 11 → Age 14	0.53***	0.01	0.50	0.51, 0.56	0.56***	0.01	0.42	0.54, 0.59	0.62***	0.01	0.63	0.60, 0.64	0.59***	0.01	0.54	0.56, 0.61
<b>Cross-sectional relationships (covariance) between paternal psychological distress and child difficulties</b>																
Age 3	0.42***	0.06	0.11	0.31, 0.54	0.51***	0.08	0.09	0.36, 0.66	0.41***	0.09	0.06	0.22, 0.59	0.31***	0.06	0.17	0.19, 0.44
Age 5	0.03	0.05	0.009	-0.07, 0.13	0.03	0.04	0.009	-0.06, 0.11	0.11	0.07	0.02	-0.02, 0.24	0.02	0.05	0.007	-0.07, 0.11
Age 7	0.07	0.05	0.02	-0.03, 0.18	0.17***	0.04	0.06	0.09, 0.25	0.21**	0.06	0.05	0.09, 0.34	0.03	0.04	0.008	-0.06, 0.11
Age 11	0.22**	0.07	0.05	0.09, 0.35	0.21***	0.05	0.06	0.11, 0.30	0.23**	0.07	0.05	0.08, 0.37	0.12*	0.06	0.03	0.01, 0.23
Age 14	0.23***	0.06	0.05	0.10, 0.35	0.20***	0.05	0.06	0.11, 0.29	0.22**	0.06	0.05	0.09, 0.34	0.11*	0.05	0.03	0.004, 0.21
<b>Cross-lagged relationships between paternal psychological distress and child difficulties</b>																
PD <sub>age3</sub> → CD <sub>age5</sub>	0.01	0.007	0.02	-0.004, 0.02	0.02**	0.006	0.04	0.008, 0.03	0.03**	0.009	0.04	0.01, 0.05	0.02**	0.006	0.05	0.01, 0.03
CD <sub>age3</sub> → PD <sub>age5</sub>	-0.01	0.03	-0.004	-0.07, 0.05	0.04*	0.02	0.03	0.004, 0.08	0.01	0.02	0.007	-0.02, 0.04	0.02	0.03	0.01	-0.03, 0.07
PD <sub>age5</sub> → CD <sub>age7</sub>	0.006	0.006	0.01	-0.006, 0.02	0.003	0.005	0.006	-0.007, 0.01	0.0004	0.008	0.0005	-0.02, 0.02	0.02**	0.005	0.04	0.006, 0.03
CD <sub>age5</sub> → PD <sub>age7</sub>	0.01	0.03	0.006	-0.04, 0.06	0.02	0.03	0.008	-0.04, 0.08	0.003	0.02	0.002	-0.03, 0.04	0.008	0.03	0.003	-0.05, 0.06
PD <sub>age7</sub> → CD <sub>age11</sub>	0.02**	0.007	0.04	0.008, 0.04	-0.002	0.005	-0.004	-0.01, 0.008	0.0002	0.008	0.0003	-0.01, 0.02	0.01	0.006	0.02	-0.0002, 0.02
CD <sub>age7</sub> → PD <sub>age11</sub>	0.02	0.03	0.008	-0.03, 0.07	-0.001	0.03	-0.004	-0.07, 0.06	0.02	0.02	0.01	-0.02, 0.05	0.06	0.03	0.02	-0.005, 0.12
PD <sub>age11</sub> → CD <sub>age14</sub>	0.03***	0.006	0.05	0.01, 0.04	0.02***	0.004	0.05	0.01, 0.03	0.02**	0.006	0.03	0.007, 0.03	0.02***	0.005	0.05	0.01, 0.03
CD <sub>age11</sub> → PD <sub>age14</sub>	0.04	0.02	0.02	-0.002, 0.08	0.01	0.03	0.005	-0.05, 0.07	0.004	0.02	0.003	-0.03, 0.04	0.08**	0.03	0.03	0.03, 0.13

\**p* < .05, \*\**p* < .01, \*\*\**p* < .001. β = standardized beta coefficient, CIs=Confidence Intervals, PD=paternal psychological distress, CD=child difficulties

#### **4.7.6. Second Sensitivity Analysis (5258 cases)**

Last, the findings for the sensitivity analysis including only families with a biological father across child ages 3-14 are displayed in Table 6. For emotional symptoms, paternal influences at the age of 7 remained, but not at 3 and 5. Moreover, a set of cross-lagged effects was found between paternal distress and emotional symptoms, across ages 11 and 14, with those links being of similar strength ( $z = -0.87$ ). For peer difficulties, paternal paths were identified for the ages of 3 and 5, while there was also a child path at the age of 7. The reciprocal relationships between ages 11 and 14 remained, with no effect being stronger than the other ( $z = 1.92$ ). With regards to conduct problems, the cross-lagged effects between ages 3 and 5 were found again, with their strength being similar ( $z = 1.36$ ). Finally, paternal distress at the age of 11 led to more conduct problems and hyperactivity at the age of 14, while paternal distress at the age of 3 led to more hyperactivity at the age of 5. Regarding the moderating role of gender, it was not possible to explore it, as models failed to converge.



**Table 6 Sensitivity analysis 5258 cases** (unstandardized coefficients, standard errors and standardized coefficients), adjusted

Regression paths	Emotional symptoms				Conduct problems				Hyperactivity				Peer relations			
	B	SE	$\beta$	95% CIs	B	SE	$\beta$	95% CIs	B	SE	$\beta$	95% CIs	B	SE	$\beta$	95% CIs
Stability in paternal psychological distress over time																
Age 3 → Age 5	0.61***	0.01	0.55	0.58, 0.63	0.61***	0.01	0.55	0.58, 0.63	0.61***	0.01	0.55	0.58, 0.63	0.61***	0.01	0.55	0.58, 0.63
Age 5 → Age 7	0.59***	0.01	0.57	0.57, 0.62	0.59***	0.01	0.57	0.57, 0.62	0.59***	0.01	0.57	0.57, 0.62	0.59***	0.01	0.57	0.57, 0.62
Age 7 → Age 11	0.63***	0.01	0.55	0.60, 0.66	0.63***	0.01	0.55	0.60, 0.66	0.63***	0.01	0.55	0.60, 0.66	0.63***	0.01	0.55	0.60, 0.66
Age 11 → Age 14	0.58***	0.01	0.61	0.56, 0.60	0.58***	0.01	0.61	0.56, 0.60	0.58***	0.01	0.61	0.56, 0.60	0.58***	0.01	0.61	0.56, 0.60
Stability in child difficulties over time																
Age 3 → Age 5	0.42***	0.01	0.39	0.39, 0.44	0.30***	0.009	0.52	0.28, 0.32	0.54***	0.01	0.54	0.51, 0.56	0.33***	0.01	0.37	0.30, 0.35
Age 5 → Age 7	0.52***	0.01	0.47	0.49, 0.55	0.53***	0.01	0.49	0.51, 0.55	0.69***	0.01	0.64	0.66, 0.71	0.53***	0.01	0.50	0.51, 0.56
Age 7 → Age 11	0.50***	0.01	0.45	0.48, 0.53	0.49***	0.01	0.54	0.46, 0.52	0.60***	0.01	0.62	0.57, 0.62	0.49***	0.01	0.45	0.46, 0.51
Age 11 → Age 14	0.53***	0.01	0.49	0.50, 0.55	0.54***	0.01	0.43	0.52, 0.57	0.62***	0.01	0.63	0.59, 0.64	0.58***	0.01	0.53	0.56, 0.61
Cross-sectional relationships (covariance) between paternal psychological distress and child difficulties																
Age 3	0.43***	0.06	0.11	0.32, 0.55	0.54***	0.08	0.10	0.39, 0.70	0.43***	0.10	0.07	0.24, 0.62	0.31***	0.07	0.07	0.19, 0.44
Age 5	0.03	0.05	0.01	-0.07, 0.13	0.03	0.04	0.01	-0.06, 0.11	0.08	0.07	0.02	-0.05, 0.21	0.03	0.05	0.01	-0.06, 0.12
Age 7	0.07	0.05	0.02	-0.03, 0.18	0.18***	0.04	0.07	0.10, 0.26	0.22***	0.07	0.05	0.09, 0.34	0.02	0.03	0.008	-0.06, 0.11
Age 11	0.22***	0.07	0.05	0.09, 0.35	0.22***	0.05	0.07	0.13, 0.31	0.24***	0.07	0.05	0.10, 0.38	0.14*	0.06	0.04	0.03, 0.25
Age 14	0.19***	0.06	0.04	0.07, 0.32	0.20***	0.04	0.06	0.11, 0.28	0.21***	0.06	0.05	0.09, 0.34	0.10	0.05	0.03	-0.002, 0.21
Cross-lagged relationships between paternal psychological distress and child difficulties																
PD <sub>age3</sub> → CD <sub>age5</sub>	0.01	0.007	0.02	-0.002, 0.03	0.02***	0.006	0.05	0.01, 0.03	0.03**	0.01	0.04	0.01, 0.05	0.02***	0.006	0.05	0.01, 0.04
CD <sub>age3</sub> → PD <sub>age5</sub>	-0.01	0.03	-0.004	-0.07, 0.05	0.05*	0.02	0.03	0.008, 0.09	0.02	0.02	0.01	-0.02, 0.05	0.04	0.03	0.02	-0.01, 0.09
PD <sub>age5</sub> → CD <sub>age7</sub>	0.006	0.006	0.01	-0.007, 0.02	0.002	0.005	-0.002	-0.008, 0.01	-0.001	0.008	-0.02	-0.02, 0.01	0.02**	0.006	0.04	0.007, 0.03
CD <sub>age5</sub> → PD <sub>age7</sub>	0.01	0.03	0.006	-0.04, 0.06	0.02	0.03	0.008	-0.04, 0.08	0.01	0.02	0.007	-0.02, 0.04	0.01	0.03	0.005	-0.04, 0.07
PD <sub>age7</sub> → CD <sub>age11</sub>	0.02**	0.007	0.04	0.008, 0.04	-0.007	0.005	0.004	-0.01, 0.009	0.0003	0.008	0.004	-0.02, 0.02	0.01	0.006	0.02	-0.0007, 0.02
CD <sub>age7</sub> → PD <sub>age11</sub>	0.02	0.03	0.009	-0.03, 0.08	-0.006	0.03	-0.002	-0.07, 0.06	0.02	0.02	0.01	-0.01, 0.06	0.07*	0.03	0.03	0.005, 0.13
PD <sub>age11</sub> → CD <sub>age14</sub>	0.02**	0.006	0.04	0.01, 0.03	0.02***	0.05	0.05	0.01, 0.03	0.02**	0.006	0.03	0.005, 0.03	0.02***	0.05	0.05	0.01, 0.03
CD <sub>age11</sub> → PD <sub>age14</sub>	0.04*	0.02	0.02	0.0007, 0.09	0.007	0.03	0.003	-0.05, 0.07	-0.006	0.02	-0.004	-0.04, 0.03	0.07**	0.03	0.03	0.02, 0.12

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .  $\beta$  = standardized beta coefficient., CIs=Confidence Intervals, PD=paternal psychological distress, CD=child difficulties

#### 4.7.7. Mediation analysis

Using the sub-sample with only biological fathers, a mediation analysis was conducted, for the paths between child ages 3 to 5 and 5 to 7. Five significant paths were identified: paternal distress at age 3 led to conduct, hyperactivity, and peer difficulties at age 5; conduct problems at age 3 led to paternal distress at age 5; and paternal distress at age 5 led to peer difficulties at age 7. For the paths between ages 3 and 5, the potential mediators examined involve paternal marital satisfaction, paternal closeness to the child, and paternal hostility towards the child (all measured at age 3). The mediators examined between ages 5 and 7 were paternal marital satisfaction and paternal involvement (both measured at age 5). The percentage of missing data was 16%, 16%, 16.8%, 10.8%, and 5.2% for paternal closeness, conflict, marital satisfaction (age 3), marital satisfaction (age 5), and involvement, respectively. Descriptive information on the mediators and correlations between the mediators, paternal distress, and child difficulties, can be found in tables A6 and A7 (Appendix 1).

Marital satisfaction, closeness, and conflict were shown to fully mediate the cross-lagged effects between paternal distress and conduct problems, ages 3 to 5 (direct, indirect, and total effects presented in Table 7). Paternal distress at the age of 3 was linked to lower marital satisfaction ( $b=-0.31$ ,  $SE=0.01$ ,  $p<0.001$ ), lower closeness ( $b=-0.12$ ,  $SE=0.01$ ,  $p<0.001$ ), and higher conflict ( $b=0.65$ ,  $SE=0.03$ ,  $p<0.001$ ). In turn, marital satisfaction ( $b=-0.02$ ,  $SE=0.008$ ,  $p<0.05$ ), closeness ( $b=-0.02$ ,  $SE=0.008$ ,  $p<0.05$ ), and conflict ( $b=0.01$ ,  $SE=0.004$ ,  $p<0.05$ ) were shown to predict more child conduct problems at 5. Similarly, conduct problems at 3 were associated with lower marital satisfaction ( $b=-0.11$ ,  $SE=0.02$ ,  $p<0.001$ ), lower closeness ( $b=-$

0.13,  $SE=0.02$ ,  $p<0.001$ ), and higher conflict ( $b=0.77$ ,  $SE=0.04$ ,  $p<0.001$ ). Paternal distress at child 5 was predicted by low levels of marital satisfaction ( $b=-0.04$ ,  $SE=0.02$ ,  $p<0.05$ ), high levels of conflict ( $b=0.03$ ,  $SE=0.008$ ,  $p<0.001$ ), as well as high levels of closeness ( $b=0.07$ ,  $SE=0.02$ ,  $p<0.001$ ).

Furthermore, the effect of paternal distress at age 3 on child hyperactivity at age 5 was fully mediated by closeness and conflict, but not marital satisfaction (direct, indirect and total effects on Table 7). Paternal distress at 3 predicted lower levels of closeness ( $b=-0.12$ ,  $SE=0.01$ ,  $p<0.001$ ) and higher levels of conflict ( $b=0.68$ ,  $SE=0.03$ ,  $p<0.001$ ). Hyperactivity at 5 was in turn negatively linked to closeness ( $b=-0.02$ ,  $SE=0.01$ ,  $p<0.05$ ), and positively linked to conflict ( $b=0.02$ ,  $SE=0.006$ ,  $p<0.01$ ). Paternal distress did also predict marital satisfaction ( $b=-0.32$ ,  $SE=0.01$ ,  $p<0.001$ ); however, marital satisfaction was not related to hyperactivity ( $b=-0.01$ ,  $SE=0.01$ ,  $p=0.301$ ).

When it comes to peer difficulties, the father path at age 3 was partially mediated by closeness (direct, indirect and total effects on Table 7). Paternal distress resulted in a less close father-child relationship ( $b=-0.12$ ,  $SE=0.01$ ,  $p<0.001$ ), which in turn resulted in more peer difficulties ( $b=-0.04$ ,  $SE=0.008$ ,  $p<0.001$ ). Conflict and marital satisfaction were not mediators, as they were linked to paternal distress ( $b=0.69$ ,  $SE=0.03$ ,  $p<0.001$ ;  $b=-0.32$ ,  $SE=0.01$ ,  $p<0.001$ ; for conflict and marital satisfaction, respectively), but not hyperactivity ( $b=0.0005$ ,  $SE=0.004$ ,  $p=0.894$ ;  $b=0.001$ ,  $SE=0.009$ ,  $p=0.870$ ; for conflict and marital satisfaction, respectively).

For peer difficulties, there was also a father path at age 5, which was partially mediated by paternal involvement. Particularly, paternal distress predicted lower levels of involvement ( $b=-0.13$ ,  $SE=0.02$ ,  $p<0.001$ ), which in turn predicted more

peer difficulties ( $b=-0.008$ ,  $SE=0.003$ ,  $p<0.05$ ). Marital satisfaction was predicted by paternal distress ( $b=-0.29$ ,  $SE=0.01$ ,  $p<0.001$ ); however, it had no effect on peer difficulties ( $b=-0.001$ ,  $SE=0.007$ ,  $p=0.844$ ).

**Table 7** Direct, indirect and total effects between child difficulties and paternal distress

	Direct Effects from IV		Indirect Effects from IV		Total Effects from IV	
	b (SE)	B	b (SE)	B	b (SE)	$\beta$
PD (age 3) → Conduct Problems (age 5)	.006 (.007)	.01	.02 (.003)***	.03	.02 (.006)**	.05
Conduct Problems (age 3) → PD (age 5)	.03 (.02)	.02	.02 (.007)**	.01	.05 (.02)*	.03
PD (age 3) → Hyperactivity (age 5)	.01 (.01)	.02	.02 (.005)***	.02	.03 (.01)**	.04
PD (age 3) → Peer Difficulties (age 5)	.02 (.007)*	.04	.004 (.003)	.009	.02 (.007)**	.05
PD (age 5) → Peer Difficulties (age 7)	.02 (.006)**	.03	.002 (.002)	.003	.02 (.006)**	.04

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , PD=Paternal Distress, IV=Independent Variable

#### 4.8. Discussion

This research examined the transactional links between paternal psychological distress and offspring internalising and externalising difficulties, from 3 to 14 years, using a large and nationally representative longitudinal sample. Using sub-group analysis, the moderating effects of the child's gender were also explored. In addition, to confirm the robustness of the results, 2 sensitivity analyses were conducted; the first involving only consistent 2-parent families, and the second, consistent 2-parent families, in which the father is also biological. Subsequently, using the subsample including only biological families, the role of potential mediators (marital conflict, father-child closeness/warmth, father-child conflict/hostility, and fathers' involvement) was evaluated between child ages 3 to 5 and 5 to 7 (which the data allowed).

Importantly, covariates such as maternal psychological distress, family income, and prior child difficulties, were controlled for in all analyses.

#### **4.8.1. Father effects**

The main analysis revealed several father paths, especially for emotional and peer difficulties, which was in line with the study's hypothesis. These outcomes agree with some of the past studies (Cioffi et al., 2018; Fanti et al., 2013; Gross et al., 2008), while not others, who either found only child paths (Villarreal & Nelson, 2018; Hou et al., 2021) or no paths at all (Hastings et al., 2020). Methodological and measurement differences may potentially account for this discrepancy, since some studies assessed paternal anxiety instead of paternal distress (Villarreal & Nelson, 2018) or investigated children in mid-adolescence, which is slightly older than the ages the present research focused on (Hastings et al., 2020; Hou et al., 2021).

In the current findings, the higher incidence of father influences in the internalising domain, compared to the externalising domain, could be attributed to the known genetic links that exist between parent and child mental well-being (Kendler et al., 2003). However, some of these influences did not persist in the sensitivity analysis involving only biological fathers, suggesting that environmental factors, such as changes in the family structure may also play a role (Fitzsimons & Villadsen, 2019).

It is also noticeable that paternal effects occurred from age 11 to age 14 in all difficulties domains and remained in both sensitivity analyses. Therefore, it can be concluded that fathers' distress may have a greater impact in adolescence, compared to earlier years. A possible interpretation is that, as children grow, their cognitive processes mature, and they become more capable of understanding and

interpreting their family environment. Hence, they are more likely to perceive the distress experienced by their fathers and to be affected by it (Shafer et al., 2017).

Another interpretation, concerning externalising difficulties, is that fathers with high distress fail to provide their adolescent offspring with sufficient monitoring. During adolescence, children start to become more independent and seek autonomy (Chen, 2010). Though autonomy-seeking is an integral element of transitioning to adulthood, it also entails greater opportunities for adolescents to be involved in delinquency (Chen, 2010). Paternal monitoring is shown to have a protective role and helps prevent risky adolescent engagement (Padilla-Walker, Coyne, & Collier, 2016). Parents who experience elevated levels of distress tend to be uninvolved and provide only limited monitoring (Kelly, Becker, & Spirito, 2017). As a result, their offspring face elevated risks of becoming deviant and of demonstrating externalising problems. Since MCS data do not provide adequate parental monitoring measures, it was not possible to evaluate this hypothesis.

#### **4.8.2. Child effects**

Child paths were noticeably fewer than father paths, indicating that it is mainly fathers who influence child well-being. This outcome is again in line with past research, and the hypothesis that had been drawn. Out of the child effects found in the present research, most concerned conduct, and hyperactivity difficulties. Compared to internalising difficulties, externalising difficulties are shown to cause more family disruption; hence, they are more likely to have an adverse impact on caregivers (Meltzer et al., 2011).

#### **4.8.3. Reciprocal links**

Bidirectional paths were found between paternal psychological distress and adolescent peer difficulties, between ages 11 and 14. These reciprocal associations persisted in both sensitivity analyses and did not differ significantly in size.

These findings are partly in line with those reported by Fanti et al., (2013). In their study, there were bidirectional paths in the internalising domain during early adolescence (11 to 12 years), but not in later adolescence (12 to 15 years), when offspring symptoms did not predict paternal distress. The cross-lagged effects yielded from the present research concern only peer difficulties, while the conclusions from Fanti et al., (2013) refer to overall internalising problems. Furthermore, in the present research, several important confounders, such as maternal distress and family poverty, were adjusted for; this was not the case in the study by Fanti et al., (2013).

Several reasons may explain the paternal links to adolescent peer difficulties. For instance, scholars suggest that fathers, through interactions and play, promote their children's independence and healthy risk-taking (Keizer et al., 2019), attributes important in helping them form successful peer relationships (Webster et al., 2013). Distressed fathers are likely to be less engaged with their children (Flouri et al., 2016; Dette-Hagenmeyer & Reichle, 2014); therefore, their offspring may miss out on the aforementioned father-child interactions and their associated benefits for social development.

Moreover, adults experiencing psychological distress often tend to be lonely (Erzen & Çikrikci, 2018), meaning that those fathers are likely to lack strong social relationships. Consequently, their children may not have had the opportunities to

observe their parents' social interactions, which could prevent them from building their own social skills, and consequently, forming relationships (Salo et al., 2020).

When it comes to the child effect, peer difficulties could indicate that the adolescent is experiencing bullying (as some items in the peer scale capture peer victimisation). In this case, it is possible that the father not only is anxious over the child's well-being but also views himself as an inadequate parent; feelings which could be linked to psychological distress (Sawyer et al., 2011). Additionally, adolescents who are victimized are likely to engage in risky behaviour, such as alcohol drinking, which could also contribute to paternal worry (Rusby et al., 2005; Topper et al., 2011).

#### **4.8.4. Moderation Results**

The results from the moderation analysis in the main sample revealed that offspring gender moderated the effect of paternal psychological distress on peer difficulties, from age 11 to age 14. Specifically, while paternal distress significantly predicted males' peer relations, with higher distress predicting more difficulties, it was not linked to females' peer relations. No such differences were observed for earlier ages or other types of difficulties. Furthermore, this moderating effect did not persist in the sensitivity analysis, possibly due to the smaller sample that was used.

There is a small amount of past research that has assessed the role of gender at similar developmental stages (Fanti et al., 2013; Flouri et al., 2019). These studies though did not report any differences between boys and girls. This discrepancy could perhaps be attributed to variations in the research designs and the measurements used.

It is noticeable that gender differences in peer relations emerged only at the age of 11, but not earlier. According to the gender intensification theory, when offspring



reach early adolescence, they begin to experience greater pressure to conform to their gender roles (Crouter et al., 1995; Galambos et al., 1990). Consequently, they may view the parent of the same gender as them as their role model and start “copying” their behaviours and attitudes (Crouter et al., 1995). This means that, at this developmental stage, boys may be more strongly influenced by their fathers than girls. Distressed fathers are likely to find social interactions stressful, leading themselves to isolation (Cui et al., 2007; Gotlib et al., 2004; Teo, Choi & Valenstein, 2013). In early adolescence, sons of those fathers may also start perceiving their social interactions negatively and may choose to withdraw from their peers, failing in this way to build healthy relationships. Girls on the other hand, who tend to identify more with their mothers, are less likely to adopt these maladaptive ways of behaving that their fathers might follow.

Moreover, it is argued that, during adolescence, girls place greater value on their peer relationships compared to boys (Gorrese & Ruggieri, 2012; Nickerson & Nagle, 2005; Song, Thompson & Ferrer, 2009; Wilkinson, 2004). Their friendships are described as more trustful and warmer, meaning that they can gain more emotional support from them (Nickerson & Nagle, 2005). Being closer to their peers could perhaps buffer the effects of paternal psychological distress for girls, a hypothesis that could be further explored by future studies.

#### **4.8.5. Sensitivity analysis**

Compared to the sensitivity analyses, slightly more father paths were observed in the main analysis. The main analysis included families which underwent changes in the father figure throughout child ages 3-14. As a result, this increased number of father paths may be partly due to these alternations in the family structure. Research

supports that, in blended families, caregivers experience higher psychological distress, compared to two-parent, biological families (Gath, 2022). Transitioning to a new family can be challenging for stepfathers, as they may struggle to adapt to the parental role and the expectations it entails (Cartwright, 2012). Therefore, the increased level of difficulties they face is likely to have a stronger impact on children.

In addition, both sensitivity analyses revealed a set of cross-lagged effects between paternal distress and conduct problems, between ages 3 and 5. The reasons explaining these cross-lagged effects are further investigated in the discussion concerning the mediation analysis. Nevertheless, it should be noted that the father path was only found in the sensitivity analyses, not in the main analysis (the child path was found in the main as well as in the sensitivity analyses). Instead, a father path for emotional difficulties, from age 3 to age 5, occurred in the main analysis, which did not prevail in the sensitivity analyses. For families in the main sample, fathers who experienced high levels of distress may have left the family. Marital dissolution is more strongly associated with internalising rather than externalising difficulties in children; this may account for why a link was observed for emotional rather than conduct problems (Fitzsimons & Villadsen, 2019). On the contrary, in both sensitivity analyses, fathers with high levels of distress remained in the family, potentially using harsh parenting towards the child, and which could be linked to conduct problems.

Furthermore, in the sensitivity analysis involving only biological fathers, another set of cross-lagged effects emerged. Specifically, there were reciprocal relationships between paternal distress and child emotional difficulties, across ages 11 to 14. In this case, while the father path existed in the main analysis, the sensitivity analysis also yielded a child path. This outcome is in line with the cross-lagged effects found

between paternal depressive symptoms and adolescent internalising difficulties in the study by Fanti et al., (2013). In the current sample, and in line with research literature (Durbeej et al., 2019), emotional difficulties peak in adolescence. Fathers (and especially fathers who have been living consistently with the child) are likely to notice this deterioration in their children's mood, which may in turn provoke their own feeling of anxiety and distress. Moreover, biological fathers whose children develop emotional difficulties are likely to share the same genetic vulnerability/predisposition towards mood disorders as them (Lopizzo et al., 2016). Hence, a stressor in their environment (witnessing their child struggle with emotional difficulties) is likely to have a significant impact on their wellbeing (Lopizzo et al., 2016).

#### **4.8.6. Mediation results**

Using the sub-sample including only biological fathers, the mediating pathways between paternal distress and child difficulties, across ages 3 to 5 and 5 to 7, were investigated. The cross-lagged effects, between paternal distress and conduct problems, across ages 3 and 5, were fully mediated by marital conflict, paternal closeness, and paternal hostility. Past research has demonstrated that there are reciprocal links between child conduct problems and marital satisfaction (Cui et al., 2007), as well as between conduct problems and parenting practices. It had also demonstrated that marital satisfaction and parenting practices mediate father paths; however, no studies have evaluated whether they may also mediate child paths. The present findings expand those conclusions by showing that both father and child influences are fully accounted for by these family factors.

It had been hypothesized that both paternal distress and conduct problems would predict lower levels of marital satisfaction, lower levels of father-child closeness and

higher levels of father-child conflict, which in turn would escalate one another. Findings mostly confirmed these hypotheses; however, there was an unexpected outcome. Particularly, though conduct problems lowered father-child closeness, lack of closeness decreased paternal distress, instead of increasing it. It may be the case that being distant from a child with conduct problems has a positive effect on parents' well-being, perhaps by "removing" their worry about how to manage their children's behaviours.

The impact of paternal distress at age 3 on child hyperactivity at age 5 was fully mediated by father-child closeness and father-child conflict. In line with past research, paternal distress led to lower closeness and higher conflict, which in turn heightened child hyperactivity. Marital conflict, on the other hand, was not related to hyperactivity.

For peer difficulties, the father path from age 3 to age 5, was partially mediated by paternal closeness. Having a close relationship with their fathers may help children acquire attributes necessary to form friendships, such as sharing (Kochanska et al., 2010). Children of distressed fathers may be lacking the opportunities at home to learn those ways of behaving which could help them build relationships outside of their family environment. Additionally, lack of involvement partially explained why paternal distress predicted peer difficulties, from age 5 to 7. Distressed fathers tend to be more disengaged; for example, they are less likely to play with their children or take them to outdoor activities (Dette-Hagenmeyer & Reichle, 2016; Flouri et al., 2016). Again, this could reduce the opportunities for children to socialize and to develop the skills necessary for forming friendships (Dette-Hagenmeyer & Reichle, 2016; Flouri et al., 2016).

While the paths between paternal distress and externalising difficulties appear to be fully accounted for by environmental factors, the same is not the case for peer difficulties. The mediating role of both closeness and involvement was partial; paternal psychological distress continued to influence peer relations directly. Other factors, unassessed in the present investigation, could perhaps account for this link further. Current findings may also suggest that paternal distress has a direct impact on children's social development. Possibly, psychological distress and peer relations could be related to one another through genetic mechanisms. For example, emerging evidence indicates that hereditary personality traits might affect people's capacity to form friendships (Horwitz, Reynolds & Charles, 2015). Individuals with more friendships are in turn more likely to elicit emotional support from their environment, which could hinder the development of psychological distress (Horwitz et al., 2015).

#### **4.8.7. Limitations and directions for future research**

There are some limitations of this research that should be considered. To start with, cross-lagged models are limited by the specific timepoints at which measures were taken. Second, the lagged parameters acquired with the cross-lagged panel modelling approach do not represent the actual within-person relationships over time, resulting in inaccurate conclusions regarding the presence, predominance, and sign of causal influences (Hamaker et al., 2015). Third, the year differences between the time-points measured were 2, 2, 4, and 3 years, respectively, meaning that they were not equidistant. Fourth, the child assessments used were completed by the main caregiver, which was most usually the mother. This could lead to some reporter-bias, particularly in cases in which the mother experienced high levels of distress or other mental health difficulties. Research suggests that, compared to

mentally healthy caregivers, caregivers with mental health difficulties are more likely to perceive their children's behaviour as problematic (Gartstein et al., 2009). Fifth, the comparison of the analytic and non-analytic samples revealed some sample selection bias, since those in the analytic sample overall reported fewer difficulties and were more likely to come from a high socio-economic status. Sixth, the mediators used were not measured in multiple sweeps. As a result, it is not possible to conclude whether their role may differ across different ages. Such outcomes would have important implication effects since, depending on the child's age, they would allow making recommendations on what family processes policies and clinicians should focus on. Seventh, due to the unavailability of measures in later sweeps, it was not possible to investigate the mediating pathways beyond the child's age of 7 years. Eighth, the effect sizes yielded from this research, even for statistically significant effects, were quite small. Another limitation is that for some of the sweeps in the emotional, conduct, and peer domain, Cronbach's alphas ranged from .5 to .6.

Finally, the limitations of the mediation approach used should be considered. Mediation was tested using the Sobel's test (Sobel, 1982). Even though considered a conservative and underpowered approach (Pan et al., 2018), Sobel's test is appropriate to use when a large sample is involved, as was the case in the current research (Özdil & Kutlu, 2019; Pan et al., 2018). However, this method still includes some limitations. Mainly, Sobel's test assumes that the variables are normally distributed, which may not be true in many cases (Fritz & MacKinnon, 2007), and could result in inaccurate confidence interval values (MacKinnon, Lockwood, & Williams, 2004). Additionally, Sobel's approach lacks some of the strengths of more advanced approaches, such as the causal mediation through potential outcomes

framework (Imai, Keele, & Tingley, 2010). For instance, rather than calculating descriptive regression links, the latter produces causal quantities, which are interpreted as probabilities (Pearl, 2014). Furthermore, the potential outcomes framework sets clear assumptions and findings can be calculated irrespectively of the choice of statistical model, mediator, and outcome (MacKinnon et al., 202). Last, this approach can be effectively applied to nonlinear models (Imai et al., 2010).

Future research could expand those conclusions by examining a more representative sample; for instance, a sample that is more ethnically diverse and in which a larger proportion of individuals with severe difficulties is included. Furthermore, an important next step is to determine the causal pathways that explain the relationships between child and father difficulties. Future studies could attempt to assess the role of different factors (such as paternal monitoring) in those relationships and perhaps with a focus on older children and adolescents.

#### **4.8.8. Conclusions and implications**

To conclude, the outcomes from this research demonstrate that paternal distress affects child wellbeing and that to a lesser extent, child wellbeing also affects paternal distress. Father paths occurred mostly (and consistently) in adolescence, highlighting that during this period, the role of fathers is most critical. In addition, transactional links were found between paternal distress and adolescent peer difficulties, showing that the 2 influence each other simultaneously. Notably, all the associations that emerged were independent of the impact of third factors, such as maternal distress and family income.

These findings underline the need to support the wellbeing of fathers, both in terms of preventing mental health difficulties but also in terms of providing treatment to

those who experience them. This will be to the benefit of fathers themselves as well as their children. Findings also underline the need to support the children of those fathers, as they are likely to face increased vulnerabilities, particularly in adolescence.

It is also important to note that child effects were also shown to take place, especially for externalising difficulties. Child behavioural problems deteriorated paternal wellbeing; these conclusions imply that clinicians working with such children should consider the welfare of other family members, as well.

Finally, results from the mediation analysis shed some light on how and why these effects may occur. Parenting practices and marital satisfaction were found to account for these effects, suggesting that perhaps, to prevent adverse family influences, clinicians should work on and provide guidance regarding these behaviours.



## **Chapter 5: Trajectories of paternal psychological distress and cognitive functioning in early adolescence (research aim 2)**

This chapter aims to investigate the effects of paternal psychological distress trajectories, from preschool years to early adolescence, on early adolescence cognitive functioning. Furthermore, it aims to investigate the influence of maternal distress trajectories in parallel, to factor out their impact on paternal psychological distress, and explore their role independent of fathers'. Cognitive functioning includes verbal abilities, visuospatial working memory, and decision-making, all of which are important elements of cognitive development, as they are associated with a range of other outcomes (Deary et al., 2007; Dias et al., 2017; Gale et al., 2010; Pearson et al., 2016). In addition, there is evidence implying that paternal distress could influence the development of those cognitive functioning skills in early adolescence, meaning that those paths should be further examined by research (Bellau et al., 2013; Mannie et al., 2015).

First, the chapter will discuss why early adolescence is a critical period to focus on, and why it is useful to explore the effects of paternal and maternal distress. It will also explain why trajectory modelling was used. Next, the chapter will introduce the research literature that has looked at paternal and maternal distress trajectories. In the case of maternal distress trajectories, research has also assessed how these are linked to offspring cognitive functioning. Thereafter, the methods and analysis undertaken to address this research aim will be presented, and finally, there will be a discussion of the results.

## **5.1. Cognitive functioning in early adolescence**

Early adolescence is an important developmental stage, as it is a time during which many changes to a child's life occur. For instance, it is when the transition from primary to secondary education commonly takes place (Evans, Borriello, & Field, 2018). Moreover, in early adolescence, children start valuing more their peer relationships (Gorrese & Ruggieri, 2012) and gradually become more independent (Steinberg & Silverberg, 1986).

Even though these changes are normal steps in a child's life, they can also be challenging and may include some risks. To be more specific, some students may have difficulty adapting to the higher academic expectations of secondary education, and as a result, may drop out (Johnson et al., 2002). Additionally, association with deviant peers is related to delinquency (Thompson, Mehari, & Farrell, 2020) and substance abuse (Trucco, Colder, & Wieczorek, 2011). What is more, many mental health disorders start to emerge during early adolescence, meaning that those difficulties faced by youth can have a negative and long-term influence on their well-being (Anglin, Cohen & Chen, 2008; Plener et al., 2015).

High cognitive functioning is shown to have a protective effect against those hazards. Particularly, it is linked to better adaptation to secondary education and higher academic performance (Jacobson, Williford, & Pianta, 2011). At the same time, it is shown to prevent the development of internalising (Harpur, Polek & van Harmelen, 2015; Weeks et al., 2014) and externalising difficulties (Flouri, Mavroveli & Panourgia, 2013), with some evidence indicating that it can also buffer the impact of environmental adversities on mental health

(Flouri et al., 2013; Harpur et al., 2015). Last, it is shown to prevent adolescent substance abuse (Khurana et al., 2017; Pentz & Riggs, 2013; Romer et al., 2009).

Overall, early adolescence cognitive functioning plays an important role in the development of different types of outcomes, some of which can have a long-lasting impact. Consequently, it is important to determine the factors that contribute to cognitive functioning at that stage.

## **5.2. The role of paternal distress**

As has been demonstrated in Chapter 2, paternal distress affects cognitive abilities in preschool years (Cheng et al., 2016; Fredriksen et al., 2019; Paulson et al., 2009). Nonetheless, no studies so far have evaluated its role in later ages. In terms of executive functions and decision-making, studies assessing the influence of paternal distress are quite scarce. Furthermore, the number of fathers included, in relation to mothers, is quite small (Halse et al., 2019; Micco et al., 2009), and commonly, fathers' and mothers' effects are reported combined (Bellau et al., 2013; Halse et al., 2019; Mannie et al., 2014; Micco et al., 2009), resulting in an unclear picture as to what the impact of fathers' distress is. Additionally, most studies have looked at outcomes for children whose fathers had a mental health disorder diagnosis; there is only one study, by Halse et al., (2019), that examined these paths in the general population. One limitation of this study though is that children's executive functions were assessed through a teacher-completed questionnaire, which may not be as accurate as a lab-based measure. Due to these limitations,

existing research does not provide a clear picture as to what the effect of paternal distress on child cognitive functioning is.

### **5.3. The role of maternal distress**

Studies exploring the effects of paternal distress on preschoolers' cognitive development did also assess the effects of maternal distress (Cheng et al., 2016; Fredriksen et al., 2019; Paulson et al., 2009). However, in all cases, maternal distress was not found to be a significant predictor. A possible explanation suggested by Paulson et al., (2009) is that mothers, due to societal expectations, will continue to engage with their children, even if they are experiencing high levels of distress. On the contrary, distressed fathers are likely to disengage and stop spending time with their children (for instance, stop reading to them). This disengagement is likely to have an adverse influence on children's abilities, as it will deprive them of opportunities to learn and exercise their cognitive skills.

When it comes to early adolescence, research studies do show that maternal distress independently predicts cognitive skills (Chae et al., 2020; Prado et al., 2021), executive functions (Oh et al., 2020), and decision-making (Flouri et al., 2017). Nonetheless, these studies did not take into account fathers. Hence, it is unclear whether paternal or maternal distress have a greater role in cognitive functioning in early adolescence. To answer this question, the current research will examine maternal distress in the same fashion as paternal. The conclusions reached from this study, considered in parallel with early years' research, will help understand whether the impact of fathers' and mothers' well-being on cognitive development varies across different ages.

#### **5.4. Trajectory modelling**

Trajectory modelling will be used in this research, as it will enable exploring how the course and variability of paternal and maternal distress across time influences early adolescence outcomes (Nguefack et al., 2020). Existing studies on maternal distress do indeed support that the timing and chronicity of maternal symptoms are crucial in determining the effects on the child (Ahun et al., 2017; Chae et al., 2020; Flouri et al., 2017). Such findings are important in helping to identify whether there are critical child ages during which maternal psychopathology is most impactful and therefore, interventions should take place. They can also help target interventions to families in which the child might be most at risk (for instance, families in which the mother is experiencing chronic levels of distress). The current research, using trajectory modelling, will attempt to show what the case is with regards to fathers' symptoms.

#### **5.5. Trajectories of paternal distress**

So far, only 1 study has examined trajectories of paternal distress from infancy until late childhood (Giallo et al., 2015), with this age range being the closest to the age range the present research will examine. In this Australian study (Giallo et al., 2015) data from 2,662 fathers were obtained from 5 waves, using the K-6 scale used to measure parental psychological distress in this thesis. Researchers identified 2 trajectories: 'minimal distress', which contained most of the sample (92%), and 'persistent and increasing distress' (8%). Belonging to the 'persistent and increasing distress' trajectory was associated with poorer parenting, including higher hostility, less warmth, and

less consistency. These findings provide important insight into the progress of paternal distress across time. However, it was beyond the scope of the study to investigate how the emerged trajectories related to cognitive functioning.

## **5.6. Trajectories of maternal distress**

On the contrary, research has explored how maternal distress trajectories relate to adolescent cognitive functioning. Specifically, Chae et al., (2020), identified 4 trajectories of maternal depression, from infancy to adolescence, which were: “infrequent symptoms”, “symptoms increasing at adolescence”, “symptoms decreasing at adolescence” and “chronic severe symptoms”. Adolescents whose mothers experienced chronically severe depression had lower verbal and mathematical skills than adolescents whose mothers experienced infrequent depression. Similarly, Flouri et al. (2017), across child ages 3-11, using MCS data, identified 4 trajectories of maternal psychological distress and examined their association with offspring decision-making at age 11. The 4 trajectories included: “consistently low distress”, “moderate-accelerating distress”, “moderate-decelerating distress” and “chronic severe distress”. Results revealed that daughters of mothers with chronic and severe distress display more risk-taking behaviours than daughters of mothers with consistently low distress. For sons, no differences were found.

No studies have so far assessed how maternal distress trajectories from early years to adolescence may be linked to adolescent executive functions. Nevertheless, Oh et al., (2020) investigated distress trajectories from pregnancy to 2 years postpartum and their relationship to child executive functions at age 9. Researchers grouped the sample into 3 trajectories; these

were 'no symptoms', 'mild symptoms', and 'moderate symptoms'. Compared to children of mothers with no symptoms, those whose mothers experienced mild or moderate symptoms demonstrated impaired executive functions.

Even though the main focus of the present research is paternal distress, it will also add some insight into the literature concerning maternal distress. As it has been highlighted, existing studies looking at paths between maternal distress trajectories and adolescent cognitive functioning did not account for paternal distress. The present research will be the first to explore both paternal and maternal effects in parallel. Furthermore, the present research will address a gap in the existing research literature, by examining how maternal distress trajectories from preschool years to adolescence are associated with adolescent executive functions.

### **5.7. Gender differences in cognitive functioning**

In early adolescence, gender differences in cognitive functioning measures have been found. Specifically, gender differences concern verbal skills (Herlitz et al., 2013; Toivainen et al., 2017), visuo-spatial working memory (Voyer et al., 2017), and decision-making (Lewis et al., 2021). In addition, as shown in the study by Flouri et al., (2017), contextual factors may contribute differently to the development of cognitive functioning in boys compared with girls (i.e., only females were influenced by maternal severe distress). To explore these gender differences further, the current study will assess the moderating role of the child's gender for any significant paths that emerge.

## **5.8. Individual and family factors related to cognitive functioning**

Apart from gender, the role of which has been discussed in the above paragraph, several additional individual and family factors are linked to cognitive functioning. For instance, ethnic differences are present, with some groups, such as Indian children, outperforming others (Hoffman, 2018; Zilanawala, Kelly, & Sacker, 2016). What is more, children with internalising and externalising difficulties are shown to have lower cognitive skills than their peers (Kuang & Flouri, 2020; Passareli-Carrazzoni, Pereira-Lima, & Loureiro, 2018). With regards to family characteristics, a plethora of evidence links socio-economic background, including income/poverty and parental education, to children's cognitive functioning (Chzhen & Bruckauf, 2019; Conway, Waldfogel, & Wang, 2018; von Stumm & Plomin, 2015). Commonly, children from lower socio-economic backgrounds demonstrate weaker cognitive skills. Furthermore, parenting practices are shown to play a role in cognitive development, with children having a higher-quality relationship with their caregivers performing better than their peers (Hertz et al., 2019). Parental reading (Paulson et al., 2009) has been also demonstrated to boost children's abilities. There is also some evidence showing that there *is* U-shaped relationship between fathers' age and offspring cognition (Gajos & Beaver, 2017). Last, there are findings suggesting that parental separation adversely affects children's cognitive development (Garriga & Pennoni, 2020).

## **5.9. Conclusions and the present study**

Cognitive functioning in early adolescence contributes to a range of different outcomes, such as mental health and academic achievement. Consequently,



it is important to determine the role that paternal distress (and maternal distress) play in its development. The current research, using MCS data, will aim to identify trajectories of paternal distress from 3 to 11 years. Maternal distress trajectories will be identified in parallel, for the same ages. In line with past studies, it is hypothesized that 2 trajectories for paternal distress and 4 trajectories for maternal distress will occur. Subsequently, the paths from these trajectories to verbal skills, visuospatial working memory, and decision-making at age 11, will be investigated, while also accounting for child and family covariates. Considering the evidence for the effects of paternal distress on cognitive outcomes in preschool years, it can be hypothesized that paternal distress will predict poorer outcomes on verbal abilities and visuospatial working memory. Drawing from the research on maternal distress (Flouri et al., 2019; Mannie et al., 2014) it is hypothesized that paternal distress will also predict decision-making. However, as the findings for the direction of the effects of maternal distress are mixed, it is not possible to speculate what the direction of the effect of paternal distress will be.

The analysis will also examine the moderating effects of the child's gender. Based on past research (Flouri et al., 2017), it is hypothesized that the effects may be stronger for daughters, since research suggests that they may be more vulnerable to the influences of their environment (Reeb & Conger, 2009).

## **5.10. Methods**

### **5.10.1. Participants**

For the present analysis, data from the MCS's child ages 3-11 (sweeps 2-5) were used. The present study's analytic sample consisted of 9846 adolescents (50.1% female) and their families, which were selected based on the following criteria: 1) Child was a singleton or a first-born twin or triplet; 2) Child had valid scores on all cognitive functioning outcomes at age 11; 3) There was at least one valid psychological distress score for the father across child ages 3-11; 4) There was at least one valid psychological distress score for the mother across child ages 3-11.<sup>1</sup>

### **5.10.2. Measures**

Across child ages 3-11, *paternal* and *maternal psychological distress* were assessed with the K-6 (described previously in Chapter 3). Cronbach's alpha, for fathers, was .81, .82, .84 and .86, across ages 3, 5, 7, and 11, respectively. For mothers, it was slightly higher (.86, .87, .87 and .89, across sweeps 3-11, respectively).

*Child cognitive functioning* at age 11 was measured in terms of verbal ability, spatial working memory, and decision-making (details for these measures are presented in Chapter 3).

*Key covariates* included both child and family characteristics that may confound associations between parent psychological distress and child cognitive functioning. The family characteristics controlled for were *family poverty, family biological status, father's age at birth<sup>2</sup>, parental education, parental reading frequency* and *child-parent relationship quality*. All parental

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<sup>1</sup> By assuring that, for each family, there was at least one valid psychological distress score for both the father and mother, consistent single-parent families across child ages 3-11 (sweeps 2-5) were excluded.

<sup>2</sup> Due to high collinearity, only paternal age (and not maternal age) was controlled for.

variables were examined separately for both fathers and mothers. *Family poverty* was assessed by the mean number of sweeps (sweeps 2-5) in which the household income was below the 60% of the UK's median household income. *Parental education* was measured with a binary variable, specifying whether the parent had a university degree by child age 11 (sweep 5) or not. Parental education was used as a proxy for parental cognitive ability (which is linked to child cognitive ability) as the latter was not measured in MCS (Guerra-Carrillo et al., 2017; Plomin & Deary, 2015). *Parental reading frequency* was evaluated at age 3, with a 6-point question, asking caregivers how frequently they read to their child. Answers ranged from "every day" (1) to "not at all" (6). *Child-parent relationship quality* was also measured at age 3, with Pianta's Child-Parent Relationship Scale (CPRS) Short Form, which was adapted from the Student-Teacher Relationship scale (STRS; Pianta & Steinberg, 1992). It includes 15 items, which were answered from caregivers on a 1-5 Likert scale. Items were reversed when needed and added, with higher scores showing a better-quality relationship. *Family biological status* was measured with a binary variable demonstrating whether the household included 2 biological parents consistently across child ages 3-11 or not.

When it comes to child characteristics, the variables assessed include *gender*, *ethnicity*, *prior cognitive ability*, *child problem behaviour* and *exact age at testing*, which was measured in months. *Ethnicity* was assessed with a set of binary variables, indicating whether the child was white, mixed, black, Indian, Pakistani/Bangladeshi or belonging to any other ethnic group. *Prior cognitive ability* was evaluated at child age 5 with the British Ability Scales II (BAS-II) sub-tests of Naming Vocabulary, Pattern Construction and Picture Similarity

(Hill, 2005). Standardized scores ( $M=50$ ,  $SD=10$ ) were added to create a general ability construct. *Problem behaviour* was measured with the Strengths and Difficulties Questionnaire (SDQ) Total difficulties scale (Goodman, 2001). The SDQ was completed by the main caregiver across child ages 3-11. The mean number of sweeps in which the child experienced severe difficulties (total score above the clinical cut-off of 18) was calculated and used for the present research (Goodman, 2001).

Finally, a binary variable was used, which indicated whether testing proceeded smoothly or whether there were any factors that might have affected the child's performance (interruptions during the test, background noise, child fatigue and technical issues) given these have been linked with performance on the CANTAB (Atkinson, 2015).

### **5.10.3. Analytic strategy**

First, the analytic and non-analytic samples were compared to detect any sample selection bias. Furthermore, for the analytic sample, bivariate correlations were run to explore the links between the main study variables, which included paternal distress (child ages 3-11), maternal distress (child ages 3-11) and cognitive functioning outcomes.

Joint Group-Based Trajectory Modelling (GBTM; Nagin & Odgers, 2010) and multiple linear modeling were conducted in STATA to examine trajectories of psychological distress and their effects on child cognitive functioning. Joint GTBM was used to identify paternal and maternal psychological distress trajectories across child ages 3-11. It is a finite mixture modeling application that uses trajectory groups as a statistical device for approximating unknown

trajectories across members of the population. Joint trajectory modelling allows for the identification of separate group trajectories for fathers and mothers, as well as the estimation of the probability of membership in each group (Nagin & Odgers, 2010). Models were fitted with the STATA plug-in *traj* command (Jones & Nagin, 2013), and missing data were handled by FIML (FIML was used only for trajectory modelling). Since there were 4 assessment points, trajectories were fitted for zero-order, linear or quadratic relationships (King et al., 2018). To establish the best-fitting model, several criteria were applied. These involved identifying the lowest Bayesian Information Criterion (BIC) value, average posterior probabilities of assignment (AvePP) greater than 0.7, odds of correct classification greater than 5, a close correspondence between the estimated, and the actual probability of group membership and finally, reasonably tight confidence intervals (Nagin & Odgers, 2010). Individuals were allocated to the group trajectory to which they had the highest probability of belonging.

Next, multiple linear regressions were run to investigate whether these paternal and maternal trajectory groups predicted each of the cognitive outcomes. Due to the high correlation between overall proportion bet and risk-taking (.96), only risk-taking was examined. The moderating role of the child's gender was also explored for any of the significant paths found, using interaction effects. Given the large sample size, which provides statistical power, and the high reliability in Kessler scores, interaction effects were considered appropriate to use (Blake & Gangestad, 2020; McClelland & Judd, 1993; Whisman & McClelland, 2005). Finally, to confirm the robustness of the findings, a sensitivity analysis was conducted, using a sample that included

only 2-parent biological families, consistently across child ages 3-11 (6056 cases).

For the regressions, missing data were handled by multiple imputation by chained equations (MICE; Royston, 2005). Specifically, all variables with no missing values (including the dependent variables of the analysis, offspring cognitive outcomes) were used as predictors, to fill the missing values of the remaining variables. Twenty imputed datasets were generated and combined for the analysis using Rubin's rules. For paternal psychological distress, missing data were 24.1%, 23.6%, 28.5%, and 22.9% and for maternal psychological distress, missing data were 13.6%, 11.3%, 13.1% and 6.5%, across child ages 3, 5, 7, and 11, respectively. For cognitive functioning, there were no missing data, as only cases with full outcome data were included in the analytic sample.

Last, to control for the over-sampling of disadvantaged groups, as well as the issues of non-response and attrition, trajectory and regression models were weighted using the `svy` STATA command (Hansen, 2014). Additionally, MCS strata (England ethnic, England advantaged, England disadvantaged, Scotland advantaged, Scotland disadvantaged, Wales advantaged, Wales disadvantaged, Northern Ireland advantaged, and Northern Ireland disadvantaged) were included in regression models as binary variables (England advantaged was the reference group).

## **5.11. Results**

### **5.11.1. Descriptive statistics**

Families in the analytic sample were generally more advantaged than those in the non-analytic sample, demonstrating some sample selection bias (Table 8). Particularly, they were less likely to experience poverty and more likely to stay intact. Additionally, parents displayed lower psychological distress, were more likely to be university-educated and to read more frequently to their children. Mothers had higher relationship quality with their children, though no such differences were observed for fathers. Children had higher cognitive scores, were more likely to be female and presented less problem behaviour. No differences were found regarding ethnicity.

**Table 8** Descriptives of study variables in the analytic sample and in the non-analytic sample (unweighted data)

	Range	Analytic sample (N=9846)		Non-analytic sample (N=9397)		
Categorical variables		N	%	N	%	$\chi^2$
Girl	NA	4935	50.1%	4412	47%	19.35***
Mixed	NA	282	3.1%	236	2.9%	.603
Indian	NA	229	2.5%	210	2.6%	.065
Pakistani or Bangladeshi	NA	666	7.4%	538	6.7%	3.386
Black	NA	349	3.9%	315	3.9%	.004
Other	NA	141	1.6%	136	1.7%	.391
Father is university-educated	NA	3598	41.1%	997	28.5%	170.34***
Mother is university-educated	NA	4423	45.6%	846	25.6%	410.24***
Biological family consistently across sweeps	NA	6056	61.5%	517	5.5%	6705.67***
Disruptions during testing	NA	3438	34.9%	7114	76.7%	3229.73***
Continuous variables						
Delay aversion	[-9, 9]	9846	.29 (.25)	2610	.30 (.26)	-2.76**
Deliberation time	[468, 19121]	9846	3318.48	2676	3382.96	-2.20*

			(1298)		(1516.12)	
Quality of decision-making	[.09, 1]	9846	.81 (.17)	2676	.78 (.18)	8.07***
Risk adjustment	[-6.43, 6]	9846	.69 (1.03)	2675	.48 (1.04)	9.36***
Risk-taking	[.05, .95]	9846	.52 (.17)	2675	.55 (.17)	-6.45***
Paternal distress age 3	[0, 24]	7476	2.85 (3.06)	2432	2.94 (3.36)	-1.14
Maternal distress age 3	[0, 24]	8504	3.04 (3.49)	4999	3.69 (4.17)	-9.26***
Paternal distress age 5	[0, 24]	7525	2.91 (3.24)	1872	3.15 (3.72)	-2.63**
Maternal distress age 5	[0, 24]	8732	2.91 (3.58)	4145	3.58 (4.21)	-8.76***
Paternal distress age 7	[0, 24]	7044	2.90 (3.29)	1270	3.27 (3.95)	-3.20**
Maternal distress age 7	[0, 24]	8558	2.87 (3.56)	3333	3.68 (4.38)	-9.57***
Paternal distress age 11	[0, 24]	7596	3.80 (3.85)	611	4.45 (4.46)	-3.51***
Maternal distress age 11	[0, 24]	9202	3.77 (4.16)	2633	5.09 (5.14)	-12.11***
Poverty	[0, 1]	8564	.20 (.32)	2135	.51 (.40)	-33.84***
Problem behaviour	[0, 1]	7866	.06 (.16)	1729	.12 (.24)	-10.46***
Age in months	[122, 148]	9846	133.97 (3.92)	3441	134.17 (4.13)	-2.59**
General cognitive ability (age 3)	[60, 240]	9329	163.12 (22.32)	5534	150.05 (24.48)	22.55***
Father's age at birth	[14, 68]	8804	32.31 (6.11)	3871	31.66 (6.59)	5.17***
Father-child relationship quality	[32, 75]	6957	62.60 (6.61)	2223	62.34 (6.86)	1.60
Mother-child relationship quality	[30, 75]	8024	64.77 (6.65)	4510	63.91 (7.25)	6.49***
Father reading frequency	[1, 6]	7680	4.34 (1.36)	2731	4.13 (1.48)	6.70***
Mother reading frequency	[1, 6]	9321	5.30 (1.10)	6004	5.00 (1.39)	15.69***

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



### **5.11.2. Correlations**

The correlations between maternal and paternal psychological distress were all significant but weak, ranging .12-.21 (Table A8, Appendix). The correlations between paternal distress and cognitive outcomes (.0001-.08) as well as between maternal distress and cognitive outcomes (.006-.013) were all weak, with some reaching statistical significance. Lastly, the correlations between different types of cognitive functioning ranged from weak to strong (.002-.66), with most being significant.

### **5.11.3. Modelling parallel trajectories for mothers and fathers using GBTM**

Different models were tested using GBTM, with the number of trajectories for fathers and mothers ranging from 2 to 6. The model that met most of the set criteria included 2 trajectories for fathers and 4 trajectories for mothers. The 2 paternal trajectories were shaped to be quadratic and linear, respectively, while the 4 maternal trajectories were shaped to be quadratic, quadratic, linear and zero-order, respectively. The BIC and AIC values were the lowest out of the models that achieved convergence; BIC=-146489.62 (N=64637), BIC=-146467.98 (N=9846) and AIC=-146385. Average posterior probabilities ranged from .83 to .96 and odds of correct classification were greater than 4.82.

Paternal trajectories are illustrated in Figure 2. The first trajectory that emerged consisted of individuals (82.8%, 'low distress') who displayed low and decreasing psychological distress across child ages 3-7. However, from age 7 to age 11 their distress scores slightly increased. Fathers in the second

trajectory (17.2%, 'moderate-increasing distress') had moderate psychological distress that escalated continuously across child ages 3-11.

**Figure 2** Paternal distress trajectories, child ages 3-11 years

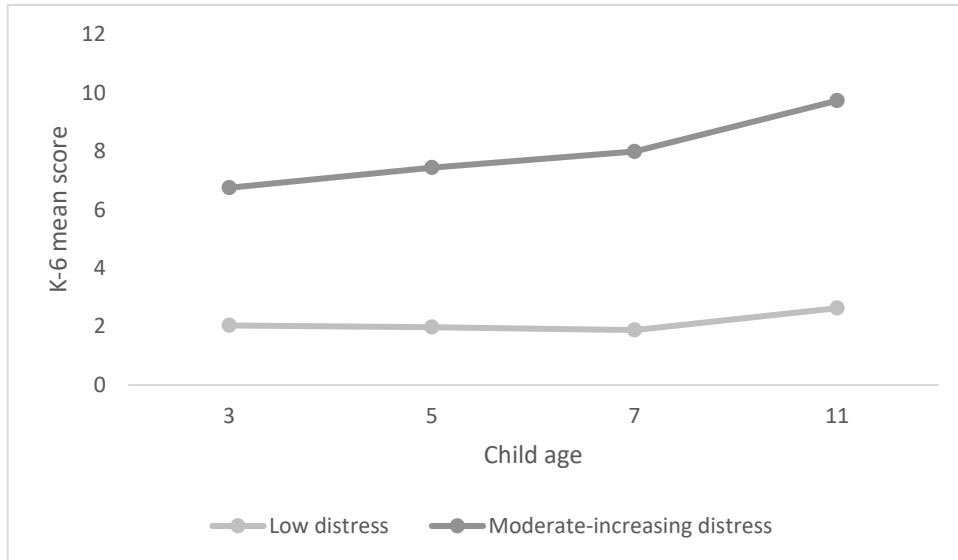
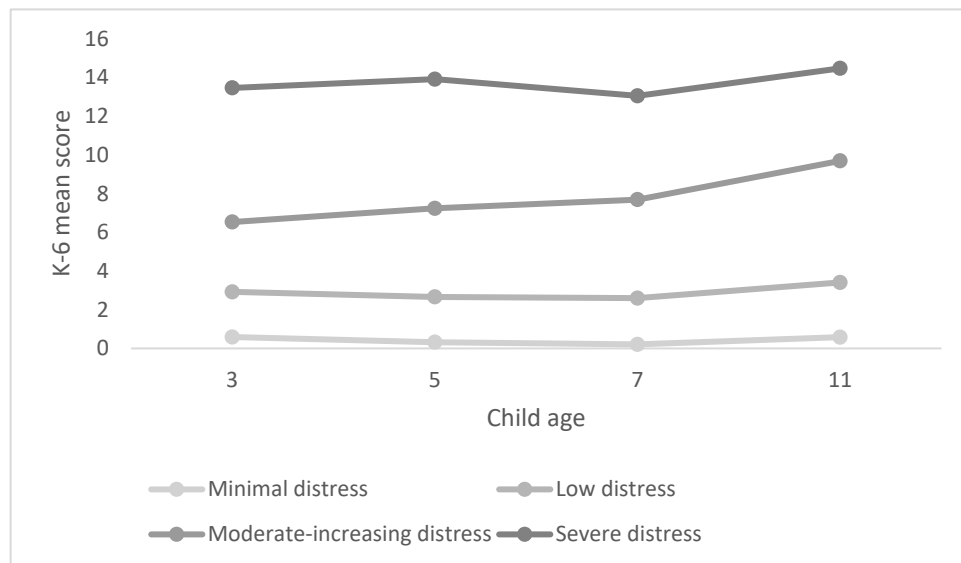


Figure 3 presents the maternal trajectories. Those in the first trajectory (25.1%, 'minimal distress') experienced negligible psychological distress, declining across child ages 3-7, but marginally increasing from age 7 to 11. The second trajectory (59.5%, 'low distress') follows the same pattern, though distress scores were slightly higher. Mothers in the third trajectory (12.4%, 'moderate-increasing distress') experienced moderate and increasing psychological distress, while those in the fourth trajectory (3%, 'severe distress') showed severe, fluctuating difficulties.

**Figure 3** Maternal distress trajectories, child ages 3-11 years



#### 5.11.4. Multiple regression results

Multiple regression was used to investigate if paternal and maternal trajectory groups predict each of the offspring outcomes. The 'low distress' group for fathers and the 'minimal distress' group for mothers were used as reference groups. Models were run first unadjusted and then adjusted with covariates and stratification variables.

Tables 9 and 10 present the results for the unadjusted and adjusted models, respectively. In the unadjusted models, "moderate-increasing distress" paternal trajectory, "moderate-increasing distress" and "severe distress" maternal trajectories were all associated with lower verbal ability, higher strategy, and higher total errors scores. Moreover, the "severe distress" maternal trajectory was linked to higher delay aversion and risk-taking scores, while the "moderate-increasing distress" maternal trajectory was linked to higher deliberation time scores. "Moderate-increasing distress" and "severe

distress” maternal trajectories were the only predictors for quality of decision making and risk adjustment, resulting in lower scores.

In the adjusted models, none of the trajectories were related to verbal ability, strategy, total errors, quality of decision making and risk adjustment. “Moderate-increasing distress” paternal trajectory predicted lower delay aversion scores, “moderate-increasing distress” maternal trajectory predicted higher deliberation time scores and “severe distress” maternal trajectory predicted higher risk-taking scores.

In terms of covariates, prior cognitive ability predicted all outcomes, while child age predicted all outcomes except risk-taking. More problem behaviour was associated with lower verbal ability and risk-adjustment as well as higher strategy and total errors. Lack of paternal and maternal higher education predicted lower verbal ability, risk adjustment, deliberation time, quality of decision-making and higher strategy and total errors. Not living in a 2-parent biological family was related to higher strategy, total errors, delay aversion, lower quality of decision-making and risk adjustment. Family poverty predicted lower verbal skills and risk adjustment, and higher total errors and risk-taking. Lower paternal age was linked to lower verbal skills, higher strategy, and total errors. Finally, less frequent paternal reading predicted lower verbal skills, lower quality of decision-making and higher strategy, while less frequent maternal reading predicted lower verbal skills and risk adjustment.

**Table 9** Multiple regression results (coefficients and standard errors) for the unadjusted models

	'Moderate-increasing distress' paternal trajectory		'Low distress' maternal trajectory		'Moderate-increasing distress' maternal trajectory		'Severe distress' maternal trajectory	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
Verbal Ability	-.79 (.26)**	[-1.32, -0.27]	-.24 (.23)	[-0.69, 0.21]	-2.37 (.34)***	[-3.05, -1.70]	-4.67 (.61)***	[-5.87, -3.48]
Strategy	.33 (.16)*	[0.03, 0.64]	.10 (.14)	[-0.17, 0.36]	.73 (.20)***	[0.33, 1.12]	.97 (.36)**	[0.27, 1.66]
Total Errors	1.79 (.51)***	[0.80, 2.79]	.37 (.44)	[-0.50, 1.24]	2.91 (.66)***	[1.62, 4.20]	6.10 (1.16)***	[3.82, 8.38]
Delay Aversion	-.01 (.007)	[-0.02, 0.002]	-.006 (.006)	[-0.02, 0.006]	.008 (.009)	[-0.009, 0.03]	.04 (.02)*	[0.008, 0.07]
Deliberation Time	-5.18 (36.02)	[-75.79, 65.42]	48.70 (31.31)	[-12.67, 110.07]	141.58 (46.49)**	[50.45, 232.72]	70.04 (82.28)	[-91.25, 231.33]
QoDM	-.004 (.005)	[-0.01, 0.005]	.0003 (.004)	[-0.008, 0.008]	-.02 (.006)**	[-0.03, -0.007]	-.03 (.01)**	[-0.05, -0.01]
Risk Adjustment	-.04 (.03)	[-0.10, 0.01]	.01 (.02)	[-0.04, 0.06]	-.13 (.04)***	[-0.20, -0.06]	-.27 (.07)***	[-0.40, -0.14]
Risk taking	.006 (.005)	[-0.004, 0.01]	.002 (.004)	[-0.006, 0.01]	.009 (.006)	[-0.003, 0.02]	.05 (.01)***	[0.03, 0.07]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, QoDM=Quality of decision-making

**Table 10** Multiple regression results (coefficients and standard errors) for the adjusted models

	'Moderate-increasing distress' paternal trajectory		'Low distress' maternal trajectory		'Moderate-increasing distress' maternal trajectory		'Severe distress' maternal trajectory	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
Verbal Ability	.30 (.25)	[0.18, 0.79]	.23 (.21)	[-0.19, 0.65]	.02 (.33)	[-0.62, 0.67]	-.30 (.57)	[-1.42, 0.83]
Strategy	.05 (.15)	[-0.57, 1.35]	-.01 (.13)	[-1.05, 0.62]	.01 (.21)	[-1.71, 0.86]	-.41 (.36)	[-2.31, 2.18]
Total Errors	.39 (.49)	[-0.25, 0.35]	-.21 (.43)	[-0.27, 0.25]	-.43 (.65)	[-0.39, 0.42]	-.07 (1.14)	[-1.11, 0.30]
Delay Aversion	-.02 (.007)*	[-0.03, -0.002]	-.01 (.006)	[-0.02, 0.001]	-.006 (.009)	[-0.02, 0.01]	.02 (.02)	[-0.01, 0.05]
Deliberation Time	-21.89 (36.75)	[-93.92, 50.15]	55.18 (31.89)	[-7.23, 117.78]	121.17 (48.84)*	[25.43, 216.91]	18.49 (85.22)	[-148.56, 185.53]
QoDM	.002 (.005)	[-0.007, 0.01]	.003 (.004)	[-0.005, 0.01]	-.003 (.006)	[-0.02, 0.009]	-.003 (.01)	[-0.02, 0.02]
Risk Adjustment	.01 (.03)	[-0.04, 0.07]	.03 (.02)	[-0.02, 0.08]	-.01 (.04)	[-0.08, 0.06]	-.05 (.07)	[-0.18, 0.08]
Risk taking	-.001 (.005)	[-0.01, 0.008]	-.002 (.004)	[-0.009, 0.006]	-.004 (.006)	[-0.02, 0.008]	.03 (.01)**	[0.007, 0.05]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, QoDM=Quality of decision-making

#### **5.11.5. Moderation by gender**

Using interaction terms, it was tested whether the child's gender moderates any of the significant relationships that were identified. Results showed that no differences occurred between boys and girls. Specifically, in terms of delay aversion, there was no interaction between 'moderate-increasing distress' paternal trajectory and gender ( $b=-.002$ ,  $SE=.01$ ,  $p=.91$ ); for deliberation time, there was no interaction between 'moderate-increasing distress' maternal trajectory and gender ( $b=68.04$ ,  $SE=78.98$ ,  $p=.39$ ); and finally, for risk-taking, there was no interaction between 'severe distress' maternal trajectory and gender ( $b=.03$ ,  $SE=.02$ ,  $p=.12$ ).

#### **5.11.6. Sensitivity analysis**

The analytic sample of this study involved households with stepparents and households with single parents for some of the assessed sweeps. A sensitivity analysis was conducted to test whether findings will remain robust for families with 2 biological parents across child ages 3-11 (6056 families), compared to the full analytic sample. Tables 11 and 12 present the results for the unadjusted and adjusted models, respectively. In the adjusted models, the significant effects of "moderate-increasing distress" maternal trajectory on deliberation time ( $b=176.73$ ,  $SE=66.34$ ,  $p=.008$ ) and "severe distress" maternal trajectory on risk taking ( $b=.05$ ,  $SE=.02$ ,  $p=.001$ ) remained. However, "moderate-increasing distress" paternal trajectory was no longer linked to delay aversion ( $b=-.009$ ,  $SE=.009$ ,  $p=.32$ ). No other significant paths were found.

The moderating role of the child's gender was again explored. It was not found to moderate the effects of 'moderate-increasing' paternal distress on delay aversion ( $b=.01$ ,  $SE=.02$ ,  $p=.56$ ); it was not found to moderate the effects of 'moderate-increasing' paternal distress on deliberation time ( $b=158.58$ ,  $SE=112.28$ ,  $p=.16$ ); and lastly, it was not found to moderate the effects of 'severe' maternal distress on risk taking ( $b=.01$ ,  $SE=.03$ ,  $p=.74$ ).

**Table 11** Multiple regression results (coefficients and standard errors) for the unadjusted models (sensitivity analysis)

	'Moderate-increasing distress' paternal trajectory		'Low distress' maternal trajectory		'Moderate-increasing distress' maternal trajectory		'Severe distress' maternal trajectory	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
Verbal Ability	-1.00 (0.34)**	[-1.67, -0.33]	0.12 (0.27)	[-0.42, 0.66]	-2.01 (0.46)***	[-2.90, 1.12]	-4.07 (0.93)***	[-5.89, -2.24]
Strategy	0.62 (0.21)**	[0.21, 1.03]	0.03 (0.17)	[-0.30, 0.36]	0.54 (0.28)	[-0.007, 1.09]	0.83 (0.57)	[-0.29, 1.95]
Total Errors	2.64 (0.67)***	[1.32, 3.96]	0.25 (0.54)	[-0.81, 1.31]	2.15 (0.90)**	[0.39, 3.91]	5.62 (1.83)**	[2.04, 9.20]
Delay Aversion	-0.008 (0.009)	[-0.03, 0.009]	-0.006 (0.007)	[-0.02, 0.008]	0.003 (0.01)	[-0.02, 0.03]	0.006 (0.02)	[-0.04, 0.05]
Deliberation Time	8.02 (47.96)	[-86.00, 102.04]	62.41 (38.51)	[-13.09, 137.90]	209.83 (64.07)**	[84.22, 335.43]	50.61 (130.25)	[-204.73, 305.95]
QoDM	-0.009 (0.006)	[-0.02, 0.003]	0.003 (0.005)	[-0.007, 0.01]	-0.02 (0.008)*	[-0.03, -0.0006]	-0.01 (0.02)	[-0.04, 0.02]
Risk Adjustment	-0.08 (0.04)*	[-0.15, -0.005]	0.04 (0.03)	[-0.02, 0.10]	-0.11 (0.05)*	[-0.21, -0.02]	-0.11 (0.10)	[-0.31, 0.09]
Risk taking	0.002 (0.006)	[-0.01, 0.01]	0.0004 (0.005)	[-0.009, 0.01]	0.009 (0.008)	[-0.007, 0.03]	0.08 (0.02)***	[0.04, 0.11]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, QoDM=Quality of decision-making

**Table 12** Multiple regression results (coefficients and standard errors) for the adjusted models (sensitivity analysis)

	'Moderate-increasing distress' paternal trajectory		'Low distress' maternal trajectory		'Moderate-increasing distress' maternal trajectory		'Severe distress' maternal trajectory	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
Verbal Ability	0.31 (0.32)	[-0.32, 0.93]	0.35 (0.26)	[-0.15, 0.85]	-0.10 (0.43)	[-0.95, 0.74]	-0.82 (0.87)	[-2.52, 0.88]
Strategy	0.27 (0.21)	[-0.14, 0.69]	0.05 (0.17)	[-0.28, 0.38]	0.01 (0.28)	[-0.54, 0.57]	-0.32 (0.57)	[-1.43, 0.80]
Total Errors	0.98 (0.65)	[-0.30, 2.26]	0.17 (0.52)	[-0.86, 1.19]	-0.57 (0.88)	[-2.30, 1.16]	0.23 (1.77)	[-3.24, 3.70]
Delay Aversion	-0.009 (0.009)	[-0.03, 0.009]	-0.008 (0.007)	[-0.02, 0.007]	-0.002 (0.01)	[-0.03, 0.02]	-0.006 (0.02)	[-0.05, 0.04]
Deliberation Time	-27.30 (48.93)	[-123.21, 68.61]	71.56 (39.23)	[-5.35, 148.47]	176.73 (66.34)**	[46.69, 306.77]	-2.99 (132.90)	[-263.52, 257.55]
QoDM	-0.002 (0.006)	[-0.01, 0.01]	0.002 (0.005)	[-0.007, 0.01]	-0.008 (0.008)	[-0.02, 0.008]	0.005 (0.02)	[-0.03, 0.04]
Risk Adjustment	-0.006 (0.04)	[-0.08, 0.07]	0.04 (0.03)	[-0.02, 0.10]	-0.03 (0.05)	[-0.13, 0.07]	0.06 (0.10)	[-0.15, 0.26]
Risk taking	-0.004 (0.006)	[-0.02, 0.008]	-0.001 (0.005)	[-0.01, 0.008]	-0.001 (0.008)	[-0.02, 0.01]	0.05 (0.02)**	[0.02, 0.09]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, QoDM=Quality of decision-making



## **5.12.Discussion**

Using a large and nationally representative sample, this research aimed to identify parallel trajectories of paternal and maternal distress, across child ages 3-11 years. Subsequently, it aimed to examine how these trajectories relate to offspring cognitive functioning at age 11. As predicted by the study's hypothesis, two trajectories of paternal distress emerged: 'low distress' and "moderate-increasing distress'. For maternal distress, in line again with the hypothesis, 4 trajectories were found: 'minimal distress', 'low distress', 'moderate-increasing distress', and 'severe distress'. 'Severe distress' is the only trajectory in which individuals experienced clinical levels of distress. Paternal and maternal trajectories were shown to predict different measures of decision-making. Specifically, children of fathers in the 'moderate-increasing distress' trajectory displayed lower delay aversion scores than children of fathers in the 'low distress' trajectory. Furthermore, adolescents whose mothers belonged to the 'moderate-increasing distress' trajectory had higher deliberation time scores than adolescents whose mothers belonged to the 'minimal distress' trajectory. Additionally, the youth of mothers in the 'severe distress' trajectory had higher risk-taking scores than the youth of mothers in the 'minimal distress' trajectory. Only the two maternal effects remained when exploring the sub-sample including only biological families. Neither paternal nor maternal trajectories predicted verbal abilities and visuospatial working memory. Moreover, child gender did not moderate any of the significant relationships found.

### **5.12.1. Trajectories of paternal and maternal distress**

Similarly to the current study, Giallo et al., (2015) also classified paternal distress into two trajectories. The description of the trajectories between the two investigations is analogous; most individuals had low symptoms, while a minority experienced higher and increasing difficulties. It is noticeable that in the present study a greater proportion of fathers belonged to the latter category (17.2%), compared to the Giallo et al.'s, (2015) study (8%).

Furthermore, the present, as well as previous studies, identified 4 trajectories of maternal distress (Chae et al., 2020; Flouri et al., 2017). Despite some slight disagreement in the characterization of those trajectories, all investigations suggest that there was a group of mothers with generally low symptoms, a group of mothers with moderate and increasing symptoms, and a group of mothers with chronic and severe symptoms. The difference in the number of trajectories between fathers and mothers is likely to reflect the fact that women do overall experience higher levels of distress than men do (Drapeau et al., 2010). In other words, more men than women are likely to report low distress symptoms, leading to most of them being grouped in the 'low distress' trajectory.

### **5.12.2. Verbal ability**

Paternal distress did not predict verbal ability at age 11, which was not in agreement with the study's hypothesis. This result contradicts the research which supports the role of paternal distress in early years' cognitive outcomes. Taken together, current and past findings indicate that the early years are a

critical period, during which paternal distress can impact children's cognition. Nonetheless, as children grow and begin to spend more time outside of the home environment, other factors, such as peers and school may play a greater role in the development of their skills (Antecol et al., 2016; Xu et al., 2022).

Chae et al., (2020) argued that maternal 'chronic severe' depressive symptoms, measured from child age 1 to 14, led to poorer offspring language, vocabulary, and reading ability at 14. This was not confirmed by the current research, as none of the maternal trajectories were associated with verbal skills. It should be underlined that the study by Chae et al., (2020) examined a larger age span (1-14 years) than the present study (3-11 years) and that researchers measured depressive symptoms, rather than overall distress.

### **5.12.3. Visuospatial working memory**

Neither paternal nor maternal trajectories predicted visuospatial working memory. These outcomes are in line with some of the past research studies (Halse et al., 2019; Micco et al., 2009), which found no links between parental mental health and child executive functions. However, support for the role of parents' well-being on executive functions has been provided by Bellau et al., (2013), who showed that children of parents with bipolar or major depressive disorder have poorer executive function skills than children of mentally healthy parents. In addition, significant paths between maternal depression and offspring executive functions were identified by Oh et al., (2020).

To assess executive functions, different types of measurements have been used across the research literature. While the current study focused on

visuospatial working memory, others have looked at executive attention (Bellau et al., 2013), or multiple aspects of executive functions (Halse et al., 2019; Micco et al., 2009; Oh et al., 2020). Moreover, inconsistency exists at the time of measurement. Specifically, while this research as well as those by Halse et al., (2019) and Oh et al., (2020) evaluated executive functions around the age of 10, the studies by Bellau et al., (2013) and Micco et al., (2009) examined children from early childhood to late adolescence. These discrepancies lead to difficulties in reaching an overall conclusion.

Despite these limitations, the present research was the first to explore the effects of paternal and maternal distress separately. Further research is needed to clarify when and for which aspects of executive functions parents' distress might be impactful. Fathers' distress was not shown to influence visuospatial working memory in early adolescence; nevertheless, like verbal ability, it may play a greater role at younger ages.

#### **5.12.4. Decision-making**

Adolescents whose fathers belonged to the 'moderate-increasing distress' trajectory had significantly lower delay aversion scores (a measure of impulsivity) than adolescents whose fathers belonged to the 'low distress' trajectory. This result supports the conclusions reached by Mannie et al., (2015), who argue that youth with familial risk of depression are likely to adopt less impulsive decision-making strategies. In both this research as well as in the study by Mannie et al., (2015), youth's internalising difficulties were accounted for, meaning that this effect occurs irrespectively of their own symptomatology.

A similar finding appeared for maternal distress. Specifically, offspring of mothers with 'moderate-increasing distress' had higher deliberation time scores (meaning that these adolescents took more time to make a box colour decision), than offspring of mothers with 'minimal distress'. Again, this outcome is in line with Mannie et al.'s, (2015) research, while it contradicts the evidence of Flouri et al., (2017) since, in this study, no paths for the trajectory classified as 'moderate-accelerating' distress were identified. One possible reason why the current findings contradict those of Flouri et al., (2017), even though both studies used MCS data, is that Flouri et al., (2017) did not adjust for paternal distress, or paternal and maternal parenting variables. Additionally, the studies used different criteria to define the analytic samples, and different trajectory modelling approaches (this study used GBTM, while Flouri et al., latent class analysis).

Moreover, the youth of mothers experiencing 'severe distress' had higher risk-taking scores than the youth of mothers experiencing 'minimal distress'. This does partly support the conclusions of Flouri et al., (2017), as researchers did report the same path, though only for females. It does, however, come in contrast with the research suggesting that children of depressed parents tend to follow conservative decision-making strategies (Mannie et al., 2015).

Overall, the results from the current research appear to indicate a pattern of effects. Specifically, while parental moderate-increasing difficulties predicted conservative decision-making, severe parental difficulties predicted risky decision-making. The reasons accounting for these different outcomes are not clear. It should be noted that this study assessed parental general distress, not a specific symptomatology. Therefore, one possibility is that

different types of psychopathologies are present in these two groups. In particular, mothers in the 'severe distress' trajectory demonstrate clinical and long-lasting symptoms, meaning that perhaps they are experiencing a chronic psychiatric disorder. Chronic psychiatric disorders include conditions such as bipolar disorder and borderline personality disorder, which typically emerge in early adulthood (Duffy et al., 2019; Zanarini et al., 2015), are lifelong (Saunders & Goodwin, 2010; Videler et al., 2019), and are described to cause significant distress (Fertuck, Karan, & Stanley, 2016; & Goodwin, 2010). Not only are these disorders associated with impulsive decision-making and riskiness (Paret et al., 2017; Ramírez-Martín et al., 2020), but also the offspring of affected parents are shown to display high impulsivity and great reward sensitivity themselves, possibly due to genetic risks or environmental influences (Sanches et al., 2014; Singh et al., 2014). On the other hand, the escalating distress observed in the 'moderate-increasing distress' maternal and paternal trajectories is more likely to mean that those individuals are experiencing depression, since depressive symptoms can arise even later in adult life (Lallukka et al., 2019; Sutin et al., 2013). As discussed, conservative decision-making strategies are found to be adopted by the offspring of parents with depression (Mannie et al., 2015).

In conclusion, the findings from trajectory modelling allow speculation that different types of psychopathologies may affect individuals in the 'severe' and 'moderate-increasing' trajectories. These psychopathologies would explain why 'severe distress' was linked to riskiness, while 'moderate-increasing' distress was linked to risk-avoidant strategies. This proposition may also account for some of the inconsistency in the results described in the existing

research literature (Flouri et al., 2017; Mannie et al., 2015). Unfortunately, MCS data do not provide detailed information on parental mental health; consequently, it can only be hypothesized that this might be the case.

#### **5.12.5. Sensitivity analysis**

A sensitivity analysis was conducted, involving only households that had 2 biological parents across child ages 3-11. While both maternal paths remained, the paternal path of 'moderate-increasing distress' on delay aversion ceased to be significant.

The neural processes corresponding to reward and punishment in adolescents with familial risk of depression are described to differ from those of adolescents with no family history of depression (McCabe et al., 2012). It is unclear if these neural alterations are inheritable or are due to environmental exposures (Foland-Ross et al., 2015). Youth whose fathers experience depression are likely to experience adversities, for instance, a lack of supportive parenting. Such adversities have been shown to physically impact brain development (Lupien et al., 2009). At the same time, some research evidence implies that genetic risk factors are likely to also play a role (Montag et al., 2009). With regards to fathers' effects, current findings appear to highlight the influence of environmental adversities; nevertheless, further research is required to understand the causes of those links.

#### **5.12.6. Gender differences**

The child's gender did not moderate any of the significant relationships found, neither in the main nor in the sensitivity analysis. This outcome contradicts the study's hypothesis and some of the past research (Flouri et al., 2017), as

authors reported that 'chronically high distress' predicted only females' risk-taking. Considering the low proportion of mothers in the 'severe distress' trajectory (3%), it is possible that the present investigation lacked the strength to detect such an effect. Furthermore, the study by Flouri et al., (2017) did not adjust for paternal distress, which could also be responsible for the different effects.

#### **5.12.7. Limitations**

This research does have some limitations. First, compared to the non-analytic sample, families in the analytic sample were more privileged; for instance, they were more affluent and more likely to stay intact. Furthermore, parents had lower levels of psychological distress and their children performed better in the cognitive tasks compared to children in the non-analytic sample. This not only decreases the generalizability of the conclusions but also implies that relationships occurring between parents and children with more severe difficulties may not have been captured. Second, with regards to fathers, trajectory modelling did not yield a group of fathers with clinical levels of distress. Hence, it was not possible to explore how having a father with clinical difficulties might impact the offspring's cognitive functioning. Third, there were missing data for most predictors, including paternal psychological distress, though this issue was handled by using multiple imputation. Fourth, because of a lack of parenting measures at child age 11, it was not possible to investigate the mediating pathways between parental distress trajectories and offspring cognition. Fifth, the effect sizes were quite small, and in the case of paternal distress, the path that emerged did not persist in the sensitivity analysis.



Last, limitations exist in the methodology used to test for the moderating role of gender. Gender differences were examined to identify whether boys or girls may be at higher risk in terms of the relationships explored. However, interaction effects have limited usage in helping researchers understand the causal mechanisms that may underlie a relationship, as well as little implications for clinical practice (Zammit et al., 2010). Furthermore, they are largely dependent on the data and the measures used. The size of interaction effects may change according to the measures' variance and the existence of outliers (McClelland & Judd, 1993). Interaction effects are also sensitive to measurement error, which can reduce statistical power, and result in misleading conclusions (Blake & Gangestad, 2020). Their usage is proposed only when there is high reliability in the measures, as was the case in the current research (Whisman & McClelland, 2005). In addition, interaction effects are sensitive to statistical power; due to the large sample of this analysis, this was unlikely to be a problem (Blake & Gangestad, 2020). Finally, scholars' recommendation, that interaction effects should be explored only when main effects have been priori identified, was followed (Whisman & McClelland, 2005).

#### **5.12.8. Conclusions and Implications**

Overall, this study indicates that the course of paternal and maternal distress across childhood is associated with decision-making in early adolescence. Nevertheless, it also raises a number of questions for future research to consider. Mainly, the reasons why moderate-increasing and severe parental distress led to contradicting outcomes could not be investigated. A possibility is that different types of parental psychopathologies are present.

Future studies could also explore whether parenting practices account for the paths between paternal (and maternal) distress and adolescent decision-making. Giallo et al. (2015) reported that paternal moderate-increasing difficulties were related to high levels of hostility and low levels of warmth and consistency. It would be interesting and useful to examine if those practices are linked to decision-making.

In adolescence, a significant lack of impulsivity is associated with mental health difficulties, such as depression and anxiety (Galván & Peris; 2014; Mannie et al., 2014). At the same time, high riskiness and impulsivity are shown to predict behaviours such as delinquency and substance abuse (Adjorlolo et al., 2018; De Bellis et al., 2013; Xiao et al., 2009). Therefore, ensuring the healthy development of adolescent decision-making can help protect them from a range of adverse outcomes. The current findings highlight that both fathers' and mothers' distress play a role and should be considered by policies that aim to promote adolescent well-being.

## **Chapter 6: Paths from paternal psychological distress to adolescent health risk engagement (research aim 3)**

The aim of this chapter is to explore the relationship between fathers' distress across childhood and adolescent engagement in health risk behaviours, including smoking, alcohol use, binge drinking, and sexual activity. These relationships will be examined separately for boys and girls, considering the different levels of risky engagement reported for the two.

As discussed in chapter 2, a scarce amount of research has so far assessed the links between paternal distress and adolescent engagement in risky behaviours (Ali et al., 2016; Essau & de la Torre-Luque, 2021; Herman-Stahl et al., 2008). Findings are mixed, with one study supporting that paternal psychopathology increases adolescent illicit drug use but is not related to alcohol misuse or nicotine dependence (Essau & de la Torre-Luque, 2021); another study demonstrated that it lowers the risk of binge drinking for black adolescents only and not for other ethnic groups (Herman-Stahl et al., 2008); and last, one study reported no links (Ali et al., 2016).

This research does though involve some limitations. For instance, in two studies (Ali et al., 2016; Essau & de la Torre-Luque, 2021) scholars looked at fathers' co-morbid mental health and substance abuse, while in the third study (Herman-Stahl et al., 2008) they explored the impact of severe psychological distress. Furthermore, in two studies the outcome was adolescent substance abuse disorder (Ali et al., 2016; Essau & de la Torre-Luque, 2021). Consequently, the conclusions reached appear to apply more to families with clinical difficulties, and potentially cannot be generalized to the general

population. Furthermore, all studies are cross-sectional, with no longitudinal research so far evaluating how paternal distress during childhood could influence adolescent risky engagement. The proportion of fathers included, compared to mothers, was relatively small. One additional limitation concerning some of the studies is that, for each household, only the father or mother was included in the analytic sample, limiting the comparisons that can be drawn on the influences between paternal and maternal distress (Ali et al., 2016; Herman-Stahl et al., 2008).

The present research will aim to address these limitations and expand current knowledge by examining longitudinal associations between paternal distress and adolescent risky engagement in the general population. The chapter will start by presenting the research concerning maternal distress and adolescent health risk behaviours. It will proceed by discussing the reasons why both paternal and maternal distress are expected to predict those behaviours. Next, the chapter will describe the gender differences that have been identified in youth risk engagement and will present the methodology and results corresponding to the current research aim. Last, there will be an evaluation of the findings.

### **6.1. Maternal distress and adolescent health risk engagement**

Adolescent engagement in health risk behaviours such as smoking, alcohol drinking, and early sexual activity are major public health concerns linked to a range of adverse outcomes. Mainly, they have been associated with later mental health difficulties, substance abuse, and poor physical health (Boden et al., 2010, 2020; Enstad et al., 2019; Levola et al., 2020). Early initiation of

adolescents is also concerning, since those adolescents are likely to continuously engage in risky behaviours, placing themselves at even greater risk (Bolland et al., 2016; Epstein et al., 2014; Leventhal et al., 2015). Hence, it is important to pinpoint the factors and the paths that are responsible for the emergence of such behaviours.

Many research studies have shown that maternal distress predicts adolescent risky engagement (Ali et al., 2016; Bohon et al., 2007; Cortes et al., 2009; Flouri & Ioakeimidi, 2018; Herman-Stahl et al., 2008, 2017; Lamis et al., 2012; Sang et al., 2016; Wickham et al., 2015). Additionally, they have provided evidence indicating that the timing of maternal symptoms is critical. Flouri and Ioakeimidi (2018) found that male adolescents whose mothers experienced high distress in late childhood (child age 11 years) were at increased risk for engagement in antisocial behaviour, including alcohol use. There were no effects for offspring whose mothers faced elevated distress at younger ages. Furthermore, Wickham et al. (2015) identified maternal depression trajectories across child ages 2-14 and assessed how these relate to youth risk engagement at the age of 16. Results suggest that, compared to adolescents experiencing consistent and low maternal symptoms (the reference group), adolescents exposed to high maternal symptoms in middle childhood are more likely to initiate smoking and drinking at a younger age. No differences were observed between the reference group and adolescents exposed to maternal symptoms in late childhood or recurrently.

The findings of these two studies contradict each other, as they suggest different child ages are critical for the impact of maternal distress. Further research is needed to explore the strength of maternal effects in relation to

their timing and to also evaluate whether the case for paternal effects is similar. In other words, whether paternal and maternal distress are equally influential during the same stages of development or whether each of them becomes most important at a different period. Research is also needed to determine whether both paternal and maternal distress affect adolescent risk engagement simultaneously, or whether adjusting for one 'cancels' the other out.

## **6.2. Reasons why paternal distress and adolescent health risk engagement are linked**

Several reasons may explain the relationship between parental distress and adolescent risky behaviour engagement. To begin with, compared to mentally healthy parents, depressed parents are more likely to engage in risky behaviours and substance abuse themselves (Åhlin et al., 2015; Boden et al., 2010). In line with the social-learning theory, offspring observe and imitate those behaviours (Bandura, 1977; Vermeulen-Smit et al., 2012). Furthermore, they may gain easier access to substances themselves or normalize dangerous practices (Danielsson et al., 2011; Ryan et al., 2010; Yap et al., 2017). Indeed, there is a plethora of evidence linking caregivers' and adolescents' substance use (Alati et al., 2014; Holst et al., 2019; Kerr et al., 2012; Mays et al., 2014; Rossow et al., 2016; Ryan et al., 2010; Sternberg et al., 2018).

Additionally, research evidence suggests that the path between maternal distress to adolescent substance abuse may be mediated by internalising and externalising difficulties (Cortes et al., 2009; Herman-Stahl et al., 2017). Youth

of distressed mothers are at increased risk for developing mental health difficulties; at the same time, youth mental health difficulties are associated with risky engagement. So far, no studies have evaluated whether the same mechanism operates for paternal distress. Youth experiencing mental health problems have low regulatory skills and poor responses to stress, characteristics associated with riskiness (Herman-Stahl et al., 2017). Moreover, they may engage in maladaptive behaviours as a way of coping with negative emotions (Herman-Stahl et al., 2017).

Last, parenting practices may be another mediator. It has been demonstrated that distressed caregivers tend to engage in poor-quality parenting practices, including low levels of monitoring, warmth, and involvement (Cheung & Theule, 2019b; Nath et al., 2016b; Shorey et al., 2019). These practices are shown to be predictors of early and risky sexual engagement, alcohol misuse, and smoking initiation (Aho et al., 2018; Coley et al., 2009; Danielsson et al., 2011; Dittus et al., 2015; Kalina et al., 2013; Ryan et al., 2010; Savioja et al., 2018; Yap et al., 2017), with some of those findings referring specifically to fathers' parenting (Coley et al., 2009; Kalina et al., 2013; Yap et al., 2017). Limited research has investigated the mediating role of parenting practices, with Herman-Stahl et al., (2008) arguing that the relationship between maternal severe distress to daughters' illicit drug use is partially accounted for by levels of involvement. Nonetheless, further research is needed to explore the role of parenting practices, for both fathers and mothers.

### **6.3. Gender differences**

Gender differences have been observed in the prevalence of adolescent risky behaviours. Specifically, girls are more likely to smoke (Lavery et al., 2019; MacArthur et al., 2012; Melotti et al., 2011) and boys are more likely to initiate alcohol drinking at a young age (Kelly et al., 2016; MacArthur et al., 2012; Melotti et al., 2011). Results regarding binge drinking are less clear, with some claiming that girls are at higher risk (Healey et al., 2014), while others support that no gender differences exist (MacArthur et al., 2012; Melotti et al., 2011). The same is the case for sexual activity; some studies found that girls are more likely to have had an early sexual intercourse (Heron et al., 2015), while others reported a similar prevalence (Kelly et al., 2019).

Not only are there different levels of engagement in risky behaviours between boys and girls, but also the pathways underlying those behaviours are described to differ (Gutman & McMaster, 2020). For instance, internalising difficulties and not living with a biological family are shown to be risk factors for smoking and early sexual engagement only for girls (Aho et al., 2018; Gutman & McMaster, 2020). In terms of parental effects, Flouri and Ioakeimidi (2018) found mothers' distress to be linked only to boys' antisocial behaviour, including alcohol use. Overall, those findings highlight that different risk mechanisms are in place for boys and girls; therefore, it may be useful to consider them separately.

#### **6.4. Conclusions and the present study**

Existing research regarding the relationship between fathers' distress and adolescent engagement in health risk behaviours includes some limitations, which the current investigation aims to address. Specifically, using MCS data,



it will explore the links between paternal distress in child ages 3, 7, and 11 on offspring risk of smoking, alcohol use, binge drinking, and sexual activity at age 14. Maternal paths will be explored in the same way, to factor out their influence and allow the comparison of maternal and paternal effects. While some past research suggests that parental psychological distress is linked to more health risk behaviours (Essau & de la Torre-Luque, 2021), others indicate the opposite (Ali et al., 2016). Therefore, though it is hypothesized that both paternal and maternal distress will be associated with those behaviours, it is not possible to predict what the direction of these effects might be.

Considering the research showing different risk pathways for engagement in those behaviours for boys and girls, the models will be examined for the full sample as well as for the 2 genders separately. Based on past research (Flouri & Ioakeimidi, 2018), it is hypothesized that maternal distress will be more strongly related to boys' than girls' behaviours. Given the limited research on fathers' distress and adolescent health risk engagement, it is not possible to predict if and how these relationships may differ for boys and girls.

## **6.5. Methods**

### **6.5.1. Participants**

For the current study, MCS data from child ages 3, 7, 11 and 14 (sweeps 2, 4, 5, and 6, respectively) were used. The analytic sample (11128 cases) consisted of children (and their families) who met the following criteria: 1) Child was a singleton or first-born twin or triplet; 2) Child had valid data on all outcomes; 3) There was at least one valid paternal distress score for child

ages 3, 5, and 11; 4) There was at least one valid maternal distress score for child ages 3, 5, and 11.

### **6.5.2. Measures**

*Adolescent health risk behaviours* were measured at age 14, as described in Chapter 3.

*Paternal and maternal psychological distress* were measured separately at child ages 3, 5, and 11, with the K-6. Cronbach's alpha for fathers was .79, .83, .97, while for mothers it was .85, .87, .97, for child ages 3, 5, and 11, respectively.

*Key family-level covariates* include factors the research literature suggests are linked to health risk behaviours; *poverty, fathers' and mothers' alcohol consumption, fathers' and mothers' educational level, and paternal biological status*. *Poverty* was assessed when children were aged 3, with a binary variable showing if the family income was above or below the 60% UK's median household income (Kipping et al., 2015; Melotti et al., 2011). *Fathers' and mothers' alcohol consumption* were each measured at child age 11, with a 6-item variable, reflecting how many alcoholic drinks caregivers consumed on an average day. Possible responses were "none", "1-2", "3-4", "5-6", "7-9", and "10 or more" (Haugland et al., 2013; McCutcheon et al., 2018). *Fathers' and mothers' educational level* were each assessed using a binary variable, indicating whether the parent had or had not obtained a university degree by the time the child was aged 14 (Melotti et al., 2011). *Fathers' biological status* was also measured with a binary variable, which demonstrated if across ages

3-14 the child lived consistently with a biological father or not (McCutcheon et al., 2018).

The *child-level variables* controlled for were *gender* (male or female), *ethnicity*, *internalising difficulties*, *externalising difficulties* as well as *pubertal status*. *Ethnicity* was assessed with a set of binary variables, specifying if the child was white, mixed, black, Indian, Pakistani/Bangladeshi, or belonged to any other ethnic group (Hale & Viner, 2016). *Internalising* and *externalising* difficulties were measured at age 11, using the parent-completed Strengths and Difficulties Questionnaire (SDQ). The emotional and peer subscales of SDQ were added to create the internalising difficulties scale, while the conduct and hyperactivity subscales were added to create the externalising difficulties scale. Both scales ranged from 0 to 20, with higher values suggesting more difficulties (Bozzini et al., 2021; Gutman & McMaster, 2020). Finally, *pubertal status* was assessed at age 11 with a binary variable, which showed if the child had displayed any signs of puberty or not. Signs of puberty for girls involved body hair, menstruation, and breast growth, while signs of puberty for boys involved body hair, facial hair, and voice change (De Azevedo et al., 2017).

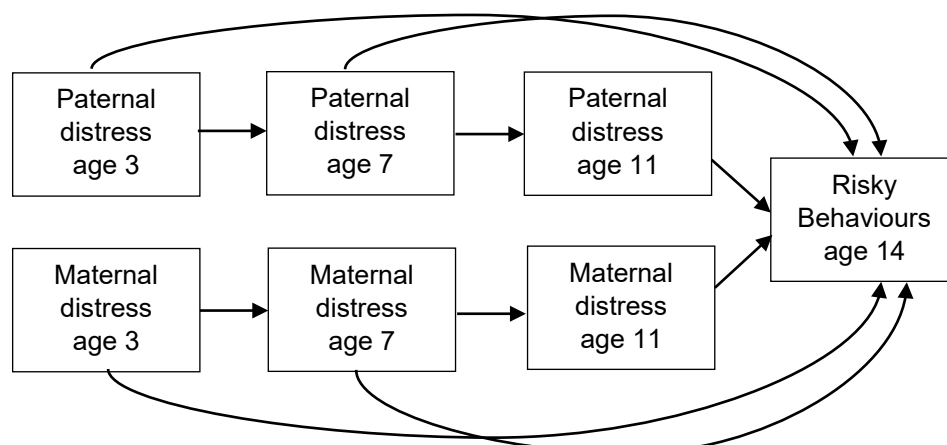
### **6.5.3. Analytic Strategy**

All analysis was conducted in STATA (version 15.0). First, to detect any potential sample selection bias, the main study variables and covariates were compared between the analytic (N=11128) and non-analytic (N=8115) samples. Next, to gain a better understanding of the data, gender differences were explored for each of the health risk behaviours (for girls, N=5,619 and for

boys, N=5,509). Subsequently, generalized structural equation modeling (GSEM) was used to investigate the paths from paternal and maternal psychological distress to adolescent health risk behaviours. GSEM is an extended version of structural equation modeling (SEM), which allows the examination of categorical outcomes with its logistic and multinomial logistic functions. The model used is illustrated in Figure 4. As shown, paternal and maternal distress were modelled simultaneously. Each of the health risk outcomes was examined separately. Models were run first unadjusted and then adjusted with covariates and stratum variables, which accounted for the stratified design of the MCS. The full sample was explored first, and then female and male adolescents separately.

Missing data were handled by multiple imputation by chained equations (MICE) (Royston, 2005). All full-data variables, included outcome variables, were used to predict the missing values. This resulted in 20 imputed datasets, combined for the analysis using Rubin's rules (Royston, 2005). For fathers' psychological distress, missing data was 37.2%, 40.3%, and 37.8%, and for mothers' psychological distress, missing data was 19.4%, 18.5%, and 14.3%, at child ages 3, 5, and 11, respectively.

**Figure 4** Path analysis model for paternal and maternal psychological distress



To evaluate the robustness of the results, a sensitivity analysis was performed, including only families with a biological father across child ages 3-14 (sweeps 2-6). This led to a sample of 5,156 cases in total, 2,583 of which involved a female child and 2,573 a male child. Models were run in the same way as in the main analysis, adjusted for covariates.

Last, to explore in more depth the relationships between fathers' distress, mothers' distress, and health risk behaviours, the paths from parental distress trajectories were also examined. Path analysis examines the associations of health risk behaviours with paternal distress measured at specific time-points. On the contrary, assessing the links with paternal distress trajectories allows exploring the effects that the course of paternal symptoms over time might have. Specifically, logistic regression was used to investigate how the trajectories that were identified in Chapter 5 relate to each of the outcomes, while accounting for covariates. Missing data were again handled by multiple imputation. There were 8199 valid cases for the full sample, 4175 for females, and 4024 for males. Findings are presented in the Appendix 2 (tables A9-A12).

## **6.6. Results**

### **6.6.1. Descriptive Statistics**

Comparisons between the analytic and non-analytic samples highlight some sample selection bias (Table 13). Specifically, regarding parental characteristics, fathers and mothers in the analytic sample had significantly lower levels of distress compared to those in the non-analytic sample. Furthermore, they were more likely to be university-educated. Families in the

analytic sample were also less likely to live in poverty and more likely to have a biological father living consistently in the household. Mothers reported consuming less alcohol, though no such differences were observed for fathers. When it comes to offspring variables, it was not possible to compare the prevalence of health risk behaviours, given missing data in the analytic sample leading to small Ns. Nevertheless, it is noticeable that adolescents in the analytic sample were more likely to be female and to have presented signs of puberty. What is more, they had significantly fewer internalising and externalising difficulties. No differences were observed regarding ethnicity.

Table 14 presents the gender differences for each of the outcomes. Boys were significantly more likely to have drunk alcohol than girls. No other differences were found.

**Table 13** Descriptives of the analytic and non-analytic samples (unweighted data)

Categorical Variables	Analytic sample (N=11128)		Non-analytic sample (N=8115)		$\chi^2$
	N	%	N	%	
Smoking	2351	21.13	557	93.14	1.6e+03***
Alcohol drinking	3870	34.78	14	2.34	NA
Binge drinking	1054	9.47	7	1.17	NA
Sexual activity	330	2.97	1	0.17	NA
Girl	5619	50.5	3728	45.9	38.972***
Mixed	322	3.2	196	2.77	3.1568
Indian	259	2.6	180	2.54	0.0724
Pakistani or Bangladeshi	726	7.3	478	6.74	1.9779
Black	385	3.9	278	3.92	0.0262
Other	160	1.6	117	1.65	0.0433

Father is university-educated	3272	46.3	76	31.28	21.2351***
Mother is university-educated	4896	45.9	155	30.10	49.6847***
Biological father	5156	46.3	146	1.80	4.8e+03***
Below 60% household median	3047	30	2039	39.02	128.5304***
Signs of puberty	6232	62.3	1483	57.82	16.8871***

Continuous Variables	Range	N	Mean (SD)	N	Mean (SD)	t
Paternal distress age 3	[0, 24]	6992	2.86 (3.05)	2916	2.91 (3.34)	0.8386
Maternal distress age 3	[0, 24]	8965	3.17 (3.62)	4538	3.51 (4.02)	4.9504***
Paternal distress age 7	[0, 24]	6646	2.92 (3.31)	1668	3.11 (3.76)	2.0446*
Maternal distress age 7	[0, 24]	9069	3.01 (3.76)	2822	3.37 (4.03)	4.3150***
Paternal distress age 11	[0, 24]	6926	3.80 (3.82)	1281	4.14 (4.32)	2.8737**
Maternal distress age 11	[0, 24]	9542	3.89 (4.29)	2293	4.77 (4.94)	8.6052***
Internalising difficulties	[0, 20]	10249	3.12 (3.07)	2537	3.74 (3.54)	8.8324***
Externalising difficulties	[0, 20]	10226	4.30 (3.45)	2536	5.38 (4.02)	13.6882***
Paternal alcohol consumption	[0, 5]	7103	1.64 (1.16)	1321	1.68 (1.24)	0.9535
Maternal alcohol consumption	[0, 5]	9809	1.28 (1.06)	2337	1.35 (1.17)	2.8081**

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

**Table 14** Gender differences in health risk behaviours

	Girls (N=5,619)		Boys (N=5,509)		$\chi^2$
	N	%	N	%	
Smoking	1,159	20.63	1,192	21.64	0.192
Alcohol drinking	1,889	33.62	1,981	35.96	0.01**
Binge drinking	533	9.49	521	9.46	0.959
Sexual activity	162	2.88	168	3.05	0.605

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

## **6.6.2. Path analysis results**

In all models, paternal and maternal distress at age 3 predicted paternal and maternal distress at age 7, respectively. Similarly, paternal and maternal distress at age 7 predicted paternal and maternal distress at age 11, respectively.

### **6.6.2.1. Smoking results**

Tables 15 and 16 present the smoking outcomes, for the unadjusted and adjusted models, respectively. Regarding the unadjusted models, paternal distress at age 11 predicted a higher likelihood of offspring smoking, in the full sample and in the boys' sub-group. Maternal distress at age 11 was linked to smoking for the full sample and females, while maternal distress at age 7 was linked to smoking for the full sample and males. When adjusting for covariates, only the maternal effects at age 7 remained.

Poverty, not living with a biological father, externalising difficulties, and signs of puberty were all related to increased smoking risk. Internalising difficulties were shown to decrease the risk. Additionally, there were significant influences for paternal and maternal alcohol drinking, except that maternal drinking was not associated with smoking for boys. There were no effects of maternal education, though lack of paternal higher education was related to a raised probability of smoking in the full sample analysis. Finally, child ethnicity did not have any impact.



**Table 15** Smoking results for the unadjusted models

	Full Sample		Girls		Boys	
	N=11,128		N=5,619		N=5,509	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → Smoking	0.03 (0.01) <sup>***</sup>	[0.001, 0.004]	0.03 (0.01) <sup>***</sup>	[0.002, 0.005]	0.03 (0.01) <sup>*</sup>	[0.002, 0.005]
MD <sub>age11</sub> → Smoking	0.02 (0.007)	[-0.001, 0.03]	0.03 (0.01) <sup>***</sup>	[0.005, 0.04]	0.002 (0.01)	[-0.02, 0.02]
PD <sub>age7</sub> → Smoking	0.01 (0.01) <sup>***</sup>	[0.001, 0.004]	0.02 (0.02) <sup>***</sup>	[0.002, 0.005]	0.009 (0.01)	[-0.001, 0.003]
MD <sub>age7</sub> → Smoking	0.03 (0.008) <sup>**</sup>	[0.009, 0.04]	0.01 (0.01)	[-0.01, 0.04]	0.04 (0.01) <sup>**</sup>	[0.02, 0.07]
PD <sub>age3</sub> → Smoking	-0.01 (0.01)	[-0.005, 0.02]	-0.03 (0.02)	[-0.001, 0.002]	0.008 (0.02)	[-0.001, 0.003]
MD <sub>age3</sub> → Smoking	0.007 (0.008)	[-0.01, 0.02]	0.01 (0.01)	[-0.01, 0.04]	-0.002 (0.01)	[-0.03, 0.02]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01) <sup>***</sup>	[0.48, 0.51]	0.51 (0.01) <sup>***</sup>	[0.48, 0.53]	0.48 (0.01) <sup>***</sup>	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01) <sup>***</sup>	[0.64, 0.68]	0.65 (0.02) <sup>***</sup>	[0.62, 0.68]	0.67 (0.01) <sup>***</sup>	[0.65, 0.70]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01) <sup>***</sup>	[0.51, 0.54]	0.52 (0.01) <sup>***</sup>	[0.49, 0.54]	0.54 (0.01) <sup>***</sup>	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01) <sup>***</sup>	[0.54, 0.58]	0.55 (0.02) <sup>***</sup>	[0.52, 0.58]	0.57 (0.01) <sup>***</sup>	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress

**Table 16** Smoking results for the adjusted models

	Full Sample		Girls		Boys	
	N=11,128		N=5,619		N=5,509	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → Smoking	0.02 (0.009)	[-0.002, 0.003]	0.02 (0.01)	[-0.001, 0.002]	0.02 (0.01)	[-0.0006, 0.003]
MD <sub>age11</sub> → Smoking	-0.003 (0.008)	[-0.02, 0.01]	0.001 (0.01)	[-0.02, 0.02]	-0.01 (0.01)	[-0.03, 0.02]
PD <sub>age7</sub> → Smoking	0.02 (0.01)	[-0.001, 0.002]	0.02 (0.02)	[-0.001, 0.003]	0.02 (0.02)	[-0.002, 0.002]
MD <sub>age7</sub> → Smoking	0.02 (0.009) <sup>*</sup>	[0.006, 0.04]	0.01 (0.01)	[-0.01, 0.04]	0.03 (0.01) <sup>**</sup>	[0.01, 0.06]
PD <sub>age3</sub> → Smoking	-0.01 (0.01)	[-0.002, 0.005]	-0.03 (0.02)	[-0.003, 0.0007]	0.006 (0.02)	[-0.002, 0.001]
MD <sub>age3</sub> → Smoking	-0.001 (0.009)	[-0.02, 0.02]	0.007 (0.01)	[-0.02, 0.04]	-0.009 (0.01)	[-0.03, 0.03]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01) <sup>***</sup>	[0.48, 0.51]	0.51 (0.01) <sup>***</sup>	[0.48, 0.53]	0.48 (0.01) <sup>***</sup>	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01) <sup>***</sup>	[0.64, 0.68]	0.65 (0.02) <sup>***</sup>	[0.62, 0.68]	0.67 (0.01) <sup>***</sup>	[0.65, 0.70]

PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01) <sup>***</sup>	[0.51, 0.54]	0.52 (0.01) <sup>***</sup>	[0.49, 0.54]	0.54 (0.01) <sup>***</sup>	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01) <sup>***</sup>	[0.54, 0.58]	0.55 (0.02) <sup>***</sup>	[0.52, 0.58]	0.57 (0.01) <sup>***</sup>	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress

### 6.6.2.2. Alcohol use results

Tables 17 and 18 display the findings for alcohol drinking, for the unadjusted and adjusted models, respectively. Maternal distress at age 3 resulted in a lower likelihood of having tried alcohol, in the full sample and in the boys' subsample, even when controlling for confounders.

Poverty and internalising difficulties were significant protective factors. Conversely, maternal alcohol drinking, paternal alcohol drinking, and child externalising difficulties were all significant risk factors. Puberty was a risk factor only for the full sample and girls. Not living with a biological father was a risk factor for girls only. Parental education and ethnicity did not have any influence.

**Table 17** Alcohol drinking results for the unadjusted model

	Full Sample		Girls		Boys	
	N=11,128		N=5,619		N=5,509	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → AU	-0.001 (0.009)	[-0.003, 0.002]	-0.01 (0.01)	[-0.002, 0.002]	0.007 (0.01)	[-0.0009, 0.002]
MD <sub>age11</sub> → AU	-0.001 (0.007)	[-0.01, 0.02]	0.01 (0.01)	[-0.004, 0.03]	-0.01 (0.01)	[-0.03, 0.01]
PD <sub>age7</sub> → AU	-0.004 (0.01)	[-0.002, 0.003]	0.004 (0.01)	[-0.002, 0.001]	-0.01 (0.02)	[-0.03, 0.0002]
MD <sub>age7</sub> → AU	0.009 (0.008)	[-0.005, 0.03]	0.006 (0.01)	[-0.02, 0.03]	0.02 (0.01)	[-0.004, 0.04]
PD <sub>age3</sub> → AU	-0.005 (0.01) <sup>**</sup>	[-0.003, -0.001]	-0.009 (0.01)	[-0.002, 0.001]	-0.0003	[-0.005, -0.002]

MD <sub>age3</sub> → AU	-0.03 (0.008)**	[-0.04, -0.009]	-0.02 (0.01)	[-0.04, 0.007]	-0.03 (0.01)**	[-0.05, -0.001]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01)***	[0.48, 0.51]	0.51 (0.01)***	[0.48, 0.53]	0.48 (0.01)***	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01)***	[0.64, 0.68]	0.65 (0.02)***	[0.62, 0.68]	0.67 (0.01)***	[0.65, 0.70]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01)***	[0.51, 0.54]	0.52 (0.01)***	[0.49, 0.54]	0.54 (0.01)***	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01)***	[0.54, 0.58]	0.55 (0.02)***	[0.52, 0.58]	0.57 (0.01)***	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, AU=Alcohol use

**Table 18** Alcohol drinking results for the adjusted model

	Full Sample		Girls		Boys	
	N=11,128		N=5,619		N=5,509	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
PD <sub>age11</sub> → AU	0.004 (0.009)	[-0.002, 0.001]	-0.009 (0.01)	[-0.004, 0.0002]	0.02 (0.01)	[-0.001, 0.003]
MD <sub>age11</sub> → AU	0.005 (0.007)	[-0.008, 0.02]	0.007 (0.01)	[-0.01, 0.03]	0.002 (0.01)	[-0.02, 0.02]
PD <sub>age7</sub> → AU	-0.0002 (0.01)	[-0.003, 0.0001]	0.006 (0.01)	[-0.003, 0.0003]	-0.006 (0.02)	[-0.004, 0.0001]
MD <sub>age7</sub> → AU	0.01 (0.008)	[-0.005, 0.03]	0.002 (0.01)	[-0.02, 0.03]	0.02 (0.01)	[-0.003, 0.04]
PD <sub>age3</sub> → AU	0.001 (0.01)	[-0.003, 0.0001]	-0.001 (0.01)	[-0.002, 0.001]	0.004 (0.01)	[-0.005, 0.0001]
MD <sub>age3</sub> → AU	-0.02 (0.008)**	[-0.03, -0.001]	-0.02 (0.01)	[-0.04, 0.006]	-0.02 (0.01)*	[-0.04, -0.0007]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01)***	[0.48, 0.51]	0.51 (0.01)***	[0.48, 0.53]	0.48 (0.01)***	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01)***	[0.64, 0.68]	0.65 (0.02)***	[0.62, 0.68]	0.67 (0.01)***	[0.65, 0.70]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01)***	[0.51, 0.54]	0.52 (0.01)***	[0.49, 0.54]	0.54 (0.01)***	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01)***	[0.54, 0.58]	0.55 (0.02)***	[0.52, 0.58]	0.57 (0.01)***	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, AU=Alcohol use

### 6.6.2.3. Binge Drinking results

#### 6.6.2.3. Binge drinking

In terms of binge drinking, table 19 shows the results for the unadjusted models and table 20 shows the results for the adjusted models. In the unadjusted models, maternal difficulties in child ages 7 and 11 were associated with a higher likelihood of binge drinking, for boys and girls, respectively. When adjusting for covariates, only the maternal path at age 7 remained. There were no father paths.

Maternal alcohol drinking, paternal alcohol drinking, not living with a biological father, signs of puberty, as well as child externalising difficulties heightened the risk of binge drinking. On the contrary, experiencing internalising difficulties lowered the risk. Living in poverty did also lower the risk for the full sample and males. The same was the case for black ethnicity, in the whole sample analysis and females. Fathers' higher education had a protective role in the full sample analysis, while mothers' higher education was a risk factor for the full sample and boys.

**Table 19** Binge drinking results for the unadjusted models

	Full Sample		Girls		Boys	
	N=11,128		N=5,619		N=5,509	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → BD	-0.003 (0.01)	[-0.0006, 0.04]	-0.003 (0.02)	[-0.001, 0.004]	-0.002 (0.02)**	[-0.001, -0.006]
MD <sub>age11</sub> → BD	0.007 (0.01)	[-0.02, 0.03]	0.03 (0.01)*	[0.005, 0.06]	-0.02 (0.02)	[-0.06, 0.003]
PD <sub>age7</sub> → BD	0.004 (0.02)	[-0.0005, 0.003]	0.01 (0.03)	[0.0005, -0.006]	-0.006 (0.03)	[-0.003, 0.002]
MD <sub>age7</sub> → BD	0.02 (0.01)	[-0.008, 0.04]	-0.001 (0.02)	[-0.04, 0.03]	0.04 (0.02)*	[0.009, 0.08]
PD <sub>age3</sub> → BD	-0.01 (0.02)	[-0.003, 0.0004]	-0.03 (0.02)	[-0.003, 0.002]	0.001 (0.02)	[-0.004, 0.0007]
MD <sub>age3</sub> → BD	-0.02 (0.01)	[-0.05, 0.005]	-0.02 (0.02)	[-0.05, 0.02]	-0.02 (0.02)	[-0.07, 0.009]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01)***	[0.48, 0.51]	0.51 (0.01)***	[0.48, 0.53]	0.48 (0.01)***	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01)***	[0.64, 0.68]	0.65 (0.02)***	[0.62, 0.68]	0.67 (0.01)***	[0.65, 0.70]

PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01)***	[0.51, 0.54]	0.52 (0.01)***	[0.49, 0.54]	0.54 (0.01)***	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01)***	[0.54, 0.58]	0.55 (0.02)***	[0.52, 0.58]	0.57 (0.01)***	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, BD=Binge drinking

**Table 20** Binge drinking results for the adjusted models

	Full Sample N=11,128		Girls N=5,619		Boys N=5,509	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
PD <sub>age11</sub> → BD	-0.003 (0.01)	[-0.003, 0.001]	-0.006 (0.02)	[-0.005, 0.0004]	0.0004 (0.02)	[-0.002, 0.003]
MD <sub>age11</sub> → BD	0.0009 (0.01)	[-0.02, 0.02]	0.02 (0.02)	[-0.01, 0.05]	-0.02 (0.02)	[-0.06, 0.006]
PD <sub>age7</sub> → BD	0.01 (0.02)	[-0.003, 0.0008]	0.02 (0.03)	[-0.003, 0.003]	0.006 (0.03)	[-0.006, 0.0002]
MD <sub>age7</sub> → BD	0.02 (0.01)	[-0.007, 0.05]	0.0001 (0.02)	[-0.04, 0.04]	0.04 (0.02)*	[0.01, 0.08]
PD <sub>age3</sub> → BD	-0.006 (0.02)	[-0.004, 0.001]	-0.02 (0.03)	[-0.004, 0.0007]	0.003 (0.02)	[-0.006, 0.0001]
MD <sub>age3</sub> → BD	-0.02 (0.01)	[-0.05, 0.007]	-0.02 (0.02)	[-0.05, 0.02]	-0.02 (0.02)	[-0.07, 0.01]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01)***	[0.48, 0.51]	0.51 (0.01)***	[0.48, 0.53]	0.48 (0.01)***	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01)***	[0.64, 0.68]	0.65 (0.02)***	[0.62, 0.68]	0.67 (0.01)***	[0.65, 0.70]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01)***	[0.51, 0.54]	0.52 (0.01)***	[0.49, 0.54]	0.54 (0.01)***	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01)***	[0.54, 0.58]	0.55 (0.02)***	[0.52, 0.58]	0.57 (0.01)***	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, BD=Binge drinking

#### 6.6.2.4. Sexual activity results

Tables 21 and 22 present the sexual activity outcomes, for the unadjusted and adjusted models, respectively. There were no significant relationships, neither for paternal nor maternal distress.

When it comes to covariates, not living with a biological father, externalising difficulties, and signs of puberty were linked to an escalated probability of

early sexual engagement. The same was the case for fathers' alcohol use in the full sample, and maternal alcohol use in the full sample and males. Internalising difficulties had a protective effect, and so was the case for maternal higher education, though only for the full sample and females. Paternal education, poverty, and ethnicity did not have any influence.

**Table 21** Sexual activity results for the adjusted models

	Full Sample N=11,128		Girls N=5,619		Boys N=5,509	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → SA	0.02 (0.02)	[-0.001, 0.005]	0.03 (0.03)	[-0.003, 0.005]	0.01 (0.03)	[-0.001, 0.007]
MD <sub>age11</sub> → SA	0.01 (0.02)	[-0.02, 0.05]	0.02 (0.02)	[-0.03, 0.06]	0.01 (0.02)	[-0.04, 0.06]
PD <sub>age7</sub> → SA	0.008 (0.03)*	[0.0006, 0.007]	0.02 (0.04)**	[0.002, 0.02]	-0.002 (0.04)	[-0.003, 0.006]
MD <sub>age7</sub> → SA	0.04 (0.02)	[-0.006, 0.07]	0.02 (0.03)	[-0.03, 0.08]	0.05 (0.03)	[-0.008, 0.10]
PD <sub>age3</sub> → SA	-0.03 (0.03)	[-0.003, 0.002]	-0.05 (0.04)	[-0.004, 0.004]	-0.01 (0.04)	[-0.005, 0.003]
MD <sub>age3</sub> → SA	-0.0002 (0.02)	[-0.03, 0.05]	0.006 (0.03)	[-0.04, 0.07]	-0.006 (0.03)	[-0.06, 0.06]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.50 (0.01)***	[0.48, 0.51]	0.51 (0.01)***	[0.48, 0.53]	0.48 (0.01)***	[0.46, 0.51]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.66 (0.01)***	[0.64, 0.68]	0.65 (0.02)***	[0.62, 0.68]	0.67 (0.01)***	[0.65, 0.70]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.53 (0.01)***	[0.51, 0.54]	0.52 (0.01)***	[0.49, 0.54]	0.54 (0.01)***	[0.52, 0.56]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.56 (0.01)***	[0.54, 0.58]	0.55 (0.02)***	[0.52, 0.58]	0.57 (0.01)***	[0.54, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, SA=Sexual activity

**Table 22** Sexual activity results for the adjusted models

	Full Sample N=11,128		Girls N=5,619		Boys N=5,509	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → SA	0.02 (0.02)	[-0.004, 0.002]	0.03 (0.03)	[-0.007, 0.002]	0.02 (0.03)	[-0.003, 0.006]
MD <sub>age11</sub> → SA	0.01 (0.02)	[-0.02, 0.05]	0.005 (0.03)	[-0.04, 0.05]	0.01 (0.03)	[-0.04, 0.06]
PD <sub>age7</sub> → SA	0.01 (0.03)	[-0.002, 0.004]	0.02 (0.04)	[-0.002, 0.006]	0.006 (0.04)	[-0.004, 0.005]

MD <sub>age7</sub> → SA	0.02 (0.02)	[-0.004, 0.002]	0.03 (0.03)	[-0.007, 0.002]	0.02 (0.03)	[-0.003, 0.006]
PD <sub>age3</sub> → SA	0.01 (0.02)	[-0.02, 0.05]	0.005 (0.03)	[-0.04, 0.05]	0.01 (0.03)	[-0.04, 0.06]
MD <sub>age3</sub> → SA	0.01 (0.03)	[-0.002, 0.004]	0.02 (0.04)	[-0.002, 0.006]	0.006 (0.04)	[-0.004, 0.005]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.02 (0.02)	[-0.004, 0.002]	0.03 (0.03)	[-0.007, 0.002]	0.02 (0.03)	[-0.003, 0.006]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.01 (0.02)	[-0.02, 0.05]	0.005 (0.03)	[-0.04, 0.05]	0.01 (0.03)	[-0.04, 0.06]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.01 (0.03)	[-0.002, 0.004]	0.02 (0.04)	[-0.002, 0.006]	0.006 (0.04)	[-0.004, 0.005]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.02 (0.02)	[-0.004, 0.002]	0.03 (0.03)	[-0.007, 0.002]	0.02 (0.03)	[-0.003, 0.006]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, SA=Sexual activity

## 6.7. Sensitivity analysis

### 6.7.1. Smoking results

A sensitivity analysis was conducted, in which only families with a biological father across child ages 3-14 were included. Smoking outcomes, adjusted for covariates, are presented in table 23. The maternal effect at age 7 that was found in the main analysis ceased to be significant, while no other paths emerged.

### 6.7.2. Alcohol use results

Table 24 shows the sensitivity analysis' results for alcohol drinking. Maternal distress at age 3 no longer had a significant impact. However, paternal distress at ages 3, 7, and 11, was linked to a lower risk of drinking, in the full sample analysis.

**Table 23** Smoking results for the adjusted models, sensitivity analysis

Full Sample	Girls	Boys
N=5,156	N=2,583	N=2,573

	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → Smoking	-0.0004 (0.002)	[-0.002, 0.006]	0.03 (0.02)	[-0.01, 0.004]	0.02 (0.02)	[-0.0003, 0.01]
MD <sub>age11</sub> → Smoking	0.01 (0.01)	[-0.01, 0.04]	0.02 (0.02)	[-0.02, 0.06]	-0.003 (0.02)	[-0.04, 0.04]
PD <sub>age7</sub> → Smoking	0.0002 (0.001)	[-0.003, 0.003]	0.02 (0.02)	[-0.003, 0.006]	0.02 (0.02)	[-0.006, 0.003]
MD <sub>age7</sub> → Smoking	0.02 (0.02)	[-0.02, 0.05]	0.008 (0.02)	[-0.03, 0.07]	0.02 (0.02)	[-0.02, 0.07]
PD <sub>age3</sub> → Smoking	-0.001 (0.001)	[-0.003, 0.002]	-0.05 (0.03)	[-0.005, 0.002]	-0.01 (0.03)	[-0.004, 0.003]
MD <sub>age3</sub> → Smoking	-0.01 (0.02)	[-0.04, 0.02]	0.004 (0.02)	[-0.05, 0.05]	-0.02 (0.02)	[-0.07, 0.03]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.18 (0.01)***	[0.15, 0.20]	0.21 (0.02)***	[0.18, 0.24]	0.14 (0.02)***	[0.11, 0.17]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.64 (0.01)***	[0.61, 0.66]	0.64 (0.02)***	[0.60, 0.68]	0.63 (0.02)***	[0.59, 0.67]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.22 (0.01)***	[0.19, 0.24]	0.20 (0.02)***	[0.17, 0.23]	0.23 (0.02)***	[0.20, 0.26]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.54 (0.01)***	[0.52, 0.57]	0.52 (0.02)***	[0.49, 0.56]	0.56 (0.02)***	[0.52, 0.60]

\**p* < .05, \*\**p* < .01, \*\*\**p* < .001, CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress

**Table 24** Alcohol drinking results for the adjusted models, sensitivity analysis

	Full Sample		Girls		Boys	
	N=5,156		N=2,583		N=2,573	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → AU	-0.004 (0.002)*	[-0.007, -0.001]	0.02 (0.02)	[-0.008, 0.002]	0.03 (0.02)	[-0.007, 0.004]
MD <sub>age11</sub> → AU	0.02 (0.01)	[-0.002, 0.05]	0.03 (0.02)	[-0.002, 0.07]	0.003 (0.02)	[-0.02, 0.04]
PD <sub>age7</sub> → AU	-0.005 (0.001)***	[-0.007, -0.002]	0.007 (0.02)	[-0.009, 0.0001]	-0.009 (0.02)	[-0.009, 0.001]
MD <sub>age7</sub> → AU	0.002 (0.01)	[-0.03, 0.02]	0.002 (0.02)	[-0.06, 0.02]	0.02 (0.02)	[-0.03, 0.05]
PD <sub>age3</sub> → AU	-0.004 (0.001)****	[-0.006, -0.001]	-0.02 (0.02)	[-0.005, 0.0007]	-0.009 (0.02)	[-0.008, 0.001]
MD <sub>age3</sub> → AU	-0.02 (0.01)	[-0.05, 0.007]	-0.02 (0.02)	[-0.06, 0.02]	-0.02 (0.02)	[-0.06, 0.02]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.18 (0.01)***	[0.15, 0.20]	0.21 (0.02)***	[0.18, 0.24]	0.14 (0.02)***	[0.11, 0.17]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.64 (0.01)***	[0.61, 0.66]	0.64 (0.02)***	[0.60, 0.68]	0.63 (0.02)***	[0.59, 0.67]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.22 (0.01)***	[0.19, 0.24]	0.20 (0.02)***	[0.17, 0.23]	0.23 (0.02)***	[0.20, 0.26]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.54 (0.01)***	[0.52, 0.57]	0.52 (0.02)***	[0.49, 0.56]	0.56 (0.02)***	[0.52, 0.60]

\**p* < .05, \*\**p* < .01, \*\*\**p* < .001, CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, AU=Alcohol usage



### 6.7.3. Binge drinking results

Sensitivity analysis' results from binge drinking are presented on table 25. Maternal distress at age 7 was no longer significant; however, maternal distress at age 11 was associated with higher likelihood for binge drinking in the full sample and in girls. Paternal paths also emerged. Specifically, for the full sample, fathers' distress at ages 3 and 11 predicted a lower binge drinking risk.

**Table 25** Binge drinking results for the adjusted models, sensitivity analysis

	Full Sample		Girls		Boys	
	N=5,156		N=2,583		N=2,573	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → BD	-0.009 (0.004)*	[-0.02, -0.001]	0.01 (0.03)	[-0.02, 0.005]	-0.02 (0.03)	[-0.02, 0.004]
MD <sub>age11</sub> → BD	0.04 (0.02)*	[0.005, 0.09]	0.06 (0.03)*	[0.02, 0.13]	0.008 (0.03)	[-0.05, 0.07]
PD <sub>age7</sub> → BD	0.0001 (0.002)	[-0.004, 0.005]	-0.003 (0.04)	[-0.004, 0.008]	0.01 (0.03)	[-0.008, 0.006]
MD <sub>age7</sub> → BD	-0.03 (0.02)	[-0.08, 0.02]	-0.06 (0.04)	[-0.14, 0.009]	0.007 (0.04)	[-0.05, 0.09]
PD <sub>age3</sub> → BD	-0.004 (0.002)*	[-0.008, -0.0003]	-0.01 (0.04)	[-0.007, 0.004]	0.002 (0.04)	[-0.01, 0.00004]
MD <sub>age3</sub> → BD	-0.03 (0.02)	[-0.08, 0.02]	-0.02 (0.03)	[-0.09, 0.04]	-0.05 (0.04)	[-0.12, 0.02]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.18 (0.01)***	[0.15, 0.20]	0.21 (0.02)***	[0.18, 0.24]	0.14 (0.02)***	[0.11, 0.17]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.64 (0.01)***	[0.61, 0.66]	0.64 (0.02)***	[0.60, 0.68]	0.63 (0.02)***	[0.59, 0.67]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.22 (0.01)***	[0.19, 0.24]	0.20 (0.02)***	[0.17, 0.23]	0.23 (0.02)***	[0.20, 0.26]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.54 (0.01)***	[0.52, 0.57]	0.52 (0.02)***	[0.49, 0.56]	0.56 (0.02)***	[0.52, 0.60]

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, BD=Binge drinking

### 6.7.4. Sexual activity results

Finally, table 26 displays the results for sexual activity. As with the main analysis, neither paternal nor maternal distress had a significant influence.

**Table 26** Sexual activity results for the adjusted models, sensitivity analysis

	Full Sample		Girls		Boys	
	N=5,156		N=2,583		N=2,573	
	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs	<i>B</i> (SE)	95% CIs
PD <sub>age11</sub> → SA	-0.03 (0.02)	[-0.08, 0.02]	-0.02 (0.03)	[-0.09, 0.04]	-0.05 (0.04)	[-0.12, 0.02]
MD <sub>age11</sub> → SA	0.18 (0.01) <sup>***</sup>	[0.15, 0.20]	0.21 (0.02) <sup>***</sup>	[0.18, 0.24]	0.14 (0.02) <sup>***</sup>	[0.11, 0.17]
PD <sub>age7</sub> → SA	0.64 (0.01) <sup>***</sup>	[0.61, 0.66]	0.64 (0.02) <sup>***</sup>	[0.60, 0.68]	0.63 (0.02) <sup>***</sup>	[0.59, 0.67]
MD <sub>age7</sub> → SA	0.22 (0.01) <sup>***</sup>	[0.19, 0.24]	0.20 (0.02) <sup>***</sup>	[0.17, 0.23]	0.23 (0.02) <sup>***</sup>	[0.20, 0.26]
PD <sub>age3</sub> → SA	0.54 (0.01) <sup>***</sup>	[0.52, 0.57]	0.52 (0.02) <sup>***</sup>	[0.49, 0.56]	0.56 (0.02) <sup>***</sup>	[0.52, 0.60]
MD <sub>age3</sub> → SA	-0.03 (0.02)	[-0.08, 0.02]	-0.02 (0.03)	[-0.09, 0.04]	-0.05 (0.04)	[-0.12, 0.02]
PD <sub>age7</sub> → PD <sub>age11</sub>	0.18 (0.01) <sup>***</sup>	[0.15, 0.20]	0.21 (0.02) <sup>***</sup>	[0.18, 0.24]	0.14 (0.02) <sup>***</sup>	[0.11, 0.17]
MD <sub>age7</sub> → MD <sub>age11</sub>	0.64 (0.01) <sup>***</sup>	[0.61, 0.66]	0.64 (0.02) <sup>***</sup>	[0.60, 0.68]	0.63 (0.02) <sup>***</sup>	[0.59, 0.67]
PD <sub>age3</sub> → PD <sub>age7</sub>	0.22 (0.01) <sup>***</sup>	[0.19, 0.24]	0.20 (0.02) <sup>***</sup>	[0.17, 0.23]	0.23 (0.02) <sup>***</sup>	[0.20, 0.26]
MD <sub>age3</sub> → MD <sub>age7</sub>	0.54 (0.01) <sup>***</sup>	[0.52, 0.57]	0.52 (0.02) <sup>***</sup>	[0.49, 0.56]	0.56 (0.02) <sup>***</sup>	[0.52, 0.60]

\**p* < .05, \*\**p* < .01, \*\*\**p* < .001, CIs=Confidence intervals, PD=Paternal distress, MD=Maternal distress, SA=Sexual activity

## 6.8. Trajectory modelling results

Appendix 2 presents the results for the links between paternal and maternal distress trajectories across child ages 3-11 (identified in Chapter 5) and adolescent engagement in health risk behaviours. Paternal ‘moderate-increasing’ distress predicted a higher likelihood of smoking for the full sample and for boys. It also predicted a higher likelihood of drinking alcohol, though only for boys. Last, the maternal ‘severe distress’ trajectory was linked to an escalated probability of smoking in the full sample and in the girls’ sub-analysis. No significant relationships emerged for binge drinking and sexual activity.

## 6.9. Discussion

This research aimed to explore the longitudinal links between paternal distress and offspring engagement in health risk behaviours in the general population. Specifically, the research looked at paths from paternal distress assessed at ages 3, 7, and 11 to adolescent health risk behaviours at age 14. The model also controlled for the links between paternal distress at ages 3, 7, and 11. Paths from maternal distress to adolescent outcomes were examined in the same way. Overall, in line with the study's hypothesis, maternal distress was associated with more health risk behaviours in boys, compared to girls. Specifically, when adjusting for covariates, maternal distress at age 7 led to higher odds of having smoked at least once, for the full sample and boys. It also led to higher odds of having engaged in binge drinking, though only for boys. Furthermore, maternal distress at age 3 was associated with a lower likelihood of having tried alcohol at 14, for the full sample and boys. No paths were predicting sexual activity, and paternal distress was not shown to have any impact.

A sensitivity analysis was performed, to investigate if findings would remain similar in a sample involving only families which had a biological father in the household across child ages 3-14. Interestingly, there were several differences in the results, with paternal paths emerging. Particularly, for smoking and alcohol outcomes, maternal distress no longer had a significant influence. Instead, paternal distress at ages 3, 7, and 11, was linked to a lower risk for alcohol use. For binge drinking, the maternal effect at age 7 was replaced by an effect at age 11, with higher maternal difficulties predicting a higher risk of binge drinking. On the contrary, paternal distress at ages 3 and

11 predicted a lower binge drinking risk. In terms of sexual activity, there were no significant relationships.

### **6.9.1. Smoking**

Paternal distress did not influence the likelihood of trying smoking; a result which is in agreement with past research (Essau & de la Torre-Luque, 2021). On the contrary, maternal distress at age 7 had a significant effect. This outcome supports the conclusions of Wickham et al., (2015), who argue that middle childhood is a critical period. Moreover, it is noticeable that this effect was present in the sub-analysis looking at boys but not girls. Parental distress is linked to decreased parental monitoring (Eckshtain et al., 2010; Kelly et al., 2017); nevertheless, monitoring is more strongly associated with boys' rather than girls' behaviour (O'Donnell et al., 2012). In other words, mothers experiencing high distress may not have effectively monitored their children, but this impacted only their sons, by increasing their riskiness.

The maternal path found did not prevail in the sensitivity analysis, possibly due to differences in the levels of psychological distress experienced by mothers in the two samples. Specifically, it is likely that mothers in the sensitivity sub-sample had lower levels of distress compared to mothers in the main analysis, as they remained in intact families (Symoens et al., 2014). Consequently, data may have lacked the 'strength' required to capture this effect.

### **6.9.2. Alcohol drinking**

In the main analysis, maternal distress at age 3 predicted a decreased risk of alcohol drinking for the full sample and boys. This finding contradicts past

research that has found that maternal distress was linked to elevated alcohol use in adolescence (Flouri & Ioakeimidi, 2018; Herman-Stahl et al., 2017; Lamis et al., 2012; Wickham et al., 2015). Methodological differences exist between the studies; for instance, the current investigation explored whether the child had ever tried alcohol, while Flouri & Ioakeimidi, (2018) explored overall antisocial behaviour, which included alcohol use. Furthermore, adjustments were different, with most of the past research not co-examining paternal distress.

In the sensitivity analysis, maternal distress was no longer significant; instead, paternal distress at ages 3, 7, and 11 was associated with lower alcohol use. Similarly to the current results, Herman-Stahl et al., (2008) found paternal distress to predict a lower likelihood of binge drinking in black families. One explanation proposed by the authors is that children of mentally ill parents adopt more 'responsible' behaviours than their peers (Herman-Stahl et al., 2008). Qualitative evidence also supports this point; these children often report that, as their parents may struggle to meet the demands of their caregiving role, they have to step up, for instance, by caring for their younger siblings (Dam & Hall, 2016). Considering the higher number of responsibilities these children may have, it is likely that they learn to behave in more mature ways. However, it should be noted that, having a greater number of responsibilities could also act as a risk factor for the development of emotional difficulties (Källquist & Salzmänn-Erikson, 2019). Further research is needed to explore this point.

While maternal paths emerged in the main analysis, in the sensitivity analysis, only paternal distress was significant. Children in the main analysis were not

as exposed to their fathers' presence as children in the sensitivity analysis; therefore, it is expected to have more paternal effects in the latter. However, it is also noticeable that in the sensitivity analysis paternal distress appears to be considerably more influential on youth alcohol usage than maternal distress. It is possible that mothers, due to societal expectations, continue to provide care for their families, even if they are experiencing distress. On the other hand, distressed fathers are more likely to withdraw, placing their role responsibilities on their children, which in turn may lead them to avoid reckless behaviours (Paulson et al., 2009).

### **6.9.3. Binge drinking**

The pattern of findings for binge drinking is similar to alcohol usage. Only maternal distress was a significant predictor in the main analysis, and it remained significant in the sensitivity analysis. Paternal paths also emerged in the sensitivity analysis only.

In line with the results for alcohol usage, paternal distress lowered the risk of engaging in binge drinking. On the contrary, maternal distress was shown to increase this risk. These outcomes indicate that, when it comes to excessive alcohol consumption, fathers' and mothers' roles are different. The meta-analysis by Yap et al., (2017) suggests that maternal parenting practices, such as monitoring and support, have a greater preventative effect than paternal parenting practices. Considering that distressed mothers tend to be non-engaged with their children (or to the contrary, engage in harsher parenting behaviour), the presence of maternal distress is likely to increase youth risky engagement, while the same may not be the case for fathers.

Moreover, it is contradictory that, while maternal distress was linked a lower likelihood for alcohol usage, when it comes to binge drinking, the opposite was the case. It should be considered that, for alcohol usage, the influence of maternal distress occurred at age 3, while for binge drinking, associations were found at later ages. This may imply that maternal distress may affect offspring differently depending on the timing of the effects. Parenting practices may again play a role; for instance, lack of monitoring in late childhood might predict delinquency, while this will not be the case in the preschool years. Nevertheless, further research is needed to explore these mechanisms.

#### **6.9.4. Sexual activity**

Past research has not explored the relationship between paternal distress and offspring sexual activity. In the present study, no link was found between the two. In terms of maternal distress, Sang et al., (2016) did examine the association with youth sexual engagement, reaching supportive conclusions. However, their study was cross-sectional. The current investigation explored longitudinal paths but did not identify any significant effects.

#### **6.9.5. The links between covariates and adolescent health risk behaviours**

Several significant associations between covariates and adolescent health risk behaviours were identified. In terms of individual factors, puberty was associated with all health risk behaviours. This finding is in line with past research (De Azevedo et al., 2017), and attributed to the hormonal and brain development changes that these adolescents undergo (Smith, Chein, & Steinberg, 2013). Furthermore, significant links were consistently found for

externalising difficulties, as deviant and hyperactive adolescents are more prone to riskiness (Cox et al., 2021; Isaksson et al., 2020; McLeod & Knight, 2010; Romero et al., 2017). On the contrary, internalising difficulties had a protective effect, which is aligned with the findings of some studies (Isaksson et al., 2020), but not others, which found internalising difficulties to be a risk factor (Hussong et al., 2011). It is argued that internalising difficulties are a risk factor only when externalising difficulties are unadjusted for, as the two may co-exist (Foster, Hicks, & Zucker, 2018).

When it comes to family-level factors, not living with a biological father was associated with all risk behaviours, a result in line with past research (McCutcheon et al., 2018; Waldron et al., 2014, 2015). It is possible that adolescents who have experienced parental separation are less monitored by their caregivers; therefore, have more opportunities for delinquency (Dargahi et al., 2018). Significant paths were also found for paternal and maternal alcohol usage, again in agreement with past studies (Cox et al., 2021; McCutcheon et al., 2018; Waldron et al., 2014, 2015; Yap et al., 2017). Multiple pathways are considered to be in place; for example, those adolescents may have easier access to substances or may normalize unhealthy behaviours.

Interestingly, poverty was a risk factor for smoking, but a protective factor for alcohol and binge drinking. It was not associated with sexual activity. Adolescents from higher socio-economic statuses are more likely to consume alcohol, possibly because it is easier for them to access it (Donath et al., 2012; Melotti et al., 2011). Research literature shows that the opposite is the



case for smoking (Melotti et al., 2011), suggesting that the pathways between socio-economic position and substance usage are complex.

Last, it is possible that some of the confounders may have had a stronger influence of health risk behaviours than paternal and maternal distress. Unfortunately, due to limitations in the model and programme used, it was not possible to explore this (STATA does not provide standardized coefficients for generalized structural equation modelling).

#### **6.9.6. Links with paternal and maternal trajectories**

A supplementary analysis examined the associations between paternal and maternal distress trajectories (as identified in Chapter 5) and health risk behaviours (findings reported in Appendix 2). In summary, paternal 'moderate-increasing' distress predicted a higher likelihood of smoking, in the full sample and boys. It also predicted a higher likelihood of smoking in boys only. Maternal 'severe distress' predicted an elevated risk for smoking in the full sample and girls. There were no other significant paths.

It is noticeable that trajectories of both fathers' and mothers' distress were related to higher odds for risk engagement. This outcome comes in contrast with many of the findings from the main and the sensitivity analysis, showing that elevated parental distress can protect against risk engagement. In other words, it appears that parental distress experienced continuously through childhood raises riskiness, while parental distress measured at specific time points lowers it. Further research is needed to explore this conclusion, and to evaluate the reasons it may be so.

### **6.9.7. Limitations**

This research includes some limitations. To begin with, families in the analytic sample were more privileged than those in the non-analytic. For instance, they were more affluent, parents experienced lower levels of distress, and adolescents were less likely to smoke (statistical comparisons for the other outcomes were not possible). This not only limits the generalizability of the conclusions but could also mean that the study did not identify relationships that may take place in more disadvantaged households. Furthermore, the study is limited by the MCS data that is available. Though the research literature implies that parental monitoring could play a mediating role in the associations found (Elgar et al., 2007), it was not possible to explore those pathways, since no reliable parental monitoring data are available in the MCS. Additionally, even for significant pathways, effect sizes were quite small. Last, though initially this investigation aimed to also examine the impact of parental clinical distress and to include an additional child outcome (drug use), the number of cases was not sufficient.

### **6.9.8. Conclusions and Implications**

This research explored the paths from paternal distress in early, middle, and late childhood on adolescent health risk behaviours. Maternal paths were explored in parallel. Overall, results indicate that both fathers' and mothers' distress affect the development of those behaviours.

The current findings, in line with past research, provide an ambiguous picture as to the direction of the effects. For instance, while some results suggest that parental distress is linked to a lower risk for alcohol drinking, others show the

opposite. Additionally, the present study indicates that the strength of fathers' and mothers' influence varies, depending on the family structure. Future research could continue to investigate those issues, to help gain a better understanding of those relationships.

As discussed previously, a limitation of the current study is that it did not have the measures available to examine the role of parenting practices, such as monitoring or hostility (which was measured only at child age 3). There is an increasing amount of research suggesting that parenting practices play a role in youth risk engagement; examining the association of those practices along with fathers' distress would be an interesting idea moving forward.

## **Chapter 7: Discussion**

### **7.1. Strengths and implications**

The scope of this thesis was to examine the associations between fathers' psychological distress and child outcomes. A strength is that a large and relatively representative sample was used, allowing to study patterns in the general population. Some of the past studies assessing the influence of paternal distress were limited by the fact that they did not adequately account for confounders. In the present research, the data enabled to control for socio-demographic confounders and maternal distress, meaning that all paths identified were robust and independent of those effects. Additionally, data allowed exploring longitudinal relationships, to understand the long-term impact of paternal distress.

Results demonstrate that paternal psychological distress is linked to child outcomes, including internalising and externalising difficulties, decision-making, and alcohol use. While a substantive number of past studies have investigated the pathways from maternal distress, the influence of paternal distress is relatively less explored. This thesis has contributed to expanding knowledge around the effects of fathers' distress on children. Moreover, it has been highlighted that fathers' distress does play a role and can shape child development. It is important that policymakers and practitioners involved with vulnerable children (i.e., children with mental health difficulties) consider how the well-being of both parents could be impacting the child. It would perhaps be beneficial to offer mental health assessments and support to mothers and well as fathers. A greater emphasis should be given to the mental health of men. There is some concern that depression and anxiety symptoms in men are underreported; raising awareness and conducting regular screening could

perhaps help support those individuals (Smith, Mouzon, & Elliott, 2018). This would not only boost the public's mental health but would also promote the well-being of children, by improving their family environment.

As highlighted, much of the past research concerning fathers' distress has focused on the early years and less is known regarding adolescence. To address this research gap, a substantive amount of this thesis examined adolescent outcomes. Importantly, this research was quite novel in investigating associations between paternal psychological distress and adolescent cognitive functioning and health risk engagement, as relevant research is very scarce. Results were unexpected, showing that overall, fathers' psychological distress is linked to less impulsivity and lower risk engagement. Risky engagement, including substance abuse, starts in adolescence (Edwards et al., 214; East et al., 2018). For policy and prevention purposes, it is crucial to know which adolescents are most likely to engage in such behaviours; current findings indicate that offspring of distressed fathers are not those at risk.

Another key conclusion reached is that, while it is mostly paternal distress that influences child mental health, child difficulties can also harm fathers' well-being. Therefore, when working with fathers with mental health problems, it would be useful for clinicians to consider how their interactions with their offspring may be affecting them. Furthermore, when designing family support interventions, the role of caregivers, as well as children, should be taken into account. Indeed, programmes promoting a whole-family approach are shown to be beneficial (Luciano et al., 2012).

Last, mediation analysis revealed that parenting practices, such as closeness, conflict, and involvement, do to some extent account for the pathways between paternal distress and child peer and externalising difficulties. Hence, to prevent

adverse influences between distressed fathers and their children, family practitioners could try working on and improving those interactions between them. For instance, it may be suggested that they allocate more time doing activities with one another. It should be noted that bidirectional links were found between paternal distress and child conduct problems, across ages 3 to 5; these links were mediated by parenting practices. Consequently, improving those practices may not only buffer negative effects but may also help fathers and children enhance their own well-being over time.

## **7.2. Limitations and directions for future research**

This thesis includes some limitations that should be discussed. To begin with, though a source of large-scale and representative UK data was used, sample selection bias was consistently detected. Specifically, bias was detected in terms of socio-demographic characteristics (i.e., family income) and paternal distress levels, with fathers excluded from the analysis experiencing higher distress than those included. Therefore, it may be the case that some effects, occurring in families with severe difficulties, may not have been captured by this research. Future studies could focus on the role of paternal distress in disadvantaged families, to understand whether the same or different relationships are in place.

In addition, due to data restrictions, it was possible to look at mediating pathways only for effects occurring in the early years. Causal mechanisms were not explored for adolescence, even though a few associations were identified. For instance, it was not possible to investigate why paternal distress predicts adolescent peer difficulties, decision-making, and alcohol usage. Parenting practices such as monitoring, or hostility, likely play a role.

Another issue to consider, is that this thesis has applied binary p-values to discern significant to non-significant effects. It should be acknowledged that the usage of p-values has been criticized by researchers for leading to inaccurate conclusions (Greenland et al., 2016; Griffiths & Needleman, 2019; Li et al., 2021). For instance, an arbitrary cut-off is used to distinguish significant from non-significant effects (Griffiths & Needleman, 2019). This distinction means that the p-value may not demonstrate the actual importance of the effect; a statistically significant effect, may not be as meaningful (Greenland et al., 2016). Likewise, especially in cases of studies with small sample sizes, a non-significant p-value does not necessarily imply the absence of an effect (Li et al., 2021). It is suggested that, instead of the p-value, emphasis should be given to effect sizes and confidence intervals (Griffiths & Needleman, 2019), which are more relevant to clinical practice (Du Prel et al., 2009). It has also been proposed that p-values as a continuous rather than a binary measure will be more informative of a study's results (Greenland et al., 2016; Li et al., 2021).

Despite the afore-mentioned limitations of the binary usage of the p-value, this was applied in the present thesis, to allow for easier comparison to past studies' findings. The thesis' results should be considered not only in terms of significance, but also in terms of effect sizes and confidence intervals. The effect sizes of this thesis, even in cases of statistically significant effects, were quite small, which should be acknowledged as a limitation.

Last, this thesis has evaluated the relationship between fathers' psychological distress and child outcomes using solely a quantitative approach. Despite the importance of the conclusions that can be drawn from quantitative data, the potential contribution of qualitative evidence should also be acknowledged. Gaining a more in-

depth and 'personal' account of these family relationships can significantly develop the current understanding and highlight directions for future research.

### **7.3. Conclusion**

This thesis aimed to examine the pathways between paternal psychological distress and child development in the general population. The child outcomes assessed included internalising and externalising difficulties, cognitive functioning, and health risk engagement. The conclusions reached have clinical and policy implications; they also provide some suggestions that upcoming studies could explore. Fathers today have a more active role in their children's upbringing; research should help pinpoint the effects that occur and the underlying causal mechanisms. Especially when it comes to vulnerable children, it is crucial to understand the interactions taking place in the family environment and to use this evidence in building effective support strategies.



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## Appendix 1

**Table A1** Bivariate correlations between paternal and maternal distress

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Age 3 Paternal distress	1									
2. Age 5 Paternal distress	.55	1								
3. Age 7 Paternal distress	.51	.58	1							
4. Age 11 Paternal distress	.43	.49	.55	1						
5. Age 14 Paternal distress	.42	.46	.52	.61	1					
6. Age 3 Maternal distress	.18	.15	.13	.13	.14	1				
7. Age 5 Maternal distress	.12	.18	.16	.15	.14	.56	1			
8. Age 7 Maternal distress	.12	.15	.19	.16	.11	.53	.59	1		
9. Age 11 Maternal distress	.14	.13	.15	.22	.17	.48	.51	.55	1	
10. Age 14 Maternal distress	.11	.13	.13	.14	.18	.46	.48	.51	.61	1

All correlation are significant at  $p < 0.001$  level.

**Table A2** Bivariate correlations between paternal, maternal distress and emotional symptoms

	Age 3 PD	Age 5 PD	Age 7 PD	Age 11 PD	Age 14 PD	Age 3 MD	Age 5 MD	Age 7 MD	Age 11 MD	Age 14 MD	Age 3 Emotio nal	Age 5 Emotio nal	Age 7 Emotio nal	Age 11 Emotio nal	Age 14 Emotio nal
Age 3 Emotional	.09	.08	.07	.09	.08	.23	.19	.17	.19	.17	1				
Age 5 Emotional	.10	.08	.09	.10	.10	.21	.27	.20	.22	.21	.43	1			
Age 7 Emotional	.10	.08	.11	.09	.08	.22	.24	.30	.24	.24	.34	.50	1		
Age 11 Emotional	.11	.09	.11	.14	.12	.23	.24	.25	.34	.30	.26	.38	.50	1	
Age 14 Emotional	.10	.09	.11	.13	.15	.20	.21	.22	.27	.31	.23	.33	.40	.54	1

All correlation are significant at  $p < 0.001$  level. Emotional=Child emotional symptoms, PD=Paternal distress, MD=Maternal distress

**Table A3** Bi-variate correlations between paternal, maternal distress and conduct problems

	Age 3 PD	Age 5 PD	Age 7 PD	Age 11 PD	Age 14 PD	Age 3 MD	Age 5 MD	Age 7 MD	Age 11 MD	Age 14 MD	Age 3 Condu ct	Age 5 Condu ct	Age 7 Condu ct	Age 11 Condu ct	Age 14 Condu ct
Age 3 Conduct	.11	.12	.11	.10	.08	.28	.23	.21	.22	.20	1				
Age 5 Conduct	.09	.11	.11	.11	.10	.21	.26	.23	.21	.20	.49	1			
Age 7 Conduct	.10	.11	.12	.11	.09	.20	.23	.27	.23	.21	.43	.59	1		
Age 11 Conduct	.09	.08	.09	.13	.12	.20	.20	.23	.29	.23	.39	.48	.56	1	
Age 14 Conduct	.09	.10	.14	.14	.16	.17	.18	.21	.25	.24	.33	.41	.48	.61	1

All correlation are significant at  $p < 0.001$  level. Conduct=Child conduct problems, PD=Paternal distress, MD=Maternal distress

**Table A4** Bi-variate correlations between paternal, maternal distress and child hyperactivity/inattention

	Age 3 PD	Age 5 PD	Age 7 PD	Age 11 PD	Age 14 PD	Age 3 MD	Age 5 MD	Age 7 MD	Age 11 MD	Age 14 MD	Age 3 Hyper.	Age 5 Hyper.	Age 7 Hyper.	Age 11 Hyper.	Age 14 Hyper.
Age 3 Hyper.	.08	.09	.09	.08	.07	.21	.17	.17	.17	.18	1				

Age 5 Hyper.	.09	.10	.11	.09	.08	.21	.23	.21	.21	.19	.57	1			
Age 7 Hyper.	.10	.09	.10	.11	.09	.18	.20	.22	.21	.18	.51	.68	1		
Age 11 Hyper.	.08	.09	.09	.13	.10	.20	.20	.22	.28	.22	.43	.57	.67	1	
Age 14 Hyper.	.09	.07	.09	.13	.13	.18	.18	.20	.24	.24	.40	.50	.58	.68	1

All correlation are significant at  $p < 0.001$  level. Hyper.=Child hyperactivity/inattention, PD=Paternal distress, MD=Maternal distress

**Table A5** Bi-variate correlations between paternal, maternal distress and child peer relations

	Age 3 PD	Age 5 PD	Age 7 PD	Age 11 PD	Age 14 PD	Age 3 MD	Age 5 MD	Age 7 MD	Age 11 MD	Age 14 MD	Age 3 Peer	Age 5 Peer	Age 7 Peer	Age 11 Peer	Age 14 Peer
Age 3 Peer	.09	.07	.07	.07	.08	.19	.16	.16	.17	.15	1				
Age 5 Peer	.08	.09	.09	.08	.10	.19	.22	.18	.20	.18	.40	1			
Age 7 Peer	.09	.09	.12	.11	.11	.20	.22	.25	.24	.19	.34	.53	1		
Age 11 Peer	.09	.09	.09	.14	.12	.20	.19	.20	.26	.22	.27	.39	.50	1	
Age 14 Peer	.08	.10	.11	.13	.14	.18	.18	.18	.34	.23	.25	.35	.43	.57	1

All correlation are significant at  $p < 0.001$  level. Peer=Child peer relation problems, PD=Paternal distress, MD=Maternal distress

**Table A6** Descriptives of mediator variables

	N	Mean (SD)
Paternal Closeness (child age 3)	4357	32.60 (2.51)
Paternal Conflict (child age 3)	4419	17.98 (5.45)
Paternal Marital Satisfaction (child age 3)	4373	6.98 (2.45)
Paternal Marital Satisfaction (child age 5)	4690	7.24 (2.59)
Paternal Involvement (child age 5)	4985	21.71 (5.23)

\* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$

**Table A7** Bi-variate correlations between paternal closeness, conflict, marital satisfaction (sweep 2), paternal distress and child difficulties (child ages 3 and 5).

	Paternal Closeness (age 3)	Paternal Conflict (age 3)	Paternal Marital Satisfaction (age 3)
Paternal Distress (age 3)	-.14***	.36***	.36***
Emotional Symptoms (age 3)	-.09***	.11***	.08***
Conduct Problems (age 3)	-.10***	.30***	.12***
Hyperactivity (age 3)	-.10***	.19***	.10***
Peer Difficulties (age 3)	-.11***	.10***	.07***
Paternal Distress (age 5)	-.05**	.25***	.24***
Emotional Symptoms (age 5)	-.09***	.10***	.08***
Conduct Problems (age 5)	-.12***	.20***	.10***

Hyperactivity (age 5)	-.11***	.17***	.13***
Peer Difficulties (age 5)	-.13***	.09***	.07***

**Table A8** Correlations between the main study variables (research aim 2)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Age 3 PD	1	0.56***	0.51***	0.44***	0.16***	0.12***	0.11***	0.13***	-0.04***	0.02	0.04***	0.004	-0.006	-0.005	-0.008	0.01
2. Age 5 PD		1	0.57***	0.49***	0.15***	0.19***	0.15***	0.13***	-0.02*	0.02	0.04**	-0.01	0.0001	-0.0002	-0.03*	0.008
3. Age 7 PD			1	0.55***	0.14***	0.16***	0.19***	0.15***	-0.04**	0.03*	0.05***	0.009	-0.004	-0.006	-0.02	0.02*
4. Age 11 PD				1	0.13***	0.14***	0.14***	0.21***	-0.07***	0.04***	0.08***	0.005	0.006	-0.02	-0.03**	0.01
5. Age 3 MD					1	0.55***	0.54***	0.48***	-0.07***	0.04***	0.06***	0.02*	0.03**	-0.03**	-0.04***	0.03**
6. Age 5 MD						1	0.59***	0.52***	-0.09***	0.05***	0.07***	0.008	0.03*	-0.05***	-0.06***	0.03**
7. Age 7 MD							1	0.56***	-0.09***	0.04***	0.05***	0.006	0.03**	-0.05***	-0.05***	0.04***
8. Age 11 MD								1	-0.13***	0.06***	0.09***	0.01	0.04***	-0.05***	-0.06***	0.04***
9. Verbal Ability									1	-0.15***	-0.18***	-0.05***	0.002	0.11***	0.14***	-0.06***
10. Strategy										1	0.66***	0.06***	0.03**	-0.14***	-0.16***	0.05***
11. Total Errors											1	0.08***	0.04***	-0.17***	-0.20***	0.10***
12. Delay Aversion												1	-0.15***	-0.06***	-0.16***	0.20***
13. Deliberation Time													1	-0.21***	-0.05***	-0.08***
14. QoDM														1	0.28***	0.09***
15. Risk Adjustment															1	-0.19***
16. Risk-Taking																1

\* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ , PD=Paternal distress, MD=Maternal distress, QoDM=Quality of Decision-Making



## Appendix 2

### Smoking

Table A9 shows the results linking paternal and maternal distress trajectories to adolescent smoking for the full sample, boys, and girls, while controlling for confounders. ‘Moderate-increasing’ paternal distress predicted a higher smoking risk, for the full sample and boys. The same was the case for maternal ‘severe distress’, for the full sample and girls.

**Table A9** Logistic regression results for smoking (adjusted models)

	Full Sample		Girls		Boys	
	N (SE)	95% CIs	N (SE)	95% CIs	N (SE)	95% CIs
‘Moderate-increasing’ paternal distress	0.21 (0.08)**	[0.009, 0.06]	0.13 (0.11)	[-0.08, 0.35]	0.28 (0.10)**	[0.07, 0.48]
‘Low’ maternal distress	0.14 (0.07)	[-0.002, 0.04]	0.15 (0.11)	[-0.06, 0.36]	0.14 (0.10)	[-0.06, 0.34]
‘Moderate-increasing’ maternal distress	0.18 (0.11)	[-0.005, 0.06]	0.13 (0.15)	[-0.17, 0.44]	0.23 (0.15)	[-0.06, 0.52]
‘Severe’ maternal distress	0.48 (0.17)**	[0.03, 0.14]	0.58 (0.24)*	[0.11, 1.04]	0.36 (0.25)	[-0.13, 0.86]

\* $p < 0.05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals

### Alcohol drinking

The findings regarding the associations between parental trajectories and alcohol drinking are displayed on table A10. Fathers’ ‘moderate-increasing’ distress predicted a higher likelihood for alcohol use in boys only. For maternal trajectories, no paths emerged.

### Binge drinking

Table A11 presents the results for binge drinking. Neither paternal nor maternal effects were identified.

**Table A10** Logistic regression results for alcohol drinking (adjusted models)

	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
'Moderate-increasing' paternal distress	0.11 (0.07)	[-0.02, 0.24]	-0.02 (0.10)	[-0.21, 0.18]	0.24 (0.10)*	[0.05, 0.43]
'Low' maternal distress	0.04 (0.06)	[-0.07, 0.16]	-0.002 (0.08)	[-0.17, 0.16]	0.10 (0.08)	[-0.07, 0.26]
'Moderate-increasing' maternal distress	0.13 (0.09)	[-0.05, 0.31]	0.10 (0.13)	[-0.16, 0.36]	0.16 (0.13)	[-0.10, 0.42]
'Severe' maternal distress	0.02 (0.17)	[-0.31, 0.35]	-0.10 (0.23)	[-0.56, 0.35]	0.14 (0.25)	[-0.35, 0.63]

\* $p < 0.05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals

**Table A11** Logistic regression results for binge drinking (adjusted models)

	Full Sample		Girls		Boys	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
'Moderate-increasing' paternal distress	-0.12 (0.12)	[-0.35, 0.11]	-0.13 (0.16)	[-0.45, 0.19]	-0.08 (0.17)	[-0.42, 0.25]
'Low' maternal distress	0.06 (0.10)	[-0.13, 0.26]	0.23 (0.15)	[-0.05, 0.51]	-0.08 (0.14)	[-0.35, 0.19]
'Moderate-increasing' maternal distress	0.01 (0.15)	[-0.29, 0.32]	0.13 (0.22)	[-0.30, 0.56]	-0.10 (0.22)	[-0.53, 0.32]
'Severe' maternal distress	0.25 (0.26)	[-0.26, 0.75]	0.19 (0.36)	[-0.52, 0.89]	0.33 (0.37)	[-0.41, 1.06]

\* $p < 0.05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals

## Sexual activity

The outcomes for sexual activity are shown on table influence. A12. Again, both fathers' and mothers' distress trajectories did not have a significant impact.

**Table A12** Logistic regression results for sexual activity (adjusted models)

	Full Sample		Girls		Boys	
	B (SE)	95% CIs	B (SE)	95% CIs	B (SE)	95% CIs
'Moderate-increasing' paternal distress	0.20 (0.18)	[-0.15, 0.54]	-0.003 (0.26)	[-0.52, 0.51]	0.37 (0.24)	[-0.10, 0.84]
'Low' maternal distress	0.16 (0.18)	[-0.19, 0.51]	0.28 (0.26)	[-0.24, 0.78]	0.10 (0.25)	[-0.39, 0.59]
'Moderate-increasing' maternal distress	0.25 (0.25)	[-0.24, 0.74]	0.23 (0.37)	[-0.49, 0.94]	0.33 (0.34)	[-0.34, 1.00]
'Severe' maternal distress	0.54 (0.37)	[-0.20, 1.27]	0.80 (0.50)	[-0.18, 1.78]	0.28 (0.60)	[-0.89, 1.44]

\* $p < 0.05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , CIs=Confidence intervals