The University Of Sheffield.

Identifying Environmental Risk Factors for Childhood and Adolescence Internalising Disorders

By:

Zeliha Ezgi Saribaz

A thesis submitted in partial fulfilment of the requirements for the degree of

Doctor of Philosophy

The University of Sheffield

Faculty of Science

Department of Psychology

December 2023

Acknowledgments

During the process of completing this thesis, I have been fortunate to receive significant encouragement and support from my supervisors, family, and friends. Firstly, I would like to express my sincere gratitude to my principal supervisor, Prof. Richard Rowe, whose valuable experience, feedback, and suggestions illuminated my path whenever I needed guidance during the complex and uncertain journey of my PhD. Also, I would like to express my gratitude to Prof. Paul Norman for adding valuable methodological perspectives to my work and for his attentive guidance throughout this process. I would also like to express my sincere gratitude to Dr. Agata Debowska. I am grateful for her always sincere and genuine approach, and for generously sharing her experiences with warmth, guiding me along the way.

I would also like to express my gratitude to my beautiful friends. To Secil, the beautiful person who witnessed every moment of my doctoral journey with its pains and joys, served as my confidante, and with whom I had the most fun; to Deniz, who never hesitated to share her warmth; and endless thanks to Mine, who is always by my side. A special thank you must also go out to my wonderful sister Gizem, who has been witness to my childhood, youth, and will hopefully be witness to my old age, and whose companionship I love very much.

A final thank you goes to my family. A very special thanks must be given to my mother, a compassionate woman whose vision opened new horizons, to my father, who is by my side at all times, and to my sister, Seline, who serves as a mirror to me.

In addition to all, I would also like to acknowledge the Ministry of National Education of Republic of Türkiye for funding my PhD.

Declaration

I, Zeliha Ezgi Saribaz, confirm that the Thesis is my own work. I am aware of the University's Guidance on the Use of Unfair Means (www.sheffield.ac.uk/ssid/unfair-means). This work has not previously been presented for an award at this, or any other, university.

Presentations arising from this thesis

Saribaz., Z.E., Speyer, L.G., Norman, P., Debowska., A., Rowe, P. (2023). *Within-person cross-lagged relationships between children and parents' mental health: Does poverty play a moderating role?*. Presented at Children of the noughties: a conference to celebrate 21 years of the Millennium Cohort Study, London, UK.

Saribaz., Z.E., Hales, G. K., Debowska., A., Rowe, P. (2022). *Which environmental risks make children anxious and depressed? A systematic review of causally informative studies* [Conference poster]. Presented at British Society for the Psychology of Individual Differences, online.

Abstract

The literature reveals a number of environmental risk factors that may contribute to the development of child internalising problems. However, the findings of these studies are usually based on cross-sectional designs, which presents methodological challenges and may not provide adequate grounds for drawing causal conclusions. The identification of causal risk factors is of great importance to the success of intervention and prevention programs. Therefore, it is necessary to investigate the environmental risk factors using sophisticated methods. The purpose of this thesis is to examine environmental risk factors associated with internalising problems in children using the best available methods that will contribute to establishing causal inference.

A systematic review was conducted for the initial study (Chapter 2), with a focus on studies using causally informative study designs to examine environmental risk factors associated with child internalising problems. The study identified several environmental risk factors for children and adolescents' internalising problems, with parental mental health problems emerging as a prominent factor. In the second study (Chapter 3), the relationship between parental mental health (distress) and child internalising problems was explored, using the random intercept cross-lagged panel model, a sophisticated methods that contributes to causal inference. These relationships were examined on the basis of the Family Stress Model, which suggests that low family income affects parent distress, which, in turn, affects child outcomes. The results showed that parental distress did not mediate the relationship between family income and child mental health problems, emphasizing the necessity for additional exploration of this relationship from different perspectives. Thus, the last study (Chapter 4) explored the relationship between parent and child mental health, examining the moderating effect of poverty. Utilizing another causally informative study design, autoregressive latent

trajectory models with structured residuals, results revealed that no differences between poverty groups at either the between or within-family levels.

These studies shed light on how environmental risk factors, particularly parental distress, are associated with internalising problems in children/adolescents from a causal perspective. It was highlighted the need to further research into environmental risk factors using sophisticated methods in order to better understand their impact on child internalising problems.

Table of Contents

List of Figures

List of Tables

Chapter 1: General Introduction

1.1 Childhood and Adolescence Internalising Symptoms and Disorders

In recent decades, there has been an increased recognition of the importance of studying the psychopathology of children, while psychiatry traditionally focuses on extensive studies related to adults (Claman, 1983; Drabick et al., 2009). The significance of the matter is underscored by the undeniable reality that the prevalence of mental health problems among children should not be underestimated. According to the World Health Organization (WHO) report, one in seven ten to nineteen-year-olds suffers from mental health problems, (WHO, 2021). Therefore, it is important to invest effort to gain greater understanding of mental health problems in young people.

Mental health problems in children and adolescents can be divided into two dimensions; namely, internalising and externalising. Internalising disorders occur when individuals try to control or regulate their internal emotions or cognitions in a maladaptive way, such as being in overcontrol, while externalising including being unbounded, try to control the external environment in a maladaptive way (Anderson, 2002).

Internalising disorders include mood disorders, anxiety disorders, obsessive-compulsive and related disorders, trauma- and stress-related disorders, and feeding and eating disorders (Goldstein & DeVries, 2017). Prevalence rates for internalising disorders are similar for boys and girls at younger ages (7-9%). In adolescents, females have a greater risk of developing internalising disorders (15.7%); four times that of males (3.9%) (Offord et al., 1987). Among them, anxiety disorders and depressive disorders are the most common, with the rate increasing with age (WHO, 2021; In-albon, 2012).

There are several types of anxiety disorders, including separation anxiety disorder, selective mutism, specific phobias, social anxiety disorder (SAD), panic disorder, panic attack

specificity, agoraphobia, and generalized anxiety disorder (GAD) (American Psychiatric Association [APA], 2022). Despite their differences, these disorders share a number of characteristics (Anderson, 2002). These shared characteristics include subjective feelings like dread or fear, avoidance of situations causing anxiety, and physiological responses such as sweating. Additionally, other symptoms include unrealistic thinking, misinterpretation of symptoms, heightened physiological arousal, fears related to specific occasions, and excessive worrying (APA, 2022).

Regarding depressive disorders, they include disruptive mood dysregulation disorder, major depressive disorder, persistent depressive disorder (dysthymia), and premenstrual dysphoric disorder (APA, 2022). The most common form is major depressive disorder (Wilkinson, 2009). Potential symptoms of major depression are depressed mood (e.g., feeling sad, hopeless); however, children, instead of feeling depressed mood, can also experience depression as feeling irritable, losing weight although not on a diet, gaining weight, decreasing or increasing appetite, sleep disturbances (sleeping for a long time or difficulty sleeping), psychomotor agitation or retardation almost daily, feelings of worthlessness, being unable to concentrate, and having recurrent thoughts about death or suicide (APA, 2022).

Clinical decisions such as treatment require diagnostic categorization. However, when diagnosing psychological disorders, relying on clear-cut thresholds based on a categorical approach, where disorders are either present or absent, can lead to oversimplification. It is possible that this oversimplified approach may hinder an understanding of the unique psychopathology of each child (Hudziak et al., 2007). Instead of focusing exclusively on a diagnosis, it is equally important to pay attention to symptomatic variations below the diagnostic cut-off threshold. This is because numerous studies have demonstrated that symptoms associated with anxiety and depressive disorders play a crucial role in impairment,

prognosis, mortality, and treatment (e.g., Angold et al., 1999; Angst et al., 2000; Broadhead et al., 1990). For example, Ford et al. (2003) found that around one-fifth of children diagnosed as "not otherwise specified" for anxiety, depression, and disruptive disorders exhibited significant impairment, even though they did not meet the DSM-IV criteria. Pickles et al. (2001), revealed that out of 476 children with subthreshold depressive symptoms, 245 (51%) displayed severe impairment scores or more. Therefore, it can be concluded that due to the continuum characteristic of psychopathology, it is crucial to study symptomatology across the entire spectrum, from non-pathological functioning to psychiatric disorders (Pickles & Angold, 2003).

As for the burden effects of internalising disorders diagnosed in childhood, research indicates that these disorders are associated with impairments in school life, peer relationships, and family functioning (Jamnik & DiLalla, 2019). In addition, depression and anxiety frequently co-occur (Essau & de la Torre-Luque, 2023; Garber & Weersing, 2010). This co-occurrence exacerbates the effects, leading to various adverse outcomes, including an increased likelihood of suicidal thoughts (Cummings et al., 2014). Left untreated, these symptoms may be indicative of mental disorders and risky behaviours in young adults, including depression, anxiety disorders, and substance abuse (Copeland et al., 2009; Liu et al, 2011). Nevertheless, initiating treatment early in life can be more cost-effective and less distressing for the individual than seeking treatments later (Seabury et al., 2019). Thus, to reduce the burden of its effects, it is imperative to strive for prevention. Understanding risk factors is one of the most important aspects of prevention studies. When these factors are recognized and addressed, it is possible to focus on reducing the risk factors and strengthening the protective factors, thus aiding the prevention of mental health problems (Gladstone et al., 2011).

1.2 Risk Factors for Child and Adolescence Internalising Problems

Internalising problems have a hereditary component (Hettema et al., 2001). A variety of studies demonstrate that anxiety disorders and depression tend to run in families, and people with first-degree relatives who have these disorders are more likely to experience them themselves (Rice et al., 2002). Polderman et al. (2015)'s meta-analysis, combined data from 2,748 twin studies conducted from 1958 to 2012, estimated heritability as 34% for depression and 40% for anxiety disorders.

Furthermore, certain studies have indicated the presence of specific genetic variants associated with the development of depression (Delli Colli et al., 2022) and anxiety (Su et al., 2021; Morris-Rosendahl, 2002). A systematic review, for example, which focused on genetic factors associated with depression, reported an association between the 5HTTLPR gene (single nucleotide polymorphism) and adolescent depression (Xia & Yao, 2015). In regard to anxiety disorders, Maron et al. (2010) reported an association between some chromosome regions, including 13q and 14q, and the transmission of panic disorder.

In contrast, although family resemblance was demonstrated to be a product of genetic factors, quantitative genetic studies indicate that not all variance in internalising problems can be explained by genetic factors (Beam et al., 2022). This highlights the importance of environmental influences, which may be categorized as either shared or non-shared (Plomin, 2011).

The shared environment encompasses environmental factors shared among family members, leading to similarities among them (Knopik et al., 2017; Plomin et al., 2001; Stoolmiller, 1999). Various environmental factors, including parenting, parent education, and neighbourhood, become components of the shared environment when they contribute to family resemblance (Knopik et al., 2017). Polderman et al. (2015)'s meta-analysis indicated

that the effect of shared environment influences on the likelihood of disorder was lower than heritability, estimated as 11% for depression and 17% for anxiety.

The non-shared environment, representing environmental components that contribute to individual differences among family members, may significantly contribute to an individual's predisposition to mental health disorders (Beam et al., 2022). In their report, Polderman et al. (2015) estimated that non-shared environments were associated with 43% and 55% of the variances for depression and anxiety, respectively.

On the other hand, in many forms of psychopathology, there is likely an interplay between genetic and environmental factors, called gene-environment interaction (GxE) (Jaffee & Price, 2012; Schiele & Domschke, 2018). A study of serotonergic gene polymorphisms (5-HTTLPR and HTR1A rs6295) found that on their own, these polymorphisms did not affect panic disorder, but their interaction with stressful life events such as parental loss increased the likelihood of panic disorder (Choe et al., 2013). Furthermore, the interaction between Neuropeptide Y and high stressor exposure, hurricane, was shown to increase the likelihood of GAD. Conversely, it was observed that high social support acted as a buffering effect on the risk of SAD associated with 5-HTTLPR (Reinelt et al., 2014). Overall, this discussion shows that environmental influences, in combination with genetic effects, perform a key role in determining variation in internalising symptomatology.

1.2.1 Environmental Correlates

1.2.1.1 Social Disadvantage

1.2.1.1.1 Family Income

Approximately 20 percent of children live below the poverty line, experiencing various challenges associated with poverty (Gershoff et al., 2007), such as inadequate food and

material deprivation which is lack of essential goods and resources necessary to maintain a decent standard of living (Gordon & Spicker, 1999).

The poverty line is the threshold at which a family's income is insufficient to meet its basic living expenses. Specifically, poverty is defined as having a net income less than 60% of the median income (Schenck-Fontaine and Panico, 2019). Numerous studies have demonstrated a positive association between poverty and child and adolescent internalizing problems, with comparable positive associations also observed in families with low income, even when not classified as poverty (e.g., McLeod & Nonnemaker, 2000; McLeod & Owens, 2004; Miller et al., 2020; Yang et al., 2023). For instance, Yang et al. (2023) explored the association between family income and child mental health problems in children aged 3 to 14, utilizing the UK-based Millennium Cohort Study. The study found that children from low-income families exhibited more internalising problems compared to their peers.

1.2.1.1.2 Neighbourhood Poverty

Neighbourhood poverty has also been studied in association with child internalising problems (Leventhal & Brooks-Gunn, 2011, Leventhal & Dupéré, 2019). For instance, the National Longitudinal Survey of Adolescent Health was employed to explore the association of community and family factors with depressive symptoms in adolescents (Wickrama & Bryant, 2003). Results indicated that neighbourhood poverty was associated with an increase in youth depression even after controlling for family and youth's characteristics. Similarly, Flouri et al. (2020) used the Millennium Cohort Study (MCS) to examine children aged 3 to 14, reporting a positive association between living in deprived neighborhoods and child emotional problems.

1.2.1.2 Parental Mental Health Problems

1.2.1.2.1 Parental Anxiety

Numerous studies have examined the impact of parental anxiety on child anxiety and depression (e.g., Beidel & Turner 1997; Biederman et al. 2006). A consensus across several studies suggests an elevated risk of anxiety disorders in children when their parents have experienced anxiety problems (Chapman et al., 2022). For instance, Burstein et al. (2010) focused on children aged 6-14 with parents both diagnosed and not diagnosed with anxiety disorders. Their findings indicated an association between parental anxiety and both child depression and anxiety. Furthermore, a systematic review of eighteen studies showed that the association between parent anxiety and child emotional problems was weak during infancy but became stronger during childhood for both maternal and paternal anxiety. This relationship was particularly pronounced for mothers during adolescence (Sweeney & Wilson, 2023).

1.2.1.2.2 Parental Depression

Several studies demonstrated that parental depression is another risk factor for internalising problems in children and adolescents (e.g., Clayborne et al., 2021; Lovejoy et al., 2000; Speyer et al., 2022). A prospective longitudinal study with four years of follow-up data revealed a significant association between major depression in mothers and psychopathology in their offspring, particularly depression (Lieb et al., 2002). In another study trajectories of maternal depressive symptoms were modelled from mid-pregnancy to three years postpartum in a large population-based cohort study. Symptoms were classified into four phases, ranging from no symptoms to high symptoms. It was reported that children with mothers having more severe depressive symptoms have shown more internalising and behaviour problems at the age of 3 years, than children with mothers with no symptoms trajectories (Cents et al., 2013). Additionally, a study using the Avon Longitudinal Study of Parents and Children (ALSPAC)

dataset examined children aged 10-13 years and reported that postnatal maternal depression was positively correlated with child anxiety at age 10 (Morales-Munoz et al., 2023).

1.2.1.3 Parental Substance Use

Substance use encompasses both illegal substances, such as cocaine, and the misuse of legal substances, including alcohol and smoking (Sen, 2007; Sithisarn et al., 2012), as well as the misuse of prescribed medications, such as benzodiazepines, which are prescribed for patients with psychiatric symptoms (Smith et al., 2022).

It has been reported that one in five children experiences exposure to the misuse of drugs or alcohol while growing up (Kulig et al., 2005). According to the Office of the Children's Commissioner data report (2020), 478,000 children (40 children per 1,000 parents) in England were exposed to parental alcohol or drug misuse between 2019 and 2020. Furthermore, this exposure may commence during the foetal development of children. Studies have demonstrated that both prenatal and postnatal exposure to substance use heightens the risk of mental health problems such as depression and anxiety in children (e.g., Ashford et al., 2008; Fergusson et al., 1998; Raitasalo et al., 2019). One reason for the elevated risk of mental health issues in children could be that parents with substance misuse may fail to create a safe environment and adequately address the physical and emotional needs of their children, making children more susceptible to mental health challenges (Orford et al., 2005).

In an eight-year longitudinal study focusing on the influence of alcohol consumption during pregnancy on child emotional problems, a positive association was found between prenatal heavy alcohol exposure in the first trimester and child emotional problems (O'Leary et al., 2009). Additionally, a meta-analysis involving 17 studies and examining 47,374 children

aged 9 to 17 years reported that parental substance use (alcohol or drugs) was associated with child internalising problems (Mcgovern et al., 2023).

1.2.1.4 Stressful life events

A stressful life event (SLE) is characterized as an occurrence that disrupts the normal course of a person's life (Rubens et al., 2013). Such events can lead to psychological stress, hindering an individual's ability to effectively cope with the associated circumstances (Lazarus & Folkman, 1984). A variety of studies reported that SLEs have been linked with child emotional problems (e.g., Furniss et al., 2009; McMahon et al., 2003; Sandberg et al., 2001). Examples of SLEs may encompass parental divorce, parental loss, or childhood maltreatment (Rubens et al., 2013).

1.2.1.4.1 Parental Divorce

Parental divorce stands as one of the second most common stressor for children (Moor et al., 2014). Approximately 30-40% of children undergo this stressful life event before reaching the age of 15 (Kennedy & Bumpass, 2008). Furthermore, the impact of divorce on children extends from the period preceding the parental divorce to the aftermath, forming it into a continuous process (Sun & Li, 2002). For example, Sun and Li (2002) investigated the influence of parental divorce on child emotional problems, conducted assessments on children three years before and three years after the divorce. The findings indicated that children who went through parental divorce demonstrated a higher prevalence of mental health problems compared to children without parental divorce, both before and after the divorce. Effect of divorce on children could be attributed to parents facing disruptions in their relationship, potentially hindering their focus on their children's needs and leading to neglect of their responsibilities. Consequently, children may feel lonely, rendering them more vulnerable to emotional problems (Spremo, 2020).

1.2.1.4.2 Parental Loss

Five percent of children in the UK encounter parental loss before the age of 16 (Parsons et al., 2011). Following the death of a parent, children often undergo a shift in family dynamics and may find themselves living with the surviving parent or other relatives, such as grandparents (Bergman et al., 2017). Furthermore, the surviving parent may grapple with additional stressors related to being single parenthood or the sole responsibility of supporting the children, making it challenging to provide adequate support. These changes can potentially render children more susceptible to mental health issues (Kwok et al., 2005).

Research suggests that children are at a significant risk of developing depression in the first year following the death of a parent (Cerel et al., 2006; Harrison & Harrington, 2001; Mack, 2001). For example, in a longitudinal study involving 360 children aged between 6-17 who experienced parental bereavement and their surviving parent, interviews were conducted over a period of 2 years. The findings indicated that the bereavement of a parent within two years was associated with child depression. However, it was observed that this relationship was mitigated by having a less depressed surviving parent and a higher socioeconomic status (SES) (Cerel et al., 2006). Similarly, Denckla et al. (2023) examined youth from the Avon dataset who experienced various types of bereavement, including the loss of a parent, and reported that bereaved youth had 0.19 points higher levels of emotional and behavioural symptoms compared to their non-bereaved peers.

1.2.1.4.3 Maltreatment

Child maltreatment involves the exposure of children under the age of 18 to maladaptive behaviours, encompassing abuse (sexual, physical or emotional) and neglect by their caregivers or parents (Sedlak et al., 2010; Van der Kolk, 2005). A child's capacity to navigate stress hinges on the nature of the experienced situation and the caregiver's- typically the parents'- skill in handling and alleviating stress in response to unfolding events (Lyons-Ruth

et al., 1999). Children who have been maltreated often lack consistent access to a supportive caregiver in stressful situations, which may undermine their ability to cope effectively and contribute to the development of mental health problems (Schore, 2001). Various studies have demonstrated that children with a history of maltreatment experience more internalising problems compared to non-maltreated children (Hildyard & Wolfe, 2002; Shonk & Cicchetti, 2001). Based on a meta-analysis of 29 studies examining the effect of child maltreatment on social anxiety problems, it was found that there was a positive relationship between those problems and child maltreatment, with an effect size of r = 0.210. This relationship was further heightened in the context of emotional maltreatment with r = 0.251 (Liu et al., 2023).

1.2.1.5 Parenting Styles

Goldin (1969) classified childrearing behaviours into three main aspects: Loving (acceptance-rejection), Demanding (Psychological Autonomy/Control), and Punishment/Harsh. Acceptance involves offering support, expressing affection, and providing positive evaluations of children. In contrast, rejection, which encompasses hostile reactions, represents the opposite end of the spectrum. The concepts of psychological autonomy and controlling parenting encompass behaviours that either support or hinder the development of a child as an individual, separate from the parent. In addition, punishment/harsh parenting refers to the use of both physical and non-physical punishment arbitrarily (Goldin, 1969). One of the most common risk factors associated with internalising problems is poor parenting styles, such as punishment/harsh treatment or authoritarian/controlling approach (Rose et al., 2018; Xu et al., 2017). Numerous studies demonstrated the association between poor parenting and various child mental health issues, including child depression (e.g., Kingsbury et al., 2020; Wang et al., 2023), and anxiety (e.g., Chong et al., 2020).

For instance, parental rejection and overcontrol were found to be associated with emotional problems in various studies (e.g., Möller et al., 2016; Bayer et al., 2006; Rapee, 1997; van der Bruggen et al., 2008). Exposure to rejection, such as criticism and disapproval, was found related to insecure attachment (Brook & Schmidt, 2008), which, in turn, was associated with childhood depression (McLeod et al., 2007). Additionally, parents exhibiting overcontrolled and overprotective behaviours are known to hinder their children from exploring the world, driven by a fear that something threatening may happen (Aktar et al., 2017). When a child's behaviour is excessively monitored and restrained by parents, it can result in the child feeling incompetent and overly dependent. This shapes their perception of the world as something beyond their control. This, in turn, was found associated with heightened levels of anxiety in children (Bögels & Brechman-Toussaint, 2006; McLeod et al., 2007).

1.2.1.6 Family functioning

Family functioning refers to the functionality of the family system, shaped by multifaceted dynamics within the family, including parent-child relations and marital satisfaction (De Los Reyes, 2013; Skinner et al., 2000). Numerous studies have established an association between family functioning and internalising problems in children and adolescents (e.g., Hammen et al., 2004; Huang et al., 2022). However, it is crucial to acknowledge that the impact of family functioning can be influenced by various mediating or moderating factors. For instance, Hammen et al. (2004) conducted a study involving 15-year-old children with both depression- and non-depressed mothers, exploring how family functioning- specifically the quality of marital and parent-child relationships- affects child depression. The results indicated that children who experienced high levels of family discord and had depressed mothers were at higher risk of depression. This underscores the notion that the impact of family functioning may vary based on specific factors. Additionally, another study by Huang

et al. (2022) highlighted the significant effect of family functioning on adolescent depression, with self-esteem mediating this relationship.

1.2.1.7 Peer Problems

Peer problems commonly including peer victimization or bullying is a significant social stressor that profoundly impacts children and young people, with enduring effects on their mental well-being even into later life (Adedeji et al., 2022; Kelly et al., 2015). In a recent study by Patalay and Gage (2019), findings revealed a significant increase in the rates of children experience peer problems. In 2015, the rates ranged from 9.7% to 17.7%, representing a substantial rise compared to 2005, where the rates were between 5.7% and 8.9%. This indicates a concerning trend of escalating peer victimization among children over the years. Research has consistently demonstrated a significant association between peer victimization and adolescent depression, both in the short term (Kaltiala-Heino et al., 2010; Zwierzynska et al., 2013) and long term (Klomek et al., 2008). In Klomek et al.'s (2008) study, which utilized a longitudinal design, the impact of exposure to peer victimization at age 8 on depression scores at age 18 was investigated. The findings revealed a significant relationship, indicating that children who experienced bullying at age 8 were four times more likely to develop depression compared to their non-bullied peers a decade later. This underscores the enduring and potentially detrimental effects of peer victimization on mental health outcomes into adolescence. It is also important to note that these relationships may occur in reverse, with depression leading to peer victimization, as reported by Kochel et al. (2012). Therefore, reciprocal influences should be taken into consideration.

1.3 Problem with Interpretation

Although there are many informative studies showing risk factors associated with child internalising problems, the findings of these studies may not be sufficient to draw causal inferences, since they mostly rely on cross-sectional designs, which pose some

methodological challenges (Foster, 2010; Rutter, 2007). The restriction of causal inferences makes it hard to develop effective intervention and treatment strategies (Jaffee et al., 2012).

In cross-sectional designs, a significant methodological challenge arises from the potential for confounding in the relationship between risk factors and internalising problems. This indicates that a third variable or a set of variables might influence both the hypothesised risk factors and the development of internalising problems simultaneously (Jaffee et al., 2012; Rutter, 2007). For example, there is a well-established relationship among SES, child internalising problems, and poor parenting (Bradley & Corwyn, 2002; Villarreal & Nelson, 2018). Any apparent association between poor parenting and child internalising problems may be due to SES confounding effects, given that SES could be correlated with both internalising problems and parenting style simultaneously. As a result, this association could lead to a cross-sectional correlation between poor parenting and child internalising problems, without implying a causal effect of parenting on internalising behaviour.

Genetic factors also can be a confounder/third variable between outcome and environmental risk factors, known as passive gene environment correlation (rGE) (Jaffee & Price, 2007). As a result of passive rGE, the same genetic variants that shape how parents interact with and raise their children are passed on to their offspring, influencing their behaviour or abilities (Knopik et al., 2017; Rutter et al., 1997). Even though we may observe a correlation between children's environments and their behaviour, children's behaviour may be more deeply influenced by traits they inherit from their parents than by their immediate environment. For instance, Goodman and Gotlib (1999) revealed that children of depressed mothers inherit their mother's depression-vulnerable DNA, potentially increasing their susceptibility to depression. Furthermore, maternal depression is known to be associated with factors related to child internalising problems, such as heightened hostility towards children (Lovejoy et al.,

2000). As a consequence, the genetics of a mother who carries depression-prone DNA may act as a confounding factor in the relationship between maternal hostility towards her children and the development of child internalising problems. This indicates that factors initially regarded as environmental risks may actually be genetic in nature.

A second challenge in cross-sectional studies is the lack of clarity regarding direction of effect, known as reverse causation. In reverse causation, some presumed effects of environmental risk factors may actually reflect children's influence on their environment (Bell, 1968). As an example, the effect of poor parenting on child anxiety disorders may be explained by the stressful nature of anxious children, which may result in parents rejecting their child (Speyer et al., 2022)

In the field of genetics, reverse causation is referred to specifically as evocative rGE. This concept implies that characteristics of children under genetic control may elicit a response from the environment (Jaffee & Price, 2007; Plomin et al., 1977). For instance, children with depressed mothers may inherit genes associated with increased vulnerability to depression, leading to heightened irritability (Vidal-Ribas & Stringaris, 2021). Consequently, these irritable children, challenging for parents to care for, may contribute to an environment that exacerbates their parent's depression.

The third challenge for cross-sectional studies is social selection. This refers to the process of choosing an environment that matches one's abilities or behaviours. In behavioural genetics terminology, active rGE refers to how individuals consciously choose or shape environments that correspond to their genetic nature (Jaffe et al., 2012; Knafo & Jaffee, 2013). It is established that introversion has a heritable component (Power & Pluess, 2015; Scarr, 1969). Several studies have demonstrated an association between social anxiety and loneliness (Halldorsson & Creswell, 2017; Van Zalk et al., 2011). For instance, Van Zalk et al. (2011)

identified an association between social anxiety and choosing to have fewer friends. Therefore, it could be that children with a predisposition for anxiety may naturally gravitate towards less social activities with peers, leading to increased feelings of loneliness. According to this study, loneliness may not be the cause of anxiety, but rather a consequence of anxiety experienced by children.

1.4 Study Designs More Suited to Causal Inference

As mentioned above, observational studies could be limited by inability to determine whether risk factors are causal, because of methodological challenges (Jaffe et al., 2012). The challenges partly arise from the fact that participants' outcomes and exposures are measured at a single point in time - cross-sectional - (Kearney, 2017; Setia, 2016), which can demonstrate correlation but is insufficient to establish causality. Using this measurement strategy, it becomes difficult to determine the temporal sequence of variables, and identify which variable precedes or follows another (Shadish & Cook, 2002).

The issue is well illustrated in a study testing whether parenting mediated an effect of maternal depression on child depression that was analysed to make a methodological point. Cross-sectional analyses of a longitudinal dataset were supportive of the hypothesised mediation. However, longitudinal analyses of the same data showed no evidence of the hypothesised effects (Maxwell et al., 2011). In short, cross-sectional studies can be informative, for example in identifying candidate factors that might be causal, but are not sufficient to make causal inferences on their own (Lahey et al., 2009). Thus, investigating of the environmental risk factors using more sophisticated methods is important.

To address the limitations of cross-sectional studies in establishing causality, it is crucial to consider study designs that offer more robust causal inferences. Accordingly, the following sections will discuss designs that are considered more rigorous than cross-sectional

approaches in establishing causality. These include experimental and quasi-experimental designs, such as fixed effects (FE) methods. These methodologies will be explored in detail, highlighting their strengths in providing more reliable evidence for causal relationships in observational research.

1.4.1 Cross-lagged Panel Modelling

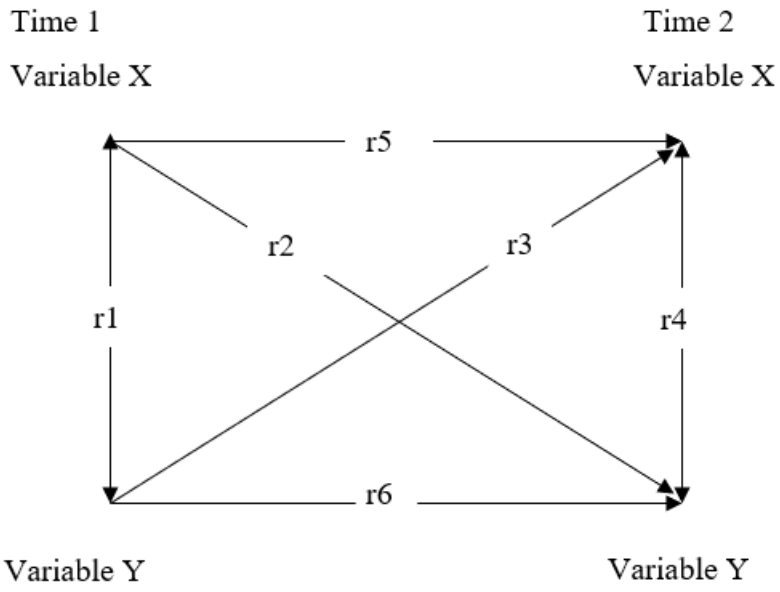
Longitudinal designs, in comparison with cross-sectionals, allow a researcher to examine data across time, which give a chance to observe temporal precedence between variables. As it may take time for causes to show their effects, longitudinal data is a better way to generate evidence relevant to causality than cross-sectional data (Maxwell et al., 2011).

To analyse longitudinal data, one of the most used longitudinal models is cross-lagged panel model (CLPM) often fitted through structural equation modelling (SEM) (Jöreskog, 1970). While still not providing definitive causal evidence this design provides much more useful evidence relevant to testing causal hypotheses compared to cross-sectional designs (Selig & Preacher, 2009) (see Figure 1.1).

The most basic CLPM includes two variables, X and Y, measured at two time points (X1, X2; Y1, Y2) in time. In this model, initial levels of risk factor and outcome are measured simultaneously, which gives the opportunity to understand change in outcome between time1 and time2. In addition, the model includes six possible intercorrelations that can be computed. The synchronous correlations, r1 and r4, refer to the correlation between X and Y, which can be seen in a typical static correlational study. Directional hypotheses may be tested by comparison of the cross-lagged correlations, r2 and r3 using the following reasoning: When X causes Y, a stronger relation should be found between X1 and Y2 (r2) and a weaker relation between Y1 and X2 (r3). On the contrary, if Y causes X, the relation between Y1 and X2 (r3) should be stronger, while the correlation of X1 and Y2 (r2) should be weaker (Lawler

& Suttle, 1973). In addition, both r2 and r3 could be strong; that may imply a reciprocal relationship between the variables; that X1 causes Y2 and that Y1 causes X2. A real-world example might be that overprotective parenting increases the chances of child anxiety and that child anxiety might lead to more overprotective parenting (Novick et al., 2023).

Figure 1. 1 *Schematic illustration of Cross-lagged Panel Model*



r1 &r4: Synchronous correlations

r2 &r3: Cross-lagged correlation

r5 &r6: Autocorrelations

Although CLPM is commonly used for causal inferences in longitudinal studies, it has limitations in adequately addressing stable-trait-level confounds. In CLPM, individuals fluctuate around a common group mean in each variable over time, suggesting no stable between-person differences in these variables (Mund & Nestler, 2019). This overlooks the possibility that the average level of a variable across time might differ between individuals. Thus, if stable between-person differences exist, CLPM includes them in estimated autoregressive and cross-lagged paths (Hamaker et al., 2015), complicating the understanding of between and within-person differences. This limitation is significant, especially for

developmental theory focusing on within-individual change in child mental health problems, making it challenging to gain clear insights into developmental relations (Berry & Willoughby, 2017; Hamaker et al., 2015).

1.4.2 Random Intercept Cross Lagged Panel Model

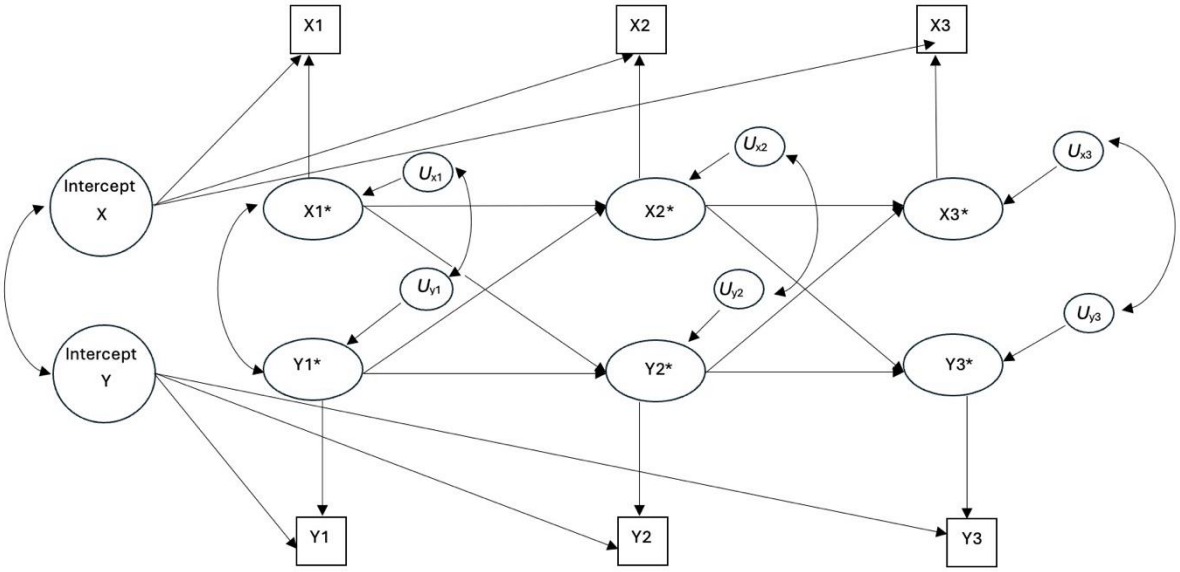
The Random Intercept Cross-Lagged Panel Model (RI-CLPM), introduced by Hamaker (2015) serves as an extension of the CLPM to address its limitations. Compare to CLPM assuming that individuals fluctuate around a common group mean in each variable over time, the RI-CLPM takes into account that each individual fluctuates around their own stable, trait-like level over time (Usami & Hamaker, 2019). Consequently, unlike the CLPM, the RI-CLPM enables the estimation of pure within-person autoregressive and cross-lagged effects.

This is achieved by incorporating random intercepts into the model, allowing for covariance between them and testing the association of the between-participants variance in the measured variables (Lüdtke & Robitzsch 2021; Mund & Nestler, 2019). Additionally, to understand within-individual dynamics, cross-lagged and autoregressive residual effects are included in the model, along with residual covariance within time points (Usami & Hamaker, 2019). Thus, RI-CLPM provide more robust causal insight compared to traditional CLPM (see Figure 1.2).

While the RI-CLPM offers advantages over the CLPM, it also has its limitations. A key limitation is that between-person differences are represented by random intercepts, which only capture correlations similar to those found in cross-sectional studies. As a result, the RI-CLPM cannot assess prospective between-person effects since it does not account for time-lagged and directional relationships across individuals (Orth et al., 2021). Another significant limitation is that the RI-CLPM requires three or more waves of data to be properly identified, whereas two-wave longitudinal designs are more common (Usami et al., 2019). Despite these

limitations, the RI-CLPM remains one of the most suitable designs for research that aims to examine between-person and within-person variance separately.

Figure 1.2 *Schematic illustration of Random Intercept Cross-lagged Panel Model*



1.4.3 Autoregressive Latent Trajectory Modelling with Structured Residuals

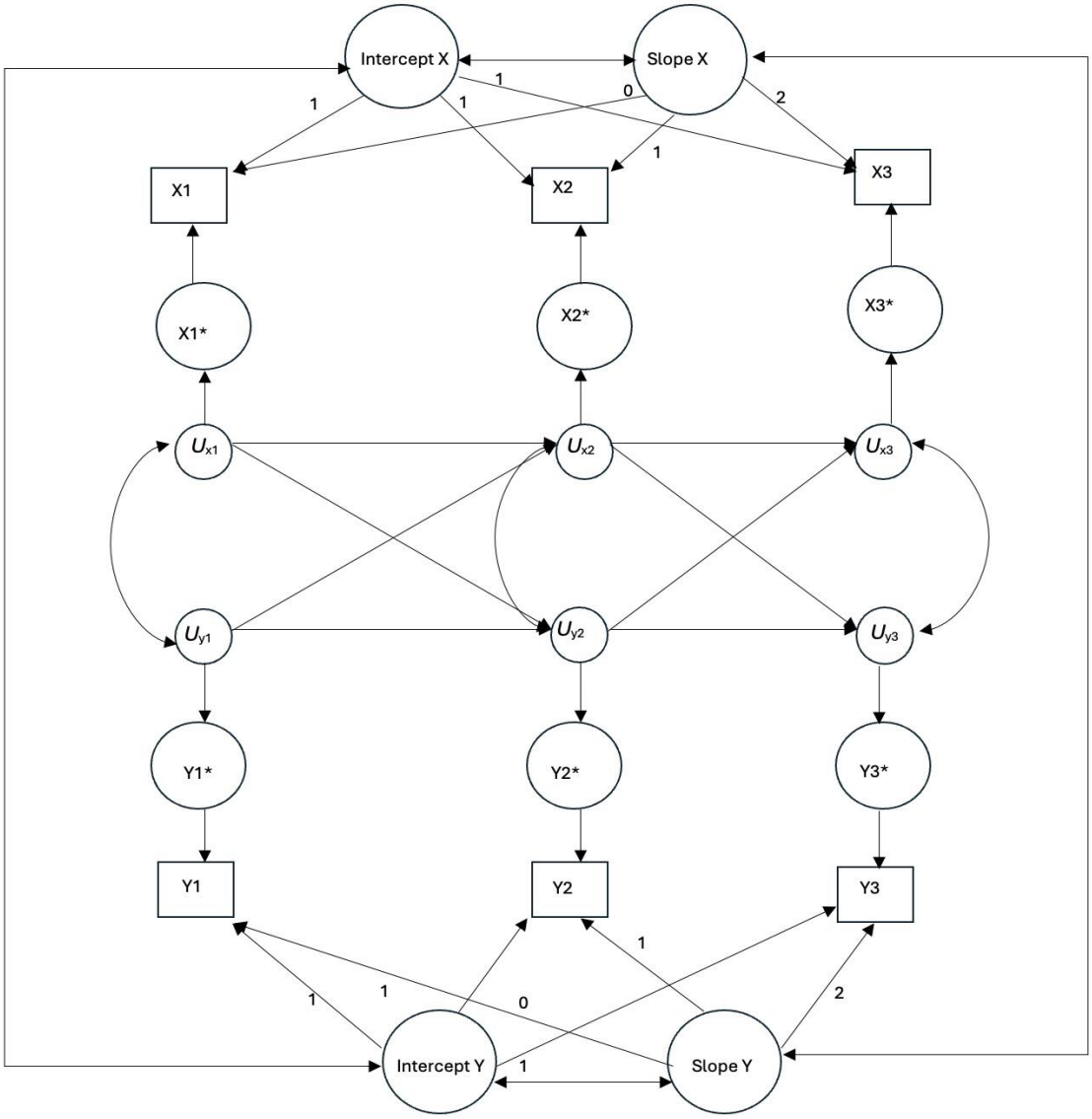
The Autoregressive Latent Trajectory Model with Structured Residuals (ALT-SR), introduced by Curran et al (2014), is another study design that addresses the limitations of CLPM. ALT-SR integrates both the Latent Growth Model (LGM) and the CLPM. The CLPM component focuses on estimating how multiple variables influence each other across sequential time points, taking into account preceding values (autoregression) for each unit. On the other hand, the LGM part aims to understand the change in developmental trajectories over time (Webb, 2021).

In the LGM part, intercepts represent initial levels in symptoms, offering insights into between-person differences at the first measurement occasion. For example, the variance of intercepts can illustrate that some individuals may experience higher levels of depression than others at the initial time point (Mund & Nestler, 2019). In the LGM part, slopes represent

changes in symptoms over time, illustrating an average development trajectory. The mean and variance of slopes provide information on how depression symptoms, for instance, change on average over time and the between-person differences in these developmental trajectories. For instance, some individuals may experience a more pronounced increase in depression symptoms over development than others (Mund & Nestler, 2019).

Concerning the CLPM part, it captures autoregressive and cross-lagged relationships between variables' residuals after estimating the LGM. This separation enables a nuanced understanding of within-individual differences. By differentiating between within-individual and between-individual differences, this approach offers insights into how variables influence each other across individuals and within the same individual over time. Thus, it provides more robust causal insights compared to traditional CLPM (Mund & Nestler, 2019) (see Figure 1.3).

Figure 1. 3 Schematic illustration of Autoregressive Latent Trajectory Modelling with Structured Residuals



1.4.4 Experimental Designs and Randomised Controlled Trials

It is well established that the best method for causal inference is experimentation (Shadish & Cook, 2002). Experiments can explore the effect of manipulated variables and definitively test causal hypotheses regarding effects on outcomes. Randomized control trials (RCT), in which participants are randomly allocated to treatment and control conditions, are seen as the "gold standard" in the assessment of causal effect (Jaffee et al., 2012; Thapar & Rutter, 2019). The reasoning behind this being the strongest method comes from the use of random

allocation of participants, providing equality between treatment and control groups, which helps make unbiased measurements of the intervention effect independently from measured and unmeasured confounders (Rutter & Thapar, 2016).

Experimental designs, including RCTs, however, have several limitations (Jaffee et al., 2012). The biggest challenge faced in RCTs is that, although they are strong in internal validity, they may have weaker external validity. This is because they are designed for just a specific population of interest (Jaffee et al., 2012; Thapar & Rutter, 2019) and are, therefore, very restrictive, which may make it difficult to draw more general causal inferences (Cartwright, 2007). Another limitation of RCTs is ethical (Thapar & Rutter, 2019). Namely, many environmental risk factors, including harsh parenting, are not suitable to be experimentally manipulated because it would be unethical to deliberately expose participants to stimuli of this sort (Thapar & Rutter, 2019)

In addition to all, the most crucial limitation is that although they are good enough to assess the effects of interventions, to improve outcomes, they cannot definitively identify their causes (Jaffe et al.,2012). For instance, an RCT study might show that using cognitive behavioural therapy to reduce maternal anxiety reduced child anxiety. While this is consistent with the hypothesis that maternal anxiety causes child depression, it is not a definitive test as it is not logically required that the mechanisms that can improve a condition are the same as those that cause it. In short, while experimental designs may appear well-suited for causal inference due to their ability to manipulate variables, in reality, they will rarely be practical tools due to their inherent limitations.

1.4.5 Quasi-Experimental Studies

Due to the infrequent opportunity of manipulating hypothesized causal variables experimentally, researchers have turned to natural experiments, in which independent

variables are manipulated in the course of daily life. Whenever ethical considerations prevent experimental designs from being applied, quasi-experimental designs, where independent variables are manipulated naturally, provide valuable insight into the influence of genetics and environmental factors on behavioural outcomes (Plomin et al., 2018).

1.4.5.1 Adoption Designs

As mentioned earlier, familial resemblance is influenced by a combination of shared genetic and environmental factors. To distinguish between these factors, the adoption design provides an opportunity to compare families that share both genetic and environmental influences with those that share only genetic or environmental factors. Adoption studies involve two types of families referred to as "genetic and environmental families." The "genetic family" comprises pairs that share the same genetic features but do not share the environment, such as adopted children and biological parents. On the other hand, the "environmental family" consists of pairs that are genetically unrelated but share the same environment, such as an adopted child with adoptive parents or adoptive siblings (Bullard et al., 2013).

By separating heritability from the rearing environment, adoption studies can eliminate passive rGE (Jaffee et al., 2012; Plomin, 1995; Rutter et al., 2001), which is one of the significant challenges to causal inference in many designs. Due to this elimination, the resemblance between the adoptive family and the child behaviour can be attributed to environmental factors, whereas the difference between them can be attributed to heritability, as the child inherits genes from the biological parents (Rutter et al., 2001). For example, Duyme et al. (1999) found that an environmental effect of SES was observed on the IQ of adopted children. Children adopted into high SES families showed greater improvement in their IQ levels compared to those adopted into low SES families.

While adoption studies offer a practical approach to environmental causal inference, they are not without limitations. One significant constraint, as highlighted by Stoolmiller (1999), is the environmental range restriction of adoptive children. Ideally, to observe the environmental impact on the development of psychopathology in adopted children, it is necessary to have adoptive families that encompass a range of environmental risks. However, adoption agencies often screen out parents with potentially risky environments, leading to a decrease in the representativeness of adoptive families for the general population (Rutter et al., 1999). Another limitation is the difficulty in understanding how the prenatal environment impacts the development of children. Due to the fact that adopted children are separated from their biological families after birth, their prenatal environment is shared with their biological rather than adoptive mothers. Therefore prenatal environment and genetic effects may be confounded (Rutter et al, 2001).

1.4.5.2 Classical Twin Design

The twin design is another important method for understanding how genetic and environmental influences affect behavioural outcomes (Thapar & Rutter, 2019; Rutter, 2005). There are various twin designs, with most stemming from the classical twin design. This design involves comparing the correlation between identical twins (monozygotic or MZ), who originate from one fertilized egg (sharing 100% of the same genes), and fraternal twins (dizygotic or DZ), who come from separately fertilized eggs (sharing 50% of their genetic features on average).

The design assumes that if genetic factors are influencing the trait under study, the correlation between siblings in MZ twin pairs should be greater than the correlation between DZ twin pairs (Plomin et al., 2018). When the similarity of DZ twins is more than half the similarity of MZ twins a contribution from shared environment effects is implicated. Thus, the design is well-suited to understand genetic and shared environmental risk factors (Plomin et al., 2018).

However, it is not without limitations. One assumption of the classical twin design is that the environments are equally similar for both MZ and DZ twins reared in the same family. However, MZ twins tend to experience more similar environments, including familial and non-familial factors, than DZ twins (O'Connor et al., 1998). Violation of the equal environment assumption may lead to an inflation of the estimation of genetic effects. Another methodological problem concerns the generalizability of the results (Plomin et al., 2018). Since twins differ from singletons in several ways, including a 19- to 54-fold greater likelihood of being born prematurely (Tingleff et al., 2022) and experiencing slower language development (Ronalds et al., 2005), the generalizability of the results has been questioned. However, despite these limitations, it is important to highlight that they are unlikely to have a substantial effect on twin study results.

1.4.5.3 Discordant Monozygotic Twin Design

This design identifies pairs of monozygotic (MZ) twins who show discordance for an experience that is believed to increase the risk of mental health problems.
This design is based on these twins being 100% genetically identical, providing a natural way to control for genetic confounders (i.e., passive-active-evocative gene- rGE). Although they grow up in the same family, making them similar in terms of the shared environment, non-shared environmental experiences are, by definition, unique to each twin. For example, this might include being bullied by a peer (Vitaro et al., 2009) if the peer only bullies one twin in the family. Therefore, differences between exposed and unexposed twins may be assumed to result from these unique/non-shared experiences, as they are matched on genetic and shared environmental effects (Rutter, 2005). This design is generally used to understand the effect of hypothesized non-shared environmental factors that can be measured separately for each twin. For instance, Caspi et al. (2004) used the discordant MZ twin design to investigate

whether the difference in receiving maternal negative emotional expression affected antisocial behaviour. They reported that maternal emotional attitudes had an environmental causal effect on childhood behavioural problems (Caspi et al., 2004).

A significant limitation of the discordant MZ twin design is the bias of the informants. Relying on a single informant for both twins may result in a reduction of differences between MZ twins. This is because informants may have an inclination to exaggerate the similarities between twins, thus introducing bias into the results. In order to address this issue, multiple informants, such as mother and father, and self-report, may provide a solution, which may improve intra-pair reliability (Vitaro et al., 2009).

1.4.5.4 Other Twin Designs

Another valuable design being able to distinguish genetic and environmental effects is the children of twins (CoT) design, an extension of previous twin designs mentioned above (D'Onofrio et al., 2003; McAdams et al., 2014; Silberg & Eaves, 2004). CoT designs consist of adult twin pairs and their children. Since children inherit 50% of their DNA from each parent, the genetic resemblance between MZ twins and their cotwin's children is 50%. As MZ twins are genetically identical, their children (cousins) resemble each other as much as half-siblings, sharing 0.25 of their genetics. Conversely, DZ twins, who share 50% of their genes, have only 25% genetic similarity with their nieces and nephews. Therefore, the rate of resemblance among the children of DZ twins is 0.125, equivalent to the rate of resemblance in normal cousins (McAdams et al., 2014). The CoT design involves comparing the children of DZ and MZ twins, along with their avuncular relations, making it well-suited for examining cross-generational genetic and environmental transmission (Thapar & Rutter, 2019).

For the first step of the design, children of affected and unaffected MZ cotwins, for instance one of cotwins diagnosed with schizophrenia, are compared with each other. If children of an unaffected MZ cotwin show lower prevalence for the disorder when compared with the children of an affected cotwin, that may indicate causal environmental transmission of risk from the parent. That is because children of the unaffected MZ twin did not live with a parent with a risk factor of interest. On the other hand, when the prevalence rates are the same with each cousin, parental psychopathology would lose its environmental causal significance (Gottesman & Bertelson, 1989).

The same rates of the disorder in children of discordant MZ twins indicate that the disorder in the parents does not exert a specific environmental influence on the children. However, this observation does not clarify the specific pathways responsible for the elevated rates of the disorder in children with schizophrenic parents (D'Onofrio et al., 2003). Thus, as another step, comparing children of unaffected MZ and DZ cotwins helps to understand the transmission of risk between the generations. When children of an unaffected MZ cotwin have the higher prevalence rate of the disorder, compared with children of the unaffected DZ cotwin, it may imply the genetic transmission of risk, as children of MZ twins are more similar to each other genetically than DZ's children. In addition, as a last pattern, differences of avuncular correlations in MZ and DZ twins could show the genetic transmission as well. As mentioned above MZ cotwins are similar as much as a first-degree relative with their nephew, but DZ cotwins and their nephews are second degree relatives to each other. Thus, similarity of the prevalence rate in the relation between unaffected DZ-MZ cotwins and their nephews, may imply the effect of shared environmental factors (D'Onofrio et al., 2003).

In addition to the CoT design, extended children of twin designs (ECoT) using adult twins and their children and child twins and their parents can be used for causal inference (Horwitz & Neiderhiser, 2008; Narusyte et al., 2008). By using twin parents, and twin children the

model may use the whole range of genetic variance. Using twin parents helps to estimate the effects of parent's genes on child behaviour, while using twin children as well as cousin comparisons helps to understand effect of children's genes on shaping their own environment. This makes it easier to distinguish evocative rGE from passive rGE (Horwitz & Neiderhiser, 2008).

In addition to ECoT designs, another extension is the longitudinal twins and parents (LTaP) design. The model includes information about twins before adulthood and in adulthood. The model assumes that twins and parents' phenotypes are affected by the same genes. Thus, the longitudinal model may measure the effect of parental phenotypes and risk factors on twin behaviour before adulthood, which may help to identify causal process (Rutter & Thapar, 2016).

However, despite the fact that CoT, ECoT, and LTaP designs could be useful to deal with gene environment correlation, they have some limitations (Jaffee et al., 2012). As the design only relies on twin parents to understand their impact on child behavior, the influence of the non-twin parent, who contributes both different genes and environmental effects for the child, might be overlooked. This is particularly relevant in situations where both parents play a role in creating a risky environment, such as marital conflict (Eaves et al., 2005). Moreover, these designs require large samples to obtain more detailed information on family relationships, however, such samples may not be readily available (Narusyte et al, 2008).

1.4.5.5 Sibling Comparison Design

Another genetically informative design is the sibling comparison design, which takes its power from the similarity rate of 50% (on average) between full biological siblings born to the same parent in a nuclear family (Thapar & Rutter, 2019). All genetic and environmental factors could be accounted via the design as researchers use the comparison of exposed and

unexposed siblings. Any differences between siblings after risk factor exposure could be attributed to non-shared environmental experiences, or if their prevalence rate is the same for an outcome, it may be interpreted as an effect of shared environment (D'Onofrio et al., 2016).

In addition, sibling comparison designs can control confounders caused by passive, active and evocative gene-environment correlations (Lahey & D'Onofrio, 2010). Because of the meiosis process, the genes of siblings received from both parents are randomly distributed, which prevents any possibility of passive rGE within families (Rutter, 2007). For example, if a mother experienced imprisonment when one of her children was an infant but not the other, the genes associated with the mother's criminal behaviours, which might also be related to the offspring's adjustment, would impact both children equally (Lahey & D'Onofrio, 2010).

Active and evocative rGE are not automatically controlled in sibling comparison designs like passive rGE, but they can be minimized under some circumstances. To meet this requirement, exposure to the candidate environment should occur before any behavioural change, ensuring that the person's behaviours do not influence the environment. (Lahey et al., 2009; Lahey & D'Onofrio, 2010)). For instance, maternal nutrition during pregnancy cannot be influenced by child's behaviours, so any association between maternal diet and offspring behaviour cannot be explained by active or evocative rGE. However, there may be hidden rGE. For instance, mother's hormone could be influenced by infant's genetic factors, so such a possibility should be taken into consideration. In addition, instead of candidate environments shaped during pregnancy, some other candidate environments could be measured via the model, but analysis may need additional assumptions. For instance, the effect of parenting practices could be measured during infancy. However, one assumption would be that controls for measured characteristics would be enough to rule out active and evocative rGE (Lahey & D'Onofrio, 2010).

As with all study designs used for causal inference, the sibling design has some limitations. The sibling design may not eliminate all possible environmental confounders which could be the driver of behavioural change (Jaffee et al., 2012). For instance, a mother with an alcohol problem might create a toxic environment by imposing harsh punishments on her child. In this scenario, the genuine reason of the child's maladjustment might be the toxic environment itself rather than the mother's alcohol use.

Another limitation is that it is assumed that exposure to a risk factor experienced by one sibling does not affect the other one. This may not match the reality of life, as children within a family may affect each other. For example, one of the siblings may decide to not to go to the school as he is exposed to bullying, and the other one affected by their sibling's experience may do not want to go there because of the same reason. In this case, although bullying was a causal environmental factor, it may not be detected by the sibling design, as the exposed sibling's behaviours affected the other child, and so caused changes to behaviour (Lahey & D'Onofrio, 2010).

1.4.5.6 Fixed Effect Methods

The fixed effect model provides researchers with the ability to compare siblings within the same family or the same individuals across different time points (Allison, 2009). The primary advantage of the fixed effect model lies in its focus on within-individual change. This approach helps eliminate the influence of time-invariant confounding variables, such as gender or heritable effects, allowing researchers to focus specifically on changes that occur within individuals over time (Kaufman, 2008; Allison, 2005). For instance, it is known that depression can be inherited genetically from parents to their children (Sullivan et al., 2000). A family's genetic predisposition to depression could result in the adoption of harsh parenting practices, as well as an escalation of depressive symptoms in the youth (Han & Grogan-Kaylor, 2013). Failure to consider this genetic background factor could lead researchers to

incorrectly attribute depressive symptoms to hostile parental practices, creating a misleading association that does not accurately reflect the causal relationship between these variables (Cherlin et al., 1998). Therefore, using fixed effect methods helps researchers control for heritable dispositions of parents, preventing the confounding of the relationship between parenting and youth depression (Grogan-Kaylor, 2004). On the other hand, the fixed effect model is limited in its capacity to control time-varying confounders, making it challenging to address reciprocal causation when both the exposure and outcome happen concurrently (Kaufman, 2008).

1.4.5.7 Propensity Score Models

Propensity score models, which is a quasi-experimental design, aim to replicate the conditions of Randomized Controlled Trials (RCTs) by creating groups of individuals with comparable characteristics existing before their exposure to a particular risk factor or treatment (D'Agostino & D'Agostino, 2007; Rosenbaum & Rubin 1983). The propensity score indicates the probability, ranging from 0 to 1, that an individual would have received the "treatment" (or have been exposed to the risk factor) based on their background characteristics (Jaffee et al., 2012).

This statistical approach seeks to equalize the distribution of variables between groups, providing a more comparable foundation for assessing the treatment's impact. Researchers use this method to separate the genuine effect of the treatment from potential confounding factors, enhancing the validity of their analyses in observational studies. Achieving this matching involves pairing study participants based on their propensity scores, facilitating the establishment of equivalence in the distribution of characteristics between treated and untreated groups (Okoli et al., 2014). It should be noted that the propensity score matching model can be limited, as it may not be possible to control for all confounders (Shah et al., 2005).

1.5 Rationale and research aims

1.5.1 Rationale

In the preceding section, an in-depth examination was provided on environmental risk factors contributing to child internalising problems. Additionally, methodological challenges related to the causal interpretation of these risk factors were explored, and various study designs were presented with the aim of addressing these interpretation challenges.

As highlighted in the preceding chapter, the investigation of environmental risk factors for child internalising problems often relies on cross-sectional study designs, which pose limitations in establishing causation (Foster, 2010; Rutter, 2007). This limitation is an obstacle to the development of effective prevention and intervention studies (Jaffee et al., 2012). Therefore, to enhance our comprehension of the development of internalising problems, there is a clear need for studies employing causally informative methods.

1.5.2 Research questions and thesis outline

The primary aim of this project is to investigate environmental risk factors that are causally linked to internalising problems in children and adolescents. This will be achieved by pursuing the following objectives:

1. To synthesize causally informative studies examining environmental risk factors for child and adolescent's internalising problems through systematic review.

2. To examine the relationship between a commonly studied risk factor-parental mental health (distress)-and internalising problems in children/adolescents by examining potential pathways with a causally informative study design (namely the RI-CLPM).

3. To further examine the relationship between parental distress and children's internalising problems by examining moderating effects with a causally informative study design (namely the ALT-SR).

Those objectives are addressed in separate chapters. Chapter 2 presents a thorough systematic review of studies exploring environmental risk factors through causally informative study designs. Building on the findings of Chapter 2, Chapter 3 examines the relationship between parental distress - highlighted in the systematic review (Chapter 2) as commonly studied risk factor – and children's internalising problems. This research explores the mediating effect of parental distress between family income and child mental health problems. The study employs a study design that is regarded as one of the best available for obtaining causal inferences, namely RI-CLPMs. In Chapter 4, the relationship between parental distress and child mental health is further investigated by exploring the moderating effect of family income through the use of ALT-SR, another study design employed to address causality more closely. Chapter 5 provides a comprehensive general discussion, encompassing the findings from the studies in the preceding chapters. It also explores the contributions of the current findings, addresses any limitations, and lays the groundwork for future research directions.

Chapter 2: Which environmental risks make children anxious and depressed? A systematic review of causally informative studies

2.1 introduction

In Chapter 1, an examination of research on environmental risk factors influencing child internalising problems was presented. Alongside this overview, the methodological challenges inherent in this type of research were explored. Notably, the limitations of cross-sectional studies, which may struggle to eliminate familial confounders and hinder the understanding of reverse causation, were discussed. As highlighted in the previous chapter, various study designs, including quasi experimental and within-individual techniques, offer viable solutions to address the interpretation challenges posed by cross-sectional and observational studies (Rutter, 2007).

In the existing literature, a few studies have systematically reviewed causally informative investigations that delve into environmental risk factors associated with child mental health problems. For instance, Jami et al. (2021) synthesized the studies examining the effects of parental characteristics, such as parenting behaviours, on offspring mental health, and using genetically sensitive designs. Jaffe et al. (2012) presented a comprehensive review of studies investigating environmental risk factors influencing children's antisocial behavior using a variety of causally informative study designs. Furthermore, Ahmadzadeh et al. (2019) scrutinized studies investigating the relationship between child internalising problems and parental anxiety, reviewing research that utilized quasi-experimental designs. Cooper and Stewart (2021) explored the relationship between poverty and child mental health by reviewing research with Randomized Controlled Trials and quasi-experiments.

However, to date, no study has comprehensively addressed all the risk factors using causally informative study designs, especially concerning children's internalising problems. Hence, the current study undertook a comprehensive systematic review of environmental risk factors,

exploring them through causally informative study designs to understand their impact on child internalising problems.

2.2 Method

Search strategy

The systematic review protocol was registered on PROSPERO CRD42021224578. PsycINFO, Medline, Web of Science and Scopus databases were systematically searched for studies published from the earliest records to 29 December 2021. Empirical and observational studies using causally informative methods to show the relation between environmental risk factors and child and adolescent's general internalising symptoms, anxious and depressed psychopathology including their symptoms and disorder subtypes has been examined.

The search terms were created under three themes including "participants", "psychopathology", and "study designs". The inclusion of search terms like "environment* influence*" or "genetic* control*" in the study design section aimed to identify studies with the capacity to establish environmental causation by mitigating genetic confounders, supplementing the focus on specific study designs. Search terms could be found in table 2.1.

Table 2.1 *Search Terms used in the systematic review.*

Themes		
Participants	**Psychopathology**	**Study designs**
child*	internalising	(twin* AND environment*)
juven*	depress*	(adopt* AND environment*)
adolescen*	anxi*	"children of twin*"
	"emotion* and behavio*	"sibling* comparison"
	problem*"	"fixed effect*"
	"emotion*/ behavio*	"quasi experiment*"
	problem*"	"genetic* inform*"
	"emotion* problem*"	"natur* experiment*"
		"genetic* sensitiv*"
		"genetic* control*"
		"environment* caus*"
		"environment* transm*"
		"environment* influenc*"
		"environment* risk*"
		"famil* confound*"
		monozygotic
		dizygotic
		"within-subject"
		"within-person"
		"within-individual"
		"within-child"
		"within-family"
		intraindividual
		"propensity scor*"
		"instrumental variable*"
		"mendelian randomization"

Selection and data extraction

The selection process followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Page et al., 2020) (see Figure 2.1 for the flow diagram). There were 12,473 records found in the initial search. Following the removal of duplicate records, 6,021 unique records remained that were screened based on their abstracts. Of these, 5,205 were excluded due to irrelevance, leaving 816 studies for full-text review. The primary reasons for exclusion were study designs that did not meet the eligibility criteria (n=308), followed by the examination of non-environmental risk factors (n=147), incorrect age range (n=118), studies not involving the general population (n=126), and irrelevant psychopathologies (n=42). A total of 98 studies met the inclusion criteria and were included in the systematic review. In addition, 10% of the 816 full-text articles, amounting to 81 articles, were randomly selected and evaluated by two raters (ZES and GH) to assess the reliability of the criteria. The data was extracted with the following information: authors, year of publication, study design, type of environmental risk factor, age range, type of population and result.

Studies were selected if they met following inclusion criteria: a) written in English; b) aiming to use general population sampling, but this was not applied to studies that used RCT design to manipulate putative risk factors and to the studies where general population is not sampled in order to allow design to work; c) children and adolescents aged 0-18, or with a mean age within the range of 0-18; d) studies measured at least one environmental risk factors which participants are exposed to; e) general internalising symptoms, anxiety and depression symptoms and disorder subtypes; f) quasi experiment/ natural experiment studies and RCT studies manipulating putative risk factors.

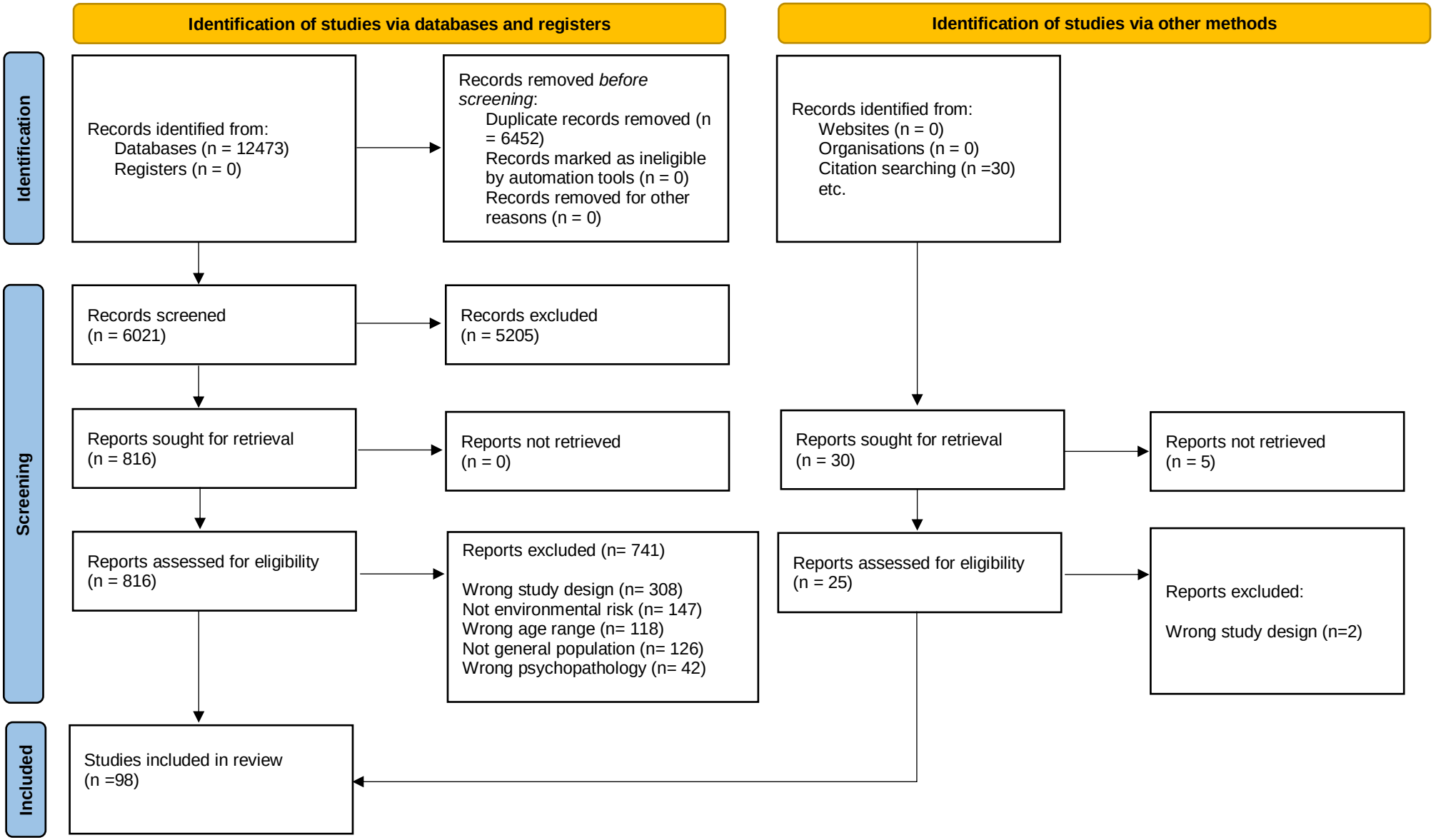
Studies were excluded if they met following exclusion criteria: a) used genetically sensitive data but did not conduct genetically sensitive analyses; or b) did not measure a specific

environmental factor. See Appendix A for the full inclusion and exclusion criteria. See Appendix A for the full inclusion and exclusion criteria.

In this study, "environmental risk factors" are defined as specific external conditions or circumstances to which participants are exposed that can potentially impact their well-being or development, such as poor parenting practices, parental psychopathology, and abuse.

In addition, a "general population" sample refers to studies that include a broad cross-section of the public without focusing on specific subgroups, such as those based on socioeconomic status or specific diagnoses. Studies involving specialized samples such as twins, siblings, or adoptees, however, are not included in this definition, as general population sampling may not be appropriate due to the study design. In addition, studies that aim to represent the general population but exhibit some degree of bias (e.g., sampling from a specific region) are included, provided their objective is to broadly reflect the general population.

Figure 2.1 *PRISMA Flow Diagram*



2.3 Results

98 studies have been published between 1977 and 2022 that were reviewed. The study characteristics for each risk factor were presented in tables corresponding to each specific risk factor. The majority of the studies were conducted in the United States (n= 44), followed by Norway (n= 11), the UK (n= 11), China (n= 8), Canada (n= 8), Sweden (n=6), Ireland (n= 2), Belgium (n= 2), the Netherlands (n=2), Iran (n=1), Greece (n= 1), and Finland (n= 1), Chile (n= 1).

1. Social Disadvantage

1.1 Family Income

Table 2.2 summarized 6 studies investigating the effect of family income on child internalising problems. The majority of these studies, including those using fixed effects (FE) designs (Mistry et al., 2009; Strohschein, 2005; Zachrisson & Dearing, 2015) and an RCT (Gennetian & Miller, 2002) design, reported that higher family income was associated with lower child internalising problems. However, Dearing et al. (2006) found no main effect of income on child internalising problems. Instead, changes in partner status and employment moderated the association between income and internalising problems with the strongest association observed when mothers of chronically poor children were employed and had a partner.

In addition, an RCT reported by Milligan& Stabile (2008) did not find a significant relationship. This discrepancy, particularly between the studies of Gennetian & Miller (2002) and Milligan & Stabile (2008), both of which are RCT studies, may be explained by the different approaches they adopted to enhance family welfare. In the study by Gennetian & Miller (2002), single mothers were randomly assigned to one of three groups: those receiving financial incentives only, those receiving financial incentives along with a requirement to

participate in employment activities, and a control group. Conversely, Milligan & Stabile (2008) provided child benefits to families. Thus, the study by Gennetian & Miller (2002) focused on family dynamics more broadly by considering both financial support for the family and the employment of mothers, which may explain the observed difference between them.

In summary, the results indicate that higher family income is generally associated with lower child internalising problems. However, the effectiveness of income-related interventions appears to vary depending on the specific context and type of financial support provided.

Table 2.2

Family income

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Mistry et al., 2009	China	444	Mean 13.0-17.2 years	FE	Youth perceptions of economic stress was associated with adolescent depression.
Strohschein, 2005	NLSY-CS/ US	7,143	4-14 years	FE	Increase in family income associated with decrease in child depression.
Zachrisson & Dearing, 2015	MOBA/ Norway	75,296	1.5-3 years	FE	Increasing family income were associated with decreasing internalising problems, especially for lower income families. In addition, childcare services decreased the adverse effect of income on children.
Gennetian & Miller, 2002	MFIP / US	879	5-12 years	RCT	Children from recipient families of the Minnesota Family Investment Program, aimed at reducing

					poverty, experienced a decrease in internalising problems, particularly among girls.
Milligan& Stabile, 2008	NLSCY/Canada	56,000	4-10 years	RCT	Child benefit programs did not influence child anxiety environmentally.
Dearing et al., 2006	NICHD/ US	1,132	2-6 years	Within-family	Although no direct association was found between family income and child internalising problems, it's noteworthy that the association became significantly stronger when chronically poor children had employed and partnered mothers.

FE= Fixed Effect; NLSY-CS= National Longitudinal Study of Youth- Child Supplement; US= United States; MOBA= Norwegian Mother, Father and Child Cohort Study; MFIP= Minnesota Family Investment Program; RCT= Randomized Controlled Trials; NCS-A = The National Comorbidity Survey Replication Adolescent Supplement; NICHD= National Institute of Child Health and Human Development.

1.2. Neighbourhood Poverty

Table 2.3 summarises five studies investigating the relationship between living in low SES neighbourhoods and child internalising problems with two RCTs (Fauth et al, 2007; Leventhal & Brooks-Gunn, 2013) and three propensity score matching (PSM) studies (Humprey& Root, 2017; Leventhal & Brooks-Gunn, 2011; Rudolph et al., 2013).

The two RCT studies focused specifically on investigating the impacts of residential mobility from impoverished to affluent neighbourhoods within the framework of housing mobility programs. These studies were the Moving to Opportunity for Fair Housing Demonstration (MTO) (Leventhal & Brooks-Gunn, 2003) and the Yonkers Family and Community Project (Fauth et al., 2007). As part of the Yonkers study, families that were randomly selected to relocate into publicly funded housing in middle-class neighbourhoods were compared with those that were not relocated (Fauth et al., 2007). Likewise, in the 3-year follow-up study of Moving to Opportunity (MTO), families from disadvantaged neighbourhoods were randomly assigned to three groups. The experimental group received housing vouchers and special assistance to move to lower-poverty neighbourhoods, the comparison group received housing vouchers without restrictions on relocation, and the control group received no intervention.

The results of the studies, however, were inconsistent. The MTO study found that internalising problems decreased significantly who moved to less-poverty neighbourhoods (Leventhal & Brooks-Gunn, 2003). In contrast, the Yonkers study showed that young people living in publicly funded housing experienced increased anxiety levels. There is a possibility that this increase is due to the fact that moving to a new neighbourhood can disrupt preexisting social networks. Families who have relocated may experience an increase in stress due to the breakdown of social networks and the loss of social support (Fauth et al., 2007).

Regarding the studies employing PSM models, the results from Leventhal & Brooks-Gunn (2011) indicated an association between change in neighbourhood poverty level and child internalising problems. The results showed that a decrease in poverty for children living in high-poverty neighbourhoods was associated with an increase in internalising problems. Conversely, an increase in poverty for children living in moderate-poverty neighbourhoods also contributed to a rise in internalising problems. This unexpected result may be due to the fact that a decrease in poverty in high-poverty neighbourhoods can lead to changes in social dynamics, leading to weaker social bonds and reduced connections among residents (Sampson, 2001), which in turn increases internalising problems. This finding is similar to the results of the Yonkers study (Fauth et al., 2007).

Furthermore, two more PSM studies confirmed that poverty has a significant impact on child internalising problems. Humphrey & Root (2017) identified that the association between neighbourhood poverty and internalising problems was most pronounced in Black children at 11 years old, with no significant influence on the relationship observed in 7-year-olds. In addition, Rudolph et al. (2013) found that residing in urban areas heightened the connection between poverty and child internalising problems compared to living in rural areas.

In summary, studies largely suggested that neighbourhood poverty is an environmental effect on child internalising problems. Results suggest that effect of the risk factor is influenced by a variety of factors, such as the level of poverty, the social dynamics of the neighbourhood, and the age and race of the child. Further, moving to less impoverished neighbourhoods may have beneficial effects on internalising problems, these benefits may not be experienced by all children and may have unexpected social consequences as well.

Table 2.3

Neighbourhood poverty

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Leventhal & Brooks-Gunn, 2011	PHDCN/ US	3,324	Mean 12.6 years	PSM	There was an environmental association between changes in neighbourhood poverty and internalising problems, particularly among boys. A decrease in poverty in high-poverty neighbourhoods was associated with an increase in internalising problems. Conversely, an increase in poverty in moderate-poverty neighbourhoods contributed to an increase in internalising problems.
Humprey& Root, 2017	NCS-A/US	21,400	7-11 years	PSM	No significant environmental relationship was found between neighbourhood disadvantages and internalising problems in 7-year-old children. At age 11, a small but significant link was observed. Additionally, being Black amplified the connection between high poverty levels and child internalising problems.

Rudolph et al., 2013	NCS-A/ US	10,074	13-17 years	PSM	Lower neighbourhood SES was found to be environmentally associated with child internalising problems among children living in urban centres. However, those relationships were no longer present for children living in rural or urban fringe areas.
Fauth et al., 2007	The Yonkers Project/US	221	8-18 years	RCT	Anxious/depressive problems increased for children allocated to publicly funded housing in middle-class neighbourhood compared to the children/youth of families who were not chosen to move. In addition, lack of frequent informal contact with neighbours was mediated the relations.
Leventhal & Brooks-Gunn, 2003	MTO	794	8-18 years	RCT	Moving from a low poverty neighbourhood to a high poverty neighbourhood reduced children's depression, particularly those aged 8 to 13 years.

PHDCN= Project on Human Development in Chicago Neighbourhoods; US= United States; PSM= Propensity score matching; NCS-A = The National Comorbidity Survey Replication Adolescent Supplement; RCT= Randomized Controlled Trials; MTO= the Moving to Opportunity for Fair Housing Demonstration.

2. Parental Psychopathology

2.1 Parental Depression

Sixteen studies investigating the effect of parental depression on child internalising problems are summarized in Table 2.4. Of these 16 studies, 6 focused solely on maternal depression (Kerr et al., 2013; Perez-Grabow et al., 2017; Lewis et al., 2011; Gjerde et al., 2019; Yan et al., 2021; Gjerde et al., 2017), 6 examined both maternal and paternal depression (Cioffi et al., 2021; Hails et al., 2019; Liskola et al., 2018; Laurent et al., 2013a; Harold et al., 2011; Laurent et al., 2013b), 1 focused solely on paternal depression (Ramchandani et al., 2008), and 3 studies, while noting the predominance of maternal reporters, investigated parental depression without distinguishing between mothers and fathers (McAdams et al., 2015; Silberg et al., 2010; Griffith et al., 2021). The studies utilized various designs, including adoption studies, sibling studies, RI-CLPM, in vitro fertilization (IVF), COT, MCoTS, and natural experiments.

Among the 12 studies focusing on maternal depression, three reported no environmental effects of maternal depression on child internalising problems (Yan et al., 2021; Hails et al., 2019; Liskola et al., 2018). All of the 9 suggesting the significance of maternal depression, eight were based on genetically sensitive designs. Of these, five identified both environmental and genetic transmission (Harold et al., 2010; Kerr et al., 2013; Laurent et al., 2013a; Lewis et al., 2011; Perez Grabow et al., 2017), while the remaining studies suggest only environmental effects (Cioffi et al., 2021; Gjerde et al., 2019; Gjerde et al., 2017; Laurent et al., 2013b). One of the most powerful studies combined a RI-CLPM with an adoption design (Cioffi et al., 2021), allowing between-person effects to be disentangled from time-specific effects. Their findings indicated that postnatal maternal depressive symptoms serve as a robust environmental risk factor. However, it's important to exercise caution when

interpreting the results of Lewis et al. (2011) and Harold et al. (2010), as their cross-sectional approach utilizing IVF design may limit their ability to determine the direction of effect.

Additionally, the 3 studies investigating parental depression, primarily based on maternal reports of parental depression, resulted in findings of environmental effects (McAdams et al., 2015; Silberg et al., 2010; Griffith et al., 2021). McAdams et al. (2015) investigated bidirectional effects between parental depression and child internalising problems in middle childhood using both adoption and twin data. While they did not find an environmental relationship between parent and child in the adoption study, they did find an environmental effect in the twin study.

Regards to paternal depression, it has received less research attention than maternal depression, being addressed in only seven addressed in eight studies identified (Cioffi et al., 2021; Hails et al., 2019; Harold et al., 2010; Laurent et al., 2013a; Laurent et al., 2013b; Liskola et al., 2018; Ramchani et al., 2008). Among these studies, only two (Cioffi et al., 2021; McAdam et al., 2015; Harold et al., 2010) did not identify a significant environmental effect. McAdam et al. (2015) and Harold et al. (2010) instead reported a genetic effect of paternal depression on child internalising problems.

In summary, the majority of studies on parental depression-whether focusing on maternal or paternal depression-highlight significant environmental effects on child internalising problems, suggesting that parental depression is a notable risk factor for these issues.

Table 2.4

Parental Depression

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Cioffi et al., 2021	EGDS/ US	561	18months- 8 years	Adoption/ RI-CLPM	Adoptive mothers, but not adoptive fathers, were found to have an environmental association between their depression and child internalising problems.
Hails et al., 2019	EGDS/ US	561	6 years	Adoption	There was an association between paternal depression and child internalising problems only when the symptoms were reported by the parents. It was not present in teacher-reported internalising problems. In addition, there was no maternal depression effect on child internalising problems.
Liskola et al., 2018	Finnish Adoption Survey/ Finland	548	9-12 years	Adoption	Significant adoptive paternal but not maternal depression effects on offspring depressive symptoms were found. In addition, girl's negative

					mood was found to influence paternal depression.
Kerr et al., 2013	EGDS/US	346	18-54 months	Adoption	Both adoptive and biological mothers' depressive symptoms were concurrently associated internalising problems of their children at age 18 months.
McAdams et al., 2015	TOSS and EGDS/ US	876-twin 361-adoption	4.5- 7 years	Adoption/ CoT	In the CoT study, association between parental depression and child internalising was found to be environmental. Conversely, in the adoption study, links between parental and child internalising problems were genetically mediated at age 7 with an additional child-to-parent environmental effect.
Perez-Grabow et al., 2017	EGDS and EPoCh/ US	541	7 years	Adoption	Adoptive mother's depression was associated with child internalising problems. In addition, there was a

					significant heritable path between biological mother depression and child outcome.
Laurent et al., 2013a	EGDS/ US	192	54 moths	Adoption	Both birth parent prenatal and rearing parent postnatal depressive symptoms reduced child cortisol levels, which in turn increasing child internalising problems. Thus, results indicated both environmental and genetical transmission.
Laurent et al., 2013b	EGDS/ US	210	18-54 months	Adoption/ Within-family	Adoptive mother's depression was associated with child internalising problems environmentally. While changes in adoptive father's depressive symptoms were not linked to changes in children's internalising problems across the whole sample, a positive association emerged for children with higher evening cortisol levels.

Silberg et al., 2010	VTSABD/ US	2,940	9-17 years	CoT	The relation between parental depression and child depression was found to be fully environmental.
Harold et al., 2011	UK and US	MGR=434; MGUR=127; FGR= 403; FGUR= 156	4-10 years	IVF	Maternal depression was associated with child internalising problems through both environmental and genetic routes, whereas paternal depression appeared to be transmitted genetically.
Lewis et al., 2011	UK and US	852	4-13 years	IVF	Both genetic and environmental associations were found to underlie the relationship between maternal depression and child internalising problems.
Gjerde et al., 2019	MOBA/Norway	35,589	1.5-5 years	MCoTS	Although for the ages of 1.5 and 3 children, passive genetic transmission of maternal depression influenced emotional problems, at 5 the relation was directly environmental.

Ramchandani et al., 2008	ALSPAC	14,541	3,5-7 years	Natural experiment	Paternal depression, when chronically elevated, in pre- and post-natal periods, predicted more severe child internalising problems in early childhood.
Griffith et al., 2021	Canada	382	11-17 years	RI-CLPM	Effect of parental depression on child internalising was found contemporaneously at the within-individual level. However, there was no within-individual effect of child on parent.
Yan et al., 2021	ALSPAC	1,178	2-15 years	RI-CLPM	The association between maternal depression and child internalising problems was largely based on between-participant correlations rather than within-participant cross-lagged effects.
Gjerde et al., 2017	MOBA/Norway	17,830	1.5- 5 years	Sibling comparison	Concurrent but not prenatal maternal depression was related

						to child internalising problems.

EGDS= Early Growth and Development Study; US= United States; TOSS= Twin and Offspring Study of Sweden; CoT= Children of Twins; EPoCh= Early Parenting of Children; VTSABD= Virginia Twin Study of Adolescent Behavioural Development; UK= United Kingdom; MGR= Mother genetically related; MGUR= Mother genetically unrelated; FGR= Father genetically related; FGUR= Father genetically unrelated; IVF= In Vitro Fertilization; MOBA= Norwegian Mother and Child Cohort Study; MCoTS= Multiple-Children-of- Twins-and-Siblings; ALSPAC= The Avon Longitudinal Study of Parents and Children; RI-CLPM= Random Intercept Cross Lagged Panel Model.

2.2. Parental Anxiety

Table 2.5 summarises seven studies that investigated the impact of parental anxiety symptoms on children's internalising problems. Among these studies, five examined postnatal, or concurrent parental anxiety during childhood and adolescence, using various research designs including adoption (Ahmadzadeh et al., 2019; Brooker et al., 2014; Field et al., 2020), CoT (Eley et al., 2015), and sibling designs (Gjerde et al., 2020). Others examined prenatal maternal anxiety (Bekkhus et al., 2018; Gjerde et al., 2020; Rice et al., 2010).

In three of the studies examining postnatal/concurrent parental anxiety the association between parental and child anxiety was found to be primarily driven by environmental factors rather than genetics (Ahmadzadeh et al., 2019; Brooker et al., 2014; Eley et al., 2015). Field et al. (2020) found both genetic and environmental effect to be very small. It is important to note that the impact of maternal and paternal anxiety varied across these studies. Ahmadzadeh et al. (2019) observed both parent-driven and child-driven effects whereas, the others who mainly found parent-driven effects. The study reported that only paternal anxiety had a significant influence on child internalising problems, while maternal anxiety had a child-driven effect; having anxious children led to maternal anxiety (Ahmadzadeh et al, 2019). In contrast, Gjerde et al. (2020) discovered that maternal anxiety had a direct influence on child anxiety.

In addition to parental anxiety during childhood and adolescence, three studies examined the effects of prenatal maternal anxiety on children. These studies consistently found no significant prenatal environmental effect on child internalisation in sibling comparison studies (Bekkhus et al., 2018; Gjerde et al., 2020) and IVF designs (Rice et al., 2010). The relationships initially identified were explained by genetic or familial confounders, indicating that they were not attributable to environmental effects.

In conclusion, most of the evidence suggests that postnatal/concurrent parental anxiety, particularly paternal anxiety, is an environmental risk factor for child internalising problems. Conversely, prenatal maternal anxiety does not appear to have a significant environmental impact, highlighting the importance of timing in the influence of parental anxiety on children's mental health.

Table 2.5

Parental anxiety

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Ahmadzadeh et al., 2019	EGDS/US	305	6- 8 years	Adoption	Father's anxiety was associated with an increase in child anxiety. This relationship was entirely attributed to environmental factors. Additionally, increases in child anxiety led to higher levels of maternal anxiety.
Brooker et al., 2014	EGDS/US	561	18- 27 months	Adoption	Adoptive parent anxiety was associated with child internalising problems environmentally. No genetic contribution was found.
Field et al., 2020	EGDS/US	561	Mean 1.5- 4.5 years	Adoption	Both inherited and environmental influences had very small effects on anxiety at 18 months, as well as negligible effects on the rate at which those symptoms change over the preschool years.
Eley et al., 2015	TOSS/ Sweden	387 Mz Twins 489 DZ Twins	11-22 years; Mean 15.7 years	CoT	Environmental transmission from parent to adolescent was found. There was no genetic transmission identified.

Rice et al., 2010	UK and US	779	4-10 years	IVF	Prenatal maternal anxiety and child anxiety were not environmentally related to each other. Their relationship was explained by the influence of concurrent maternal anxiety.
Bekkhus et al., 2018	MOBA/ Norway	21980	6 months-3 years	Sibling comparison	Prenatal maternal anxiety was not environmentally linked to child internalising problems. The observed relationship between them was attributed to familial and genetic confounds.
Gjerde et al.,2020	MOBA/ Norway	17,724	6 months- 5 years	Sibling comparison	Concurrent maternal anxiety was environmentally associated with child internalising problems. No environmental effect of prenatal anxiety was observed. Familial confounds were present for the impact of prenatal anxiety.

EGDS= Early Growth and Development Study; US= United States; TOSS = Twin and Offspring Study of Sweden; CoT = Children of Twin; UK= United Kingdom; IVF = In Vitro Fertilisation; MOBA= Norwegian Mother and Child Cohort Study.

2.3. Parental Internalising and Externalising Problems

Table 2.6 summarizes findings from seven studies investigating the impact of parental internalising problems measured in the combining scores of both anxiety and depression (3 studies) (Marceau et al., 2013; Ross et al., 2016; Schulz et al., 2021)) and parental distress (1 study) (Speyer et al., 2022); externalising problems, including parental antisocial behaviours (2 studies) (Kerr et al., 2013; Silberg et al., 2012); and general psychopathology (1 study) (Xerxa et al., 2020) on child internalising problems.

Within the studies exploring parental internalising problems, the results were inconsistent. Studies using adoption designs found that adoptive parents' internalising problems were environmentally associated with children's internalising symptoms (Marceau et al., 2013; Ross et al., 2016). Additionally, interactions between biological parents' and adoptive parents' internalising symptoms were associated with child internalising problems in Ross et al.'s (2016) study, while genetic transmission was also observed in Marceau et al.'s (2013) study. These findings indicate the existence of both environmental and heritable effects of parental internalising symptoms in adoption studies. Similarly, a study using the ALT-SR within-individual design by Speyer et al. (2022) also supported the effect of parental distress on child and adolescent internalising problems.

In contrast, the study of Schulz et al. (2021), another within-individual design (RI-CLPM), there was no parent-driven effect on child internalising problems at the within-family level; instead, only child-driven effects on maternal internalising problems were found. One possible explanation for the differences between adoption studies and the study of Schulz et al., 2021 would be the developmental stages examined. Adoption studies focused on early childhood, during which children are more dependent on their parents and parents play a central role in the lives of their children. On the other hand, Schulz et al. (2021) examined

adolescence, a period of increasing independence and shifting family dynamics that is often characterized by children's dependence on their parents (Schepman et al., 2011). Thus, adolescents may be less affected by their parents' mental health.

However, it is also notable that Speyer et al. (2022) also examined adolescents and found significant effects. The discrepancy between these findings and those of Schulz et al. (2021) may be due to differences in how internalising problems were measured. Specifically, Speyer et al. (2022) focused on parental distress, while Schulz et al. (2021) employed a broader assessment of internalising problems.

Regarding the effect of parental antisocial behaviours on children's internalising problems, in an adoption study belongs to Kerr et al. (2013) was found both heritable and environmental effect. Silberg et al. (2012), however, found a fully environmental effect using ECoT.

In short, the studies yielded mixed results regarding the impact of measured mental health problems. The effect of parental internalising problems on child internalising problems varies based on study design, developmental stage, and the specific types of problems assessed. On the other hand, studies on parental externalising problems provided more consistent results, generally supporting the environmental effects of these mental health problems of parents.

Table 2.6

Parental internalising and externalising problems

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Kerr et al., 2013	EGDS/US	346	1.5 -4.5 years	Adoption	Mother's antisocial behaviour was related to child internalising problems through genetic and environmental pathways.
Marceau et al., 2013	EGDS/US	561	27 months	Adoption	Both genetic and environmental effect of maternal internalising problems were found.
Ross et al., 2016	EGDS/US	293	6- 7 years	Adoption	With both biological and adoptive parents' internalising symptoms interacting, a heritable effect was evident. Furthermore, the environmental effect of adoptive parents' internalisation was examined.
Speyer et al., 2021	MCS	10,734	3 -17 years	ALT-SR	At the within-family level, maternal distress affected both genders, while paternal distress only affected boys during the early adolescent years. In addition, child to parent effect was observed.
Xerxa et al., 2020	Generation R Study/ Dutch	5,536	10 years	ALT-SR	Parental psychopathology had an effect on child internalising problems at the within-family level. In addition, child to parent effect was observed.
Silberg et al., 2012	MATR and VTSABD	2,454 2,826	9-17 years 8-18 years	E-CoT	The effect of parental antisocial behaviours on child depression was fully environmental.

Schulz et al., 2021	RADAR-Y/ Dutch	497	13- 18 years	RI-CLPM	Maternal internalising symptoms did not have an environmental effect on child internalising problems in within-person level. However, child-driven effect was found on maternal internalising problems.

EGDS= Early Growth and Development Study; US= United States; MCS= Millennium Cohort Study; ALT-SR= auto-regressive latent trajectory model with structured residuals; MATR= Mid-Atlantic Twin Registry; VTSABD= Virginia Twin Study of Adolescent Behavioural Development; E-COT = Extended Children of Twins study; RADAR-Y= Research on Adolescent Development and Relationships Young; RI-CLPM= Random intercept cross lagged model.

3. Parental Substance Misuse and Exposing to Prenatal Psychotropic Medications

3.1 Parental Alcohol and Smoking Use

Table 2.7 provides a summary of four studies investigating the impact of prenatal exposure to smoking and alcohol on children and adolescents' internalising problems (Knudsen et al., 2015; Lund Et al., 2019; McCrory & Layte, 2012; Sen & Swaminathan, 2007). The studies examining the effects of smoking during pregnancy have yielded inconsistent results (McCrory & Layte, 2012; Sen & Swaminathan, 2007).

McCrory & Layte (2012) focused on the effect of prenatal maternal smoking on child internalising problems during late childhood (aged 9) - by comparing mothers who smoked during pregnancy with those who did not, using PSM model. They found that children whose mothers smoked during pregnancy exhibited more internalising symptoms than those whose mothers did not smoke, as reported by parents. Furthermore, this significant relationship became more pronounced with an increase in the amount of maternal smoking. Sen and Swaminathan (2007) combined both PSM and FE methods to investigate the prenatal maternal smoking effect on child internalising problems. Their study did not find a significant effect during both early and later childhood. It is worth noting that although both McCrory & Layte (2012) and Sen and Swaminathan (2007) utilized similar methods, PSM and focused on similar age ranges, they produced different results. The inclusion of the FE method in Sen and Swaminathan's (2007) study, in addition to PSM, may explain the disparity in results, as it provides additional control over unobservable confounders (Sen & Swaminathan, 2007).

Regarding the impact of parental alcohol consumption on internalising problems, the results also exhibit inconsistencies (Lund et al., 2019; Knudsen et al., 2015; Sen & Swaminathan, 2007). Sen and Swaminathan's (2007) study found a significant relationship between heavy maternal alcohol use and child internalisation problems. On the other hand, Knudsen et al.

(2015) and Lund et al. (2019) examined the same sample (MOBA) and the same age range (toddlerhood) and reported that the relation found between parental alcohol consumption and child internalising problems was explained by unmeasured confounding variables. Lund et al. (2019) found a significant effect of maternal alcohol use on somatic complications and emotional reactivity, but not on anxiety or depression. Overall, there is mixed evidence regarding the impact of prenatal exposure to smoking and alcohol on child internalising problems. The discrepancies between results may be related to differences in study methodologies or other factors, such as unmeasured confounders.

Table 2.7

Parental alcohol and smoking use

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Knudsen et al., 2015	MoBa/ Norway	51,115	1,5 and 3 years	FE	There was no environmental effect of maternal heavy alcohol use on children's internalising problems.
Lund et al., 2019	MoBa/ Norway	25,744	1.5, 3 and 5 years	IV and Sibling comparison	Compared with unexposed siblings, children exposed to maternal hazardous drinking were more emotionally reactive and had more somatic complaints at 3 years only. This association was not present for symptoms of anxiety/depression.
Sen & Swaminathan, 2007	CNLSY/ US	8,395	4-6.5 years 8-10.5 years	PSM /FE	There was no relationship between prenatal smoking exposure and anxiety or depression symptoms. Prenatal alcohol use had an environmental effect on internalising problems.
McCrory & Layte, 2012	Growing Up in Ireland/ Ireland	8,568	9 years	PSM	A relationship was found between prenatal maternal smoking exposure and child internalising problems.

MOBA= Norwegian Mother and Child Cohort Study; FE= Fixed Effects; IV= Instrumental Variable; CNLSY= Children of the National Longitudinal Survey; US= United States; PSM= Propensity Score Matching.

3.2 Prenatal Exposure to Psychotropic Medications

Table 2.8 summarises 4 studies investigating the effect of prenatal psychotropic medication exposure including both antidepressants (Brandlistuen et al., 2015; Park et al., 2020) and depressants including benzodiazepines (BZDs)/ z-hypnotics (Brandlistuen et al., 2017; Sundbakk et al., 2019) on children's internalising problems.

Park et al. (2020) and Brandlistuen et al. (2015) reported that prenatal exposure to antidepressants was associated with increased anxiety symptoms in children. Brandlistuen et al. (2015), for instance, compared mothers who continued antidepressant treatment during pregnancy with those who discontinued it. Their analysis revealed that children born to mothers who discontinued antidepressant treatment exhibited significantly fewer anxiety symptoms.

However, regarding the studies on the effects of BZDs and z-hypnotics, inconsistencies arise between the findings of Sundbakk et al. (2019) and Brandlistuen et al. (2017). Brandlistuen et al.'s (2017) sibling comparison design found children exposed to BZDs during prenatal development displayed more internalising problems compared to their non-exposed siblings. This effect persisted even after accounting for familial factors, suggesting an environmentally mediated association. However, in the same study, the use of z-hypnotics did not lead to an increase in internalising problems after adjusting for familial confounders. In contrast, Sundbakk et al. (2019), using a propensity score design, reported that children did not exhibit a significant increase in internalising problems after prenatal exposure to both BZDs and z-hypnotics.

It should be noted that in both the studies conducted by Park et al. (2020) and Sundbakk et al. (2019), a critical limitation was the absence of dose information for antidepressants and BZDs/z-hypnotics. This limitation made it impossible to assess whether higher doses of these

treatments could be associated with adverse effects. Additionally, Brandlistuen et al. (2015) could not exclude the possibility that the effect of prenatal antidepressant exposure might be influenced by mothers who continued treatment having more severe mental illnesses than those who discontinued.

In summary, while these studies have some limitations, they collectively suggest that prenatal antidepressant exposure is associated with an increased risk of children's internalising problems, particularly anxiety, when compared to exposure to BZDs and z-hypnotics. However, the absence of dose-related information and other potential confounders should be considered when interpreting these findings.

Table 2.8

Prenatal exposure to psychotropic medications

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Brandlistuen et al., 2015	MoBa/ Norway	14,435	1.5- 3 years	FE	Prenatal antidepressant exposure was associated with child internalising problems through an environmental path.
Brandlistuen et al., 2017	MoBa/ Norway	71,996	1.5- 3 years	FE	There was no association between z-hypnotics and children's internalising problems. There was an environmental association between internalising and BZDs.
Park et al., 2020	Canada	94,712	Mean 5.65 years	PSM	Antidepressant exposure was environmentally associated with child internalising problems.
Sundbakk et al., 2019	MoBa/ Norway	35, 629	5 years	PSM	Neither BZDs nor z-hypnotics were associated with children's internalising problems.

MOBA= Norwegian Mother and Child Cohort Study; FE= Fixed Effects PSM= Propensity Score Matching.

4. Stressful life Events

4.1 Stressful life Events- General Perspective

Table 2.9 provides a summary of four studies investigating the impact of exposure to stressful life events (SLEs) on internalising problems using within-individual designs. These studies consistently found a significant association between SLEs and internalising problems. Additionally, they revealed moderation effect of higher average levels of SLEs (Jenness et al., 2019), emotion differentiation (Nook et al., 2021) on the relation between SLE and child internalising problems. Mediation variables including the frequency of phone communication (Rodman et al., 2021) and variability in sleep duration (Bustamante et al., 2020) were revealed on that relation also.

Notably, Jennes et al. (2019) reported that the level of SLEs at the between-individual level moderated the relationship between within-individual SLEs and depression. Adolescents with higher cumulative exposure to SLEs experienced more depression symptoms during times when they reported greater SLEs, in contrast to adolescents with lower mean levels of SLEs. This suggests that the impact of SLEs on depressive symptoms was more pronounced among adolescents with higher cumulative exposure to stressors. However, it's important to mention that the identified studies, with the exception of Jennes et al. (2019), included had a limited sample size, which may hinder the generalizability of the results.

In conclusion, the studies consistently support the environmental effect of SLEs on internalising problems. Additionally, the findings on moderation and mediation effects provide further insight into this relationship. However, the generalizability of these findings should be considered carefully due to the small sample sizes in most studies, except for Jenness et al. (2019).

Table 2.9

Stressful life Events- General Perspective

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Bustamante et al., 2020	US	30	15-17 years	Within-individual	SLEs were environmentally associated with internalising problems. The relationship was mediated by sleep duration.
Jenness et al., 2019	Canada	382	9-15 years	Within-individual	SLE were environmentally associated with internalising problems. The relationship between within-individual level SLEs and depression was stronger among adolescents who had higher SLE level in between-individual level.
Nook et al., 2021	US	30	15-17 years	Within-individual	Perceived stress was environmentally associated with internalising problems. The relationship was stronger for adolescents with low emotion differentiation-Being

					able to identify one's feelings.
Rodman et al., 2021	US	30	15-17 years	Within-individual	SLEs were associated with anxiety and depression via changes in frequency of phone communication in young people.

US= United States; SLE= Stressful life events.

4.2 Divorce

Table 2.10 provides a summary of two studies investigating the effect of parental divorce on child internalising problems. In the study by O'Connor et al. (2000), both adoptive and biological families were examined separately, and then divorced adoptive families were compared to divorced biological families. In both biological and adoptive families, children with divorced parents exhibited more internalising problems when compared to children from intact families. Additionally, adoptive children from divorced families showed a higher level of internalising problems than biological children from divorced families. This indicates an environmental impact of parental separation on children's internalising problems. However, it is important to note a limitation of this study, as it did not consider the level of children's internalising problems before the divorce. Therefore, it's uncertain whether the internalising problems were a result of the divorce or if they preceded it.

Weaver and Schofield (2015) overcame this limitation by examining the long-term effects of divorce on children's behaviour problems including measurement before and after divorce. This study revealed that, in addition to experiencing more internalising problems compared to a propensity score-matched sample of intact families, these children exhibited increased in internalising problems after divorce when compared to their pre-divorce levels. Furthermore, the study reported that mother's pre-divorce sensitivity and child's intelligence had protective effects on the child's internalising symptoms. Children with highly sensitive mothers and high IQ levels experienced fewer internalising problems following parental separation. Additionally, the study utilized both maternal and teacher reports to assess these effects, and the observed impact on internalising symptoms was consistent both at home and at school. This consistency suggests that the findings were not influenced by the setting or potential informant bias (Weaver and Schofield, 2015).

In conclusion, the results consistently demonstrate the adverse impact of parental divorce on internalising problems, with Weaver and Schofield (2015) providing more comprehensive insight by addressing pre-divorce conditions and employing multiple informants. However, it should be noted that divorce has only been examined in two studies, so further research would be beneficial to gain a more comprehensive understanding of this risk factor.

Table 2.10

Divorce

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
O'Connor et al., 2000	CAP/US	398	12	Adoption	Divorce associated with internalising problems environmentally.
Weaver & Schofield, 2015	NICHD/US	260	5- 15 years	PSM	Divorce increased internalisation problems in children. A sensitive mother and a high child IQ were protective against the impact of divorce.

CAP = Colorado Adoption Project; US= United States; NICHD = Study of Early Child Care and Youth Development; PSM= Propensity Score Matching.

4.3 Parental Loss

Table 2.11 summarizes findings from four studies that utilized PSM (Bradshaw et al., 2019; William & Amber, 2015; Zhou et al., 2018) and twin study designs (Eley & Stevenson, 2000) to investigate the impact of parental loss due to bereavement, parental migration, and parental incarceration on child mental health. There was a consensus among these studies that these risk factors had adverse effects on the mental health of children, except in the study of Williams & Amber (2015) which reported null results.

William & Amber (2015) formed a treatment group consisting of children who experienced the loss of a parent between kindergarten and 3rd grade spring. They compared this group to a control group of children who did not experience parental bereavement up to the end of 3rd grade. Initially, they found that bereaved children tended to exhibit higher levels of internalising behaviours compared to the control group. However, demographic and family characteristics that existed before the parental bereavement occurred explained this association.

In contrast to William & Amber (2015), Eley & Stevenson (2000) found that loss events, particularly those involving the death of a family member, were associated with an increased likelihood of depressive symptoms in children. They compared twins within-family and found that when one twin experienced a loss event, e.g., grandparent or other shared loss, it was more likely that their co-twin also experienced similar depressive symptoms. This suggests a shared influence within twin pairs in response to such loss events. This shared influence was specific to depression and did not apply to anxiety.

The evidence indicates that both parental migration and parental incarceration are environmentally associated with negative outcomes for children. Bradshaw et al. (2019) found that parental incarceration was linked to higher anxiety levels at age 9, lower happiness

at age 13, and increased emotional difficulties reported by caregivers at age 13. In contrast, Zhou et al. (2018) reported that parental migration exacerbated depressive symptoms in affected children, primarily due to decreased caregiving and emotional support resulting from parental absence. Increased household income resulting from parental migration did not alleviate the negative impact on depressive symptoms, highlighting the limitations of economic compensation in addressing the emotional and psychological challenges faced by these children.

Except for the study belongs to William & Amber (2015), most studies emphasize the significant environmental impact of parental loss on child internalisation. It is important to note, however, that the types of parental loss examined varied, including those caused by bereavement, incarceration, and migration. For a more comprehensive understanding, more studies focusing parental loss would be beneficial.

Table 2.11

Parental loss

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Eley & Stevenson, 2000	UK	795 twins	8-16 years	MZ/ DZ twin	Parental loss was environmentally associated with child depression rather than anxiety.
Bradshaw et al., 2019	GUI/ Ireland	8,568	9-13 years	PSM	Having a parent in prison has been found to be environmentally associated with adverse developmental outcomes for their children.
Williams & Amber, 2015	ECLS-K/ US	21, 410	5-14 years	PSM	There was no relationship between parental bereavement and child emotional problems.
Zhou et al., 2018	CFPS/ China	442	10-15 years	PSM	Parental migration led to increased depression in children.

UK= United Kingdom; MZ= Monozygotic; DZ= Dizygotic; GUI = Growing up in Ireland National Longitudinal Study of Children; PSM Propensity score matching; ECLS-K = Early Childhood Longitudinal Study-Kindergarten Class of 1998-1999; US = United States; CFPS = China Family Panel Studies.

4.4 Maltreatment

Table 2.12 presents the findings of four studies that employed Fixed Effect designs (Emery et al., 2011; Ma et al., 2022) and RI-CLPM (Li et al., 2021; Yang et al., 2021). These studies explored the impact of childhood maltreatment, including parental physical abuse, intimate partner violence (IPV) against children, emotional maltreatment, and general maltreatment, on children's internalising problems. These studies consistently identified that childhood maltreatment was environmentally associated with child internalising problems.

While most of the studies had relatively short follow-up periods, typically lasting only 2 years (e.g., Li et al. (2021), Emery et al. (2011) studied children from ages 3 to 15 years. This extended duration allowed examination of how maltreatment affects internalising problems over an extended period. Emery et al. (2011) found a relationship between child internalising and IPV. In addition, they reported that the relationship diminished as the child's age increased.

In summary, while there is growing evidence supporting the association between maltreatment and child internalising problems, gaining a comprehensive understanding of the long-term relationship remains a work in progress. In addition, it is essential to exercise caution when interpreting these consistent yet diverse findings, as the studies investigate various forms of maltreatment, ranging from emotional to physical abuse forms of maltreatment, ranging from emotional to physical abuse.

Table 2.12

Maltreatment

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Emery, 2011	PHDCN/ US	1,816	3- 15 years	FE	Intimate partner violence against children led to increase in children's internalising problems environmentally.
Ma et al., 2022	China	2,180	8-13 years	FE	Parental physical abuse was associated with increasing depression environmentally.
Li et al., 2021	China	4273	8-11 years	RI-CLPM	Emotional maltreatment environmentally increased the risk of depression at the within-family level.
Yang et al., 2021	China	4110	8-11 years	RI-CLPM	Childhood maltreatment was environmentally associated with increasing depression at the within-family level.

PHDCN= Project on Human Development in Chicago Neighbourhoods; US= United States; FE= Fixed Effect; RI-CLPM = Random Intercept Cross-lagged Panel Model.

5. Parenting and Family Functioning

5.1 Parental Rejection/Hostility/Negative feelings

Table 2.13 summarises eight studies investigating the effect of parental rejection/hostility/negative feelings on child internalising problems. The results were inconsistent; half of the included studies reported an environmental relationship between parental rejection/negativity and child internalising (Asbury et al.,2003; Chen et al., 2016; Ross et al., 2016; van der Voort et al., 2013), while the other half did not support an environmental association (Asbury et al., 2006; Glover et al., 2010; Mullineaux et al., 2009; Lin et al., 2021).

Among the studies using the MZ twin differences design, Chen et al. (2016) and Asbury et al. (2003) identified an environmental association between parental harshness, hostility, or negativity and child internalisation problems. Chen et al. (2016) also found genetic influences on this association. Chen et al. (2016) conducted a cross-sectional study, limiting the ability to establish the direction of causation. In contrast, Mullineaux et al. (2009) used a longitudinal MZ twin difference design and found no relationship between maternal negativity and child outcomes. However, due to the correlational nature of the MZ difference method, Mullineaux et al. (2009) also noted that their study does not provide definitive evidence regarding the direction of causation.

Regarding adoption studies, Roos et al (2016) and van der Voort et al. (2013) found that parental sensitivity/involvement was environmentally associated with child internalising. In addition, Ross et al. (2010) reported that a combination of birth mother's slow processing speed and adoptive mother's high internalising symptoms predicted internalising symptoms, suggesting that genetic predispositions may make children more sensitive to social environmental stressors (Reiss et al., 2013).

Glover et al (2010) examined families with both adopted and biological children the relation between adopted children internalising and negative parenting was not significant. It is important to consider that adoption studies have limited sample sizes, which may increase the likelihood of spurious results (Glover et al., 2010; Roos et al., 2016; Voort et al., 2013).

All in all, despite some evidence supporting the effect of parental rejection/hostility/negative feelings on internalising problems in children, the results are not consistent across studies. Therefore, reaching a definitive conclusion regarding the environmental effects of parental hostility on child internalising problems based on existing evidence is challenging.

Table 2.13

Parental Rejection/Hostility/Negative feelings

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Glover et al., 2010	Northeast/ US	170	4 to 16 years	Adoption	Parental negativity/positivity and child's internalising problems were not associated environmentally.
Ross et al., 2016	EGDS/ US	293	6-7 years	Adoption	The combination of birth mother's slow processing speed and adoptive mother's high internalising symptoms predicted internalising problems environmentally.
van der Voort et al., 2013	Leiden/ Netherlands	160	7-14 years	Adoption	More maternal sensitivity was environmentally associated with less anxiety/depression in adolescents.
Asbury et al., 2006	TEDS/UK	4,034 twins	7 years	MZ twin	There was no environmental relationship between negative parental feelings and children’s anxiety symptoms.

Asbury et al.,2003	TEDS/UK	4,706 twins	4 years	MZ twin	Negative parental feelings and child's anxiety symptoms were related environmentally. The relationship was stronger in the context of greater family chaos and maternal depression.
Chen et al., 2016	BeTwiSt/ China	1608	10-18 years	MZ twin	Parental hostilities were environmentally associated with youth anxiety, in addition to genetic influences.
Mullineaux et al., 2009	NEAD/ US	154 twins	Mean 4.33–7.92years	MZ twin	There was no environmental relation between maternal negativity and child internalising problems.
Lin et al., 2021	ABCD/ US	4,951	9 and 10 years	Within-individual	There was no environmental relation between increasing parental acceptance and child internalising problems.

The Northeast =Northwest Collaborative Adoption Project; US = United States; EGDS= The Early Growth and Development Study; TEDS = Twins Early Development Study; UK = United Kingdom; MZ= Monozygotic; BeTwiSt= Beijing Twin Study;NEAD = Non-Shared Environment and Adolescent Development; ABCD= The Adolescent Brain and Cognitive Development.

5.2 Harsh and Overreactive Parenting

Table 2.14 provides a summary of 5 studies investigating the effect of overreactive parenting referring to propensity to react with anger, frustration, and harshness to children's challenging behavior (Marceau et al., 2015; Marceau et al., 2013) and harsh parental discipline (Asbury et al., 2006; Asbury et al.,2003; Ma et al., 2022) on child internalising problems. Both studies on overreactive parenting found an environmental association with child internalising problems in adoption studies. Marceau et al. (2015) reported that adoptive fathers' overreactive parenting, measured from 9 months to 4.5 years, predicted more internalising problems at age 6. Additionally, child's lower morning cortisol levels mediated the relationship between father's overreactive parenting and child internalising problems.

It is important to note that these studies examined internalising problems at different developmental stages, specifically infancy (Marceau et al., 2013) and middle childhood (Marceau et al., 2013). This suggests that the impact of overreactive parenting may persist across different stages of development. Nonetheless, it is crucial to exercise caution in drawing definitive conclusions, as only two studies may not provide sufficient evidence to make such claims.

Regarding the effect of parental harsh discipline on child's internalising problems, the results of studies using MZ twin difference (Asbury et al., 2006; Asbury et al., 2003) and FE (Ma et al., 2022) designs are inconsistent. Notably, Asbury et al. (2003) and Asbury et al. (2006) reached different conclusions, despite using the same data and study designs. While Asbury et al. (2003) reported that within-pair differences in harsh parenting were correlated with child anxiety, no such correlation was found in Asbury et al. (2006). It has been suggested that this discrepancy in results could be attributed to the use of a cross-rater design, involving

teacher-reporters in Asbury et al. (2006), as opposed to relying solely on parent reporters as in Asbury et al. (2003).

In summary, overreactive parenting has consistently been shown to have an environmental effect, while harsh discipline has also been found to have an environmental impact, though the results on harsh discipline are mixed. The differences between harsh discipline studies may be explained by methodological variations, such as the sample size and the choice of informants. Considering the limited number of studies available, further research would be beneficial in order to obtain a more comprehensive understanding.

Table 2.14

Harsh and overreactive parenting

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Marceau et al., 2015	EGDS/US	361	9 months- 6 years	Adoption	Fathers' overreactive parenting was environmentally associated with higher child internalising problems, both directly and indirectly through elevated morning cortisol levels in the child. Overreactive parenting increased cortisol levels, which in turn heightened the child's internalising problems.
Marceau et al., 2013	EGDS/US	561	18 moths	Adoption	There was an environmental association between over-reactive parenting and internalising problems.
Ma et al., 2022	KCYPS/China	2,180	8-13 years	FE	There was an environmental association between physical punishment and physical abuse and higher levels of depression in children.

Asbury et al.,2003	TEDS/UK	4,706	4 years	MZ twin	An environmental correlation was found between harsh discipline and anxiety.
Asbury et al., 2006	TEDS/UK	4,034	7 years	MZ twin	There was no environmental effect of harsh discipline on child anxiety.

EGDS= Early Growth and Development Study; US= United States; KCYPS= The Korean Children and Youth Panel Survey; FE = Fixed Effects; TEDS= Twins Early Development Study; UK= United Kingdom; MZ = Monozygotic.

5.3 Over- controlling Parenting

Table 2.15 provides a summary of three studies with RI-CLPM (Ching et al., 2021; Nelemans & Keijsers, 2020) and sibling comparison (Dunn et al., 1990) designs investigating the relationship between over- controlling parenting and child internalising problems. The results of these studies are inconsistent. Nelemans and Keijsers (2020) found child-driven effects rather than mother-driven effects, with mothers changing their behaviour in response to their child's symptoms. At the within-individual level parental psychological control did not affect adolescent anxiety symptoms. Parents reported a decrease in their own controlling attitudes during periods of increased social anxiety symptoms in adolescents.

On the other hand, both Dunn et al. (1990) and Ching et al. (2021) found environmental effects associated with over-controlling parenting. Ching et al. (2021) identified reciprocal relationships between perceived maternal achievement-oriented psychological control and children's mathematics anxiety at the within-family level. Additionally, they found that academic contingent self-esteem mediated those relationships. However, for both studies the identified within-person cross-lagged effects were small. Thus, Ching et al. (2020) suggested the results should not be over-interpreted and that the cross-lagged effects, with 2 years between measurement, may not be able to capture overtime effects that happen intensively such as weekly, monthly.

The majority of the studies, two out of three, highlighted an environmental effect of over-controlling parenting. However, given the limited number of studies and methodological limitations, such as limited measurement frequency (e.g., Ching et al., 2021), further research is needed to provide a more comprehensive understanding of this relationship.

Table 2.15

Over- controlling parenting

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Nelemans & Keijsers, 2019	STRATEGIES/ Belgium	819	Mean 13.4 years	RI-CLPM	Only child-driven effect found between maternal controlling parenting and child anxiety symptoms.
Ching et al., 2021	China	336	13-17 years	RI-CLPM	A reciprocal, environmental relationship was found between maternal achievement oriented psychological control and children's mathematics anxiety through academic contingent self-esteem at the within-individual level.
Dunn et al., 1990	CAP/ US	67 sibling dyads	4-7 years	Sibling	Greater maternal control was associated with child internalising problems environmentally.

STRATEGIES: Studying Transactions in Adolescence: Testing Genes in Interaction with Environments; RI-CLPM = Random Intercept Cross-lagged Panel Model; CAP = Colorado Adoption Project.

5.4 Family Functioning

Table 2.16 summarises 6 studies investigating the effect of family functioning on child internalising problems. Studies including RI-CLPM (Lougheed et al., 2020; Mastrotheodoros et al., 2019) and MZ twin difference designs (Guimond et al., 2016) suggest that there was no environmental relation between family functioning and child internalising problems. Conversely, studies using the CoT design (Ahmadzadeh et al., 2022; Hannigan et al., 2017; Schermerhorn et al., 2011) have found an environmental association between family functioning, including parent- offspring and parent-parent relationships, and child internalising problems.

While both studies using RI-CLPM found a significant relationship between family functioning and child internalising problems at the between-individual level, this relationship was no longer significant after random intercepts were included in the model. One explanation for the lack of association is that the timescale those studies used to collect the data may not be appropriate to capture dynamic association between family functioning and child internalising, thus shorter time scales (e.g, weeks) may be needed (Lougheed et al., 2020; Mastrotheodoros et al., 2019). In addition, Guimond et al (2016) found only child-driven effect was observed in genetically sensitive analysis capitalising on the MZ twin difference design.

Based on these findings, it can be concluded that the majority of studies-four out of seven-did not find an environmental influence on family functioning. In order to gain a deeper understanding of this risk factor, it would be beneficial to conduct further research that addresses the limitations of previous studies, including timescales that may not adequately capture dynamic associations.

Table 2.16

Family functioning

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Ahmadzadeh et al., 2022	TOSS/ Sweden	1,030 twins	Mean 15.75 years	CoT	Parental criticism was associated with adolescent internalising problems environmentally.
Hannigan et al., 2017	TCHAD and TOSS/ Sweden	1120 twin 909 twins	Mean 13.67 years; Mean 15.75 years	CoT	Parent-offspring relationship quality environmentally influenced child internalising.
Schermerhorn et al., 2011	TOSS/ Sweden	867 twins	Mean 15.75 years	CoT	Family conflict was associated with child internalising problems solely through environmental factors according to parental reports. When assessed using child reports, a genetic effect was also identified.
Guimond et al., 2016	Quebec Newborn Twin Study/ Canada	326 twins	13 -14 years	MZ twin	Relation between maternal support and child depression was environmental, but child-driven rather than parent-driven.

Lougheed et al., 2020	SECCYD/ US	1,364	8-13 years	RI-CLPM	There was no association between mother-child conflict and child internalising problems at the within-family level.
Mastrotheodoros et al., 2019	Greece	480	Mean 15.73 years	RI-CLPM	There was no relationship between family functioning and adolescent internalising problems at the within-family level.

TOSS= Twin and Offspring Study in Sweden; CoT= Children of Twin; TCHAD = Twin Study of Child and Adolescent Development; MZ = Monozygotic; SECCYD = The Study of Early Child Care and Youth Development; US= United States; RI-CLPM = Random Intercept Cross Lagged Panel Model

6. Peer Bullying

Table 2.17 summarizes nine studies that investigated the impact of exposure to bullying on children's internalising problems. The majority of these studies employed twin difference (Arseneault et al., 2008; Bowes et al., 2010; Silberg et al., 2016; Singham et al., 2017; Vitaro et al., 2011) and within-individual study designs (Babaee et al., 2021; Davis et al., 2019; Davis et al., 2018; Li et al., 2021). These studies consistently found that peer victimization had a significant environmental impact on children's internalising problems, along with evidence of some genetic influences in one study (Silberg et al., 2016).

Silberg et al. (2016) reported that an environmental effect of bullying on social and separation anxiety from the comparison of bullied and non-bullied MZ twins. They also found that genetic factors contributed to both bullying victimization and social anxiety. Additionally, Bowes et al. (2010) found a moderation effect such that twins with warmer maternal relationships tended to experience fewer emotional problems when confronted with bullying.

These studies mentioned in this section covered a range of age groups, including mid and late childhood (Arseneault et al., 2008; Babaee et al., 2021; Li et al., 2021; Vitaro et al., 2011), early adolescence (Bowes et al., 2010; Davis et al., 2019), mid-adolescence (Davis et al., 2018; Singham et al., 2017), and late adolescence (Silberg et al., 2016). This suggests that the environmental effects of peer victimization persist across different stages of development.

Furthermore, Singham et al. (2017) and Vitaro et al. (2011) stand out for their use of multiple informants and multiple scales to measure anxiety and depression, enhancing the robustness of their results. These comprehensive approaches contribute to a stronger understanding of the impact of peer victimization on children's mental health.

A significant environmental factor had consistently been found in all studies investigating the effect of peer bullying on children's internalising problems. Some studies utilized multiple informants, which enhanced the robustness of their findings. Additionally, this risk factor was studied across a range of age groups, emphasizing its significance across the entire development span, from childhood through adolescence.

Table 2.17

Peer bullying

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Davis et al., 2018	US	1,875	11-13 and 13-15 years	ALT-SR	Adolescents reporting higher peer victimization was environmentally related to higher depression symptoms.
Davis et al., 2019	US	2,177	11-15 years	ALT-SR	Both peer victimization and adolescent depression environmentally affected each other bidirectionally at the within-individual level.
Babaee et al., 2021	CASPIAN/Iran	16,064	6-10 years	FE	Being bullied was associated with both anxiety and depression environmentally.
Arseneault et al., 2008	E- Risk/ UK	1,116 twin pairs	5 years	MZ twin	Being bullied was associated with child internalising problems environmentally.
Bowes et al., 2010	E- Risk/ UK	1,116 twins	10-12 years	MZ twin	Those with warmer maternal relationships showed fewer emotional problems when bullied.
Silberg et al., 2016	VTSABD/ US	2824 twins	8-17 years	MZ twin	Being bullied was associated with social and separation anxiety both genetically and environmentally.
Singham et al., 2017	TEDS/ UK	11, 108 twins	11-16 years	MZ/DZ twin	Bullied twin showed more concurrent anxiety and depression compared to non-exposed.
Vitaro et al., 2011	QNTS/Canada	446 twins	Mean 6.7- 7.5 years	MZ twin	Twin differences in perceived peer victimization were associated with depressive symptoms environmentally.

Li et al., 2021	China	4,273	Mean 9.90 years	RI-CLPM	There was an environmental association between peer victimization and depressive symptoms at the within-family level.

US= United States; ALT-SR= Autoregressive Latent Trajectory Model with Structured Residuals; CASPIAN= Childhood & adolescence surveillance and prevention of adult non-communicable disease; FE= Fixed effect; E- Risk = Environmental Risk Longitudinal Twin Study; MZ= Monozygotic; VTSABD = Virginia Twin Study of Adolescent Behavioural Development; TEDS= Twins Early Development Study; DZ= Dizygotic; QNTS= the Quebec New-born Twin Study; UK= United Kingdom; RI-CLPM= Random Intercept Cross Lagged Panel Model.

7. Extras

Table 2.18 presents 11 studies featuring environmental risk factors that do not align with any of the risk factors mentioned in the results above. These risk factors include parental education, having tardy classmates, ability-based grouping in a class, stressors associated with schoolwork, having anxious friends, multiple relocations during childhood, being placed in out-of-home care, breastfeeding, having fathers working evening and night shifts, high-noise environments, and teacher-child relationship quality. Except for the risk factor of high noise (Rudolph et al., 2019) and parental education (Torvik et al., 2020), the results consistently demonstrated an environmental correlation with child internalising problems.

There was a significant effect on internalising problems in the majority of studies. It would not be appropriate, however, to draw a general conclusion regarding the impact of each risk factor since each study examined different risk factors.

Table 2.18

Extras

Author	Study name/Country	Sample size	Age	Study designs	Summary of Results
Torvik et al., 2020	MoBa/ Norway	34,958	8 years	CoT	Parental education and child depression symptoms were explained by shared familial risk factors. There was no evidence of an environmental correlation.
Gottfried, 2014	ECLS-K/ US	21,765	5 years	FE	The presence of late classmates was found to be environmentally associated with an increase in internalising problems.
McDool,2019	MCS/ UK	27,512	7- 11 years	FE	There was an environmental association between the ability-based grouping in math class and increased internalising problems, particularly among boys.
Eley & Stevenson, 2000	UK	795	8-16 years	MZ and DZ	Stressors associated with schoolwork had an environmental influence on internalising problems.

Poirier et al., 2015	Canada	521	Mean 10.04 years	MZ	There was an environmental association between child anxiety and having anxious friends.
Anderson & Leventhal, 2016	NICHD/ US	1,056	12–15 years	PSM	An environmental relationship was found between multiple relocations during childhood and adolescence and internalising problems.
Averdijk et al., 2018	z-proso/ Sweden	1,675	13-17 years	PSM	There was an environmental association between placed in out-of-home care and internalising problems.
Girard & Farkas, 2019	ELPI/ Chile	1282	58 month- 7 years	PSM	There was an environmental relation between breastfeeding during the first 6 months, and even up to 12 months, and a reduction in emotional problems in children.

Han, 2017	China	2282	6 years	PSM	An environmental relation was found between children whose fathers work evening and night shifts and internalising problems.
Rudolph et al., 2019	US	4,508	13-18 years	PSM	No environmental evidence was found between high-noise and emotional problems.
Maldonado-Carren˜o & Votruba-Drzal, 2011	NICHD/ US	1,364	9-11 years	Within-person	Increasing relationship quality between teacher and child was associated with child internalising problems, environmentally.

Note: MoBa= Norwegian Mother, Father and Child Cohort Study; CoT= Children of Twins; ECLS-K = Early Childhood Longitudinal Studies Kindergarten; US = United States; FE = Fixed effect; MCS = Millennium Cohort Study; UK= United Kingdom; MZ = Monozygotic twins; DZ= Dizygotic twins; NICHD = National Institute of Child; PSM = Propensity score matching; z-proso= Zurich Project on the Social Development from Childhood into Adulthood; ELPI = Encuesta Longitudinal de la Primera Infancia cohort. This table primarily includes risk factors associated with child internalising problems. However, protective factors are also included where their reverse form may reflect a risk factor. This dual consideration helps provide a comprehensive view of the factors' potential impacts.

2.4 Conclusion

This study reviewed the literature that tests environmental risk factors for child internalising problems. In total, this review encompasses 98 articles, covering 6 broad categories of environmental risk factors, including 17 subtypes, as well as additional studies that do not fall into any of these categories.

The most extensively studied category of risk factors is parental mental health problems, including parental anxiety, depression, and other mental health issues, which was addressed in 30 studies. Following closely is "parenting and family functioning", the second most studied category, with a total of 16 studies. In contrast, the least studied category of the risk factors is "parental substance misuse, psychotropic medications" with 9 studies. Among the subtypes within each category, parental depression, in particular, is the most frequently studied subtype, addressed in 16 studies. Conversely, the least studied subtype of environmental risk factor, except for the studies under the "extras" part, is divorce (which is included in the category of stressful life events), with only 2 studies.

There was evidence that many of the risk factors studied had causal effects on internalising problems in children and adolescents. For example, out of 16 studies addressing parental depression 15 of them identified environmental effects in internalising problems (see Gjerde et al., 2019; Harold et al., 2011; Lewis et al., 2011). Similarly, out of 7 studies on parental anxiety, 4 of them were associated with environmental factors (see Ahmadzadeh et al., 2019; Brooker et al., 2014; Eley et al., 2015; Gjerde et al.,2020). However, prenatal maternal anxiety was the only risk factor for which no environmental influence was found; instead, there was evidence of familial and genetic transmission (Bekkhus et al., 2018; Gjerde et al.,2020; Rice et al., 2010). In addition, studies within the SLE category, which investigated parental loss, divorce, maltreatment and general SLE, consistently highlighted the presence of

environmental effects, except for one study examining parental bereavement (see Williams & Amber, 2015).

Likewise, within the social disadvantages category, 4 out of 5 studies addressing neighbourhood risk (Fauth et al., 2007; Leventhal & Brooks-Gunn, 2013; Leventhal & Brooks-Gunn, 2011; Rudolph et al., 2013) and 4 out of 6 studies addressing family income (Mistry et al., 2009; Gennetian & Miller, 2002; Strohschein, 2005; Zachrisson & Dearing, 2015) reported the presence of environmental effects. In the parenting category, 4 out of 5 studies on harsh parenting (Asbury et al.,2003; Ma et al., 2022; Marceau et al., 2015; Marceau et al., 2013) and 2 out of 3 studies on controlling parenting (Ching et al., 2021; Dunn et al., 1990) identified environmental influences. Additionally, 7 out of 8 studies on peer bullying found evidence of environmental influences. The exception was Silberg et al. (2016) who found both genetic and environmental effect together. Moreover, 2 out of 3 studies on the parental alcohol use risk factor also indicated the presence of environmental effects (Lund et al., 2019; McCrory & Layte, 2012; Sen & Swaminathan, 2007).

Conversely, there was evidence that the relationship between internalising problems and many risk factors is not predominantly environmental. For instance, among the studies on the parental rejection risk factor, four out of eight did not find environmental effects, while the remaining four did. Similarly, in the family functioning risk factor, only three out of six studies reported environmental effects.

Some risk factors have been examined within only a limited developmental span or exclusively during the prenatal period. For instance, parental alcohol use (Knudsen et al., 2015; Lund et al., 2019) and smoking were only assessed during toddlerhood (McCrory & Layte, 2012; Sen & Swaminathan, 2007). In addition, parental smoking has only been investigated for its prenatal effects (McCrory & Layte, 2012; Sen & Swaminathan, 2007),

even though various observational studies (that did not meet the inclusion criteria of this review) have observed the effect of postnatal smoking on children's emotional problems (Chastang et al., 2015). This limited observation makes it challenging to form a comprehensive understanding of the effects of risk factors during child development.

On the other hand, for all other risk factors, there are studies that have explored broader age ranges. Depression, for example, is a risk factor that has been investigated across a wide age range, spanning from childhood to adolescence (Griffith et al., 2021; Lewis et al., 2011; Liskola et al., 2018; Silberg et al., 2010; Yan et al., 2021). However, the evidence based regarding risk factors other than depression contains a limited number of causally informative studies, often over specific age range. Therefore, reaching definitive conclusions about the impact of these risk factors at any specific age period can be challenging. It underscores the need for more causally informative research covering a wider developmental span, which can help us comprehensively examine the effects of these risk factors.

Furthermore, it's worth noting that studies should take into account potential measurement bias. Many studies have predominantly depended on a single reporter, often the mother (e.g., Brandlistuen et al., 2015; Sen & Swaminathan, 2007; Silberg et al., 2012). Nevertheless, the objectivity of mothers as reporters of children's behavioural problems is the subject of debate (Sen & Swaminathan, 2007). For instance, depressed mothers may exhibit a "cognitive bias" that affects their judgments, including their assessments of their child's emotions and behaviours (Najman et al., 2001). Depressed mothers may tend to perceive their children as more problematic compared to non-depressed mothers, as suggested by Goodman et al. (2011). Hails et al. (2019) found the link between parental depression and child internalising problems was evident only when relying on parental reports of symptoms, particularly from adoptive fathers. This association was not observed when considering teacher-reported results. This discrepancy raises questions about the impact of reporter bias on the outcomes

of these studies. Given these concerns, it is crucial for researchers to incorporate the perspectives of multiple reporters whenever possible.

Almost half of the studies included in this review, totalling 44, were based on samples from the United States. Other countries have either been significantly less researched or not studied at all. This indicates that adopting a multicultural perspective is essential for achieving a more comprehensive understanding on causally related environmental risk factors.

Additionally, the majority of studies examining the effects of risk factors on child internalising problems neglected to examine moderation, with a few exceptions (e.g., Bowes et al., 2010; Dearing et al., 2006; Humphrey & Root, 2017; Jenness et al., 2019; Leventhal & Brooks-Gunn, 2011; Marceau et al., 2015; Nook et al., 2021; Rudolph et al., 2013; Strohschein, 2005). Similarly, the exploration of mediation effects was also lacking, with only a few studies addressing this aspect (e.g., Ching et al., 2021; Laurent et al., 2013a; Rodman et al., 2021; Vidal Bustamante et al., 2020), highlighting the need for more comprehensive investigations on the risk factors. Exploring these aspects can contribute to a deeper understanding of the relationships, providing valuable insights for more effective prevention and treatment strategies. Notably, even the most studied risk factor, parental depression, has limited research on its moderating effects, with only one study (Laurent et al., 2013b) examining the moderating effect of HPA activity between parental depression and child internalising problems. Future studies should consider mediating and moderating effects on the relationship between risk factors and child mental health, with causally informative methods. Thus, the aim of the next chapter is to build a better understanding of the relation between those risk factors (mainly parental depression) and child mental health problems, by examining mediation and moderation processes.

Finally, while the limitations of the included studies have been discussed, it is also important to address the quality assessment process. Given the diverse range of study designs included in this review, such as twin, sibling, adoption, within-individual, and in vitro fertilization studies, no single comprehensive checklist was available to assess the quality of all these studies. As a result, rigorous inclusion criteria were applied, and the methodological rigor of each study was critically evaluated without relying on a formal checklist. Despite the fact that this approach ensured a robust evaluation, the absence of a standardized tool for quality appraisal is a limitation. In the future, the development of a more adaptable quality appraisal tool in order to accommodate various study designs may be beneficial to future research.

To sum up, this review has highlighted the diverse environmental risk factors that influence child internalising problems and identified challenges within the studies reviewed. The next chapter will delve deeper into understanding how these environmental risk factors, especially parental depression- the most extensively studied factor-relate to child internalising problems by examining mediation and moderation processes, providing valuable insights into these complex relationships.

Chapter 3. The relationship between family Income, child mental health problems and parental distress: A within-family examination of the family stress model

3.1 Introduction

The preceding chapter provided a systematic review of the environmental risk factors investigated in the context of child internalising problems. The study highlighted that many risk factors have a causal impact on children environmentally. Among these risk factors, parental mental health problems emerged as prominent factor influencing the child's internalising problems.

Given the significant burden of mental health problems in children (Odgers et al., 2008), a thorough understanding of the risk factors that contribute to these problems is important for developing comprehensive preventive and therapeutic interventions. As demonstrated in the systematic review (Chapter 2) and discussed in Chapter 1, gaining a deeper understanding of parental mental health problems together with causally informative studies, would serve a valuable purpose in advancing this overarching goal.

The current chapter aims to provide a deeper understanding of the relationship between parental mental health problems and internalising problems. It should be noted that, in line with this objective, externalising problems were also considered. Externalising problems show a high co-occurrence with internalising problems (Pesenti-Gritti et al., 2008). Inclusion of externalising problems facilitates a comparative analysis and identifies factors specific to internalising problems (Pesenti-Gritti et al., 2008).

In addition to the evidence showing a close relationship between child and parental mental health, many studies also highlight that family income is a significant risk factor for both parent and child mental health. Numerous studies have found that decreasing family income leads to increase in child and parental mental health problems (e.g., Zachrisson & Dearing,

2015). Thus, family income as a risk factor associated with both child and parental mental health problems may help us understand the relationship between child and parental mental health.

It may be possible to gain some deep understanding into the relationship between mental health problems in parents and children by using the family stress model (FSM) (Conger et al., 2010). FSM provides a framework that incorporates parental and child mental health while considering family income as a closely related risk factor. This helps to gain a deep understanding of the relationship between mental health problems in parents and children.

The FSM aims to explain the relationship between family economic hardships and child psychopathology, by modelling parental mental health as a mediating factor. According to the FSM, parents who are experiencing economic hardship face greater economic pressure, which in turn contributes to greater emotional distress (e.g., parental depression, anxiety). The increased level of parental distress leads to conflict within the family, including harsh and cold parenting, leading to youth mental health problems - both internalising (e.g., depression) and externalising (e.g., aggression) (Masarik & Conger, 2017). The FSM is supported by a range of evidence (e.g., Conger et al., 2010; Conger & Conger, 2002; Kiernan et al., 2008). For example, Kiernan et al. (2008) examined children at 3 and 5 years of age using data from the Millennium Cohort Study. In the study, economic deprivation was associated with children's emotional and behavioural problems and the relationships were partly explained by maternal depression symptoms as well as parenting factors, such as discipline methods adopted by mothers. In a longitudinal study conducted by Rijlaarsdam et al. (2013), where 2,139 children were assessed from the prenatal period until they reached the age of 3, it was discovered that maternal depressive symptoms, parenting stress, and harsh

discipline acted as mediators of the effect of economic disadvantage on child mental health problems.

Despite the extensive body of research examining the FSM, many studies have been based on cross-sectional designs (e.g., Conger et al., 2002; Scaramella et al., 2008). As discussed in Chapter 1, cross-sectional studies measure constructs at the same time point, so they are unable to inform on temporal ordering and are subject to confounding effects (Piotrowska et al., 2022; Shelleby et al., 2014). Because of the limitations in establishing the direction of the effect, it is impossible to determine whether decreased family income is a result or a cause of child mental health problems (Shelleby et al., 2014). It has been demonstrated that children with mental health problems can affect family income by negatively affecting parents' working skills, as caring for these children is more demanding for parents, which indicates the existence of reverse causality (e.g., Coley et al., 2011; Powers, 2001). In addition, some studies show that mental health problems in children also adversely affect parental mental health, indicating the presence of reverse causality in relationships between parent and child mental health problems (e.g., Piotrowska et al., 2022; Speyer et al. 2022). That's why, as an alternative to cross-sectional studies, longitudinal studies can be used, especially those using a cross-lagged approach, since they offer the opportunity to investigate the direction and stability of the effects (Preacher, 2015). The only study to date employing the cross-lagged panel model (CLPM) approach to examine FSM is that of Piotrowska et al. (2022), which reported no mediational effect of parental distress on externalising problems, while internalising problems were not studied.

However, it is important to acknowledge that the cross-lagged approach also has limitations when it comes to establishing robust causal inferences, as mentioned in Chapter 1. This is because a CLPM does not distinguish within-individual and between-individual effects from each other, leading to ambiguous results regarding the within- family dynamics. Since

developmental theory predominantly focuses on within-individual change to understand child mental health problems (Berry & Willoughby, 2017; Hamaker et al., 2015), a statistical model that effectively disentangles within-individual effects from between-individual effects would provide more robust results compared to traditional CLPM. As mentioned in the chapter 1, Random intercept cross-lagged panel model (RI-CLPM) extend CLPM for this purpose (Hamaker et al. 2015; Mund & Nestler, 2019; Usami et al. 2019). The present study applies a RI-CLPM to test the FSM model in the Millennium Cohort Study with children aged between ages 3 and 14.

To the best of our knowledge and as observed in the studies examined in the systematic review, the causal relationship between parental mental health and child mental health within the framework of FSM has not been previously explored using a causally informative method. The current study represents the first investigation of the mediating role of parental distress in the association between family income and child internalising and externalising problems, that focuses on within-family dynamics, with a causally informative study design, RI-CLPM.

In this study models are fitted separately for boys and girls as it is possible that relationships between child and parental mental health differ by child gender (e.g., Speyer et al., 2022; Xerxa et al., 2021). As the first study to explore within-individual fluctuations in FSM and considering the limited research on parent-child mental health relations at the within-family level, we refrain from formulating specific hypotheses regarding child gender or any differences between internalising and externalising mental health problems.

In addition, children in the present study were examined through a wide developmental period spanning three to fourteen years of age. Although it has been found that the effect of decreased family income was more evident in older children than in younger children (Case

et al., 2002; Duncan et al., 1998), there are some convincing studies suggesting that the effect of family income was visible even as a child was growing up (Rijlaarsdam et al., 2013; Votruba-Drzal, 2006). Based on the Millennium Cohort Study (MCS) data, for instance, Kiernan and Huerta (2008) found that children's well-being at age 3 was negatively affected by low income in the family. Considering that studies provide mixed results regarding the age groups of children who are affected by family income, we refrain from formulating a specific hypothesis in this regard as well. Thus, we hypothesised that within-family change in family income and child mental health problems would affect each other reciprocally, in addition within- family change in parental distress would mediate the relation between them.

3.2 Method

Participants

The study utilized data from the MCS, a nationally representative UK-based birth cohort study that followed approximately 19,000 children born between September 2000 and January 2002 (www.cls.ioe.ac.uk/mcs). Data began to be collected at the age of 9 months and there were further collections when the children were three, five, eleven, fourteen, and seventeen years of age. A stratified sampling method was employed for selecting the sample, in which disadvantaged families and ethnic minorities were oversampled (Joshi & Fitzsimons, 2016). The study also provides sampling weights, stratification and clustering variables to account for oversampling, attrition, and non-random dropouts. The study included all children who participated up to age 14 (N= 11,845, female= 5,907, male=5,938). Informed written consent was obtained from parents of the cohort children during the data collection, and the NHS Multi-Centre Ethics Committees approved the data collection (Shepherd & Gilbert, 2019). The current study was also approved as a secondary data analysis research project by the Department of Psychology Research Ethics Committee at the University of Sheffield.

Measurements

Child Mental Health Problems: The parent reported Strengths and Difficulties Questionnaire (SDQ) was used to measure child internalising and externalising problems (Goodman & Goodman, 2009) with data collected from age of 3 to 14 years old children. The SDQ is a 25-item questionnaire including 5 subscales called conduct disorders (e.g., generally obedient), prosocial behaviour (e.g., considerate of other feelings), hyperactivity (e.g., restless, overreactive) and emotional problems (e.g., often complains of headaches). Each subscale is composed of five items measured with response options ranging from 'not true' to 'somewhat true' and 'definitely true'. For internalising problems, the score is calculated based on the sum of emotional and peer problems, whereas for externalising difficulties, the score is calculated based on the sum of behaviour and hyperactivity problems. The result of this is a score of 0-20, with a higher score indicating more difficulties. It has been demonstrated that the factor structure of the SDQ is invariant longitudinally and across gender (Murray et al., 2022), as well as across ethnicity and socioeconomic status (Toseeb et al., 2022).

Parent Psychological Distress: Parental distress was measured with the 6-item, Kessler 6 (K6) Scale at 3, 5, 7, 11 and 14 years (Kessler et al., 2002). Within the past 30 days, both parents were asked how often they felt "so depressed that nothing could lift you up", "nervous, hopeless, restless, or fidgety", "everything was an effort" and "worthless." These items were measured on a 5-point Likert scale with options ranging from "all the time" to "none of the time". In this study, all items have been reversed and rescaled from 0 to 4. Psychological distress scores were calculated by summing the item scores to provide a total score range 0-24, higher score indicates higher parental distress. The Kessler scale was found to have good internal consistency and reliability in the MCS (Flouri et al., 2019).

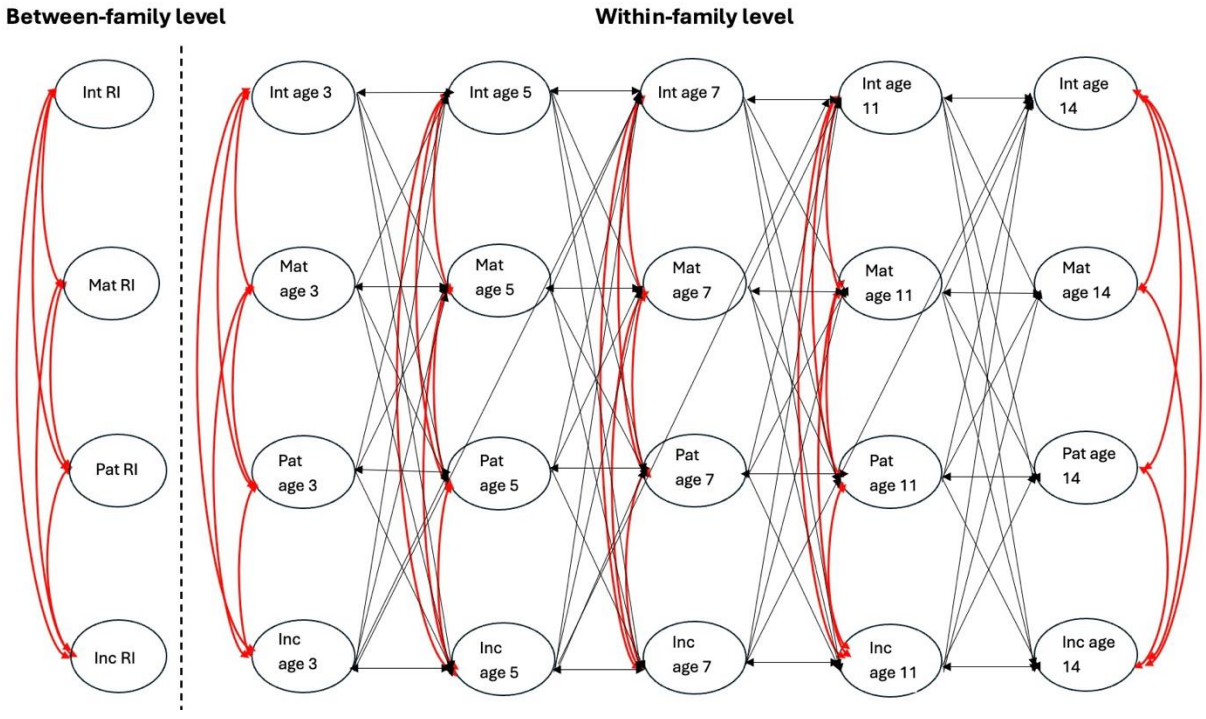
Family income: A predefined income band list was used to ask respondents about their family's total income. A continuous measure of income was imputed by using interval regression techniques (Stewart, 1983). By applying the OECD household equivalence scale (OECD, 2009), which adjusts the measure of household income for size and composition, the MCS team converted these imputed incomes into OECD equivalised income.

Statistical Analysis

To investigate the within-person effects of family income on child mental health problems via parental distress, random intercept cross-lagged panel (RI-CLPM) models were fitted. Separate models were fitted regarding child internalising and externalising problems. We illustrate the approach with regards to internalising below; the same modelling approach was used for externalising. Through the inclusion of random intercepts for each repeatedly measured variable, RI-CLPM disaggregates within- and between-person effects. The models allow covariance between the random intercepts, testing the association of the between-participants variance in the measured variables. To understand the within-individual dynamics, we also incorporated cross-lagged and autoregressive residual effects in addition to residual covariance within time points (for further explanation see Hamaker et al., 2015). To investigate longitudinal mediation, we incorporated second-order cross-lagged effects, which consider the influence of variables from two preceding time points, in examining the path from family income to child internalising problems through parental distress (Lüdtke & Robitzsch, 2021). Second order cross-lagged effects were included from family income at age 3 to internalising problems at age 11; in addition, from family income at age 7 to internalising problems at age 14 and from family income at age 11 to internalising problems at age 14. Models were tested separately for males and females (Moffitt et al., 2001; Murray et al., 2019). Bootstrapped 95% confidence intervals were used to assess the statistical significance

of indirect effects. Our modelling approach was based on Mulder and Hamaker's (2021) suggestions (see: jeroendmulder.github.io/RI-CLPM).

Models were fitted in Mplus 8.8(Muthén et al., 2017) with the robust maximum likelihood estimator (MLR), so that missing data was addressed using full information maximum likelihood estimation, which yields unbiased estimates assuming that the data are missing at random (Enders, 2001). To account for the complex sampling design of the MCS and non-random dropout, all models included stratification and clustering variables as well as attrition weights. Tucker Lewis Index (TLI) and Comparative Fit Index (CFI) >.90 and Root Mean Squared Error of Approximation (RMSEA) <.05 (Kline, 2005) were used as cut-off values to indicate good model fit. Figure 3.1 shows schematic representation of the model for internalising problems.



Note: RI= Random intercept, Int = Internalising, Mat= Maternal psychological distress, Pat= Paternal psychological distress, Inc= Family income.

3.3 Results

Descriptive Statistics

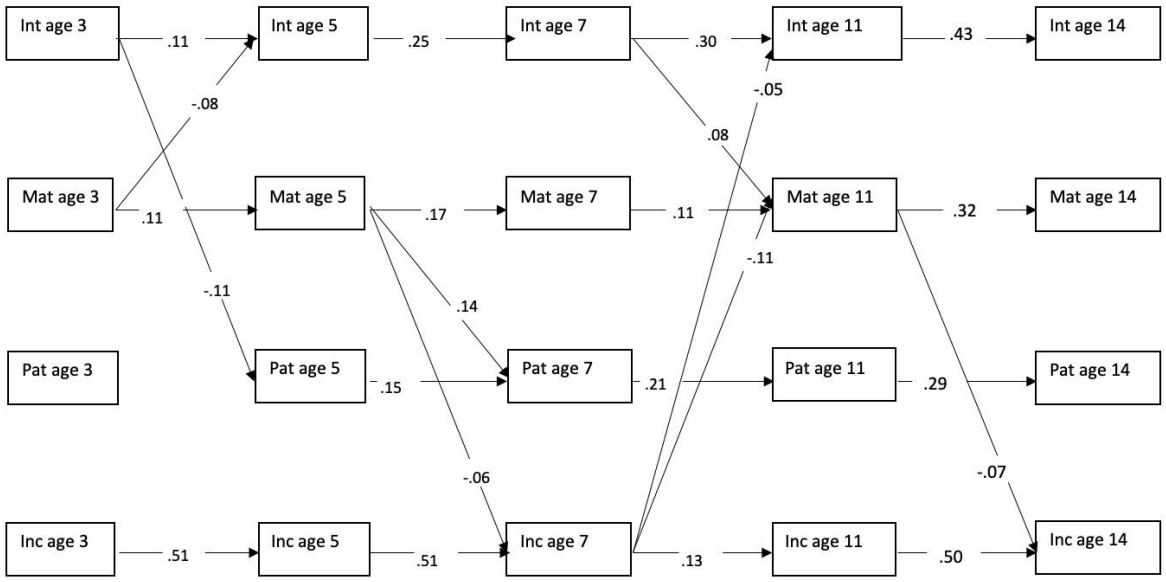
Table B1 provides descriptive statistics (see Appendix B).

The effect of income on internalising via parental distress in girls

The RI-CLPM model provided excellent fit (RMSEA = .024, CFI = .987 and TLI=.971). In Figure 3.2 standardized autoregressive and cross-lagged parameters are summarized. Higher child internalising problems at ages 3 predicted decrease in paternal distress at age 5. In addition, child internalising problems at age 7 was associated with increase in maternal distress at age 11. In terms of the parent to child effect, there was no effect of paternal distress on child internalising but increased maternal distress at age 3 predicted decreased internalising problems at age 5. Maternal distress at age 5 and 11 was associated with a decrease in family income at ages 7 and 14, respectively. Additionally, lower family income at age 7 predicted higher internalising problems and maternal distress at age 11. Table B2, in Appendix B, shows the parameters in full.

In examining the indirect effects of family income on child internalising problems through parental distress, the results indicated that the indirect effect was non-significant for ages 3 to 7 (β = 0.00, 95% CI= -0.00, 0.00, p = 0.86), ages 5 to 11 (β = 0.00, 95% CI= -0.00, 0.00, p = 0.96), and ages 7 to 14 (β = 0.00, 95% CI= -0.01, 0.00, p = 0.26) (see Appendix B, Table B6).

Figure 3.2 *Standardized autoregressive and cross- lagged parameters for the relation between family income and internalising via parental distress, for girls*



Note. Paths are shown only if they are statistically significant. Random intercept and covariance parameter have been omitted for clarity. Int = Internalising, Mat= Maternal psychological distress, Pat= Paternal psychological distress, Inc= Family income.

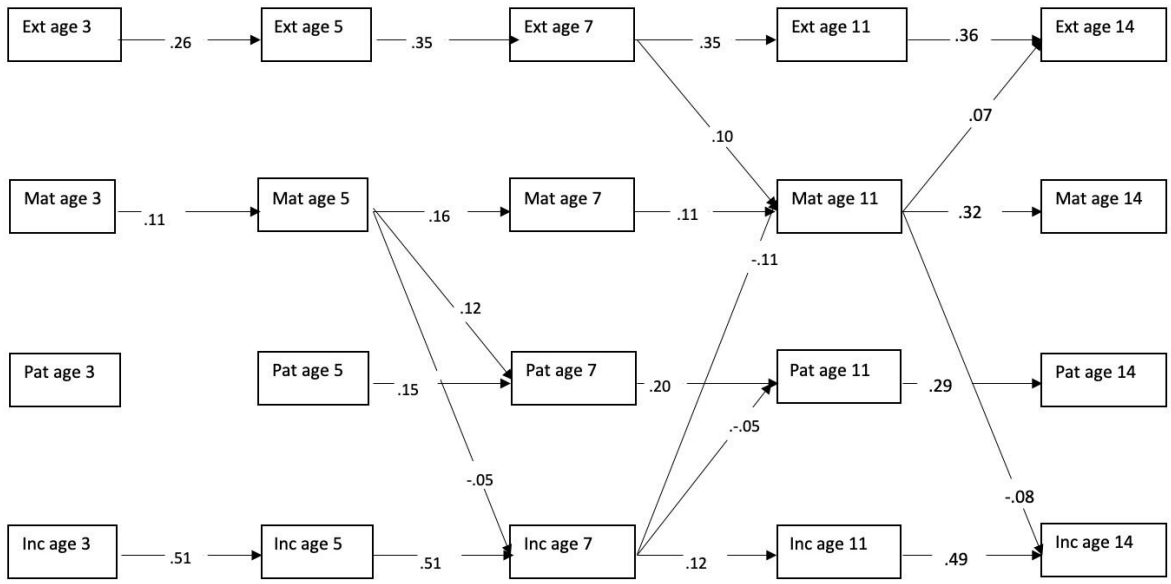
The effect of income on externalising via parental distress in girls

Model fit indices for the RI- CLPM showed good fit according to RMSEA = .023, CFI = .989 and TLI = .974. In Figure 3.3, significant standardized autoregressive and cross-lagged parameters are presented. None of the cross-lagged paths were significant between ages 3 and 5. The only significant effect of a child variable on a parent was found between externalising problems at age 7 and maternal distress at age 11. Furthermore, the only relationship between parental distress and externalising problems occurred between the ages of 11 and 14; higher maternal distress at age 11 resulted in lower higher externalisation problems at age 14. Higher maternal distress between the ages of 5 and 11 was associated with lower family income between the ages of 7 and 14, respectively. In addition, a decrease in family income

at age 7 led to increases in maternal and paternal distress at age 11. Table B3 shows the parameters in full (see Appendix B).

Analyses of indirect effects indicated that maternal distress at age 11 mediated the relation between family income at age 7 and girl's externalising problems at age 14 (β = 0.00, 95% CI= -0.01, 0.00, p < 0.05). On the other hand, indirect effects were non-significant for ages 3 to 7 (β = 0.00, 95% CI= -0.00, 0.00, p= 0.87), and ages 5 to 11 (β = 0.00, 95% CI= -0.00, 0.01, p= 0.42) (See Table B7 for the indirect effect, in Appendix B).

Figure 3.3 *Standardized autoregressive and cross- lagged parameters for the relation between family income and externalising via parental distress, for girls*



Note. Paths are shown only if they are statistically significant. Random intercept and covariance parameter have been omitted for clarity. Ext = Externalising, Mat= Maternal psychological distress, Pat= Paternal psychological distress, Inc= Family income.

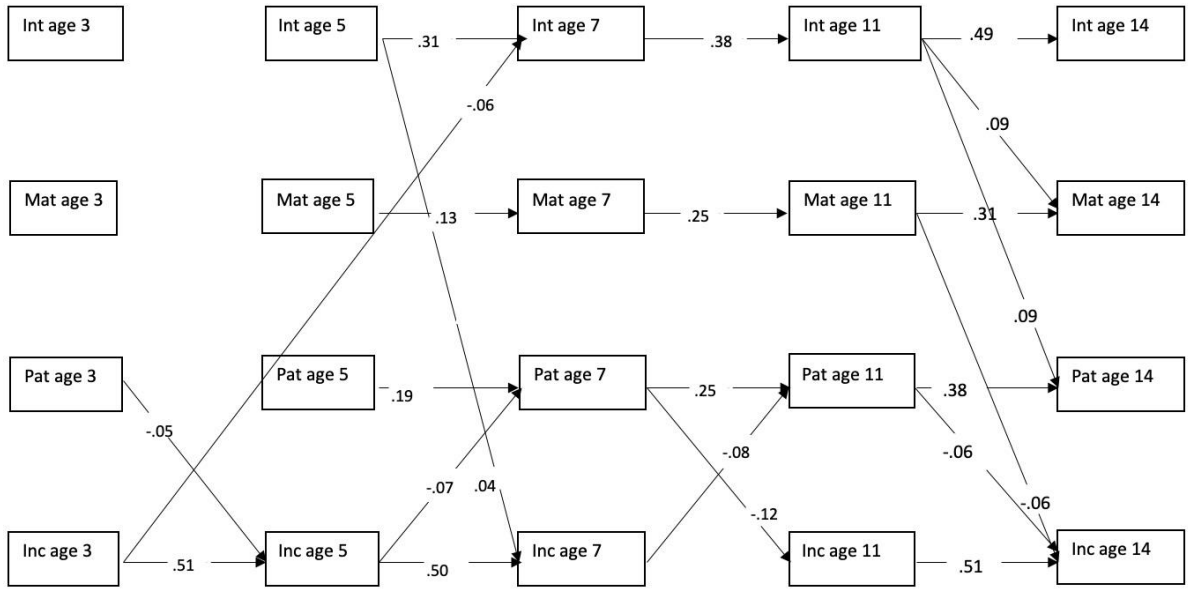
The effect of income on internalising via parental distress in boys

The model fitted well regards to RMSEA =.021, CFI = .990 and TLI = .977. Figure 3.4 shows significant standardized autoregressive and cross-lagged parameters. There were no

significant cross- lagged effects of maternal and paternal distress on internalising problems. On the other hand, internalising problems at age 11 was associated with increasing maternal and paternal distress at age 14. There was no effect of internalising on income, but family income at age 3 had a negative cross-lag effect on internalising at age 7, indicating that decreasing family income at age 3 was associated with higher internalising problems at age 7. Decrease in family income at age 5 and 7 was associated with increase in paternal distress at age 11 and 14 respectively. In addition, increasing paternal distress at age 7 and 11 was associated with a decrease in family income at ages 11 and 14, respectively. Table B4 shows the parameters in full (see Appendix B).

Regards to the indirect effect, results showed that internalising problems were not indirectly affected by family income for ages 3 to 7 (β = 0.00, 95% CI= -0.00, 0.00, p = 0.74), ages 5 to 11 (β = -0.00, 95% CI= -0.01, 0.00, p = 0.37), and ages 7 to 14 (β = -0.00, 95% CI= -0.01, 0.01, p = 0.67) (see Table B8, in Appendix B).

Figure 3.4 *Standardized autoregressive and cross- lagged parameters for the relation between family income and internalising via parental distress, for boys*



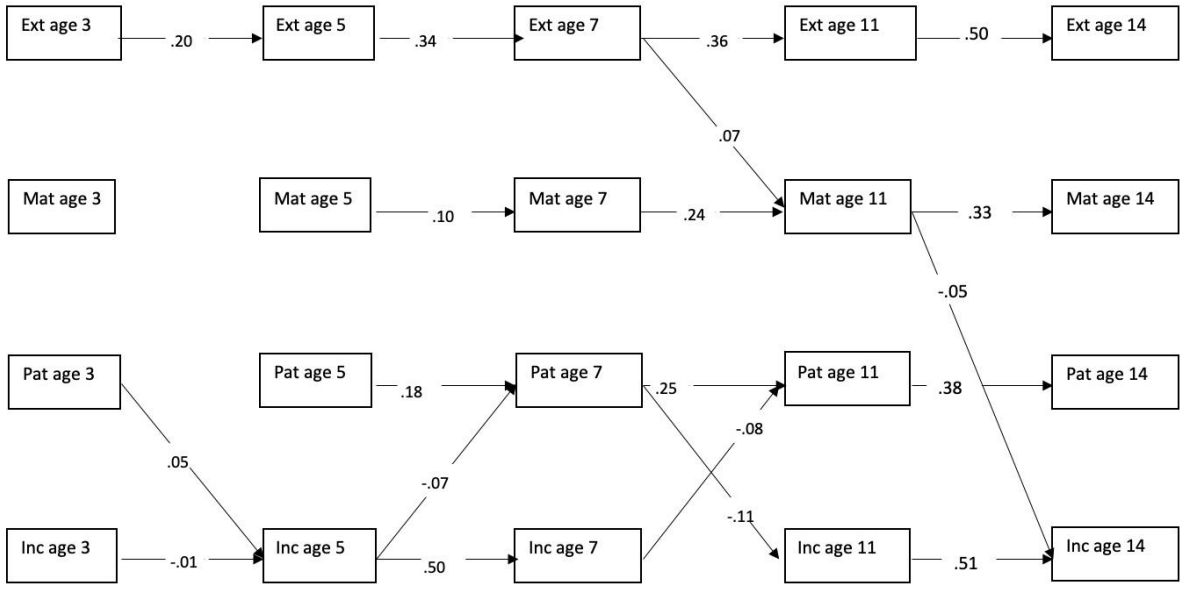
Note. Paths are shown only if they are statistically significant. Random intercept and covariance parameter have been omitted for clarity. Int = Internalising, Mat= Maternal psychological distress, Pat= Paternal psychological distress, Inc= Family income.

The effect of income on externalising via parental distress in boys

In terms of RMSEA = .021, CFI = .991 and TLI =.980, the RI-CLPM's model fit indices were good. See Figure 3.5 for significant cross-lagged effects, and Table B5 shows the parameters in full (see Appendix B). Similar to the results for internalising problems, externalising problems were not affected by either maternal or paternal distress. Considering the child-to-parent effect, maternal depression at age 11 was exacerbated by externalising at age 7. Regarding family income, a negative cross-lagged relationship emerged between family income levels at ages 5 and 7 and paternal distress at ages 7 and 11, respectively. This indicates that a decrease in family income was associated with an increase in paternal distress. In addition, increased externalising problems at age 7 resulted with increased income at age 11.

Analysis of indirect effects found that family income did not affect externalising problems indirectly for ages 3 to 7 ($\beta = 0.00$, 95% CI= -0.00, 0.01, $p = 0.44$), ages 5 to 11 ($\beta = -0.00$, 95% CI= -0.01, 0.00, $p = 0.42$), and ages 7 to 14 ($\beta = -0.00$, 95% CI= -0.01, 0.00, $p = 0.46$). Table B9 shows indirect effects (see Appendix B).

Figure 3.5 *Standardized autoregressive and cross- lagged parameters for the relation between family income and externalising via parental distress, for boys*



Note. Paths are shown only if they are statistically significant. Random intercept and covariance parameter have been omitted for clarity. Ext = Externalising, Mat= Maternal psychological distress, Pat= Paternal psychological distress, Inc= Family income.

3.4 Discussion

The study aim was to investigate the relationship between parent and child mental health using the FSM framework in children aged 3 to 14 years. We hypothesized that family income and child mental health problems would affect each other reciprocally and that parental distress would mediate this relationship. We found some evidence supporting both direct relationships between family income and child mental health problems and indirect relationships via maternal distress (e.g., Coley et al., 2011; Martin et al., 2010; Neppl et al., 2015). However, those relations varied according to the child's developmental stage, gender, and by mental health outcome (internalising vs externalising).

For girls, the direct relationship between income and internalising is evident in the association between income at age 7 and internalising at age 11. Furthermore, despite no

direct relation between income and externalisation, an indirect effect supporting FSM was observed between income at age 7 and externalisation at age 14, which was mediated by maternal distress at age 11. Table 3.1 gives a summary of significant associations between income and internalising problems for girl's subsample.

Table 3.1. Standardised autoregressive and cross-lagged parameters from RICLMP for female's internalising outcome

Parameter	***Estimate***	***SE***	***P***
Age 11 int on age 7 inc	-0.045	0.021	**0.034***

Notes: int, internalising problems; inc, family income; Correlations are significant at $p < .05$. Children's ages are in accordance with their respective median ages at the time of data collection.

In boys, the effect of internalising on income was identified between internalising at age 5 and income at 7, with a positive correlation between them. The effect of income on internalising was observed between income at age 3 and internalising at age 7, with a negative correlation between them. Nevertheless, no relationship was found between income and externalising problems. Additionally, in contrast to girls, there was no mediation effect of parental distress observed in boys. However, in line with our hypothesis, we found evidence of reciprocal effects between boys' internalising problems and family income. This is evident in the relationships found between family income problems at age 3 and internalising problems at age 7, as well as between internalising problems at age 5 and family income at age 7. Tables 3.2 show a summary of significant associations between income and internalising problems for the boys' subsample.

Table 3.2. Standardised autoregressive and cross-lagged parameters from RICLMP for male's internalising outcome

Parameter	***Estimate***	***SE***	***P***
Age 7 int on age 3 inc	-0.056	0.021	**0.008***
Age 7 inc on age 5 int	0.039	0.019	**0.040***

Notes: int, internalising problems; inc, family income; Correlations are significant at $p < .05$. Children's ages are in

accordance with their respective median ages at the time of data collection.

Results of our study contradict previous research which has suggested a potentially stronger relation between family income and externalising problems (Costello et al., 2003; Piotrowska et al., 2015) compared to internalising problems in children (Dearing et al., 2006; Duncan & Brooks-Gunn, 1994). Contrary to previous findings, there was no evidence of a direct relationship between income and externalising problems at any developmental stage for children of any gender. Dearing et al. (2006), for example, explored how variations in family income influenced externalising and internalising problems in children and concluded that family income had a stronger association with externalising problems. However, they pointed out that the reporters in their study were childcare providers and teachers, who might encounter difficulties in identifying internalising problems compared to externalising problems when compared to parents (e.g., Stanger & Lewis, 1993). Thus, the discrepancy between the current study's results and those of Dearing et al. (2006) may be linked to the methodology employed. The current study primarily relied on parent reports, especially from mothers, to describe children's mental health problems. This choice could contribute to differences in observed outcomes compared to studies employing outside observers like teachers.

Additionally, the current findings indicate an increase in child internalising problems corresponding to a decrease in family income. This aligns with the findings of Coley et al. (2015), who reported that higher child distress is associated with a decrease in maternal employment, which, in turn, leads to a decrease in family income.

The current study extends and confirm those from previous studies that have examined the FSM, particularly in relation to family income and child externalising problems, as maternal distress was found to mediate these relationships in our study. For instance, Parke et al.

(2004) reported that economic pressure was related to increasing child adjustment problems via parental distress, couple conflict and harsh parenting. In addition, Gershoff et al. (2007) and Yeung et al. (2002) found similar results for the relationship between economic adversity and child behavioural outcomes which was mediated through parental distress and poor parenting.

On the other hand, the results of the current study do not align with those of Piotrowska et al. (2022), who did not find that parental distress mediated the relationship between family income and conduct problems. This discrepancy between the findings reported here and those by Piotrowska et al. (2022) needs further consideration. One possible explanation is that, in the current study, the RI-CLPM has been applied, while Piotrowska et al. (2022) used the traditional CLPM. The primary distinction between these models lies in the fact that the RI-CLPM allows for the differentiation between- and within-individual differences, enabling the estimation of pure within-person autoregressive and cross-lagged effects, as opposed to the CLPM (Mund & Nestler, 2018). This methodological difference may account for the variations observed between the current study and Piotrowska et al. (2022). Another explanation is that Piotrowska et al. (2022) investigated only conduct problems, while the current study examined externalising problems more broadly, incorporating both conduct problems and symptoms of attention deficit and hyperactivity disorder. Thus, the discrepancy in our results regarding externalising problems could be attributed to differences in how externalising problems were assessed.

The results of the current study are also inconsistent with some other studies investigating the FSM. Kiernan and Huerta (2008) examined the FSM using structural equation modelling (SEM) for children aged 9 months to 3 years from MCS datasets. Results of their study supported the FSM for both internalising and externalising problems, compared to the current study which only supported the FSM for externalising problems. However, it should be

noted that the study design they used (i.e., SEM), was not able to differentiate within- and between-person effects from each other, which may explain the difference between the current study and Kiernan and Huerta (2008). Furthermore, in their study, Kiernan and Huerta (2008) concurrently assessed maternal depression and family income at 9 months. Yet, it is acknowledged that mediational processes unfold gradually over time (Maxwell et al., 2011). The absence of control for previous evaluations of the mediator and the outcome variable introduces bias into the results (Judd & Kenny, 1981), which may further explain the difference between the current study and Kiernan and Huerta (2008).

In another study by Conger et al. (1994), who conducted a three-year study on adolescents, it was found that economic pressure was linked to parental conflicts, which, in turn, contributed to harsh parenting. The potential reasons for the disparity between our study and Conger et al. (1994) might be that Conger et al. (1994) investigated mediation pathways from family income to child mental health by taking into account a broader array of mediating factors, including parental depressed mood, marital conflict, and harsh parenting. Therefore, it is possible that there are other pathways that could help explain the impact of income on internalising problems, which we did not investigate here.

In addition to all discussed, some relationships in the study require further explanation as they are counterintuitive. These unexpected findings include increased internalising problems at age 5 leading to higher family income at age 7 (boys); increased internalising problems at age 3 reducing paternal distress at age 5 (girls); and increased maternal distress at age 3 lowering internalising problems at age 5.

One possible explanation for the positive correlation between increased internalising problems at age 5 and increased income at age 7 is that children with internalising problems are less outwardly disruptive than those with externalising problems and so less visible

(Aguilar-Yamuza et al., 2023). Therefore, parents may perceive these matters to be less pressing or demanding, allowing them to devote more time and energy to activities that contribute to their income. This shift in parental attention could lead to an increase in family income as parents prioritize financial stability over immediate intervention in their child's less overtly problematic behaviours.

Regarding the counterintuitive results between increased internalising problems at age 3 and decreased paternal distress at age 5, one possible explanation is that, in many families, mothers are the primary caregivers during early childhood, especially in the formative years (ages 3-5) (Brooks-Gunn et al., 2014). When the mother assumes a larger role in managing the child's internalising problems, the father may experience less direct stress related to these issues.

Lastly, to explain the counterintuitive result where increased maternal distress at age 3 is associated with reduced child internalizing problems at age 5, an underlying reason could be that distressed mothers seek help from their partners to step in and take on a more active caregiving role (Fisher & Glangeaud-Freudenthal, 2023; Katayama et al., 2022). By redistributing caregiving responsibilities, the child could benefit from a more balanced and supportive environment, thereby reducing their mental health problems.

In addition to these counterintuitive results, it's important to note that in all our models, the coefficients between family income at ages 7 and 11 are either very small or non-existent. This could be partly due to the economic instability caused by the global financial crisis of 2007 (Barrel & Davis, 2008), which led to significant fluctuations in family income during the period when data was collected at age 7. Such economic disruptions may have impacted income stability, affecting the observed correlation between family income at different ages.

Strengths and limitations

In accordance with the FSM, the results of our study affirm the significance of income in predicting child mental health, although its impact may vary depending on age of children and the specific mental health problem. The present study is the first investigation of within-family dynamics between family income and child mental health problems based on the FSM. One of the strengths of the study is the investigation of the reciprocal relationship between household income and child mental health problems while considering parent distress as a mediating factor, in a large, nationally representative sample. Especially, examination of this relationship using statistical models that disaggregate between- and within-person effects is helpful in providing a deeper understanding of the causal relationships in the family income, parental distress and child mental health triangle (Hamaker et al., 2015). Furthermore, examining children from early childhood to late adolescence across a broad span of their developmental process provides a comprehensive perspective for testing this theory, setting it apart from many longitudinal studies that focus on brief periods of developmental span (e.g., Kiernan et al., 2008).

It is also important to note that the study has several limitations. Marital conflict and parenting quality, which are other components of the FSM, were not included in the current study. In the FSM, economic hardships result in parental distress, which is connected to marital dissatisfaction. In turn, marital dissatisfaction is associated with poor parenting, ultimately impacting children's mental health (Conger et al., 1994). Therefore, adding other paths related to this model may provide us with a deeper understanding of how internalisation problems occur that could not be explained by the mediation effect of parental distress. However, other paths are out of scope of this study, as we aim to expand our understanding of the pathways through which parental distress contributes to child mental health problems in this study.

This study faced data attrition as a limitation (Speyer, 2021). To manage missing data, FIML was employed, which assumes that data are missing at random (Enders, 2001). In addition, attrition weights provided by the MCS were used to adjust for non-random dropout, helping to ensure that underrepresented groups are more accurately reflected in the analysis. This approach reduces bias and improves the dataset's accuracy and representativeness (Schermelleh-Engel et al., 2003).

In addition, it is important to highlight the potential contribution of common method variance. This arises from the fact that children's problems were primarily evaluated by their mothers, who might be experiencing distress and, as a result, could tend to overemphasize their child's behavioural and emotional difficulties (Kroes et al., 2003). However, considering that the study covers a lengthy developmental period of children's, at least two years, the potential influence of parental mental health problems on the reporting of child mental health is likely to be less confounded in subsequent reports (Speyer et al., 2022).

Nonetheless, despite these limitations, this study holds significant implications. When considering the impact of income on children, the importance of poverty-reduction policies becomes evident. Implementing policies aimed at reducing child poverty through public interventions would undoubtedly contribute to substantial enhancements in children's health, development, and overall well-being (e.g., Costello et al., 2003). Furthermore, when considering the mediating effect of maternal distress in this relationship, interventions aimed at reducing maternal mental health problems could potentially mitigate the influence of family income on children (Leventhal & Brooks-Gunn, 2003).

Conclusion and future directions

The current study reported that both family income and child mental health problems influence each other. In addition, maternal distress mediated this relation, for externalising

problems in girls. Although the results support the FSM regarding child externalising and help us to understand how parental distress can be associated with child externalising problems, internalising problems was not explained through this model. Therefore, in future studies, it may be worthwhile to explore the relationship between parental and child mental health problems from other theoretical perspectives, which may help to explain internalising problems more fully. Thus, the next study, reported in Chapter 4, examined the relationship between parental distress and child mental health problems, considering family income as a moderator.

Chapter 4: Within-person cross-lagged relationships between children and parent's mental health: Does poverty play a moderating role?

4.1 Introduction

In the previous chapter (Chapter 3), we explored the relationship between parental and child mental health problems in the light of the Family Stress Model (FSM). However, contrary to our expectations, the results only demonstrated a significant mediating effect of parental distress on child externalising problems, and not on internalising problems, which is the primary focus of this thesis. Hence, this chapter was developed with the aim of further exploring alternative possibilities regarding the relationship between parent and child mental health.

A moderating influence of poverty on the effects of other risks on psychopathology may also contribute to understanding the association between parent and child mental health problems. McLoyd's (1990) Context of Stress model may offer insights into the complex dynamics of poverty and its impact on mental health problems. The model suggests that stressful life conditions can exhaust emotional, social, and financial resources, making it more likely that additional risk factors will become overwhelming (McLoyd et al., 1990). Thus, poverty, as a stressful life condition, heightens individuals' vulnerability to chronic and discrete stressors by diminishing coping capacity and limiting access to social support (see e.g., Chai et al., 2022; McLoyd et al., 1994; Wadsworth et al., 2005).

A number of studies showed that the burden of familial risk stressors varies according to family income (see e.g., Chai & Schieman, 2022; Dearing et al., 2006; Dearing et al., 2001). For instance, Chai and Schieman (2022) investigated the effect of parents struggling to balance their parenting roles due to work-family conflict on the health of their children. They found children living in lower income households had more health problems as a result of

work-family conflict associated with problematic parenting practices than children living in families with higher incomes (Chai et al., 2022).

Parental depression, as a well-documented risk factor for child internalising problems in Chapter 2, can also be understood through the lens of the Context of Stress model. In addition, due to the reciprocal nature of parent-child relations, child mental health problems can also function as stressors for parents (see e.g., Xerxa et al., 2021; Tyrell et al., 2019; Sameroff et al., 1975). Based on the Context of Stress model (McLoyd et al., 1990), it might be expected that parents living in poverty may have fewer emotional resources to cope with the emotional pressure of their children's mental health difficulties, leaving them more vulnerable to mental health problems. Likewise, as a result of poverty, children may be more vulnerable to the effect of their parents' mental health problems, which may strengthen the association between parental mental health problems and child internalising/externalising problems (Ryan et al., 2015). Goodman et al. (2011) conducted a meta-analysis of 193 studies examining the strength of the association between maternal depression and children's internalising/externalising problems. This study found that the relationship between parent and child mental health problems was stronger in studies focussing on low-income households that those focussing on middle, high or mixed-income households.

The majority of studies investigating the relationship between SES and child and parent mental health face the methodological challenges discussed in previous sections. These challenges typically involve the use of cross-sectional designs, and traditional Cross-Lagged Panel Models which fail to distinguish variance within families from variance between families (see e.g., Mennen et al., 2018). As mentioned in the previous chapters, failing to account for these distinct sources of variance may result in misleading patterns of effect, especially when processes differ within and between families (Hamaker et al., 2015). If research is focused on understanding the dynamics of people rather than populations, then

examining associations “within families” offers a more robust test of causality (Keijsers et al., 2015).

There are convincing studies using within-family designs and testing the prospective reciprocal relations between parent and child mental health problems (see e.g., Speyer et al., 2022; Xerxa et al., 2021; Yan et al., 2021). Speyer et al. (2022) tested the within-family relations between parental mental health problems (distress) and child mental health problems, using autoregressive latent trajectory modelling with structured residuals (ALT-SR), in children aged 9 months to 17 years from the Millennium Cohort Study (MCS). There was a reciprocal relationship between parent and child mental health problems. However, to date, no study using a within-family design to examine child and parent mental health relation has tested moderation of these relations by family income.

The present study addresses this gap. We built on the ALT-SR approach, following Speyer et al. (2022), testing whether the strength of reciprocal links between parental distress and child psychopathology varies as a function of poverty. In line with the Context of Stress model (McLoyd, 1990), we hypothesise that between and within-family reciprocal relations between parental distress and child mental health will be stronger for families in poverty than families with higher income levels. In addition, as some differences in paths have been reported between boys and girls in the association between parental and child mental health (see e.g., Speyer et al., 2022; Xerxa et al., 2021) models were fitted separately for both genders. However, considering the limited research available on parent-child mental health relations at the within-family level, we refrain from formulating any specific hypotheses regarding the gender of the child (Speyer et al., 2022).

4.2 Methods

Sample

The study utilized the same dataset, MCS, as in Chapter 3 (www.cls.ioe.ac.uk/mcs). In the current study we focus on seven data collection sweeps which were conducted when the children were aged 9 months, 3, 5, 7, 11, 14 and 17 years. Our study included all children who participated up to the age of 17 whose families reported the poverty status of the household at the 9 months contact (N=10309; females = 5161; males = 5148). All sweeps received ethical approval from the National Health Service Research Ethics Committee and parents provided informed consent. Additionally, the Department of Psychology Research Ethics Committee at the University of Sheffield approved the current study as a secondary data analysis research project.

Measures

Child Emotional and Behavioural Problems: The SDQ questionnaire, which was used to measure child internalising and externalising problems in Chapter 3, was employed again for this study (Goodman et al., 1997).

Parent Psychological Distress: In this study, the Kessler (K6) Scale (Kessler et al., 2002), which was used in Chapter 3 to measure parental distress, was used again.

Family poverty: The MCS uses banded response questions to measure household income. Interval regression techniques have been used to impute a continuous measure of income (Stewart, 1983). These imputed incomes are equivalised based on the OECD household equivalence scale (OECD, 2009), which adjusts the income measure for the household size and composition. Families were identified by the MCS team as being below the poverty threshold using a binary variable when their equivalised net family income was less than 60%

of national median household income when the child was 9 months old (see e.g., Fitzsimons et al., 2017)

Statistical Analysis

To examine the between and within-family processes related to maternal, paternal, and child mental health in the context of being in poverty and non-poverty, an ALT-SR multigroup analysis was conducted for boys and girls separately. The ALT-SR model combines a latent growth curve model and a cross-lagged panel model (Bollen & Zimmer, 2010). The growth curve part of the model captures between-family differences in child and parent mental health problems. The cross-lagged part is fitted to the growth curve's residuals and as such captures within-family dynamics. Specifically, an individual's "within-person" deviation from their own typical trajectory can be observed through analysing these residuals (Mund et al., 2019; Curran et al., 2014).

In all models, stratification variables, clustering variables, and attrition weights were employed to address the complex sampling design and account for non-random dropouts. Models were fitted in Mplus 8.8 (Muthén et al., 2017) using the robust maximum likelihood estimator (MLR) which utilizes full information maximum likelihood (FIML) to account for missing data, assuming that data is missing at random (Enders, 2001). The proportion of missing data for child internalising problems in the analytic sample was 12.4%, 8.4%, 10.2%, 9.2%, 11.3%, and 12.6% across sweeps 2 to 7, respectively. For child externalising problems, the percentages were the same as those for internalising problems, except for sweep 7, where the missing data was 12.5%. Regarding maternal distress, the percentage of missing data was 20.4%, 12%, 13.7%, 13.4%, 17.3%, and 31.3% across sweeps 2 to 7, respectively. Finally, there were 38.2%, 35.0%, 39.6%, 41.9%, 47.6%, and 57.2% of missing data for paternal distress across sweeps 2 to 7 respectively.

The Tucker Lewis Index (TLI) and Comparative Fit Index (CFI) >.90 and Root Mean Squared Error of Approximation (RMSEA) <.05 were used as cut-off values to indicate acceptable model fit (Kline et al., 2023). To evaluate competing models in our large sample, we adhered to Cheung and Rensvold's (2002) recommendation (Cheung et al., 2002) to accept the more parsimonious model unless the CFI shows an increase of 0.01 or more in a more complex model. Mplus codes and full model results are available at https://osf.io/kmwzf/?view_only=99297c92f33a4d108c835ba38ef7b686.

An unconstrained model was initially fitted in which parameter estimates were allowed to vary between the poverty and non-poverty groups. To test whether the between-family component of the model differed by poverty status, the unconstrained model was then compared with a constrained model, wherein intercept covariances were constrained across poverty groups. The model with superior fit was selected to test whether the within-family relationships differed across poverty. This involved comparing a model where the within-families components (both autoregressive and cross-lagged parameters) were fixed equal across poverty groups to one in which they were unconstrained.

As a sensitivity analysis to check that results were consistent across a different definition of poverty, an additional model was tested in which poverty was re-classified as chronic or non-chronic rather than only on the basis of poverty status at 9 months. Chronic poverty was defined as meeting the poverty threshold at each of the age 9 months, 3- and 5-years assessments whereas others were coded as non-chronically poor.

4.3 Results

Girls subsample between-family associations

A fully unconstrained model in which parameter estimates were allowed to vary across the poverty (N =1651) and non-poverty (N =3510) groups fitted the data well ($\chi 2$ =1558.192, p

< .001, CFI = .962, TLI = .933, and RMSEA = .039). A second model that constrained the intercept covariances to be equal across the poverty groups also fit the data well ($\chi 2$ = 1594.362, p < .001, CFI =.961, TLI = .932, and RMSEA =.039). There was no substantial loss of fit in the constrained model (ΔCFI =.001), indicating that poverty did not moderate the between-family associations of child and parent mental health. Full results for the intercept covariances for both groups are displayed in Table 4.1.

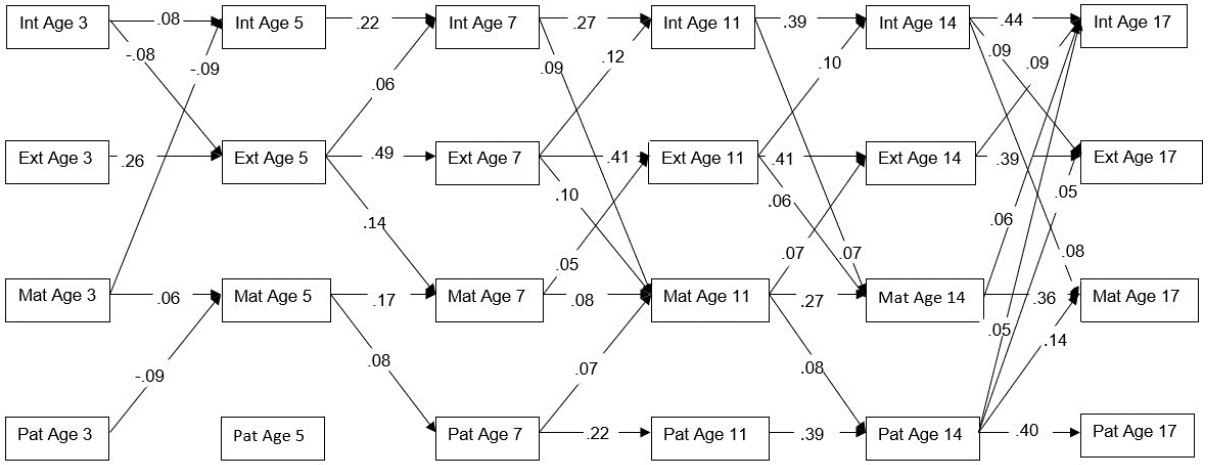
Table 4.1 *Intercept covariances for girls in the poverty and non-poverty groups*

	Non-poverty			**Poverty**		
	Est.	***SE***	***p***	***Est.***	***SE***	***p***
Internalising WITH						
Externalising	0.531	0.031	<.001*	0.647	0.040	<.001*
Maternal distress	0.474	0.026	<.001*	0.575	0.036	<.001*
Paternal distress	0.192	0.030	<.001*	0.294	0.064	<.001*
Externalising WITH						
Maternal distress	0.329	0.027	<.001*	0.381	0.040	<.001*
Paternal distress	0.099	0.028	<.001*	0.213	0.066	0.001
Maternal Distress WITH						
Paternal distress	0.243	0.027	<.001*	0.344	0.058	<.001*

Girls subsample within-family associations

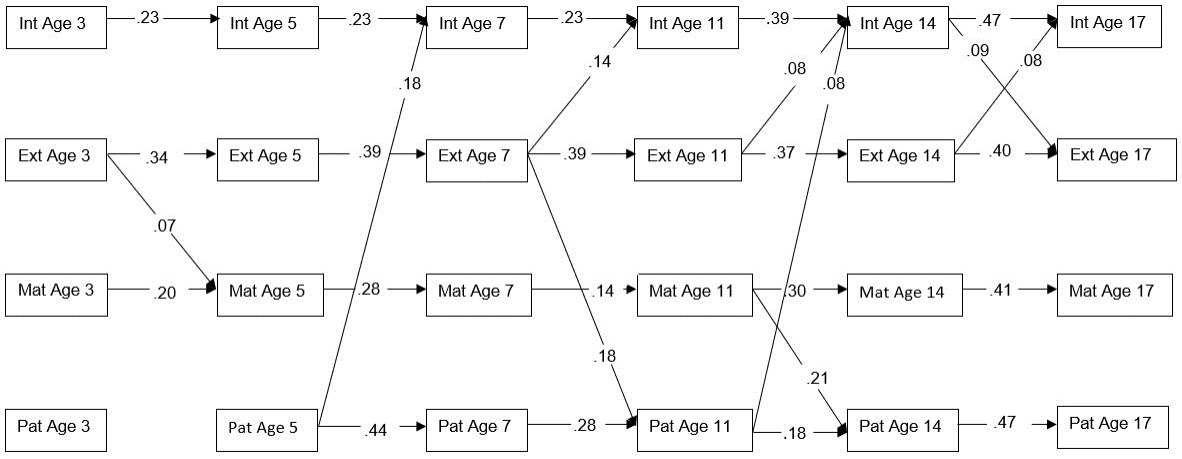
The within-family associations were examined in the model fixing the between-family intercept covariances equal across poverty groups. Standardized significant autoregressive and cross-lag parameters in poverty and non-poverty groups are displayed in Figure 4.1 and 4.2, respectively.

Figure 4.1 *Multi-group ALT-SRs with* unconstrained *autoregressive and cross-lagged paths for girls, non-poverty group*



Note. Int = internalising, Ext = externalising, Mat = maternal distress, Pat = paternal distress. Only paths with statistically significant results ($p < .05$) are presented. Children's ages are in accordance with their respective median ages at the time of data collection.

Figure 4.2 *Multi-group ALT-SRs with* unconstrained *autoregressive and cross-lagged paths for girls, poverty group.*



Note. Int = internalising, Ext = externalising, Mat = maternal distress, Pat = paternal distress. Children's ages correspond to their median ages at the time of data collection. Statistically significant paths ($p <. 05$) are presented only.

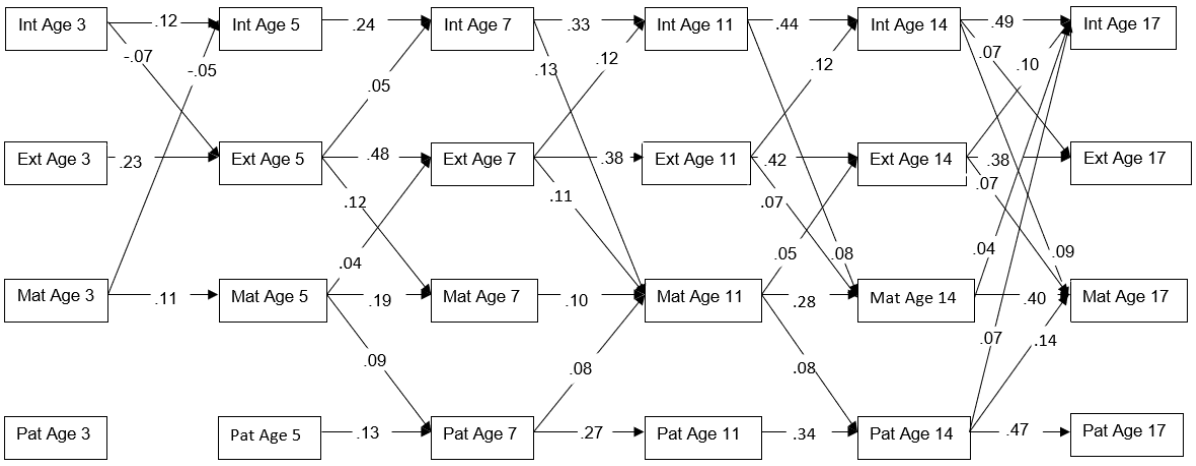
In the non-poverty group, the only positive cross-lagged effect of maternal distress on child internalising problems was observed between the ages of 14 and 17 with higher maternal distress related to higher internalising problems. The cross-lagged effect between maternal distress at age 3 and child internalising problems at age 5 was, however, negative, indicating that maternal distress was related to a decrease in internalising problems. In addition, there was an association between paternal psychological distress at the age of 14 and increase in internalising problems at the age of 17. Regarding externalising problems, children aged 11 and 14 were consistently affected by maternal distress experienced at ages 7 and 11, respectively. In addition, paternal distress at age 14 was associated with an increase in externalising problems at age 17. Regarding the effect of child mental health on parental distress, within-family cross-lagged effects of internalising problems on maternal distress were found from the ages of 7 to 17. In addition, there was a positive cross-lagged relationship between externalising problems at ages 5, 7 and 11 and within-family changes in maternal distress at ages 7, 11 and 14. In contrast, paternal distress was not affected by children's mental health problems.

In the poverty group, an effect from parent to child, particularly where paternal distress at ages 5 and 11 was associated with an increase in the child's internalising problems at ages 7 and 14 was observed, respectively. Additionally, a child-to-parent effect was also observed, indicating that the child's externalising behaviour at age 3 effected an increase in maternal distress at age 5. Furthermore, the child's externalising behaviour at age 7 was associated with an increase in paternal distress at age 11.

A model which additionally constrained the autoregressive and cross-lagged paths to be equal in the poverty and non-poverty groups also fit the data well ($\chi 2 = 1687.049$, $p < .001$, CFI = .960, TLI =. 945, and RMSEA =. 035). Comparison of the fit indices of those models (ΔCFI = .001) shows that poverty did not moderate the within-family associations between

child and parent mental health measures. Significant autoregressive and cross-lagged parameters from the constrained model are presented in Fig 4.3. Full results with confidence intervals are showing in Tables C4-C8, Appendix C.

Figure 4.3 *Multi-group ALT-SRs with unstandardized constrained autoregressive and cross-lagged paths for girls*



Note. Int = internalising, Ext = externalising, Mat = maternal distress, Pat = paternal distress. Children's ages correspond to their median ages at the time of data collection. Statistically significant paths ($p < .05$) are presented only.

Boys subsample between-family associations

A fully unconstrained model, allowing parameter estimates to vary between poverty (N =1636) and non-poverty (N =3512) groups, demonstrated a good fit to the data ($\chi 2$ =1530.148, $p < .001$, CFI =. 965, TLI =.938, and RMSEA =.039). A second model, where the intercept covariances were constrained to be equal across the poverty groups, also showed a good fit to the data ($\chi 2$ =1544.276, $p < .001$, CFI =.965, TLI =.939, and RMSEA =.039). CFI did not differ between those models, indicating that poverty did not significantly moderate the relation between parent and child mental health between families. Full results for the intercept covariances for both groups are displayed in the table 4.2.

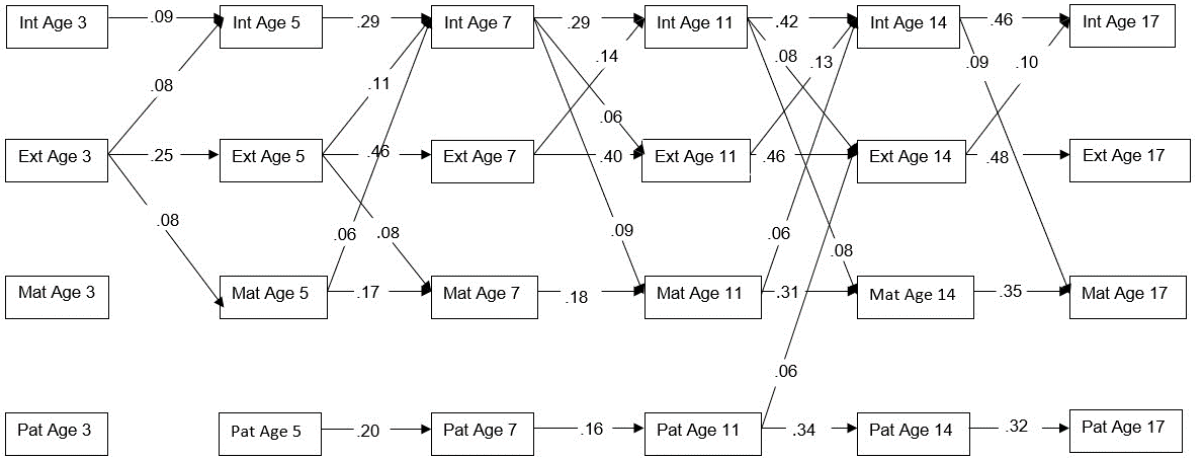
Table 4.2 *Intercept covariances for boys in the non-poverty and poverty groups*

	Non-poverty			**Poverty**		
	Est.	***SE***	***p***	***Est.***	***SE***	***p***
Internalising WITH						
Externalising	0.526	0.027	<.001*	0.555	0.040	<.001*
Maternal distress	0.482	0.026	<.001*	0.515	0.045	<.001*
Paternal distress	0.224	0.033	<.001*	0.247	0.084	.003*
Externalising WITH						
Maternal distress	0.371	0.024	<.001*	0.334	0.041	<.001*
Paternal distress	0.176	0.030	<.001*	0.253	0.063	<.001*
Maternal Distress WITH						
Paternal distress	0.209	0.028	<.001*	0.273	0.067	<.001*

Boys subsample within-family associations

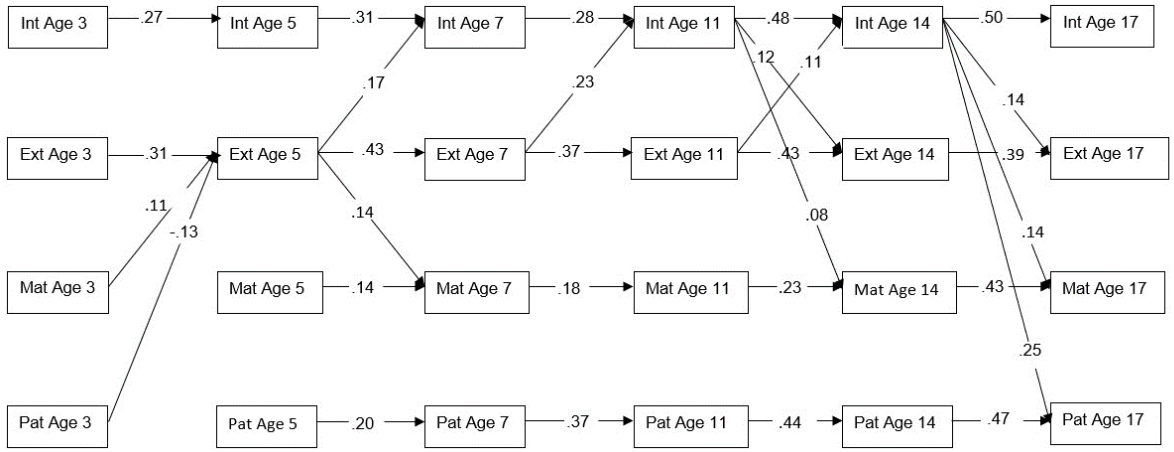
The model, which held the between-family intercept covariances constant across poverty groups, was used to analyse within-family associations. Figures 4.4 and 4.5 show the standardized significant autoregressive and cross-lagged parameters for non-poverty and poverty groups, respectively.

Figure 4.4 *Multi-group ALT-SRs with* unconstrained *autoregressive and cross-lagged paths for boys, non-poverty group*



Note. Int = internalising, Ext = externalising, Mat = maternal psychological distress, Pat = paternal mental health. Children's ages are in accordance with their respective median ages at the time of data collection. Statistically significant paths (p <. 05) are presented only.

Figure 4.5 *Multi-group ALT-SRs with* unconstrained *autoregressive and cross-lagged paths for boys, poverty group*



Note. Int = internalising, Ext = externalising, Mat = maternal psychological distress, Pat = paternal mental health. Children's ages are in accordance with their respective median ages at the time of data collection. Statistically significant paths ($p < .05$) are presented only.

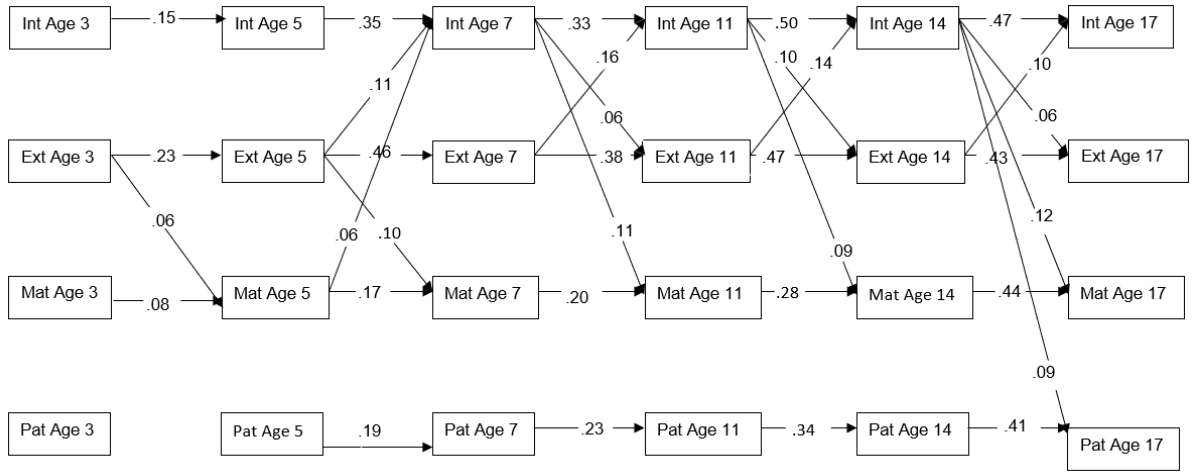
In the non-poverty group, a within-family association was found between maternal distress at ages 5 and 11 and an increase in internalising problems at ages 7 and 14, respectively. Additionally, paternal distress at age 11 was associated with higher externalising problems at age 14. When examining the child to parent effect, internalising problems that occur between the ages of 7 and 14 were consistently associated with a within-family increase in maternal distress during adolescence. In addition, externalising problems at age 3 and 5 were associated with an increase in maternal distress at age 5 and 7, respectively.

Regarding the poverty group, the cross-lagged associations between paternal distress at age 3 and externalising problems at age 5 were negatively correlated with each other, while maternal distress at age 3 and externalising problems at age 5 were positively correlated with each other. A child-to-parent effect was observed in the relation between child internalising problems at age 11 and maternal distress at age 14. Furthermore, child internalising problems

at age 14 were related to increased maternal and paternal distress at age 17. Additionally, externalising problems at age 5 were associated with maternal distress at age 7.

Also, a model that included additional constraints on the autoregressive and cross-lagged paths to be equal for poverty and nonpoverty groups fits the data well ($\chi 2 = 1619.065$, $p < .001$, CFI =. 965, TLI =. 951, and RMSEA =. 034). The model fit indices for these models indicated no differences, suggesting that poverty did not moderate the within-family associations between child and parent mental health problems. Fig 4.6. present the significant constrained autoregressive and cross-lag parameters. Full results with confidence intervals are shown in Tables C9-C13 in Appendix C.

Figure 4.6 *Multi-group ALT-SRs with unstandardized constrained autoregressive and cross-lagged paths for girls*



Note. Int = internalising, Ext = externalising, Mat = maternal distress, Pat = paternal distress. Children's ages are in accordance with their respective median ages at the time of data collection. Statistically significant paths ($p <. 05$) are presented only.

Sensitivity Analyses

Sensitivity analysis repeated the modelling categorising families as being in chronic poverty (between child ages 9 months and 5 years) or not rather than only defining poverty in terms

of family income at 9 months. The model fixing between-family intercept covariances across poverty groups was chosen as fit indices indicated no loss of fit when compared to an unconstrained model, for boys or girls. The results were consistent with the main multigroup analyses. For females, the model that constrained within-family parameter estimates to be equal in the chronic poverty and non-chronic poverty groups fitted the data well ($\chi2$ = 1629.477, $p < .001$, CFI = .949, TLI = .929, RMSEA = .037), with fit statistics similar to the unconstrained model ($\chi2$ = 1569.072, $p < .001$, CFI = .948, TLI =. 910 and RMSEA = .042). Competing models did not show a substantial loss of fit (ΔCFI= 0.001), indicating that being in chronically poverty was not a moderator.

The pattern was similar in males. Both unconstrained and constrained models fit the data well. Model fit for the unconstrained model was $\chi2$ = 1327.153, $p < .001$, CFI = .958, TLI = .927 and RMSEA = .038, while model fit for the constrained model was $\chi2$ = 1427.570, $p < .001$, CFI = .957, TLI = .941 and RMSEA = .034. A substantial loss of fit was not observed (ΔCFI= 0.001), indicating that chronic poverty status did not moderate the within-family relation between parent and child mental health problems.

4.4 Discussion

This is the first study to explicitly compare within-family relations between parent-child mental health difficulties between families in poverty and non-poverty, using a large nationally representative sample. As the study built on an ALT-SR model, a statistical design that offers more direct insights into within-family dynamics, it was possible to make more robust conclusions compared to traditional cross lagged model that conflate within- and between-family effects (Hamaker et al., 2015).

This study examined whether the strength of the between and within-family relations between maternal, paternal and child mental health over child and adolescent development differ

between families living in poverty and those that are not living in poverty. It was hypothesized that there would be a stronger association for families living in poverty on the basis of the Context of Stress model (Hamaker et al., 2015). However, no difference between the groups in terms of the strength of parent-child mental health relations was found. This was found when poverty was based on family circumstances when the child was aged 9 months and in the sensitivity analysis where poverty was defined in terms of chronic poverty, and nonchronic poverty.

In the poverty group, it was expected to observe stronger cross-lag relations than in the non-poverty group in within-family level. Not only were the differences between poverty and non-poverty groups not significant; but inspection of the coefficients in the unconstrained model showed that the pattern of results was opposite to our prediction, further emphasising the incompatibility of the results with our hypothesis. The results of the study also did not indicate any differences between the poverty groups at the between- family level. These results are contrary to McLoyd's (1990)'s Context of Stress framework that suggests that financial hardship depletes the resources of individuals to cope with other difficulties in their lives (Hamaker et al., 2015). In addition, the current results are inconsistent with the results of the meta-analysis presented by Goodman et al. (2011) showing that the effect sizes of the relations between maternal depression and child mental health problems are larger in low-income families. The results are also not consistent with previous longitudinal research examining poverty moderation between risk stressors and child mental health problems as well (see e.g., Chai et al., 2022; Dearing et al., 2006; Dearing et al, 2001).

A number of reasons may account for the differences between the results of current study and the findings of previous studies. Using a study design that differentiates within and between person differences, we applied a rigorous methodological approach. This may explain the difference between the current results and Goodman et al.'s (2011) meta-analysis, which

contains a large number of cross-sectional studies. While some studies that have examined moderation of environmental risk factors for child mental health by family poverty using longitudinal designs, they have not focused on parental mental health problems as a risk factor focussing instead on other stressors such as work – family conflict (see e.g., Chai et al., 2022).

Although the primary focus of the study was to understand within-family relationships between parent-child mental health difficulties, the analysis also provided insights into the relationships between internalising and externalising problems in children. In line with existing literature, these results indicate that internalising and externalising problems generally co-occur (Murray et al., 2020). Furthermore, the relationship between these problems appears to be unaffected by poverty, suggesting that the psychological processes linking internalising and externalising difficulties remain stable regardless of socioeconomic context. However, other factors (e.g., parenting practices) may still play a significant role. These influences should be explored in future research to gain a deeper understanding of this relationship.

The study has a number of strengths, such as a statistical approach that disentangles between-family effects from within-family effects, and a large representative sample of the population and an examination of children over an extended developmental span. However, some limitations of the study must be considered. It is important to note that while ALT-SRs control for stable differences in mental health problems between families, they are susceptible to time-varying confounds such as daily stressors. It should also be noted that poverty is a time-varying factor. Main analyses of the study were based only on poverty at age 9, however sensitivity analyses of the study incorporated variations in chronic and non-chronic poverty. In addition, a potential issue is also common method variance. This is because children's problems were rated by their parents, and parents who are in distress may

inflate the level of their children's behavioural and emotional problems (Kroes et al., 2003). Work involving multiple raters will be required to investigate this possibility.

Another limitation is that reporting of paternal distress was substantially lower. Several factors may contribute to this trend. One primary reason could be that caregiving responsibilities are often perceived as primarily falling on mothers, which may reduce fathers' engagement in reporting their distress. In addition, as a child matures, fathers might perceive they have less direct caregiving responsibility, leading to decreased engagement and, consequently, lower reported distress levels. To address this limitation, attrition weights were used to account for missing data. By assigning weights based on an individual's estimated response probability, this method corrects for non-random dropouts. In particular, observations from participants who were less likely to respond received a higher weighting than observations from participants who were more likely to respond. Taking this approach mitigates bias caused by underrepresented cases, thereby improving data accuracy and representativeness (Schermelleh-Engel et al., 2003).

The current study found that poverty did not moderate the between parental distress and child psychopathology at both between and within-family level. This implies that policymakers should be equally concerned about the relations between parent and child mental health problems, without regard to the level of family poverty. On the other hand, although we cannot say that children in poverty are more sensitive to the changes in their parents' mental health problems, it is well-documented that the prevalence of mental health problems is higher in low SES samples than in higher status samples (Brooks-Gunn & Duncan, 1997). Therefore, it is still important to prioritize that group when conducting prevention and treatment efforts.

Chapter 5: General discussion

This thesis addressed causally related environmental risk factors for child and adolescent internalising problems. To achieve this goal, the initial step involved conducting a comprehensive synthesis of empirical tests of environmental risk factors for child internalising problems. The review focussed on research designs that provide stronger opportunities for causal insight than are common in the field (e.g., cross-sectional or simple cohort studies). Based on the results of the study, further investigations looked into the association between a prominent risk factor- parental mental health- and child internalising problems. Within-family study designs were used to examine this relationship and to test mediation and moderation in order to provide a more nuanced understanding of the complicated causal relationships involved.

5.1 Summary of main findings

As reviewed in Chapter 1, numerous studies have examined environmental risk factors associated with internalising problems in children were investigated, primarily using cross-sectional designs and traditional CLPMs. As mentioned in the Chapter 1, there are some methodological limitations to these approaches when it comes to interpreting causal relationships. The limitations of these methods are that they are unable to determine the direction of the effect (cross-sectional), as well as to distinguish between within-individual and between-individual effects (CLPM). Therefore, these methodological shortcomings may result in misleading interpretations of identified risk factors.

To address this gap, in Chapter 2, a comprehensive systematic review was conducted, focusing specifically on research designs which provide more robust causal insights than cross-sectional or CLPM studies. In this research, a number of environmental risk factors have been environmentally associated with child internalising problems. The results showed

that parents' mental health problems had more examination compared to other risk factors. In addition, it was shown that except for parental mental health problems, other risk factors have been studied in very limited numbers. Moreover, those inventions were predominantly U.S.-based, and there was very limited examination from other countries. Furthermore, there was a noticeable gap in multivariate examination of risk factors, particularly regarding mediation and moderation.

Following up on the systematic review (Chapter 2), the second study (Chapter 3) explored the relationship between parent mental health and child internalisation problems in a multivariate perspective. This investigation was built on the concept of FSM, which posits that lower family income leads to parental distress, which in turn contributes to child internalising problems (Conger et al., 2010). By using the RI-CLPM design, stable trait factors such as genetics were accounted for, allowing the focus to be placed on within-family differences, thus increasing the probability of making causal inferences (Usami, 2020). An analysis of the MCS dataset revealed unexpected results; maternal distress did not mediate the relationship between family income and child internalising problems. This might be a result of the use of RI-CLPM which focusses on within-family change; an approach that has not previously been applied to examine this issue. To provide a comparison with internalising, effects on externalising problems were also examined. In contrast to internalising problems, maternal distress mediated the effect of income on child externalising problems, particularly among girls. Therefore, it was demonstrated that the processes hypothesised in the FSM may take place at the within-family level. Furthermore, there was an association between child internalisation and income at the within-family level. Considering these findings, it appears that the relationship between parental distress and internalising problems has not been clarified by FSM, which illustrates the need for further examination.

Chapter 4 examined an alternative conceptualisation of the interplay between family income and parental mental health in relation to child internalising problems. It investigated whether the relationship between parental, and child mental health differed between families living in poverty and those not living in poverty, at both within and between family levels. An ALT-SR multi-group study was conducted, enabling examination of the dynamics at within-family level. The study reported that the relation between parental mental health and child mental health problems was associated with each other reciprocally at the within-family level. However, no differences between the poverty groups were found at either between or within-family levels.

5.2 Theoretical and practical implications

The most significant implication of this study is its contribution to our understanding of genuine environmental risk factors in children's internalising problems through the use of sophisticated methods. Each study within this framework will be discussed below, highlighting its unique practical and theoretical implications.

In the context of Study One (Chapter 2), previous systematic reviews have either focused on a restricted range of risk factors (e.g., Ahmadzadeh et al., 2021; Jami et al., 2021) or different mental health problems (e.g., Jaffee et al., 2012). However, this study stands out as the first comprehensive examination of environmental risk factors for children's internalising problems within a causally informative study design. Moreover, the study's identification of numerous risk factors associated with children's internalising problems, along with the relatively limited research on these risk factors outside of parental depression, emphasizes the need for additional studies to investigate risk factors using causally informative methods. Additionally, as mentioned in the main results, the studies are predominantly U.S.-based, underscoring the necessity for more diverse international research to achieve a broader and

more representative understanding of environmental risk factors. Furthermore, the exploration of the gap in the multivariate examination of risk factors highlights the need for further research within the contexts of mediation and moderation.

Consequently, the findings of the systematic review provided significant insights for future research directions, urging researchers to adopt more comprehensive study designs, diversify samples globally, and explore mediation and moderation mechanisms in greater depth. In light of the study's practical implications, it can be recommended that interventions related to risk factors identified in the systematic review should be implemented with high-risk families in order to decrease child internalising problems. Studies, for instance, have shown that cognitive-behavioural and psychoeducational interventions can significantly reduce the adverse effects of parents with mental health problems on their children (Peris et al., 2021).

With respect to the implications of the studies in Chapters 3 and 4, they provide some contributions to the existing literature. Contrary to the existing literature, these studies focus on within-family dynamics and employing the FSM (Conger et al., 2010) and Context of Stress Model (McLoyd, 1990) to understand the parent-child mental health relationship through mediation and moderation. These studies represent pioneering efforts in this area, distinguishing between dynamics within and between- families using a method that provides more robust causal insights.

Furthermore, these analyses were conducted using the MCS dataset, which is a UK based nationally representative longitudinal dataset. Thus, these studies have contributed to a multicultural perspective of causally environmental risk factors by including the UK sample in an area predominantly characterized by US samples- as mentioned in Chapter 2. On the other hand, in many countries, there is still a scarcity or absence of studies on environmental risk factors using methods closely aligned with causality. This underscores the need for future

studies to take this into account. Particularly in light of the fact that the United States and the United Kingdom are high income countries, studies from countries with lower incomes are also necessary.

In Chapter 3 (Study Two), it was found that the mediation effect of maternal distress on family income and child mental health was significant only for child externalising problems and not for internalising problems. However, despite the lack of an FSM-supported relationship for internalising problems in this study, the co-occurrence of internalising as well as externalising problems (Pesenti-Gritti et al., 2008) suggests that interventions targeting externalising problems may be useful for alleviating internalising problems as well (Zarakoviti et al., 2021).

In addition, this study found that an increase in family income had a reducing effect on children's internalising problems. Therefore, it is important to develop policies that target income in order to enhance the well-being of children. Implementing such policies through public interventions would contribute to substantial improvements in children's health, development, and overall well-being, as indicated in previous research (e.g., Boccia et al., 2023; Costello et al., 2003).

According to the findings of the last study (Chapter 4), poverty does not moderate the relationship between parental distress and child psychopathology, both between and within-family levels. As noted in Chapter 4, in light of these findings, policymakers should give priority to addressing the association between parent and child mental health problems, regardless of the poverty level of the family. In spite of this, it remains critical to pay particular attention to children in poverty, since there is well-documented evidence that low SES samples are more likely to experience mental health problems than high SES samples (Brooks-Gunn & Duncan, 1997).

Furthermore, as the children's mental health and their parents' mental health had a reciprocal relationship at the within-family level it is important to implement interventions aimed at improving parental mental health to reduce child internalising problems (e.g., Reedtz et al., 2019). In addition, intervening with child internalising problems can also improve parental mental health. In a positive parenting programme, Triple P, parents participated a variety of interventions improving their parenting skills. As a result of the program, children exhibited fewer emotional problems, and parents experienced reduced depression and stress, demonstrating how interventions aimed at reducing internalising problems can also benefit parents (Sanders et al., 2008).

5.3 Limitations and future work

In spite of the fact that these studies are enhancing our understanding of genuine environmental risk factors for internalising problems in children and adolescents through sophisticated causal methods, it is important to consider their limitations when interpreting their findings. The presence of a shared-rater bias is a notable limitation of the studies in this thesis. There was a significant influence of maternal reports on the assessment of children's problems in the systematic review and the studies presented in Chapters 3 and 4. Using maternal reporting introduces the potential for reporter bias, as suggested by Goodman et al. (2011), where depressed mothers may perceive their children as more problematic. It has been highlighted in the literature that results can vary depending on the type of reporter utilized, emphasizing the necessity of using multiple reporters. Several studies have reported disagreements between parents and teachers with respect to the reporting of children's mental health issues (Kroes et al., 2003). Nonetheless, it's essential to acknowledge that the MCS dataset used in Chapters 3 and 4 employed longitudinal measurements spanning at least two years. While concurrent reports are more susceptible to be confounded by reporter bias, later reports are less likely to be affected by such biases (L. Speyer, 2021). However, future

research involving multiple raters would be beneficial in order to better understand the mental health problems of children.

Additionally, the findings from Chapters 3 and 4 are subject to limitations due to their models, namely, the RI-CLPM and the ALT-SR. Although these models are effective in capturing stable effects between individuals, such as genetic influences, they are insufficient in controlling time-varying factors, such as significant life events, which can have significant impacts on individual symptoms (Lüdtke & Robitzsch, 2021; Speyer, 2021). Even though adding more time-varying confounders could be a viable solution, it presents several computational challenges, and an excessive number of confounders may complicate model estimation. Nevertheless, future research should focus on developing existing methods to analyse within-person developmental relations, so that time-varying confounders can be seamlessly accounted for in the models (Speyer, 2021)

In this thesis, parenting distress as well as family income/poverty have been specifically examined as factors affecting child internalisation. However, as described in the systematic review (Chapter 2), there are a lot of other environmental risk factors that contribute to child internalising problems. Thus, future research should explore additional risk factors and investigate possible interventions.

In addition, the FSM study (Chapter 3) only examined parental distress as a mediating factor in Chapter 3. It is important to note, however, that FSM encompasses additional mediating variables like marital dissatisfaction and poor parenting (Conger et al., 2010). Despite this, the MCS dataset has limitations in exploring these potential mediating factors. Future research could address this gap by considering other variables in their investigations.

A further disadvantage of MCS is that it measures over a period of two or more years, which may make it challenging to capture short-term fluctuations and immediate changes, which are

necessary for understanding dynamic phenomena in psychology. This is particularly important for young children to experience rapid changes in their social, and emotional development (Edwards, 2018). In this regard, using shorter time intervals, such as weeks or months, would allow a more accurate picture of the relationships under study to be captured. In the study presented in Chapter 4, no differences were found in the relationship between parents and children regarding poverty levels. However, it is important to consider whether different results might emerge if poverty, child and parental mental health were measured over shorter periods of time. No study has yet examined these relationships using a within-individual design, so future research should explore these relationships over shorter intervals.

5.3 Conclusion

In this thesis, the primary objective was to explore genuine environmental risk factors that contribute to internalising problems among children and adolescents, with the use of sophisticated methods in order to gain a deeper understanding of causal relationships. In this thesis it was illustrated that various environmental risk factors contribute to child/adolescent internalising problems (Study 1). Additionally, the relationship between parental distress and child internalising problems remained unexplained by FSM. Results were attributed to the use of advanced modelling approaches examining these relations at the within-family level (Study 2). Lastly, the interplay between income and parental mental health problems did not clarify the relationship between parental distress and child internalisation, emphasizing the need for further investigation into this relationship (Study 3). The importance of further research into environmental risk factors using sophisticated methods has been underscored in order to increase our understanding of their effect on child internalising problems.

References

Adedeji, A., Otto, C., Kaman, A., Reiss, F., Devine, J., & Ravens-Sieberer, U. (2022). Peer relationships and depressive symptoms among adolescents: results from the German BELLA study. *Frontiers in Psychology*, *12*, 767922–767922. https://doi.org/10.3389/fpsyg.2021.767922

Aguilar-Yamuza, B., Herruzo-Pino, C., Lucena-Jurado, V., Raya-Trenas, A. F., & Pino-Osuna, M. J. (2023). Internalizing Problems in Childhood and Adolescence: The Role of the Family. *Alpha Psychiatry : (Online)*, *24*(3), 87–92. https://doi.org/10.5152/alphapsychiatry.2023.221086

Ahmadzadeh, Y. I., Eley, T. C., Hannigan, L., Creswell, C., Lichtenstein, P., Spotts, E., Ganiban, J., Neiderhiser, J., Rijsdijk, F., & McAdams, T. A. (2022). Parental criticism and adolescent internalising symptoms: using a Children-of-Twins design with power calculations to account for genetic influence. *Journal of Child Psychology and Psychiatry*, *63*(5), 599–607. https://doi.org/10.1111/jcpp.13498

Ahmadzadeh, Y. I., Eley, T. C., Leve, L. D., Shaw, D. S., Natsuaki, M. N., Reiss, D., Neiderhiser, J. M., & McAdams, T. A. (2019). Anxiety in the family: a genetically informed analysis of transactional associations between mother, father and child anxiety symptoms. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *60*(12), 1269–1277. https://doi.org/10.1111/jcpp.13068

Ahmadzadeh, Y. I., Schoeler, T., Han, M., Pingault, J. B., Creswell, C., & McAdams, T. A. (2021). Systematic review and meta-analysis of genetically informed research: associations between parent anxiety and offspring internalizing problems. *Journal of the American Academy of Child & Adolescent Psychiatry*, *60*(7), 823-840.

Aktar, E., Nikolić, M., & Bögels, S. M. (2017). Environmental transmission of generalized anxiety disorder from parents to children: worries, experiential avoidance, and intolerance of uncertainty. *Dialogues in Clinical Neuroscience, 19*(2), 137–147. https://doi.org/10.31887/DCNS.2017.19.2/eaktar

Allison, P. D. (2006). Fixed effects regression methods in SAS. *SUGI 31 Proceedings, 1*, 20.

American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). https://doi.org/10.1176/appi.books.9780890425787

Anderson, D. W. (2002). Helping Students Overcome Depression and Anxiety: A Practical Guide. *Kappa Delta Pi Record, 38*(4), 149. https://www.proquest.com/scholarly-journals/helping-students-overcome-depression-anxiety/docview/232051612/se-2

Anderson, C. A., & Hammen, C. L. (1993). Psychosocial Outcomes of Children of Unipolar Depressed, Bipolar, Medically Ill, and Normal Women. *Journal of Consulting and Clinical Psychology*, *61*(3), 448–454. https://doi.org/10.1037/0022-006X.61.3.448

Anderson, S., & Leventhal, T. (2017). Residential Mobility and Adolescent Achievement and Behavior: Understanding Timing and Extent of Mobility. *Journal of Research on Adolescence*, *27*(2), 328–343. https://doi.org/10.1111/jora.12288

Angold, A., Costello, E. J., Farmer, E. M., Burns, B. J., & Erkanli, A. (1999). Impaired but undiagnosed. *Journal of the American Academy of Child & Adolescent Psychiatry*, *38*(2), 129-137.

Angst, J., Sellar, R., & Merikangas, K. R. (2000). Depressive spectrum diagnoses. *Comprehensive Psychiatry*, *41*(2), 39–47. https://doi.org/10.1016/S0010-440X(00)80007-3

Arseneault, L., Milne, B. J., Taylor, A., Adams, F., Delgado, K., Caspi, A., & Moffitt, T. E. (2008). Being bullied as an environmentally mediated contributing factor to children's

internalizing problems: a study of twins discordant for victimization. *Archives of pediatrics & adolescent medicine*, *162*(2), 145-150.

Asbury, K., Dunn, J. F., Pike, A., & Plomin, R. (2003). Nonshared Environmental Influences on Individual Differences in Early Behavioral Development: A Monozygotic Twin Differences Study. *Child Development*, *74*(3), 933–943. https://doi.org/10.1111/1467-8624.00577

Asbury, K., Dunn, J. F., & Plomin, R. (2006). Birthweight-discordance and differences in early parenting relate to monozygotic twin differences in behaviour problems and academic achievement at age 7. *Developmental Science*, *9*(2), F22–F31. https://doi.org/10.1111/j.1467-7687.2006.00469.x

Ashford, Janka, M.Sc, Lier, P. A. C. V., Timmermans, Maartje, M.Sc, Cuijpers, P., & Koot, H. M. (2008). Prenatal Smoking and Internalizing and Externalizing Problems in Children Studied From Childhood to Late Adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(7), 779–787. https://doi.org/10.1097/CHI.0b013e318172eefb

Averdijk, M., Ribeaud, D., & Eisner, M. (2018). The long-term effects of out-of-home placement in late adolescence: a propensity score matching analysis among swiss youths. *Longitudinal and Life Course Studies*, *9*(1), 30–57. https://doi.org/10.14301/llcs.v9i1.450

Babaee, E., Nojomi, M., Asadi-Aliabadi, M., & Eshrati, B. (2021). Bullying and being bullied; how much can it increase the risk of depression and anxiety in students? A multilevel fixed-effect model analysis. *Iranian Journal of Psychiatry and Behavioral Sciences*, *15*(2). https://doi.org/10.5812/ijpbs.109730

Barrell, R., & Davis, E. P. (2008). THE EVOLUTION OF THE FINANCIAL CRISIS OF 2007–8. *National Institute Economic Review, 206*(206), 5–14. https://doi.org/10.1177/0027950108099838

Bayer, J. K., Sanson, A. V., & Hemphill, S. A. (2006). Parent influences on early childhood internalizing difficulties. *Journal of Applied Developmental Psychology*, *27*(6), 542–559. https://doi.org/10.1016/j.appdev.2006.08.002

Beam, C. R., Pezzoli, P., Mendle, J., Burt, S. A., Michael, C., Boker, S. M., Keel, P. K., & Klump, K. L. (2022). *heredity*. *34*(1), 321–333. https://doi.org/10.1017/S0954579420001017.

Beidel, D. C., & Turner, S. M. (1997). At risk for anxiety: I. Psychopathology in the offspring of anxious parents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36* (7), 918–924.

Bekkhus, M., Lee, Y., Nordhagen, R., Magnus, P., Samuelsen, S. O., & Borge, A. I. H. (2018). Re-examining the link between prenatal maternal anxiety and child emotional difficulties, using a sibling design. *International Journal of Epidemiology*, *47*(1), 156–165. https://doi.org/10.1093/ije/dyx186

Bell. (1968). Effects in studies of socialization. *Psychological Review*, *75*(2), 81–95.

Bergman, A.-S., Axberg, U., & Hanson, E. (2017). When a parent dies - A systematic review of the effects of support programs for parentally bereaved children and their caregivers. *BMC Palliative Care*, *16*(1), 1–39. https://doi.org/10.1186/s12904-017-0223-y

Berry, D., & Willoughby, M. T. (2017). On the Practical Interpretability of Cross-Lagged Panel Models: Rethinking a Developmental Workhorse. *Child Development*, *88*(4), 1186–1206. https://doi.org/10.1111/cdev.12660

Biederman, J., Petty, C., Hirshfeld-Becker, D. R., Henin, A., Faraone, S. V., Dang, D., Jakubowski, A., & Rosenbum, J. F. (2006). A controlled longitudinal 5-year follow-up study of children at high and low risk for panic disorder and major depression. *Psychological Medicine, 36*(8), 1141–1152. https://doi.org/10.1017/S0033291706007781

Bogels, S. M., & Brechman-Toussaint, M. L. (2006). Family issues in child anxiety: attachment, family functioning, parental rearing and beliefs. *Clinical Psychology Review, 26*(7), 834–856. https://doi.org/10.1016/j.cpr.2005.08.001

Bollen, K. A., & Zimmer, C. (2010). An overview of the autoregressive latent trajectory (ALT) model. In K. van Montfort, J. Oud, & A. Satorra (Eds.). Longitudinal research with latent variables (pp. 153–176). New York: Springer.

Brooks-Gunn, J., & Duncan, G. J. (1997). The Effects of Poverty on Children. *The Future of Children, 7*(2), 55–71. https://doi.org/10.2307/1602387

Brooks-Gunn, J., Han, W.-J., & Waldfogel, J. (2010). First-Year Maternal Employment and Child Development in the First Seven Years. *Monographs of the Society for Research in Child Development, 75*(2), 7–9. https://doi.org/10.1111/j.1540-5834.2010.00562.x

Boccia, D., Maritano, S., Pizzi, C., Richiardi, M. G., Lioret, S., & Richiardi, L. (2023). The impact of income - support interventions on life course risk factors and health outcomes during childhood : a systematic review in high income countries. *BMC Public Health*, 1–18. https://doi.org/10.1186/s12889-023-15595-x

Bowes, L., Maughan, B., Caspi, A., Moffitt, T. E., & Arseneault, L. (2010). Families promote emotional and behavioural resilience to bullying: evidence of an environmental effect. *Journal of Child Psychology and Psychiatry, 51*(7), 809–817. https://doi.org/10.1111/j.1469-7610.2010.02216.x

Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, *53*(1), 371–399. https://doi.org/10.1146/annurev.psych.53.100901.135233

Brandlistuen, R. E., Ystrom, E., Eberhard-Gran, M., Nulman, I., Koren, G., & Nordeng, H. (2015). Behavioural effects of fetal antidepressant exposure in a Norwegian cohort of discordant siblings. *International Journal of Epidemiology*, *44*(4), 1397–1407. https://doi.org/10.1093/ije/dyv030

Brandlistuen, R. E., Ystrom, E., Hernandez-Diaz, S., Skurtveit, S., Selmer, R., Handal, M., & Nordeng, H. (2017). Association of prenatal exposure to benzodiazepines and child internalizing problems: A sibling-controlled cohort study. *PLoS ONE*, *12*(7). https://doi.org/10.1371/journal.pone.0181042

Bradshaw, D., Hannigan, A., Creaven, A., & Muldoon, O. T. (2020). Longitudinal associations between parental incarceration and children's emotional and behavioural development: Results from a population cohort study. *Child: Care, Health & Development*, *46*(2), 195–202. https://doi.org/10.1111/cch.12732

Broadhead, W. E., Blazer, D. G., George, L. K., & Tse, C. K. (1990). Depression, disability days, and days lost from work in a prospective epidemiologic survey. *JAMA: the Journal of the American Medical Association*, *264*(19), 2524–2528. https://doi.org/10.1001/jama.1990.03450190056028

Brook, C. A., & Schmidt, L. A. (2008). Social anxiety disorder: a review of environmental risk factors. *Neuropsychiatric Disease and Treatment*, *4*(1 A), 123–143. https://doi.org/10.2147/NDT.S1799

Brooker, R. J., Neiderhiser, J. M., Ganiban, J. M., Leve, L. D., Shaw, D. S., & Reiss, D. (2014). Birth and adoptive parent anxiety symptoms moderate the link between infant

attention control and internalizing problems in toddlerhood. *Development and Psychopathology*, *26*(2), 347–359. https://doi.org/10.1017/S095457941300103X

Bullard, S. E., Griss, M., Greene, S., & Gekker, A. (2013). Encyclopedia of Clinical Neuropsychology. *Archives of Clinical Neuropsychology*, *28*(1), 92–92. https://doi.org/10.1093/arclin/acs103

Burstein, M., Ginsburg, G. S., & Tein, J.-Y. (2010). Parental Anxiety and Child Symptomatology: An Examination of Additive and Interactive Effects of Parent Psychopathology. *Journal of Abnormal Child Psychology*, *38*(7), 897–909. https://doi.org/10.1007/s10802-010-9415-0

Capistrano, C. G., Bianco, H., & Kim, P. (2016). Poverty and internalizing symptoms: The indirect effect of middle childhood poverty on internalizing symptoms via an emotional response inhibition pathway. *Frontiers in Psychology*, *7*, 1242–1242. https://doi.org/10.3389/fpsyg.2016.01242

Cartwright, N. (2007). Are RCTs the gold standard? *BioSocieties*, *2*(1), 11–20. https://doi.org/10.1017/s1745855207005029

Case, A., Lubotsky, D., & Paxson, C. (2002). Economic status and health in childhood: The origins of the gradient. *American Economic Review*, *92*(5), 1308-1334.

Chai, L., & Schieman, S. (2022). Work-to-Family Conflict and Children's Problems with School, Friends, and Health: Household Economic Conditions and Couple Relationship Quality as Contingencies. *Journal of Family Issues*, *43*(6), 1555–1578. https://doi.org/10.1177/0192513X211026953

Cheung, G. W., & Rensvold, R. B. (2002). Evaluating Goodness-of-Fit Indexes for Testing Measurement Invariance. *Structural Equation Modeling*, *9*(2), 233–255. https://doi.org/10.1207/S15328007SEM0902_5

Caspi, A., Moffitt, T. E., Morgan, J., Rutter, M., Taylor, A., Arseneault, L., … Polp-Tomas, M. (2004). Maternal expressed emotion predicts children's antisocial behavior problems: using monozygotic-twin differences to identify environmental effects on behavioral development. *Developmental Psychology*, *40*(2), 149–161. https://doi.org/10.1037/0012-1649.40.2.149

Cents, R. A. M., Diamantopoulou, S., Hudziak, J. J., Jaddoe, V. W. V., Hofman, A., Verhulst, F. C., Lambregtse-van den Berg, M. P., & Tiemeier, H. (2013). Trajectories of maternal depressive symptoms predict child problem behaviour: the generation R study. *Psychological Medicine*, *43*(1), 13–25. https://doi.org/10.1017/S0033291712000657

Cerel, J., Fristad, M. A., Verducci, J., Weller, R. A., & Weller, E. B. (2006). Childhood bereavement: psychopathology in the 2 years post parental death. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(6), 681–690. https://doi.org/10.1097/01.chi.0000215327.58799.05

Chapman, L., Hutson, R., Dunn, A., Brown, M., Savill, E., & Cartwright-Hatton, S. (2022). The impact of treating parental anxiety on children's mental health: an empty systematic review. *Journal of Anxiety Disorders, 88,* 102557–102557. https://doi.org/10.1016/j.janxdis.2022.102557

Chen, J., Yu, J., & Zhang, J. (2016). Investigating Unique Environmental Influences of Parenting Practices on Youth Anxiety: A Monozygotic Twin Differences Study. *International Journal of Behavioral Development*, *40*(3), 205–212. https://doi.org/10.1177/0165025415611261

Cherlin, A. J., Chase-Lansdale, P. L., & McRae, C. (1998). Effects of parental divorce on mental health throughout the life course. *American Sociological Review, 63,* 239-249. https://doi.org/10.2307/2657325

Children's Commissioner. Childhood Vulnerability in England. (2023, June). https://www.childrenscommissioner.gov.uk/chldrn/.

Ching, B. H. H., Wu, H. X., & Chen, T. T. (2021). Maternal Achievement-Oriented Psychological Control: Implications for Adolescent Academic Contingent Self-Esteem and Mathematics Anxiety. *International Journal of Behavioral Development*. https://doi.org/10.1177/0165025420981638

Choe, A. Y., Kim, B., Lee, K. S., Lee, J. E., Lee, J. Y., Choi, T. K., & Lee, S. H. (2013). Serotonergic genes (5-HTT and HTR1A) and separation life events: gene-by-environment interaction for panic disorder. *Neuropsychobiology*, *67*(4), 192–200. https://doi.org/10.1159/000347084

Chong, L. J., Mirzadegan, I. A., & Meyer, A. (2020). The association between parenting and the error-related negativity across childhood and adolescence. *Developmental Cognitive Neuroscience*, *45*, 100852. https://doi.org/10.1016/j.dcn.2020.100852

Cioffi, C. C., Leve, L. D., Natsuaki, M. N., Shaw, D. S., Reiss, D., Ganiban, J. M., & Neiderhiser, J. M. (2021). Examining reciprocal associations between parent depressive symptoms and child internalizing symptoms on subsequent psychiatric disorders: An adoption study. *Depression and Anxiety*, *38*(12), 1211–1224. https://doi.org/10.1002/da.23190

Claman, L. (1983). Handbook of Child Psychopathology. In *American Journal of Psychotherapy* (Vol. 37, Issue 3). https://doi.org/10.1176/appi.psychotherapy.1983.37.3.444a

Clayborne, Z. M., Nilsen, W., Torvik, F. A., Gustavson, K., Bekkhus, M., Gilman, S. E., Khandaker, G. M., Fell, D. B., & Colman, I. (2023). Prenatal maternal stress, child internalizing and externalizing symptoms, and the moderating role of parenting:

findings from the Norwegian mother, father, and child cohort study. *Psychological Medicine, 53*(6), 2437–2447. https://doi.org/10.1017/S0033291721004311

Coley, R. L., Lynch, A. D., & Kull, M. (2015). Early exposure to environmental chaos and children's physical and mental health. *Early Childhood Research Quarterly*, *32*, 94–104. https://doi.org/10.1016/j.ecresq.2015.03.001

Coley, R. L., Ribar, D., & Votruba-Drzal, E. (2011). Do Children's Behavior Problems Limit Poor Women's Labor Market Success? *Journal of Marriage and Family*, *73*(1), 33–45. https://doi.org/10.1111/j.1741-3737.2010.00787.x

Conger, R. D., & Elder, G. H. (1994). Families in Troubled Times: Adapting to Change in Rural America.

Conger, R. D., & Conger, K. J. (2002). Resilience in Midwestern Families: Selected Findings from the First Decade of a Prospective, Longitudinal Study. *Journal of Marriage and Family*, *64*(2), 361–373. https://doi.org/10.1111/j.1741-3737.2002.00361.x

Conger, R. D., Conger, K. J., & Martin, M. J. (2010). Socioeconomic Status, Family Processes, and Individual Development. *Journal of Marriage and Family*, *72*(3), 685–704. https://doi.org/10.1111/j.1741-3737.2010.00725.x

Conger, K. J., Rueter, M. A., & Conger, R. D. (2000). The role of economic pressure in the lives of parents and their adolescents: The family stress model. In L. J. Crockett & R. K. Silbereisen (Eds.), Negotiating adolescence in times ofsocial change (pp. 201–223). New York: Cambridge University Press.

Cooper, K., & Stewart, K. (2021). Does Household Income Affect children’s Outcomes? A Systematic Review of the Evidence. *Child Indicators Research*, *14*(3), 981–1005. https://doi.org/10.1007/s12187-020-09782-0

Copeland, W. E., Shanahan, L., Costello, E. J., & Angold, A. (2009). Childhood and adolescent psychiatric disorders as predictors of young adult disorders. *Archives of General Psychiatry*, *66*(7), 764–772. https://doi.org/10.1001/archgenpsychiatry.2009.85

Costello, E. J., Compton, S. N., Keeler, G., & Angold, A. (2003). Relationships Between Poverty and Psychopathology: A Natural Experiment. *JAMA: the Journal of the American Medical Association*, *290*(15), 2023–2029. https://doi.org/10.1001/jama.290.15.2023

Croft, S., Stride, C., Maughan, B., & Rowe, R. (2015). Validity of the strengths and difficulties questionnaire in preschool-aged children. *Pediatrics (Evanston)*, *135*(5), e1210–e1219. https://doi.org/10.1542/peds.2014-2920

Cummings, C. M., Caporino, N. E., & Kendall, P. C. (2014). Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychological Bulletin*, *140*, 816–845. https://doi.org/10.1037/a0034733

Curran, P. J., Howard, A. L., Bainter, S. A., Lane, S. T., & McGinley, J. S. (2014). The separation of between-person and within-person components of individual change over time: A latent curve model with structured residuals. *Journal of Consulting and Clinical Psychology*, *82*(5), 879–894. https://doi.org/10.1037/a0035297

D'Agostino, R. B., Jr., & D'Agostino, R. B., Sr. (2007). Estimating treatment effects using observational data. *Journal of the American Medical Association*, *297*, 314–316. https://doi.org/10.1001/jama.297.3.314

Davis, J. P., Dumas, T. M., Merrin, G. J., Espelage, D. L., Tan, K., Madden, D., & Hong, J. S. (2018). Examining the Pathways Between Bully Victimization, Depression, Academic Achievement, and Problematic Drinking in Adolescence. *Psychology of Addictive Behaviors*, *32*(6), 605–616. https://doi.org/10.1037/adb0000394

Davis, J. P., Merrin, G. J., Ingram, K. M., Espelage, D. L., Valido, A., & El Sheikh, A. J. (2019). Examining Pathways between Bully Victimization, Depression, & School Belonging Among Early Adolescents. *Journal of Child and Family Studies*, *28*(9), 2365–2378. https://doi.org/10.1007/s10826-019-01340-9

Dearing, E., McCartney, K., & Taylor, B. A. (2001). Change in Family Income-to-Needs Matters More for Children with Less. *Child Development*, *72*(6), 1779–1793. https://doi.org/10.1111/1467-8624.00378

Dearing, E., McCartney, K., & Taylor, B. A. (2006). Within-Child Associations Between Family Income and Externalizing and Internalizing Problems. *Developmental Psychology*, *42*(2), 237–252. https://doi.org/10.1037/0012-1649.42.2.237

Delli Colli, C., Borgi, M., Poggini, S., Chiarotti, F., Cirulli, F., Penninx, B. W. J. H., Benedetti, F., Vai, B., & Branchi, I. (2022). Time moderates the interplay between 5-HTTLPR and stress on depression risk: gene x environment interaction as a dynamic process. *Translational Psychiatry*, *12*(1), 1–12. https://doi.org/10.1038/s41398-022-02035-4

De Los Reyes, A., Ehrlich, K. B., Swan, A. J., Luo, T. J., Van Wie, M., & Pabón, S. C. (2013). An experimental test of whether informants can report about child and family behavior based on settings of behavioral expression. *Journal of Child and Family Studies*, *22*(2), 177–191. https://doi.org/10.1007/s10826-012-9567-3

Denckla, C. A., Espinosa Dice, A. L., Slopen, N., Koenen, K. C., & Tiemeier, H. (2023). Mental health among bereaved youth in the ALSPAC birth cohort: Consideration of early sociodemographic precursors, cognitive ability, and type of loss. *Development and Psychopathology*, 1–12. https://doi.org/10.1017/S0954579423000512

Derlan, C. L., Umaña-Taylor, A. J., Updegraff, K. A., Jahromi, L. B., & Fuentes, S. (2019). A Prospective Test of the Family Stress Model with Mexican-Origin Adolescent Mothers. *Journal of Latina/o Psychology*, *7*(2), 105–122. https://doi.org/10.1037/lat0000109

D'Onofrio, B. M., Class, Q. A., Rickert, M. E., Sujan, A. C., Larsson, H., Kuja-Halkola, R., Sjölander, A., Almqvist, C., Lichtenstein, P., & Oberg, A. S. (2016). Translational epidemiologic approaches to understanding the consequences of early-life exposures. *Behavior Genetics*, *46*(3), 315–328. https://doi.org/10.1007/s10519-015-9769-8

D'Onofrio, B. M., Turkheimer, E. N., Eaves, L. J., Corey, L. A., Berg, K., Solaas, M. H., & Emery, R. E. (2003). The role of the Children of Twins design in elucidating causal relations between parent characteristics and child outcomes. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *44*(8), 1130–1144. https://doi.org/10.1111/1469-7610.00196

Drabick, D. A. G. (2009). Can a Developmental Psychopathology Perspective Facilitate a Paradigm Shift Toward a Mixed Categorical-Dimensional Classification System? *Clinical Psychology (New York, N.Y.)*, *16*(1), 41–49. https://doi.org/10.1111/j.1468-2850.2009.01141.x

Duncan, G. J., Magnuson, K., Kalil, A., & Ziol-Guest, K. (2012). The Importance of Early Childhood Poverty. *Social Indicators Research*, *108*(1), 87–98. https://doi.org/10.1007/s11205-011-9867-9

Duncan, G. J., Yeung, W. J., Brooks-Gunn, J., & Smith, J. R. (1998). How much does childhood poverty affect the life chances of children?. *American sociological review*, 406-423.

Dunn, J., Stocker, C., & Plomin, R. (1990). Nonshared experiences within the family: Correlates of behavioral problems in middle childhood. *Development and Psychopathology*, *2*(2), 113-126.

Duyme, M., Dumaret, A. C., & Tomkiewicz, S. (1999). How can we boost IQs of "dull children"? a late adoption study. *Proceedings of the National Academy of Sciences of*

the United States of America, *96*(15), 8790–8794. https://doi.org/10.1073/pnas.96.15.8790

Eaves, L. J., Silberg, J. L., & Maes, H. H. (2005). Revisiting the children of twins: can they be used to resolve the environmental effects of dyadic parental treatment on child behavior? *Twin Research and Human Genetics*, *8*(4), 283–290. https://doi.org/10.1375/1832427054936736

Edwards, N. M. (2018). *Early social-emotional development : your guide to promoting children's positive behavior* (1st ed.). Paul H. Brookes Publishing Co.

Eley, T. C., McAdams, T. A., Rijsdijk, F. V., Lichtenstein, P., Narusyte, J., Reiss, D., ... & Neiderhiser, J. M. (2015). The intergenerational transmission of anxiety: a children-of-twins study. *American Journal of Psychiatry*, *172*(7), 630-637.

Eley, T. C., & Stevenson, J. (2000). Specific life events and chronic experiences differentially associated with depression and anxiety in young twins. *Journal of Abnormal Child Psychology*, *28*(4), 383–394. https://doi.org/10.1023/A:1005173127117

Emery, C. R. (2011). Controlling for Selection Effects in the Relationship Between Child Behavior Problems and Exposure to Intimate Partner Violence. *Journal of Interpersonal Violence*, *26*(8), 1541–1558. https://doi.org/10.1177/0886260510370597

Enders, C. K. (2001). The performance of the full information maximum likelihood estimator in multiple regression models with missing data. *Educational and Psychological Measurement*, *61*(5), 713–740. https://doi.org/10.1177/00131640121971482

Essau, C. A., & de la Torre-Luque, A. (2023). Comorbidity Between Internalising and Externalising Disorders Among Adolescents: Symptom Connectivity Features and Psychosocial Outcome. *Child Psychiatry and Human Development*, *54*(2), 493–507.

Evans, G. W., & Kim, P. (2007). Childhood Poverty and Health: Cumulative Risk Exposure and Stress Dysregulation. *Psychological Science*, *18*(11), 953–957. https://doi.org/10.1111/j.1467-9280.2007.02008.x

Evans, G. W., & Kim, P. (2013). Childhood Poverty, Chronic Stress, Self-Regulation, and Coping. *Child Development Perspectives*, *7*(1), 43–48. https://doi.org/10.1111/cdep.12013

https://doi.org/10.1007/s10578-021-01264-w

Fauth, R. C., Leventhal, T., & Brooks-Gunn, J. (2007). Welcome to the Neighborhood? Long-Term Impacts of Moving to Low-Poverty Neighborhoods on Poor Children's and Adolescents' Outcomes. *Journal of Research on Adolescence*, *17*(2), 249–284. https://doi.org/10.1111/j.1532-7795.2007.00522.x

Fergusson, D. M., Woodward, L. J., & Horwood, L. J. (1998). Maternal Smoking During Pregnancy and Psychiatric Adjustment in Late Adolescence. *Archives of General Psychiatry*, *55*(8), 721–727. https://doi.org/10.1001/archpsyc.55.8.721

Field, A. P., Lester, K. J., Cartwright-Hatton, S., Harold, G. T., Shaw, D. S., Natsuaki, M. N., Ganiban, J. M., Reiss, D., Neiderhiser, J. M., & Leve, L. D. (2020). Maternal and paternal influences on childhood anxiety symptoms: A genetically sensitive comparison. *Journal of Applied Developmental Psychology*, *68*. https://doi.org/10.1016/j.appdev.2020.101123

Fisher, S. D., & Glangeaud-Freudenthal, N. (2023). Fathers as assets to support maternal mental health and family wellbeing. *Archives of Women's Mental Health*, *26*(1), 87–88. https://doi.org/10.1007/s00737-023-01292-6

Fitzsimons, E., Goodman, A., Kelly, E., & Smith, J. P. (2017). Poverty dynamics and parental mental health: Determinants of childhood mental health in the UK. *Social Science & Medicine (1982)*, *175*, 43–51. https://doi.org/10.1016/j.socscimed.2016.12.040

Flouri, E., Midouhas, E., & Francesconi, M. (2020). Neighbourhood deprivation and child behaviour across childhood and adolescence. *Longitudinal and Life Course Studies*, *11*(2), 203–227. https://doi.org/10.1332/175795919X15722477076216

Flouri, E., Sarmadi, Z., & Francesconi, M. (2019). Paternal psychological distress and child problem behavior from early childhood to middle adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*, *58*(4), 453-458.

Ford, T., Goodman, R., & Meltzer, H. (2003). The British Child and Adolescent Mental Health Survey 1999: The Prevalence of DSM-IV Disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*(10), 1203–1211. https://doi.org/10.1097/00004583-200310000-00011

Foster, E. M. (2010). Causal inference and developmental psychology. *Developmental Psychology*, *46*(6), 1454–1480. https://doi.org/10.1037/a0020204

Furniss, T., Beyer, T., & Müller, J. M. (2009). Impact of life events on child mental health before school entry at age six. *European Child & Adolescent Psychiatry*, *18*(12), 717–724. https://doi.org/10.1007/s00787-009-0013-z

Garber, J., & Weersing, V. R. (2010). Comorbidity of anxiety and depression in youth: implications for treatment and prevention. *Clinical Psychology: Science and Practice, 17*(4), 293–306. https://doi.org/10.1111/j.1468-2850.2010.01221.x

Gennetian, L. A., & Miller, C. (2002). Children and welfare reform: A view from an experimental welfare program in Minnesota. *Child Development*, *73*(2), 601–620. https://doi.org/10.1111/1467-8624.00426

Girard, L.-C., & Farkas, C. (2019). Breastfeeding and behavioural problems: Propensity score matching with a national cohort of infants in Chile. *BMJ Open*, *9*(2), e025058–e025058. https://doi.org/10.1136/bmjopen-2018-025058

Gjerde, L. C., Eilertsen, E. M., Eley, T. C., McAdams, T. A., Reichborn-Kjennerud, T., Røysamb, E., & Ystrom, E. (2020). Maternal Perinatal and Concurrent Anxiety and Mental Health Problems in Early Childhood: A Sibling-Comparison Study. *Child Development*, *91*(2), 456–470. https://doi.org/10.1111/cdev.13192

Gjerde, L. C., Eilertsen, E. M., Hannigan, L. J., Eley, T., Roysamb, E., Reichborn-Kjennerud, T., Rijsdijk, F. V., McAdams, T. A., & Ystrom, E. (2019). Associations between maternal depressive symptoms and risk for offspring early-life psychopathology: The role of genetic and non-genetic mechanisms. *Psychological Medicine*, *51*(3), 441–449. https://doi.org/10.1017/S0033291719003301

Gjerde, L. C., Eilertsen, E. M., Reichborn-Kjennerud, T., McAdams, T. A., Zachrisson, H. D., Zambrana, I. M., Røysamb, E., Kendler, K. S., & Ystrom, E. (2017). Maternal perinatal and concurrent depressive symptoms and child behavior problems: a sibling comparison study. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *58*(7), 779–786. https://doi.org/10.1111/jcpp.12704

Gladstone, T. R. G., Beardslee, W. R., & O'Connor, E. E. (2011). The prevention of adolescent depression. *Psychiatric Clinics of North America*, *34*(1), 35–52. https://doi.org/10.1016/j.psc.2010.11.015

Glover, M. B., Mullineaux, P. Y., Deater-Deckard, K., & Petrill, S. A. (2010). Parents' feelings towards their adoptive and non-adoptive children. *Infant and Child Development*, *19*(3), 238–251. https://doi.org/10.1002/icd.664

Gershoff, E. T., Aber, J. L., Raver, C. C., & Lennon, M. C. (2007). Income is not enough: incorporating material hardship into models of income associations with parenting and child development. *Child Development*, *78*(1), 70–95. https://doi.org/10.1111/j.1467-8624.2007.00986.x

Goldin, P. C. (1969). A review of children's reports of parent behaviors. *Psychological Bulletin*, *71*(3), 222–236. https://doi.org/10.1037/h0027017

Goldstein & DeVries. (2017). *Handbook of DSM-5 Disorders in Children and Adolescents*. https://doi.org/10.1007/978-3-319-57196-6

Gollob, H. F., & Reichardt, C. S. (1987). Taking Account of Time Lags in Causal Models. *Child Development*, *58*(1), 80–92. https://doi.org/10.2307/1130293

Goodman, A., & Goodman, R. (2009). Strengths and Difficulties Questionnaire as a Dimensional Measure of Child Mental Health (vol 48, pg 400, 2009). *Journal of the American Academy of Child and Adolescent Psychiatry*, *48*(5), 581–581.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A Research Note. *Journal of Child Psychology and Psychiatry*, *38*(5), 581–586. https://doi.org/10.1111/j.1469-7610.1997.tb01545.x

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychological review, 106*(3), 458. https://doi.org/10.1037/0033-295x.106.3.458

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal Depression and Child Psychopathology: A Meta-Analytic Review. *Clinical Child and Family Psychology Review, 14*(1), 1–27. https://doi.org/10.1007/s10567-010-0080-1

Gordon, D., Spicker, P., & Comparative Research Programme on Poverty. (1999). *The International glossary on poverty*. Zed.

Gottesman, I. I., & Bertelsen, A. (1989). Confirming Unexpressed Genotypes for Schizophrenia: Risks in the Offspring of Fischer's Danish Identical and Fraternal

Discordant Twins. *Archives of General Psychiatry*, *46*(10), 867–872. https://doi.org/10.1001/archpsyc.1989.01810100009002

Gottfried, M. A. (2014). The influence of tardy classmates on students' socio-emotional outcomes. *Teachers College Record (1970)*, *116*(3), 1–35. https://doi.org/10.1177/016146811411600305

Grabow, A. P., Khurana, A., Natsuaki, M. N., Neiderhiser, J. M., Harold, G. T., Shaw, D. S., Ganiban, J. M., Reiss, D., & Leve, L. D. (2017). Using an adoption-biological family design to examine associations between maternal trauma, maternal depressive symptoms, and child internalizing and externalizing behaviors. *Development and Psychopathology*, *29*(5), 1707–1720. https://doi.org/10.1017/S0954579417001341

Grogan-Kaylor, A. (2004). The effect of corporal punishment on antisocial behavior in children. *Social Work Research*, *28*(3), 153–162. https://doi.org/10.1093/swr/28.3.153

Griffith, J. M., Young, J. F., & Hankin, B. L. (2021). Longitudinal Coupling of Depression in Parent–Adolescent Dyads: Within- and Between-Dyads Effects Over Time. *Clinical Psychological Science*, *9*(6), 1059–1079. https://doi.org/10.1177/2167702621998313

Guimond, F.-A., Laursen, B., Vitaro, F., Brendgen, M., Dionne, G., & Boivin, M. (2016). Associations between mother-child relationship quality and adolescent adjustment: Using a genetically controlled design to determine the direction and magnitude of effects. *International Journal of Behavioral Development*, *40*(3), 196–204. https://doi.org/10.1177/0165025415620059

Hails, K. A., Shaw, D. S., Leve, L. D., Ganiban, J. M., Reiss, D., Natsuaki, M. N., & Neiderhiser, J. M. (2019). Interaction between adoptive mothers' and fathers' depressive symptoms in risk for children's emerging problem behavior. *Social Development (Oxford, England)*, *28*(3), 725–742. https://doi.org/10.1111/sode.12352

Halldorsson & Creswell. (2017). Social anxiety in pre-adolescent children: what do we know about maintenance? *Behaviour Research and Therapy*, *99*, 19–36. https://doi.org/10.1016/j.brat.2017.08.013

Hamaker, E. L., Kuiper, R. M., & Grasman, R. P. P. P. (2015). A Critique of the Cross-Lagged Panel Model. *Psychological Methods*, *20*(1), 102–116. https://doi.org/10.1037/a0038889

Hammen, C., Brennan, P. A., & Shih, J. H. (2004). Family discord and stress predictors of depression and others in adolescent children of depressed and nondepressed women. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*, 994–1002. https://doi.org/10.1097/01.chi.0000127588.57468.f6

Han, W. J. (2020). A first look at parental work schedules and children's well-being in contemporary China. *Community, Work & Family*, *23*(3), 286–304. https://doi.org/10.1080/13668803.2018.1531826

Han, Y., & Grogan-Kaylor, A. (2013). Parenting and Youth Psychosocial Well- Being in South Korea using Fixed-Effects Models. *Journal of Family Issues*, *34*(5), 689–715. https://doi.org/10.1177/0192513X12440091

Hannigan, L. J., Rijsdijk, F. V., Ganiban, J. M., Reiss, D., Spotts, E. L., Neiderhiser, J. M., Lichtenstein, P., McAdams, T. A., & Eley, T. C. (2018). Shared genetic influences do not explain the association between parent–offspring relationship quality and offspring internalizing problems: results from a Children-of-Twins study. *Psychological Medicine*, *48*(4), 592–603. https://doi.org/10.1017/S0033291717001908

Hansen K, editor. Millennium Cohort Study: A Guide to the Datasets (Eighth Edition) First, Second, Third, Fourth and Fifth Surveys. London: Centre for Longitudinal Studies; 2014. Available from: https://doc.ukdataservice.ac.uk/doc/5795/mrdoc/pdf/mcs1-5_user_guide_ed9_2020-08-07.pdf.

Harrison, L., & Harrington, R. (2001). Adolescents' bereavement experiences. prevalence, association with depressive symptoms, and use of services. *Journal of Adolescence, 24*(2), 159–169. https://doi.org/10.1006/jado.2001.0379

Hettema, J. M., Neale, M. C., & Kendler, K. S. (2001). A Review and Meta-Analysis of the Genetic Epidemiology of Anxiety Disorders. *The American Journal of Psychiatry*, *158*(10), 1568–1578. https://doi.org/10.1176/appi.ajp.158.10.1568

Hildyard, K. L., & Wolfe, D. A. (2002). Child neglect: developmental issues and outcomes. *Child Abuse & Neglect*, *26*(6), 679–695. https://doi.org/10.1016/S0145-2134(02)00341-1

Horwitz, B. N., & Neiderhiser, J. M. (2011). Gene-Environment Interplay, Family Relationships, and Child Adjustment. *Journal of Marriage and Family*, *73*(4), 804–816. https://doi.org/10.1111/j.1741-3737.2011.00846.x

Huang, X., Hu, N., Yao, Z., & Peng, B. (2022). Family functioning and adolescent depression: a moderated mediation model of self-esteem and peer relationships. *Frontiers in Psychology, 13*, 962147–962147. https://doi.org/10.3389/fpsyg.2022.962147

Hudziak, J. J., Achenbach, T. M., Althoff, R. R., & Pine, D. S. (2007). A dimensional approach to developmental psychopathology. *International Journal of Methods in Psychiatric Research*, *16*(S1), S16–S23. https://doi.org/10.1002/mpr.217

Humphrey, J. L., & Root, E. D. (2017). Spatio-temporal neighborhood impacts on internalizing and externalizing behaviors in U.S. elementary school children: Effect modification by child and family socio-demographics. *Social Science and Medicine, 180*, 52–61. https://doi.org/10.1016/j.socscimed.2017.03.014

In-Albon, T. (2012). State of Research on Internalizing Disorders in Children and Adolescents: Is It Still in Its Infancy?. *Verhaltenstherapie, 22*, 246-257.

Jaffee, S. R., & Price, T. S. (2007). Gene-environment correlations: a review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry*, *12*(5), 432–442. https://doi.org/10.1038/sj.mp.4001950

Jaffee, S. R., & Price, T. S. (2008). Genotype–environment correlations: implications for determining the relationship between environmental exposures and psychiatric illness. *Psychiatry (Abingdon, England)*, *7*(12), 496–499. https://doi.org/10.1016/j.mppsy.2008.10.002

Jaffee, S. R., Strait, L. B., & Odgers, C. L. (2012). From correlates to causes: can quasi-experimental studies and statistical innovations bring us closer to identifying the causes of antisocial behavior? *Psychological Bulletin*, *138*(2), 272–295. https://doi.org/10.1037/a0026020

Jami, E. S., Hammerschlag, A. R., Bartels, M., & Middeldorp, C. M. (2021). Parental characteristics and offspring mental health and related outcomes: a systematic review of genetically informative literature. *Translational Psychiatry*, *11*(1), 197–38. https://doi.org/10.1038/s41398-021-01300-2

Jamnik, M. R., & DiLalla, L. F. (2019). Health outcomes associated with internalizing problems in early childhood and adolescence. *Frontiers in Psychology*, *10*(JAN), 1–11. https://doi.org/10.3389/fpsyg.2019.00060

Jenness, J. L., Peverill, M., King, K. M., Hankin, B. L., & McLaughlin, K. A. (2019). Dynamic Associations Between Stressful Life Events and Adolescent Internalizing Psychopathology in a Multiwave Longitudinal Study. *Journal of Abnormal Psychology (1965)*, *128*(6), 596–609. https://doi.org/10.1037/abn0000450

Jöreskog, K. G. (1970). Estimation and Testing of Simplex Models. *ETS Research Bulletin Series*, *1970*(2), i–45. https://doi.org/10.1002/j.2333-8504.1970.tb00599.x

Judd, C. M., & Kenny, D. A. (1981). Process Analysis. *Evaluation Review*, *5*(5), 602–619. https://doi.org/10.1177/0193841X8100500502

Kaltiala-Heino, R., Fröjd, S., & Marttunen, M. (2010). Involvement in bullying and depression in a 2-year follow-up in middle adolescence. *European Child & Adolescent Psychiatry*, *19*(1), 45–55. https://doi.org/10.1007/s00787-009-0039-2

Katayama, M., Kitaoka, K., & Aijo, R. (2022). Mothers with depressed mood: help-seeking from husbands and child-rearing behaviors. *BMC Women's Health*, *22*(1), 25–25. https://doi.org/10.1186/s12905-022-01604-5

Kathleen Kiernan, & M. Carmen Huerta. (2008). Economic deprivation, maternal depression, parenting and children's cognitive and emotional development in early childhood1. *The British Journal of Sociology*, *59*, 783–806. https://doi.org/10.1111/j.1468-4446.2008.00219.x

Kaufman, J. S. (2008). Commentary: why are we biased against bias? *International Journal of Epidemiology*, *37*(3), 624. https://doi.org/10.1093/ije/dyn035

Keijsers, L. (2016). Parental monitoring and adolescent problem behaviors. *International Journal of Behavioral Development*, *40*(3), 271–281. https://doi.org/10.1177/0165025415592515

Kelly, E. V., Newton, N. C., Stapinski, L. A., Slade, T., Barrett, E. L., Conrod, P. J., & Teesson, M. (2015). Suicidality, internalizing problems and externalizing problems among adolescent bullies, victims and bully-victims. *Preventive Medicine*, *73*, 100–105. https://doi.org/10.1016/j.ypmed.2015.01.020

Kennedy, S., & Bumpass, L. (2008). Cohabitation and children's living arrangements. *Demographic Research*, *19*, 1663–1692. https://doi.org/10.4054/DemRes.2008.19.47

Kerr, D. C. R., Leve, L. D., Harold, G. T., Natsuaki, M. N., Neiderhiser, J. M., Shaw, D. S., & Reiss, D. (2013). Influences of Biological and Adoptive Mothers' Depression and Antisocial Behavior on Adoptees' Early Behavior Trajectories. *Journal of Abnormal Child Psychology*, *41*(5), 723–734. https://doi.org/10.1007/s10802-013-9711-6

Kersten, P., Czuba, K., McPherson, K., Dudley, M., Elder, H., Tauroa, R., & Vandal, A. (2016). A systematic review of evidence for the psychometric properties of the Strengths and Difficulties Questionnaire. *International Journal of Behavioral Development*, *40*(1), 64–75. https://doi.org/10.1177/0165025415570647

Kessler, R., Andrews, G., Colpe, L., Hiripi, E., Mroczek, D., Normand, S., Walters, E., & Zaslavsky, A. (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychological Medicine*, *32*(6), 959–976. https://doi.org/10.1017/S0033291702006074

Kessler, R. C., Barker, P. R., Colpe, L. J., Epstein, J. F., Gfroerer, J. C., Hiripi, E., Howes, M. J., Normand, S.-L. T., Manderscheid, R. W., Walters, E. E., & Zaslavsky, A. M. (2003). Screening for Serious Mental Illness in the General Population. *Archives of General Psychiatry*, *60*(2), 184–189. https://doi.org/10.1001/archpsyc.60.2.184

Kline, R. B. (2016). *Principles and practice of structural equation modeling* (Fourth edition.). The Guilford Press.

Klomek, A. B., Sourander, A., Kumpulainen, K., Piha, J., Tamminen, T., Moilanen, I., Almqvist, F., & Gould, M. S. (2008). Childhood bullying as a risk for later depression and suicidal ideation among Finnish males. *Journal of Affective Disorders*, *109*(1), 47–55. https://doi.org/10.1016/j.jad.2007.12.226

Knudsen, A. K., Ystrom, E., Skogen, J. C., & Torgersen, L. (2015). Maternal heavy alcohol use and toddler behavior problems: a fixed effects regression analysis. *European Child*

and Adolescent Psychiatry, *24*(10), 1269–1277. https://doi.org/10.1007/s00787-015-0677-5

Kochel, K. P., Ladd, G. W., & Rudolph, K. D. (2012). Longitudinal associations among youth depressive symptoms, peer victimization, and low peer acceptance: an interpersonal process perspective. *Child Development*, *83*(2), 637–650. https://doi.org/10.1111/j.1467-8624.2011.01722.x

Kroes, G., Veerman, J. W., & De Bruyn, E. E. J. (2003). Bias in parental reports? Maternal psychopathology and the reporting of problem behavior in clinic-referred children. *European Journal of Psychological Assessment: Official Organ of the European Association of Psychological Assessment*, *19*(3), 195–203. https://doi.org/10.1027//1015-5759.19.3.195

Kulig, J. W., & Committee on Substance Abuse. (2005). Tobacco, alcohol, and other drugs: the role of the pediatrician in prevention, identification, and management of substance abuse. *Pediatrics (Evanston)*, *115*(3), 816–821. https://doi.org/10.1542/peds.2004-2841

Kwok, O., Haine, R. A., Sandler, I. N., Ayers, T. S., Wolchik, S. A., & Tein, J. (2005). Positive parenting as a mediator of the relations between parental psychological distress and mental health problems of parentally bereaved children. Journal of Clinical Child and Adolescent Psychology, 34,260–271.

Lahey, B. B., & D'Onofrio, B. M. (2010). All in the Family: Comparing Siblings to Test Causal Hypotheses Regarding Environmental Influences on Behavior. *Current Directions in Psychological Science*, *19*(5), 319–323. https://doi.org/10.1177/0963721410383977

Lahey, B. B., D'Onofrio, B. M., & Waldman, I. (2009). Using epidemiologic methods to test hypotheses regarding causal influences on child and adolescent mental disorders.

Journal of Child Psychology and Psychiatry and Allied Disciplines, *50*(1–2), 53–62. https://doi.org/10.1111/j.1469-7610.2008.01980.x

Langrock, A., Compas, B., Keller, G., Merchant, M., & Copeland, M. (2002). Coping with the stress of parental depression: Parents' reports of children's coping, emotional, and behavioral problems. *Journal of Clinical Child and Adolescent Psychology*, *31*(3), 312–324. https://doi.org/10.1207/153744202760082577

Laurent, H. K., Leve, L. D., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Fisher, P. A., Marceau, K., Harold, G. T., & Reiss, D. (2013). Effects of Parental Depressive Symptoms on Child Adjustment Moderated by Hypothalamic Pituitary Adrenal Activity: Within- and Between-Family Risk. *Child Development*, *84*(2), 528–542. https://doi.org/10.1111/j.1467-8624.2012.01859.x

Laurent, H. K., Leve, L. D., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Harold, G. T., & Reiss, D. (2013). Effects of prenatal and postnatal parent depressive symptoms on adopted child HPA regulation: Independent and moderated influences. *Developmental Psychology*, *49*(5), 876–886. https://doi.org/10.1037/a0028800

Lazarus, S., Folkman, R. S., & Folkman, Susan. (1984). *Stress, appraisal, and coping*. Springer.

Lawler, E. E., & Suttle, J. L. (1973). Expectancy theory and job behavior. *Organizational Behavior and Human Performance*, *9*(3), 482–503. https://doi.org/10.1016/0030-5073(73)90066-4

Lemstra, M., Neudorf, C., D'Arcy, C., Kunst, A., Warren, L. M., & Bennett, N. R. (2008). A Systematic Review of Depressed Mood and Anxiety by SES in Youth Aged 10-15 Years. *Canadian Journal of Public Health*, *99*(2), 125–129. https://doi.org/10.1007/BF03405459

Leventhal, T., & Brooks-Gunn, J. (2003). Moving to Oppurtunity: An Experimental Study of Neighborhood Effects on Mental Health. *American Journal of Public Health, 93*(9), 1576–1582. https://doi.org/10.2105/AJPH.93.9.1576

Leventhal, T., & Brooks-Gunn, J. (2011). Changes in neighborhood poverty from 1990 to 2000 and youth's problem behaviors. *Developmental Psychology, 47*(6), 1680–1698. https://doi.org/10.1037/a0025314

Leventhal, T., & Dupéré, V. (2019). Neighborhood Effects on Children's Development in Experimental and Nonexperimental Research. *Annual Review of Developmental Psychology, 1*(1), 149–176. https://doi.org/10.1146/annurev-devpsych-121318-085221

Lewis, G., Rice, F., Harold, G. T., Collishaw, S., & Thapar, A. (2011). Investigating environmental links between parent depression and child depressive/anxiety symptoms using an assisted conception design. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*(5), 451-459.e1. https://doi.org/10.1016/j.jaac.2011.01.015

Li, X., Huebner, E. S., & Tian, L. (2021). Vicious cycle of emotional maltreatment and bullying perpetration/victimization among early adolescents: Depressive symptoms as a mediator. *Social Science & Medicine (1982), 291*, 114483–114483. https://doi.org/10.1016/j.socscimed.2021.114483

Lieb, R., Isensee, B., Höfler, M., Pfister, H., & Wittchen, H. U. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: a prospective-longitudinal community study. *Archives of general psychiatry, 59*(4), 365-374. https://doi.org/10.1001/archpsyc.59.4.365

Lin, S. Y., Schleider, J. L., & Eaton, N. R. (2021). Family Processes and Child Psychopathology: A Between- and Within-Family/Child Analysis. *Research on Child and Adolescent Psychopathology, 49*(3), 283–295. https://doi.org/10.1007/s10802-020-00749-x

Liskola, K., Raaska, H., Lapinleimu, H., & Elovainio, M. (2018). Parental depressive symptoms as a risk factor for child depressive symptoms; testing the social mediators in internationally adopted children. *European Child and Adolescent Psychiatry*, *27*(12), 1585–1593. https://doi.org/10.1007/s00787-018-1154-8

Liu, J., Chen, X., & Lewis, G. (2011). Childhood internalizing behaviour: analysis and implications. *Journal of psychiatric and mental health nursing, 18*(10), 884-894. 10.1111/j.1365-2850.2011.01743.x

Liu, J., Deng, J., Zhang, H., & Tang, X. (2023). The relationship between child maltreatment and social anxiety: a meta-analysis. *Journal of Affective Disorders*, *329*, 157–167. https://doi.org/10.1016/j.jad.2023.02.081

Lougheed, J. P., Duncan, R. J., Keskin, G., & Marceau, K. (2020). Longitudinal associations between mother-child conflict and child internalizing problems in mid-childhood. *Development and Psychopathology*, 1–10. https://doi.org/10.1017/S0954579420000863

Lovejoy MC, Graczyk PA, O'Hare E, Neuman G (2000). Maternal depression and parenting behavior: a meta-analytic review. *Clinical Psychology Review 20*,561–592. https://doi.org/10.1016/s0272-7358(98)00100-7

Lund, I. O., Moen Eilertsen, E., Gjerde, L. C., Røysamb, E., Wood, M., Reichborn-Kjennerud, T., & Ystrom, E. (2019). Is the association between maternal alcohol consumption in pregnancy and pre-school child behavioural and emotional problems causal? Multiple approaches for controlling unmeasured confounding. *Addiction*, *114*(6), 1004–1014. https://doi.org/10.1111/add.14573

Lüdtke, O. R. (2021). A Critique of the Random Intercept Cross-Lagged Panel Model. *Psychological Methods*, *20*(1), 102–116. https://doi.org/10.1037/a0038889

Lyons-Ruth, K., Bronfman, E., & Atwood, G. (1999). A relational diathesis model of hostile-helpless states of mind: Expressions in mother–infant interaction.

Kearney. (2017). Cross-lagged panel analysis. *the SAGE Encyclopedia of Communication Research Methods*, 1–6. https://doi.org/10.4135/9781483381411.n117

Kingsbury, M., Sucha, E., Manion, I., Gilman, S. E., & Colman, I. (2020). Adolescent mental health following exposure to positive and harsh parenting in childhood. *Canadian Journal of Psychiatry*, *65*(6), 392–400. https://doi.org/10.1177/0706743719889551

Knafo, A., & Jaffee, S. R. (2013). Gene-environment correlation in developmental psychopathology. *Development and Psychopathology*, *25*(1), 1–6. https://doi.org/10.1017/S0954579412000855

Knopik, V. S., Neiderhiser, J. M., DeFries, J. C., & Plomin, R. (2017). *Behavioral genetics* (pp. p93-110). New York: Worth Publishers, Macmillan Learning.

Ma, J., Han, Y., & Kang, H. R. (2022). Physical punishment, physical abuse, and child behavior problems in South Korea. *Child Abuse & Neglect*, *123*, 105385–105385. https://doi.org/10.1016/j.chiabu.2021.105385

Mack, K. (2001). Childhood family disruptions and adult well-being: the differential effects of divorce and parental death. *Death Studies*, *25*(5), 419–443. https://doi.org/10.1080/07481180125745

Maldonado-Carreño, C., & Votruba-Drzal, E. (2011). Teacher-Child Relationships and the Development of Academic and Behavioral Skills During Elementary School: A Within- and Between-Child Analysis. *Child Development*, *82*(2), 601–616. https://doi.org/10.1111/j.1467-8624.2010.01533.x

Marceau, K., Hajal, N., Leve, L. D., Reiss, D., Shaw, D. S., Ganiban, J. M., Mayes, L. C., & Neiderhiser, J. M. (2013). Measurement and associations of pregnancy risk factors with genetic influences, postnatal environmental influences, and toddler behavior.

International Journal of Behavioral Development, *37*(4), 366–375. https://doi.org/10.1177/0165025413489378

Marceau, K., Laurent, H. K., Neiderhiser, J. M., Reiss, D., Shaw, D. S., Natsuaki, M., Fisher, P. A., & Leve, L. D. (2015). Combined influences of genes, prenatal environment, cortisol, and parenting on the development of children's internalizing vs. externalizing problems. *Behavior Genetics*, *45*(3), 268–282. https://doi.org/10.1007/s10519-014-9689-z

Maron, E., Hettema, J. M., & Shlik, J. (2010). Advances in molecular genetics of panic disorder. *Molecular Psychiatry*, *15*(7), 681–701. https://doi.org/10.1038/mp.2009.145

Martin, M. J., Conger, R. D., Schofield, T. J., Dogan, S. J., Widaman, K. F., Donnellan, M. B., & Neppl, T. K. (2010). Evaluation of the interactionist model of socioeconomic status and problem behavior: A developmental cascade across generations. *Development and Psychopathology*, *22*(3), 695–713. https://doi.org/10.1017/S0954579410000374

Masarik, A. S., & Conger, R. D. (2017). Stress and child development: A review of the Family Stress Model. *Current Opinion in Psychology*, *13*, 85–90. https://doi.org/10.1016/j.copsyc.2016.05.008

Mastrotheodoros, S., Canário, C., Cristina Gugliandolo, M., Merkas, M., & Keijsers, L. (2020). Family Functioning and Adolescent Internalizing and Externalizing Problems: Disentangling between-, and Within-Family Associations. *Journal of Youth and Adolescence*, *49*(4), 804–817. https://doi.org/10.1007/s10964-019-01094-z

Maxwell, S. E., Cole, D. A., & Mitchell, M. A. (2011). Bias in Cross-Sectional Analyses of Longitudinal Mediation: Partial and Complete Mediation Under an Autoregressive Model. *Multivariate Behavioral Research*, *46*(5), 816–841. https://doi.org/10.1080/00273171.2011.606716

McAdams, T. A., Neiderhiser, J. M., Rijsdijk, F. V., Narusyte, J., Lichtenstein, P., & Eley, T. C. (2014). Accounting for genetic and environmental confounds in associations between parent and child characteristics: a systematic review of children-of-twins studies. *Psychological Bulletin, 140*(4), 1138–1173. https://doi.org/10.1037/a0036416

McAdams, T. A., Rijsdijk, F. V., Neiderhiser, J. M., Narusyte, J., Shaw, D. S., Natsuaki, M. N., Spotts, E. L., Ganiban, J. M., Reiss, D., Leve, L. D., Lichtenstein, P., & Eley, T. C. (2015). The relationship between parental depressive symptoms and offspring psychopathology: Evidence from a children-of-twins study and an adoption study. *Psychological Medicine, 45*(12), 2583–2594. https://doi.org/10.1017/S0033291715000501

McCrory, C., & Layte, R. (2012). Prenatal exposure to maternal smoking and childhood behavioural problems: A quasi-experimental approach. *Journal of Abnormal Child Psychology, 40*(8), 1277–1288. https://doi.org/10.1007/s10802-012-9640-9

McDool, E. (2019). Ability grouping and children's non-cognitive outcomes. *Applied Economics, 52*(28), 3035-3054.

McGovern, R., Bogowicz, P., Meader, N., Kaner, E., Alderson, H., Craig, D., Geijer-Simpson, E., Jackson, K., Muir, C., Salonen, D., Smart, D., & Newham, J. J. (2023). The association between maternal and paternal substance use and child substance use, internalizing and externalizing problems: a systematic review and meta-analysis. *Addiction (Abingdon, England), 118*(5), 804–818. https://doi.org/10.1111/add.16127

McLeod, B. D., Weisz, J. R., & Wood, J. J. (2007). Examining the association between parenting and childhood depression: a meta-analysis. *Clinical Psychology Review, 27*(8), 986–1003. https://doi.org/10.1016/j.cpr.2007.03.001

McLeod, J. D., & Nonnemaker, J. M. (2000). Poverty and child emotional and behavioral problems: racial/ethnic differences in processes and effects. *Journal of Health and Social Behavior*, *41*(2), 137–161. https://doi.org/10.2307/2676302

McLeod, J. D., & Owens, T. J. (2004). Psychological well-being in the early life course: variations by socioeconomic status, gender, and race/ethnicity. *Social Psychology Quarterly*, *67*(3), 257–278. https://doi.org/10.1177/019027250406700303

McLoyd, V. C. (1990). impact of economic hardship on black families and children: psychological distress, parenting, and socioemotional development. *Child Development*, *61*(2), 311–346. https://doi.org/10.2307/1131096

McLoyd, V. C., & Wilson, L. (1994). The strain of living poor: Parenting, social support, and child mental health. In A. C. Huston (Ed.), Children in poverty: Child development and public policy. Cambridge, UK: Cambridge University Press.

McMahon, S. D., Grant, K. E., Compas, B. E., Thurm, A. E., & Ey, S. (2003). Stress and psychopathology in children and adolescents: is there evidence of specificity? *Journal of Child Psychology and Psychiatry*, *44*(1), 107–133. https://doi.org/10.1111/1469-7610.00105

Mennen, F. E., Negriff, S., Schneiderman, J. U., & Trickett, P. K. (2018). Longitudinal Associations of Maternal Depression and Adolescents' Depression and Behaviors: Moderation by Maltreatment and Sex. *Journal of Family Psychology*, *32*(2), 240–250. https://doi.org/10.1037/fam0000394

Anderson, D. W. (2002). Helping Students Overcome Depression and Anxiety: A Practical Guide. *Kappa Delta Pi Record*, *38*(4), 149. https://www.proquest.com/scholarly-journals/helping-students-overcome-depression-anxiety/docview/232051612/se-2

Miller, P., Betancur, L., Whitfield, K., & Votruba-Drzal, E. (2020). Examining income dynamics and externalizing and internalizing trajectories through a developmental

psychopathology lens: a nationally representative study. *Development and Psychopathology*, 1–17. https://doi.org/10.1017/S0954579419001494

Milligan, K., & Stabile, M. (2008). Do child tax benefits affect the well-being of children? Evidence from Canadian child benefit expansions. *American Economic Journal: Economic Policy*, *3*(3), 175–205. https://doi.org/10.1257/pol.3.3.175

Mistry, R. S., Benner, A. D., Tan, C. S., & Kim, S. Y. (2009). Family Economic Stress and Academic Well-Being Among Chinese-American Youth. *Journal of Family Psychology*, *23*(3), 279–290. https://doi.org/10.1037/a0015403

Moffitt, Terrie E., & Moffitt, Terrie E. (2001). *Sex differences in antisocial behaviour : conduct disorder, delinquency, and violence in the Dunedin longitudinal study*. Cambridge University Press.

Moller, E. L., Nikolic, M., Majdandzic, M., & Bogels, S. M. (2016). Associations between maternal and paternal parenting behaviors, anxiety and its precursors in early childhood: A meta-analysis. *Clinical Psychology Review*, *45*, 17–33. https://doi.org/10.1016/j.cpr.2016.03.002

Morales-Munoz, I., Ashdown-Doel, B., Beazley, E., Carr, C., Preece, C., & Marwaha, S. (2023). Maternal postnatal depression and anxiety and the risk for mental health disorders in adolescent offspring: Findings from the Avon Longitudinal Study of Parents and Children cohort. *Australian and New Zealand Journal of Psychiatry*, *57*(1), 82–92. https://doi.org/10.1177/00048674221082519

Morris-Rosendahl, D. J. (2002). Are there anxious genes? *Dialogues in Clinical Neuroscience*, *4*(3), 251–260. https://doi.org/10.31887/dcns.2002.4.3/dmrosendahl

Mulder, J. D., & Hamaker, E. L. (2021). Three Extensions of the Random Intercept Cross-Lagged Panel Model. *Structural Equation Modeling*, *28*(4), 638–648. https://doi.org/10.1080/10705511.2020.1784738

Mund, M., & Nestler, S. (2019). Beyond the Cross-Lagged Panel Model: Next-generation statistical tools for analyzing interdependencies across the life course. *Advances in Life Course Research*, *41*, 100249. https://doi.org/10.1016/j.alcr.2018.10.002

Murray, A. L., Booth, T., Eisner, M., Auyeung, B., Murray, G., & Ribeaud, D. (2019). Sex differences in ADHD trajectories across childhood and adolescence. *Developmental Science*, *22*(1), e12721–n/a. https://doi.org/10.1111/desc.12721

Mullineaux, P. Y., Deater-Deckard, K., Petrill, S. A., & Thompson, L. A. (2009). Parenting and child behaviour problems: a longitudinal analysis of non-shared environment. *Infant and Child Development*, *18*(2), 133–148. https://doi.org/10.1002/icd.593

Murray, A. L., Booth, T., Eisner, M., Auyeung, B., Murray, G., & Ribeaud, D. (2019). Sex differences in ADHD trajectories across childhood and adolescence. *Developmental Science*, *22*(1), e12721–n/a. https://doi.org/10.1111/desc.12721

Murray, A. L., Speyer, L. G., Hall, H. A., Valdebenito, S., & Hughes, C. (2022). A Longitudinal and Gender Invariance Analysis of the Strengths and Difficulties Questionnaire Across Ages 3, 5, 7, 11, 14, and 17 in a Large U.K.-Representative Sample. *Assessment (Odessa, Fla.)*, *29*(6), 1248–1261. https://doi.org/10.1177/10731911211009312

Murray, A. L., Eisner, M., & Ribeaud, D. (2020). Within‐person analysis of developmental cascades between externalising and internalising problems. Journal of Child Psychology and Psychiatry, 61(6), 681–688. https://doi.org/10.1111/jcpp.13150

Muthén, L. K., & Muthén, B. O. (1998–2017). Mplus user's guide. 8th ed. Los Angeles, CA: Muthén & Muthén.

Narusyte, J., Neiderhiser, J. M., D'Onofrio, B. M., Reiss, D., Spotts, E. L., Ganiban, J., & Lichtenstein, P. (2008). Testing different types of genotype-environment

correlation. *Developmental Psychology*, *44*(6), 1591–1603. https://doi.org/10.1037/a0013911

Nelemans, S. A., Keijsers, L., Colpin, H., van Leeuwen, K., Bijttebier, P., Verschueren, K., & Goossens, L. (2020). Transactional Links Between Social Anxiety Symptoms and Parenting Across Adolescence: Between- and Within-Person Associations. *Child Development*, *91*(3), 814–828. https://doi.org/10.1111/cdev.13236

Neppl, T. K., Senia, J. M., & Donnellan, M. B. (2016). Effects of Economic Hardship: Testing the Family Stress Model Over Time. *Journal of Family Psychology*, *30*(1), 12–21. https://doi.org/10.1037/fam0000168

Nook, E. C., Flournoy, J. C., Rodman, A. M., Mair, P., & McLaughlin, K. A. (2021). High Emotion Differentiation Buffers Against Internalizing Symptoms Following Exposure to Stressful Life Events in Adolescence: An Intensive Longitudinal Study. *Clinical Psychological Science*, *9*(4), 699–718. https://doi.org/10.1177/2167702620979786

Novick, D. R., Meyer, C. T., Wagner, N. J., Rubin, K. H., Danko, C. M., Dougherty, L. R., Druskin, L. R., Smith, K. A., & Chronis-Tuscano, A. (2023). Testing reciprocal associations between child anxiety and parenting across early interventions for inhibited preschoolers. *Journal of Child Psychology and Psychiatry*, *64*(12), 1665–1678. https://doi.org/10.1111/jcpp.13879

O'Connor, T. G., Caspi, A., DeFries, J. C., & Plomin, R. (2000). Are associations between parental divorce and children's adjustment genetically mediated? An adoption study. *Developmental Psychology*, *36*(4), 429–437. https://doi.org/10.1037/0012-1649.36.4.429

O'Connor, T. G., Deater-Deckard, K., & Plomin, R. (1998). Contributions of behavioral genetics research to clinical psychology. *Comprehensive Clinical Psychology*, 87–114. https://doi.org/10.1016/b0080-4270(73)00175-9

Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., Poulton, R., Sears, M. R., Thomson, W. M., & Caspi, A. (2008). Female and male antisocial trajectories: From childhood origins to adult outcomes. *Development and Psychopathology*, *20*(2), 673–716. https://doi.org/10.1017/S0954579408000333

OECD. What are equivalence scales?. (2009). http://www.oecd.org/eco/growth/OECD-Note-EquivalenceScales.pdf.

Offord, D. R., Boyle, M. H., Szatmari, P., Rae-Grant, N. I., Links, P. S., Cadman, D. T., Byles, J. A., Crawford, J. W., Blum, H. M., Byrne, C., Thomas, H., & Woodward, C. A. (1987). Ontario Child Health Study: II. Six-Month Prevalence of Disorder and Rates of Service Utilization. *Archives of General Psychiatry*, *44*(9), 832–836. https://doi.org/10.1001/archpsyc.1987.01800210084013

Okoli, G. N., Sanders, R. D., & Myles, P. (2014). Demystifying propensity scores. *British Journal of Anaesthesia*, *112*(1), 13–15. https://doi.org/10.1093/bja/aet290

O'Leary, C. M., Nassar, N., Zubrick, S. R., Kurinczuk, J. J., Stanley, F., & Bower, C. (2010). Evidence of a complex association between dose, pattern and timing of prenatal alcohol exposure and child behaviour problems. *Addiction (Abingdon, England)*, *105*(1), 74–86. https://doi.org/10.1111/j.1360-0443.2009.02756.x

Orford J, Natera G, Copello A et al (2005) *Coping with alcohol and drug problems: the experiences of family members in three contrasting cultures.* Routledge, London and NewYork

Orth, U., Clark, D. A., Donnellan, M. B., & Robins, R. W. (2021). Testing Prospective Effects in Longitudinal Research: Comparing Seven Competing Cross-Lagged Models. *Journal of Personality and Social Psychology*, *120*(4), 1013–1034. https://doi.org/10.1037/pspp0000358

Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71.

Park, M., Hanley, G. E., Guhn, M., & Oberlander, T. F. (2021). Prenatal antidepressant exposure and child development at kindergarten age: a population-based study. *Pediatric Research*, *89*(6), 1515–1522. https://doi.org/10.1038/s41390-020-01269-6

Parke, R. D., Coltrane, S., Duffy, S., Buriel, R., Dennis, J., Powers, J., French, S., & Widaman, K. F. (2004). Economic Stress, Parenting, and Child Adjustment in Mexican American and European American Families. *Child Development*, *75*(6), 1632–1656. https://doi.org/10.1111/j.1467-8624.2004.00807.x

Parsons, S. (2011). Long-term impact of childhood bereavement: Preliminary analysis of the 1970 British Cohort Study (BCS70). London: Childhood Wellbeing Research Centre. CWRC Working Paper.

Patalay, P., & Gage, S. H. (2019). Changes in millennial adolescent mental health and health-related behaviours over 10 years: a population cohort comparison study. *International Journal of Epidemiology*, *48*(5), 1650–1664. https://doi.org/10.1093/ije/dyz006

Patrick J. Curran, Andrea L. Howard, Sierra A. Bainter, Stephanie T. Lane, & James S. McGinley. (2013). The separation of between-person and within-person components of individual change over time: A latent curve model with structured residuals. *Journal of Consulting and Clinical Psychology*, *82*, 879–894. https://doi.org/10.1037/a0035297

Pickles & Angold. (2003). Natural categories or fundamental dimensions: on carving nature at the joints and the rearticulation of psychopathology. *Development and Psychopathology*, *15*(3), 529–551. https://doi.org/10.1017/S0954579403000282

Piera Pi-Sunyer, B., Andrews, J. L., Orben, A., Speyer, L. G., & Blakemore, S. (2023). The relationship between perceived income inequality, adverse mental health and

interpersonal difficulties in UK adolescents. *Journal of Child Psychology and Psychiatry*, *64*(3), 417–425. https://doi.org/10.1111/jcpp.13719

Piotrowska, P. J., Stride, C. B., Croft, S. E., & Rowe, R. (2015). Socioeconomic status and antisocial behaviour among children and adolescents: A systematic review and meta-analysis. *Clinical Psychology Review, 35*, 47–55. https://doi.org/10.1016/j.cpr.2014.11.003#

Piotrowska, P. J., Stride, C. B., Maughan, B., Ford, T., McIntyre, N. A., & Rowe, R. (2023). Understanding the relationship between family income and conduct problems: findings from the mental health of children and young people survey. *Psychological Medicine, 53*(9), 3987–3994. https://doi.org/10.1017/S0033291722000654

Plomin, R. (1995). Genetics and Children's Experiences in the Family. *Journal of Child Psychology and Psychiatry*, *36*(1), 33–68. https://doi.org/10.1111/j.1469-7610.1995.tb01655.x

Plomin, R. (2011). Commentary: why are children in the same family so different? Non-shared environment three decades later. *International Journal of Epidemiology*, *40*(3), 582–592. https://doi.org/10.1093/ije/dyq144

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin, 84*(2), 309–322. https://doi.org/10.1037/0033-2909.84.2.309

Polderman, T. J. C., Benyamin, B., De Leeuw, C. A., Sullivan, P. F., Van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics*, *47*(7), 702–709. https://doi.org/10.1038/ng.3285

Poirier, C. S., Brendgen, M., Girard, A., Vitaro, F., Dionne, G., & Boivin, M. (2016). Friendship Experiences and Anxiety Among Children: A Genetically Informed Study. *Journal of Clinical Child and Adolescent Psychology*, *45*(5), 655–667. https://doi.org/10.1080/15374416.2014.987382

Power, R. A., & Pluess, M. (2015). Heritability estimates of the big dive personality traits based on common genetic variants. *Translational Psychiatry, 5*(7), e604-e604. https://doi.org/10.1038/tp.2015.96

Preacher, K. J. (2015). Advances in mediation analysis: A survey and synthesis of new developments. *Annual review of psychology, 66*, 825-852.

Raitasalo, K., Holmila, M., Jääskeläinen, M., & Santalahti, P. (2019). The effect of the severity of parental alcohol abuse on mental and behavioural disorders in children. *European Child & Adolescent Psychiatry*, *28*(7), 913–922. https://doi.org/10.1007/s00787-018-1253-6

Ramchandani, P. G., O'Connor, T. G., Evans, J., Heron, J., Murray, L., & Stein, A. (2008). The effects of pre- and postnatal depression in fathers: A natural experiment comparing the effects of exposure to depression on offspring. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *49*(10), 1069–1078. https://doi.org/10.1111/j.1469-7610.2008.02000.x

Rapee, R. M. (1997). Potential role of childrearing practices in the development of anxiety and depression. *Clinical Psychology Review*, *17*(1), 47–67. https://doi.org/10.1016/S0272-7358(96)00040-2

Reedtz, C., van Doesum, K., Signorini, G., Lauritzen, C., van Amelsvoort, T., van Santvoort, F., Young, A. H., Conus, P., Musil, R., Schulze, T., Berk, M., Stringaris, A., Piché, G., & de Girolamo, G. (2019). Promotion of Wellbeing for Children of Parents With

Mental Illness: A Model Protocol for Research and Intervention. *Frontiers in Psychiatry*, *10*. https://doi.org/10.3389/fpsyt.2019.00606

Reinelt, E., Aldinger, M., Stopsack, M., Schwahn, C., John, U., Baumeister, S. E., Grabe, H. J., & Barnow, S. (2014). High social support buffers the effects of 5-HTTLPR genotypes within social anxiety disorder. *European Archives of Psychiatry and Clinical Neuroscience*, *264*(5), 433–439. https://doi.org/10.1007/s00406-013-0481-5

Rice, F., Harold, G., & Thapar, A. (2002). The genetic aetiology of childhood depression: a review. *Journal of Child Psychology and Psychiatry*, *43*(1), 65–79. https://doi.org/10.1111/1469-7610.00004

Rice, F., Harold, G. T., Boivin, J., van den Bree, M., Hay, D. F., & Thapar, A. (2010). The links between prenatal stress and offspring development and psychopathology: disentangling environmental and inherited influences. *Psychological Medicine*, *40*(2), 335–345. https://doi.org/10.1017/S0033291709005911

Rijlaarsdam, J., Stevens, G. W. J. M., van der Ende, J., Hofman, A., Jaddoe, V. W. V., Mackenbach, J. P., Verhulst, F. C., & Tiemeier, H. (2013). Economic Disadvantage and Young Children's Emotional and Behavioral Problems: Mechanisms of Risk. *Journal of Abnormal Child Psychology*, *41*(1), 125–137. https://doi.org/10.1007/s10802-012-9655-2

Rodman, A. M., Vidal Bustamante, C. M., Dennison, M. J., Flournoy, J. C., Coppersmith, D. D. L., Nook, E. C., Worthington, S., Mair, P., & McLaughlin, K. A. (2021). A Year in the Social Life of a Teenager: Within-Persons Fluctuations in Stress, Phone Communication, and Anxiety and Depression. *Clinical Psychological Science*, *9*(5), 791–809. https://doi.org/10.1177/2167702621991804

Ronalds, G. A., De Stavola, B. L., & Leon, D. A. (2005). The cognitive cost of being a twin: evidence from comparisons within families in the Aberdeen children of the 1950s

cohort study. *British Medical Journal*, *331*(7528), 1306–1309. https://doi.org/10.1136/bmj.38633.594387.3A

Roos, L. E., Fisher, P. A., Shaw, D. S., Kim, H. K., Neiderhiser, J. M., Reiss, D., Natsuaki, M. N., & Leve, L. D. (2015). Inherited and environmental influences on a childhood co-occurring symptom phenotype: Evidence from an adoption study. *Development and Psychopathology*, *28*(1), 111–125. https://doi.org/10.1017/S0954579415000322

Rose, J., Roman, N., Mwaba, K., & Ismail, K. (2018). The relationship between parenting and internalizing behaviours of children: a systematic review. *Early Child Development and Care*, *188*(10), 1468–1486. https://doi.org/10.1080/03004430.2016.1269762

Rosenbaum, P. R., & Rubin, D. B. (1983). The central role of the propensity score in observational studies for causal effects. *Biometrika*, *70*, 41–55. https://doi.org/10.1093/biomet/70.1.41

Rubens, S. L., Fite, P. J., Gabrielli, J., Evans, S. C., Hendrickson, M. L., & Pederson, C. A. (2013). Examining Relations Between Negative Life Events, Time Spent in the United States, Language Use, and Mental Health Outcomes in Latino Adolescents. *Child & Youth Care Forum*, *42*(5), 389–402. https://doi.org/10.1007/s10566-013-9205-2

Rudolph, K. E., Shev, A., Paksarian, D., Merikangas, K. R., Mennitt, D. J., James, P., & Casey, J. A. (2019). Environmental noise and sleep and mental health outcomes in a nationally representative sample of urban US adolescents. *Environmental Epidemiology*, *3*(4), e056. https://doi.org/10.1097/EE9.0000000000000056

Rudolph, K. E., Stuart, E. A., Glass, T. A., & Merikangas, K. R. (2014). Neighborhood disadvantage in context: The influence of Urbanicity on the association between neighborhood disadvantage and adolescent emotional disorders. *Social Psychiatry and Psychiatric Epidemiology*, *49*(3), 467–475. https://doi.org/10.1007/s00127-013-0725-8

Rutter, M. (2005). Environmentally Mediated Risks for Psychopathology: Research Strategies and Findings. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*(1), 3–18. https://doi.org/10.1097/01.chi.0000145374.45992.c9

Rutter, M. (2007). Proceeding from observed correlation to causal inference: the use of natural experiments. *Perspectives on Psychological Science*, *2*(4), 377–395. https://doi.org/10.1111/j.1745-6916.2007.00050.x

Rutter, M., Dunn, J., Plomin, R., Simonoff, E., Pickles, A., Maughan, B., ... & Eaves, L. (1997). Integrating nature and nurture: Implications of person–environment correlations and interactions for developmental psychopathology. *Development and psychopathology*, *9*(2), 335-364.

Rutter, M. L. (1999). Psychosocial adversity and child psychopathology. *British Journal of Psychiatry*, *174*(6), 480–493. https://doi.org/10.1192/bjp.174.6.480

Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behavior. *Psychological bulletin, 127*(3), 291. https://doi.org/10.1037/0033-2909.127.3.291

Rutter, M. L., & Thapar, A. (2016). Using Natural Experiments to Test Environmental Mediation Hypotheses. *Developmental Psychopathology*, 1–27. https://doi.org/10.1002/9781119125556.devpsy104

Ryan, R. M., Claessens, A., & Markowitz, A. J. (2015). Associations Between Family Structure Change and Child Behavior Problems: The Moderating Effect of Family Income. *Child Development*, *86*(1), 112–127. https://doi.org/10.1111/cdev.12283

Moore, K., Sacks, V., Bandy, T., Murphy, D. (2014). Adverse childhood experiences: National and state-level prevalence. https://cms.childtrends.org/wp-content/uploads/2014/07/Fact-sheet-adverse-childhood-experiences_FINAL.pdf

Scaramella, L. V., Sohr-Preston, S. L., Callahan, K. L., & Mirabile, S. P. (2008). A Test of the Family Stress Model on Toddler-Aged Children's Adjustment Among Hurricane Katrina Impacted and Nonimpacted Low-Income Families. *Journal of Clinical Child and Adolescent Psychology*, *37*(3), 530–541. https://doi.org/10.1080/15374410802148202

Sameroff AJ, Chandler MJ: Reproductive risk and the continuum of caretaking casualty, in Review of Child Development Research. Edited by Horowitz FD. Chicago, IL, University of Chicago Press, 1975, pp 187–244

Sanders, M. R., Ralph, A., Sofronoff, K., Gardiner, P., Thompson, R., Dwyer, S., et al. (2008). Every family: A population approach to reducing behavioral and emotional problems in children making the transition to school. *The Journal of Primary Prevention*, *29*, 197–222.

Sandberg, S., Rutter, M., Pickles, A., McGuinness, D., & Angold, A. (2001). Do High-threat Life Events Really Provoke the Onset of Psychiatric Disorder in Children? *Journal of Child Psychology and Psychiatry*, *42*(4), 523–532. https://doi.org/10.1111/1469-7610.00746

Sampson, R. J. (2001). How do communities undergird or undermine human development? Relevant contexts and social mechanisms. In A. Booth & N. Crouter (Eds.), *Does it take a village? Community effects on children, adolescents, and families* (pp. 3–30). Mahwah, NJ: Erlbaum.

Scarr, S. (1969). Social Introversion-Extraversion as a Heritable Response. *Child Development*, *40*(3), 823–832. https://doi.org/10.2307/1127191

Schenck-Fontaine, A., & Panico, L. (2019). Many Kinds of Poverty: Three Dimensions of Economic Hardship, Their Combinations, and Children's Behavior

Problems. *Demography*, *56*(6), 2279–2305. https://doi.org/10.1007/s13524-019-00833-y

Schepman, K., Collishaw, S., Gardner, F., Maughan, B., Scott, J., & Pickles, A. (2011). Do changes in parent mental health explain trends in youth emotional problems? *Social Science & Medicine (1982)*, *73*(2), 293–300. https://doi.org/10.1016/j.socscimed.2011.05.015

Schermelleh-Engel, K., Moosbrugger, H., & Müller, H. (2003). Evaluating the Fit of Structural Equation Models: Tests of Significance and Descriptive Goodness-of-Fit Measures. In *Methods of Psychological Research Online* (Vol. 8, Issue 2).

Schermerhorn, A. C., D'Onofrio, B. M., Turkheimer, E., Ganiban, J. M., Spotts, E. L., Lichtenstein, P., Reiss, D., & Neiderhiser, J. M. (2011). A genetically informed study of associations between family functioning and child psychosocial adjustment. *Developmental Psychology*, *47*(3), 707–725. https://doi.org/10.1037/a0021362

Schiele, M. A., & Domschke, K. (2018). Epigenetics at the crossroads between genes, environment and resilience in anxiety disorders. *Genes, Brain and Behavior*, *17*(3). https://doi.org/10.1111/gbb.12423

Schore, A. N. (2001). The effects of early relational trauma on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, *22*(1-2), 201–269. https://doi.org/10.1002/1097-0355(200101/04)22:1<201::AID-IMHJ8>3.0.CO;2-9

Schulz, S., Nelemans, S. A., Oldehinkel, A. J., Meeus, W., & Branje, S. (2021). Examining intergenerational transmission of psychopathology: Associations between parental and adolescent internalizing and externalizing symptoms across adolescence. *Developmental Psychology*, *57*(2), 269–283. https://doi.org/10.1037/dev0001144

Shelleby, E. C., Votruba-Drzal, E., Shaw, D. S., Dishion, T. J., Wilson, M. N., & Gardner, F. (2014). Income and Children's Behavioral Functioning: A Sequential Mediation

Analysis. *Journal of Family Psychology*, *28*(6), 936–946. https://doi.org/10.1037/fam0000035

Shepherd P, Gilbert E. (2019, February). *Millennium Cohort Study Ethical Review and Consent*. Retrieved March 6, 2022 from https://cls.ucl.ac.uk/wp-content/uploads/2017/07/MCS-Ethical-Approval-and-Consent-2019.pdf

Seabury, S. A., Axeen, S., Pauley, G., Tysinger, B., Schlosser, D., Hernandez, J. B., Heun-Johnson, H., Zhao, H., & Goldman, D. P. (2019). Measuring the lifetime costs of serious mental illness and the mitigating effects of educational attainment. *Health Affairs*, *38*(4), 652–659. https://doi.org/10.1377/hlthaff.2018.05246

Sedlak, Andrea J., Jane Mettenburg, Monica Basena, Ian Petta, Karla McPerson, Angela Green, and Spencer Li. 2010. "Fourth National Incidence Study of Child Abuse and Neglect ðNIS- 4Þ." Report. US Department ofHealth and Human Services, Washington, DC.

Selig, J. P., & Preacher, K. J. (2009). Mediation Models for Longitudinal Data in Developmental Research. *Research in Human Development*, *6*(2–3), 144–164. https://doi.org/10.1080/15427600902911247

Sen, B., & Swaminathan, S. (2007). Maternal prenatal substance use and behavior problems among children in the US. *The Journal of Mental Health Policy and Economics, 10,* 189-206.

Setia, M. S. (2016). Methodology series module 3: Cross-sectional studies. *Indian Journal of Dermatology*, *61*(3), 261–264. https://doi.org/10.4103/0019-5154.182410

Shadish, W. R., Cook, T. D., Campbell, D. T., & Reichardt, C. S. (2002). Experimental and quasi-experimental designs for generalized causal inference. *Social Service Review*, *70*(3), 510–514.

Shah, B. R., Laupacis, A., Hux, J. E., & Austin, P. C. (2005). Propensity score methods gave similar results to traditional regression modeling in observational studies: a systematic review. *Journal of Clinical Epidemiology*, *58*(6), 550–559. https://doi.org/10.1016/j.jclinepi.2004.10.016

Shepherd P, Gilbert E. (2019, July). *Millennium Cohort Study Ethical Review and Consent*. https://cls.ucl.ac.uk/wp-content/uploads/2017/07/MCS-Ethical-Approval-and-Consent-2019.pdf

Shonk, S. M., & Cicchetti, D. (2001). Maltreatment, competency deficits, and risk for academic and behavioral maladjustment. *Developmental Psychology*, *37*(1), 3–17. https://doi.org/10.1037/0012-1649.37.1.3

Sifaki, M., Midouhas, E., Papachristou, E., & Flouri, E. (2021). Reciprocal relationships between paternal psychological distress and child internalising and externalising difficulties from 3 to 14 years: a cross-lagged analysis. *European Child & Adolescent Psychiatry*, *30*(11), 1695–1708. https://doi.org/10.1007/s00787-020-01642-0

Silberg, J. L., Copeland, W., Linker, J., Moore, A. A., Roberson-Nay, R., & York, T. P. (2016). Psychiatric outcomes of bullying victimization: a study of discordant monozygotic twins. *Psychological Medicine*, *46*(9), 1875–1883. https://doi.org/10.1017/S0033291716000362

Silberg, J. L., & Eaves, L. J. (2004). Analysing the contributions of genes and parent-child interaction to childhood behavioral and emotional problems: a model for the children of twins. *Psychological Medicine*, *34*(2), 347–356. https://doi.org/10.1017/S0033291703008948

Silberg, J. L., Maes, H., & Eaves, L. J. (2010). Genetic and environmental influences on the transmission of parental depression to children's depression and conduct disturbance: an

extended Children of Twins study. *Journal of Child Psychology and Psychiatry*, *51*(6), 734–744. https://doi.org/10.1111/j.1469-7610.2010.02205.x

Silberg, J. L., Maes, H., & Eaves, L. J. (2012). Unraveling the effect of genes and environment in the transmission of parental antisocial behavior to children's conduct disturbance, depression and hyperactivity. *Journal of Child Psychology and Psychiatry*, *53*(6), 668–677. https://doi.org/10.1111/j.1469-7610.2011.02494.x

Singham, T., Viding, E., Schoeler, T., Arseneault, L., Ronald, A., Cecil, C. M., McCrory, E., Rijsdijk, F., & Pingault, J.-B. (2017). Concurrent and Longitudinal Contribution of Exposure to Bullying in Childhood to Mental Health The Role of Vulnerability and Resilience. *JAMA Psychiatry (Chicago, Ill.)*, *74*(11), 1112–1119. https://doi.org/10.1001/jamapsychiatry.2017.2678

Sithisarn, T., Granger, D. T., & Bada, H. S. (2012). Consequences of prenatal substance use. *International Journal of Adolescent Medicine and Health*, *24*(2), 105–112. https://doi.org/10.1515/IJAMH.2012.016

Skinner, H., Steinhauer, P., & Sitarenios, G. (2000). Family assessment measure (FAM) and process model of family functioning. *Journal of Family Therapy*, *22*(2), 190–210. https://doi.org/10.1111/1467-6427.00146

Smith, V. C., & Wilson, C. R. (2016). Families affected by parental substance use. *Pediatrics (Evanston)*, *138*(2), e1. https://doi.org/10.1542/peds.2016-1575

Speyer, L. G. (2021*) Co-occurring Mental Health Problems across Childhood and Adolescence: Within-Person Developmental Relations, Mediators and Antecedents* (Doctoral thesis, The University of Edinburgh). Edinburgh Research Archive. https://era.ed.ac.uk/handle/1842/38421

Speyer, L. G., Hall, H. A., Hang, Y., Hughes, C., & Murray, A. L. (2022). Within-family relations of mental health problems across childhood and adolescence. *Journal of Child Psychology and Psychiatry*. https://doi.org/10.1111/jcpp.13572

Speyer, L. G., Hang, Y., Hall, H. A., & Murray, A. L. (2022). The role of harsh parenting practices in early-to middle-childhood socioemotional development: An examination in the Millennium Cohort Study. *Child Development, 93*(5), 1304-1317.

Spremo, M. (2020). Children and divorce. *Psychiatria Danubina, 32*(suppl. 3), 353–359.

Stanger, C., & Lewis, M. (1993). Agreement among parents, teachers, and children on internalizing and externalizing behavior problems. *Journal of clinical child psychology, 22*(1), 107-116.

Stewart, M. B. (1983). On Least Squares Estimation when the Dependent Variable is Grouped. *The Review of Economic Studies*, *50*(4), 737–753. https://doi.org/10.2307/2297773

Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. *Psychological Bulletin*, *125*(4), 392–409. https://doi.org/10.1037/0033-2909.125.4.39

Su, X., Li, W., Lv, L., Li, X., Yang, J., Luo, X. J., & Liu, J. (2021). Transcriptome-wide association study provides insights into the genetic component of gene expression in anxiety. *Frontiers in Genetics*, *12*, 1–12. https://doi.org/10.3389/fgene.2021.740134

Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic Epidemiology of Major Depression: Review and Meta-Analysis. *The American Journal of Psychiatry*, *157*(10), 1552–1562. https://doi.org/10.1176/appi.ajp.157.10.1552

Sun, Y., & Li, Y. (2002). Children's well-being during parents' marital disruption process: a pooled time-series analysis. *Journal of Marriage and Family*, *64*(2), 472–488. https://doi.org/10.1111/j.1741-3737.2002.00472.x

Sundbakk, L. M., Wood, M., Gran, J. M., & Nordeng, H. (2019). Impact of prenatal exposure to benzodiazepines and z-hypnotics on behavioral problems at 5 years of age: A study from the Norwegian Mother and Child Cohort Study. *PLoS ONE*, *14*(6), 1–15. https://doi.org/10.1371/journal.pone.0217830

Sweeney, S., & Wilson, C. (2023). Parental anxiety and offspring development: a systematic review. *Journal of Affective Disorders*, *327*, 64–78. https://doi.org/10.1016/j.jad.2023.01.128

Thapar, A., & Rutter, M. L. (2019). Do natural experiments have an important future in the study of mental disorders? *Psychological Medicine*, *49*(7), 1079–1088. https://doi.org/10.1017/S0033291718003896

Torvik, F. A., Eilertsen, E. M., McAdams, T. A., Gustavson, K., Zachrisson, H. D., Brandlistuen, R., Gjerde, L. C., Havdahl, A., Stoltenberg, C., Ask, H., & Ystrom, E. (2020). Mechanisms linking parental educational attainment with child ADHD, depression, and academic problems: a study of extended families in The Norwegian Mother, Father and Child Cohort Study. *Journal of Child Psychology and Psychiatry*, *61*(9), 1009–1018. https://doi.org/10.1111/jcpp.13197

Toseeb, U., Oginni, O., Rowe, R., & Patalay, P. (2022). Measurement invariance of the strengths and difficulties questionnaire across socioeconomic status and ethnicity from ages 3 to 17 years: A population cohort study. *Plos One*, *17*(12), e0278385. https://doi.org/10.1371/journal.pone.0278385

Tingleff, T., Räisänen, S., Vikanes, Å., Sandvik, L., Sugulle, M., Murzakanova, G., & Laine, K. (2023). Different pathways for preterm birth between singleton and twin pregnancies: a population-based registry study of 481 176 nulliparous women. *BJOG : An International Journal of Obstetrics and Gynaecology*, *130*(4), 387–395. https://doi.org/10.1111/1471-0528.17344

Tyrell, F. A., Yates, T. M., Reynolds, C. A., Fabricius, W. V., & Braver, S. L. (2019). The unique effects of maternal and paternal depressive symptoms on youth's symptomatology: Moderation by family ethnicity, family structure, and child gender. *Development and Psychopathology*, *31*(4), 1213–1226. https://doi.org/10.1017/S0954579418000846

Usami, S., Murayama, K., & Hamaker, E. L. (2019). A Unified Framework of Longitudinal Models to Examine Reciprocal Relations. *Psychological Methods*, *24*(5), 637–657. https://doi.org/10.1037/met0000210

Usami, S. (2021). On the Differences between General Cross-Lagged Panel Model and Random-Intercept Cross-Lagged Panel Model: Interpretation of Cross-Lagged Parameters and Model Choice. *Structural Equation Modeling*, *28*(3), 331–344. https://doi.org/10.1080/10705511.2020.1821690

Van Der Bruggen, C. O., Stams, G. J. J. M., & Bögels, S. M. (2008). Research review: the relation between child and parent anxiety and parental control: a meta-analytic review. *Journal of Child Psychology and Psychiatry*, *49*(12), 1257–1269. https://doi.org/10.1111/j.1469-7610.2008.01898.x

Van der Kolk, B. A. (2005). Developmental trauma disorder: toward a rational diagnosis for children with complex trauma histories. *Psychiatric Annals*, *35*(5), 401–408. https://doi.org/10.3928/00485713-20050501-06

van der Voort, A., Linting, M., Juffer, F., Bakermans-Kranenburg, M. J., Schoenmaker, C., & van IJzendoorn, M. H. (2014). The Development of Adolescents' Internalizing Behavior: Longitudinal Effects of Maternal Sensitivity and Child Inhibition. *Journal of Youth and Adolescence*, *43*(4), 528–540. https://doi.org/10.1007/s10964-013-9976-7

Van Zalk, N., Van Zalk, M., Kerr, M., & Stattin, H. (2011). Social anxiety as a basis for friendship selection and socialization in adolescents' social networks. *Journal of Personality*, *79*(3), 499–526. https://doi.org/10.1111/j.1467-6494.2011.00682.x

Vidal Bustamante, C. M., Rodman, A. M., Dennison, M. J., Flournoy, J. C., Mair, P., & McLaughlin, K. A. (2020). Within-person fluctuations in stressful life events, sleep, and anxiety and depression symptoms during adolescence: a multiwave prospective study. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *61*(10), 1116–1125. https://doi.org/10.1111/jcpp.13234

Vidal-Ribas, P., & Stringaris, A. (2021). How and why are irritability and depression linked?. *Child and Adolescent Psychiatric Clinics*, *30*(2), 401-414.

Villarreal, D. L., & Nelson, J. A. (2018). Longitudinal associations between family members' internalizing symptoms across middle childhood. *Journal of Family Psychology*, *32*(3), 419–424. https://doi.org/10.1037/fam0000399

Vitaro, F., Brendgen, M., & Arseneault, L. (2009). The discordant MZ-twin method: one step closer to the holy grail of causality. *International Journal of Behavioral Development*, *33*(4), 376–382. https://doi.org/10.1177/0165025409340805

Vitaro, F., Brendgen, M., Boivin, M., Cantin, S., Dionne, G., Tremblay, R. E., Girard, A., & Pérusse, D. (2011). A Monozygotic Twin Difference Study of Friends' Aggression and Children's Adjustment Problems. *Child Development*, *82*(2), 617–632. https://doi.org/10.1111/j.1467-8624.2010.01570.x

Votruba-Drzal, E. (2006). Economic Disparities in Middle Childhood Development. *Developmental Psychology*, *42*(6), 1154–1167. https://doi.org/10.1037/0012-1649.42.6.1154

Wadsworth, M. E., & Achenbach, T. M. (2005). Explaining the Link Between Low Socioeconomic Status and Psychopathology. *Journal of Consulting and Clinical Psychology*, *73*(6), 1146–1153. https://doi.org/10.1037/0022-006X.73.6.1146

Wadsworth, M. E., Raviv, T., Compas, B. E., & Connor-Smith, J. K. (2005). Parent and adolescent responses to poverty-related stress: tests of mediated and moderated coping models. *Journal of Child and Family Studies*, *14*(2), 283–298. https://doi.org/10.1007/s10826-005-5056-2

Wadsworth, M. E., Raviv, T., Santiago, C. D., & Etter, E. M. (2011). Testing the Adaptation to Poverty-Related Stress Model: Predicting Psychopathology Symptoms in Families Facing Economic Hardship. *Journal of Clinical Child and Adolescent Psychology*, *40*(4), 646–657. https://doi.org/10.1080/15374416.2011.581622

Wang, J., Li, M., Sun, W., Ma, P., & Gong, H. (2023). The relationship between harsh parenting and depression in adolescents: the chain mediation effect of rumination and bullying. *Scientific Reports*, 1–9. https://doi.org/10.1038/s41598-023-48138-w

Weaver, J. M., & Schofield, T. J. (2015). Mediation and Moderation of Divorce Effects on Children's Behavior Problems. *Journal of Family Psychology*, *29*(1), 39–48. https://doi.org/10.1037/fam0000043

Webb, C. (2023). In Defence of Ordinary Help: Estimating the effect of Early Help/Family Support Spending on Children in Need Rates in England using ALT-SR. *Journal of Social Policy*, *52*(2), 396–423. https://doi.org/10.1017/S0047279421000696

Wickrama, K. A. S., & Bryant, C. M. (2003). Community Context of Social Resources and Adolescent Mental Health. *Journal of Marriage and Family*, *65*(4), 850–866. https://doi.org/10.1111/j.1741-3737.2003.00850.x

Wilkinson, P. (2009). Conceptualization about internalizing problems in children and adolescents. *Ciência & Saude Coletiva*, *14*(2), 373–381. https://doi.org/10.1590/S1413-81232009000200007

Williams, L. D., & Lawrence Aber, J. (2016). Testing for plausibly causal links between parental bereavement and child socio-emotional and academic outcomes: a propensity-score matching model. *Journal of abnormal child psychology*, *44*, 705-718.

World Health Organization. (2021, October 12). *Mental health of adolescents.* https://www.who.int/news-room/fact-sheets/detail/adolescent-mental-health#:~:text=Globally%2C%20it%20is%20estimated%20that,remain%20largely%20unrecognized%20and%20untreated.

Xerxa, Y., Rescorla, L. A., Ende, J., Hillegers, M. H. J., Verhulst, F. C., & Tiemeier, H. (2021). From Parent to Child to Parent: Associations Between Parent and Offspring Psychopathology. *Child Development*, *92*(1), 291–307. https://doi.org/10.1111/cdev.13402

Xia, L., & Yao, S. (2015). The Involvement of Genes in Adolescent Depression: A Systematic Review. *Frontiers in Behavioral Neuroscience*, *9*(December), 1–9. https://doi.org/10.3389/fnbeh.2015.00329

Xu, J., Ni, S., Ran, M., & Zhang, C. (2017). The relationship between parenting styles and adolescents' social anxiety in migrant families: a study in Guangdong, China. *Frontiers in Psychology*, *8*, 626–626. https://doi.org/10.3389/fpsyg.2017.00626

Yan, N., Liu, Y., Ansari, A., Li, K., & Li, X. (2021). Mothers' Depressive Symptoms and Children's Internalizing and Externalizing Behaviors: Examining Reciprocal Trait-State Effects from Age 2 to 15. *Child Development*, *92*(6), 2496–2508. https://doi.org/10.1111/cdev.13609

Yang, J., Huebner, E. S., & Tian, L. (2021). Transactional processes between childhood maltreatment and depressive symptoms from middle childhood to early adolescence: Locus of control as a mediator. *Journal of Affective Disorders*, *295*, 216–224. https://doi.org/10.1016/j.jad.2021.08.040

Yang, M., Carson, C., Creswell, C., & Violato, M. (2023). Child mental health and income gradient from early childhood to adolescence: Evidence from the UK. *SSM - Population Health*, *24*, 101534–101534. https://doi.org/10.1016/j.ssmph.2023.101534

Yeung, W. J., Linver, M. R., & Brooks-Gunn, J. (2002). How Money Matters for Young Children's Development: Parental Investment and Family Processes. *Child Development*, *73*(6), 1861–1879. https://doi.org/10.1111/1467-8624.t01-1-00511

Zachrisson, H. D., & Dearing, E. (2015). Family Income Dynamics, Early Childhood Education and Care, and Early Child Behavior Problems in Norway. *Child Development*, *86*(2), 425–440. https://doi.org/10.1111/cdev.12306

Zarakoviti, E., Shafran, R., Papadimitriou, D., & Bennett, S. D. (2021). The Efficacy of Parent Training Interventions for Disruptive Behavior Disorders in Treating Untargeted Comorbid Internalizing Symptoms in Children and Adolescents : A Systematic Review. *Clinical Child and Family Psychology Review*, *24*(3), 542–552. https://doi.org/10.1007/s10567-021-00349-1

Zhou, M., Sun, X., Huang, L., Zhang, G., Kenny, K., Xue, H., Auden, E., & Rozelle, S. (2018). Parental migration and left-behind children's depressive symptoms: Estimation based on a nationally-representative panel dataset. *International Journal of Environmental Research and Public Health*, *15*(6), 1069. https://doi.org/10.3390/ijerph15061069

Zwierzynska, K., Wolke, D., & Lereya, T. S. (2013). Peer victimization in childhood and internalizing problems in adolescence: a prospective longitudinal study. *Journal of*

Abnormal Child Psychology, *41*(2), 309–323. https://doi.org/10.1007/s10802-012-9678-8

Appendix A.

Inclusion and Exclusion Criteria.

1) English language only

- Full text of an article in English.

2) Empirical and quantitative studies

- Exclude qualitative studies.
- Exclude conference papers.
- Exclude reviews.
- Exclude non-peer-reviewed articles.

3) General population-based samples

- Studies trying to recruit general population sampling (studies selected based on sex or ethnicity, age or number of children in the family will be included) will be included.
- Studies aiming to make general population but still including some biases will be included.
- Studies selecting participants regarding to specific characteristics that is of interest to them (e.g., low SES, children with a diagnosis) will be excluded. However, this was not applied to studies that used RCT designs to manipulate putative risk factors and to the studies where the general population is not sampled in order to allow the design to work (e.g, twin, sibling, adoption studies).

4) Age range: 0-18

- Only studies involving children and adolescents aged 0-18, or with a mean age within the range of 0-18, will be included.

5) Measurement of environmental risk factor(s)

- Included studies must measure one or more environmental risk factors (e.g., parental divorce, poor parenting, parental psychopathology, abuse, maternal smoking etc.) Studies not measuring at least one of the specific environmental risk factors will be excluded. This includes studies inferring unspecified environmental effects from twin correlations.
- Environmental risk factors included will be the factors participants are exposed to rather than those which depend on the participants' behaviour, such as substance misuse.

6) Measurement of depression, anxiety and related psychopathology

- Depressive and anxious psychopathology, their constituent symptoms, and related measures, for example emotional problems, depressed mood, worrying excessively.
- Anxious or depressive psychopathology should be measured separately from other any disorders (e.g., not mixed into a total psychopathology score). However, any score including both anxious and depressive psychopathology, or related symptoms such as withdrawn, and somatic symptoms will be included.

7) Study designs included:

- Quasi experiment /natural experiment studies (e.g., twin, adoption, fixed effect, etc.), and within-family designs testing the relation between at least one specific environmental risk factor (as in [5], above) and the children or adolescents' anxious or depressive psychopathology, as in [6].
- Studies using genetically sensitive data such as twin data, however not making genetically sensitive analyses, or not measuring a specific environmental factor, will be excluded.

- Experimental studies including randomized control trials manipulating environmental risk factors will be included.

Appendix B

Table B1. Descriptive statistics

	N	*Mean*	*SD*	*Min*	*Max*	*Skew*	*Kurtosis*	*Omega*
Age 3 internalising problems	10385	2.85	2.49	0	18	1.24	1.88	.61
Age 5 internalising problems	10847	2.51	2.52	0	18	1.54	3.05	.79
Age 7 internalising problems	10620	2.72	2.78	0	18	1.49	2.57	.67
Age 11 internalising problems	10797	3.18	3.12	0	19	1.40	2.10	.80
Age 14 internalising problems	11393	3.79	3.42	0	19	1.24	1.46	.72
Age 3 externalising problems	10386	6.57	3.77	0	20	.59	.03	.82
Age 5 externalising problems	10844	4.67	3.38	0	20	.87	.58	.78
Age 7 externalising problems	10621	4.61	3.55	0	20	.90	.51	.83
Age 11 externalising problems	10797	4.38	3.52	0	20	1.02	.89	.78
Age 14 externalising problems	11392	4.40	3.57	0	20	1.06	1.08	.82
Age 3 maternal distress	9389	3.22	3.67	0	24	1.892	4.30	NA
Age 5 maternal distress	10372	3.12	3.75	0	24	1.960	4.74	NA
Age 7 maternal distress	10168	3.08	3.80	0	24	1.958	4.58	NA
Age 11 maternal distress	10234	3.95	4.31	0	24	1.663	3.04	NA
Age 14 maternal distress	10553	4.35	4.22	0	24	1.459	2.32	NA
Age 3 paternal distress	7231	2.89	3.07	0	24	1.755	4.28	NA
Age 5 paternal distress	7485	2.96	3.29	0	24	1.896	4.95	NA
Age 7 paternal distress	6948	2.95	3.34	0	24	1.775	3.82	NA
Age 11 paternal distress	6733	3.79	3.85	0	24	1.628	3.28	NA
Age 14 paternal distress	6320	3.67	3.65	0	24	1.716	4.07	NA
Age 3 family income	10755	341.93	223.20	14	1362	1.188	.024	NA
Age 5 family income	11072	361.62	221.62	12	1283	1.124	.023	NA
Age 7 family income	10733	395.09	231.92	13	1283	1.059	.024	NA
Age 11 family income	11231	415.68	178.60	66	1163	.349	.023	NA
Age 14 family income	11847	409.86	177.72	80	1154	.414	.023	NA

Omega, McDonalds Omega. Sum-score provided by the Millennium Cohort Study were used for parental distress value, thus a value of internal consistency for the parental mental health measure was not available. Kessler (K6) scale, however, has reported good reliability in Flouri et al (2019).

Table B2. Standardised autoregressive and cross-lagged parameters from RICLMP for female's internalising outcome

Parameter	***Estimate***	***SE***	***P***
Age 14 int on age 11 int	0.429	0.026	**<.001***
Age 14 int on age 11 mat	0.022	0.024	0.360
Age 14 int on age 11 pat	0.027	0.036	0.465
Age 14 int on age 11 inc	-0.020	0.028	0.489
Age 14 int on age 7 inc	-0.014	0.019	0.453
Age 14 mat on age 11 int	0.050	0.026	**0.052**
Age 14 mat on age 11 mat	0.323	0.036	**<.001***
Age 14 mat on age 11 pat	-0.028	0.052	0.589
Age 14 mat on age 11 inc	-0.028	0.032	0.374
Age 14 pat on age 11 int	0.025	0.037	0.498
Age 14 pat on age 11 mat	0.020	0.045	0.660
Age 14 pat on age 11 pat	0.290	0.057	**<.001***
Age 14 pat on age 11 inc	-0.052	0.039	0.184
Age 14 inc on age 11 int	0.034	0.028	0.226
Age 14 inc on age 11 mat	-0.073	0.029	**0.011***
Age 14 inc on age 11 pat	-0.037	0.027	0.179
Age 14 inc on age 11 inc	0.495	0.044	**<.001***
Age 11 int on age 7 int	0.298	0.027	**<.001***
Age 11 int on age 7 mat	0.019	0.031	0.543
Age 11 int on age 7 pat	0.022	0.031	0.468
Age 11 int on age 7 inc	-0.045	0.021	**0.034***
Age 11 int on age 5 inc	-0.022	0.024	0.345
Age 11 mat on age 7 int	0.084	0.028	**0.002***
Age 11 mat on age 7 mat	0.108	0.045	**0.017***
Age 11 mat on age 7 pat	-0.023	0.042	0.584
Age 11 mat on age 7 inc	-0.106	0.019	**<.001***
Age 11 pat on age 7 int	0.052	0.041	0.211
Age 11 pat on age 7 mat	0.025	0.058	0.674
Age 11 pat on age 7 pat	0.210	0.057	**<.001***
Age 11 pat on age 7 inc	-0.048	0.025	0.059
Age 11 inc on age 7 int	0.045	0.036	0.208
Age 11 inc on age 7 mat	0.025	0.037	0.499
Age 11 inc on age 7 pat	-0.038	0.044	0.377
Age 11 inc on age 7 inc	0.126	0.039	**0.001***
Age 7 int on age 5 int	0.253	0.042	**<.001***
Age 7 int on age 5 mat	0.014	0.037	0.710
Age 7 int on age 5 pat	-0.026	0.035	0.457
Age 7 int on age 5 inc	-0.001	0.024	0.982
Age 7 int on age 3 inc	0.019	0.023	0.410
Age 7 mat on age 5 int	-0.009	0.043	0.835
Age 7 mat on age 5 mat	0.174	0.048	**<.001***
Age 7 mat on age 5 pat	0.034	0.047	0.473
Age 7 mat on age 5 inc	0.021	0.024	0.370
Age 7 pat on age 5 int	-0.004	0.047	0.924
Age 7 pat on age 5 mat	0.142	0.050	**0.005***

Age 7 pat on age 5 pat	0.148	0.063	**0.020***
Age 7 pat on age 5 inc	-0.021	0.027	0.437
Age 7 inc on age 5 int	-0.011	0.019	0.574
Age 7 inc on age 5 mat	-0.057	0.016	**<.001***
Age 7 inc on age 5 pat	-0.007	0.026	0.778
Age 7 inc on age 5 inc	0.506	0.034	**<.001***
Age 5 int on age 3 int	0.110	0.041	**0.007***
Age 5 int on age 3 mat	-0.078	0.038	**0.037***
Age 5 int on age 3 pat	0.036	0.042	0.386
Age 5 int on age 3 inc	-0.029	0.023	0.217
Age 5 mat on age 3 int	-0.045	0.042	0.292
Age 5 mat on age 3 mat	0.108	0.052	**0.037***
Age 5 mat on age 3 pat	0.016	0.049	0.747
Age 5 mat on age 3 inc	-0.015	0.018	0.406
Age 5 pat on age 3 int	-0.106	0.048	**0.027***
Age 5 pat on age 3 mat	0.018	0.050	0.720
Age 5 pat on age 3 pat	0.008	0.076	0.918
Age 5 pat on age 3 inc	-0.001	0.033	0.983
Age 5 inc on age 3 int	-0.013	0.016	0.434
Age 5 inc on age 3 mat	0.006	0.020	0.756
Age 5 inc on age 3 pat	0.021	0.020	0.305
Age 5 inc on age 3 inc	0.507	0.036	**<.001***

Notes: int, internalising problems; inc, family income; mat, maternal distress; pat, paternal distress; Correlations are significant at $p < .05$. Children's ages are in accordance with their respective median ages at the time of data collection.

Table B3. Standardised autoregressive and cross-lagged parameters from RICLMP for female's externalising outcome

Parameter	***Estimate***	***SE***	***P***
Age 14 ext on age 11 ext	0.355	0.036	**<.001***
Age 14 ext on age 11 mat	0.065	0.027	**0.017***
Age 14 ext on age 11 pat	-0.021	0.044	0.640
Age 14 ext on age 11 inc	-0.032	0.033	0.331
Age 14 ext on age 7 inc	-0.030	0.022	0.170
Age 14 mat on age 11 ext	0.057	0.035	0.108
Age 14 mat on age 11 mat	0.323	0.034	**<.001***
Age 14 mat on age 11 pat	-0.043	0.051	0.403
Age 14 mat on age 11 inc	-0.029	0.032	0.371
Age 14 pat on age 11 ext	0.030	0.041	0.459
Age 14 pat on age 11 mat	0.016	0.046	0.730
Age 14 pat on age 11 pat	0.285	0.058	**<.001***
Age 14 pat on age 11 inc	-0.050	0.040	0.205
Age 14 inc on age 11 ext	0.043	0.034	0.211
Age 14 inc on age 11 mat	-0.080	0.030	**0.008***
Age 14 inc on age 11 pat	-0.041	0.028	0.137
Age 14 inc on age 11 inc	0.487	0.046	**<.001***
Age 11 ext on age 7 ext	0.352	0.030	**<.001***
Age 11 ext on age 7 mat	0.046	0.036	0.201
Age 11 ext on age 7 pat	0.000	0.035	0.998
Age 11 ext on age 7 inc	-0.020	0.024	0.397
Age 11 ext on age 5 inc	-0.009	0.024	0.688
Age 11 mat on age 7 ext	0.097	0.029	**0.001***
Age 11 mat on age 7 mat	0.110	0.045	**0.014***
Age 11 mat on age 7 pat	-0.038	0.042	0.355
Age 11 mat on age 7 inc	-0.107	0.020	**<.001***
Age 11 pat on age 7 ext	0.065	0.040	0.108
Age 11 pat on age 7 mat	0.028	0.059	0.629
Age 11 pat on age 7 pat	0.199	0.058	**0.001***
Age 11 pat on age 7 inc	-0.049	0.025	**0.047***
Age 11 inc on age 7 ext	0.069	0.040	0.088
Age 11 inc on age 7 mat	0.045	0.039	0.250
Age 11 inc on age 7 pat	-0.038	0.045	0.397
Age 11 inc on age 7 inc	0.118	0.041	**0.004***
Age 7 ext on age 5 ext	0.352	0.030	**<.001***
Age 7 ext on age 5 mat	0.027	0.031	0.378
Age 7 ext on age 5 pat	-0.045	0.039	0.247
Age 7 ext on age 5 inc	-0.025	0.025	0.321
Age 7 ext on age 3 inc	0.000	0.020	0.995
Age 7 mat on age 5 ext	0.008	0.039	0.835
Age 7 mat on age 5 mat	0.161	0.046	**0.001***
Age 7 mat on age 5 pat	0.015	0.048	0.750
Age 7 mat on age 5 inc	0.027	0.025	0.277
Age 7 pat on age 5 ext	0.008	0.042	0.845
Age 7 pat on age 5 mat	0.121	0.053	**0.022***

Age 7 pat on age 5 pat	0.145	0.065	**0.026***
Age 7 pat on age 5 inc	-0.015	0.028	0.594
Age 7 inc on age 5 ext	-0.025	0.023	0.274
Age 7 inc on age 5 mat	-0.051	0.017	**0.002***
Age 7 inc on age 5 pat	-0.010	0.026	0.716
Age 7 inc on age 5 inc	0.505	0.034	**<.001***
Age 5 ext on age 3 ext	0.259	0.029	**<.001***
Age 5 ext on age 3 mat	-0.014	0.036	0.700
Age 5 ext on age 3 pat	0.005	0.043	0.910
Age 5 ext on age 3 inc	-0.023	0.025	0.358
Age 5 mat on age 3 ext	-0.009	0.030	0.773
Age 5 mat on age 3 mat	0.105	0.052	**0.042***
Age 5 mat on age 3 pat	0.011	0.051	0.832
Age 5 mat on age 3 inc	-0.016	0.018	0.389
Age 5 pat on age 3 ext	-0.051	0.042	0.226
Age 5 pat on age 3 mat	0.018	0.050	0.709
Age 5 pat on age 3 pat	0.009	0.076	0.910
Age 5 pat on age 3 inc	-0.003	0.033	0.917
Age 5 inc on age 3 ext	0.006	0.017	0.736
Age 5 inc on age 3 mat	0.004	0.021	0.838
Age 5 inc on age 3 pat	0.024	0.020	0.234
Age 5 inc on age 3 inc	0.507	0.036	**<.001***

Notes: ext, externalising problems; inc, family income; mat, maternal distress; pat, paternal distress; Correlations are significant at $p < .05$. Children's ages are in accordance with their respective median ages at the time of data collection.

Table B4. Standardised autoregressive and cross-lagged parameters from RICLMP for male's internalising outcome

Parameter	***Estimate***	***SE***	***P***
Age 14 int on age 11 int	0.494	0.020	**<.001***
Age 14 int on age 11 mat	-0.020	0.028	0.477
Age 14 int on age 11 pat	0.036	0.051	0.482
Age 14 int on age 11 inc	-0.060	0.043	0.161
Age 14 int on age 7 inc	-0.026	0.026	0.326
Age 14 mat on age 11 int	0.087	0.022	**<.001***
Age 14 mat on age 11 mat	0.312	0.037	**<.001***
Age 14 mat on age 11 pat	-0.003	0.047	0.957
Age 14 mat on age 11 inc	-0.024	0.027	0.360
Age 14 pat on age 11 int	0.094	0.039	**0.016***
Age 14 pat on age 11 mat	0.012	0.043	0.776
Age 14 pat on age 11 pat	0.381	0.056	**<.001***
Age 14 pat on age 11 inc	0.028	0.039	0.469
Age 14 inc on age 11 int	-0.005	0.027	0.855
Age 14 inc on age 11 mat	-0.058	0.024	**0.018***
Age 14 inc on age 11 pat	-0.062	0.030	**0.041***
Age 14 inc on age 11 inc	0.505	0.040	**<.001***
Age 11 int on age 7 int	0.377	0.028	**<.001***
Age 11 int on age 7 mat	0.033	0.028	0.251
Age 11 int on age 7 pat	0.030	0.036	0.413
Age 11 int on age 7 inc	-0.006	0.022	0.802
Age 11 int on age 5 inc	-0.018	0.020	0.371
Age 11 mat on age 7 int	0.063	0.037	0.086
Age 11 mat on age 7 mat	0.251	0.043	**<.001***
Age 11 mat on age 7 pat	-0.001	0.052	0.990
Age 11 mat on age 7 inc	-0.044	0.026	0.085
Age 11 pat on age 7 int	0.022	0.046	0.637
Age 11 pat on age 7 mat	0.067	0.046	0.144
Age 11 pat on age 7 pat	0.251	0.057	**<.001***
Age 11 pat on age 7 inc	-0.080	0.025	**0.002***
Age 11 inc on age 7 int	-0.069	0.046	0.137
Age 11 inc on age 7 mat	-0.051	0.030	0.089
Age 11 inc on age 7 pat	-0.115	0.050	**0.022***
Age 11 inc on age 7 inc	0.053	0.044	0.230
Age 7 int on age 5 int	0.315	0.040	**<.001***
Age 7 int on age 5 mat	0.037	0.035	0.296
Age 7 int on age 5 pat	0.019	0.034	0.580
Age 7 int on age 5 inc	-0.028	0.024	0.241
Age 7 int on age 3 inc	-0.056	0.021	**0.008***
Age 7 mat on age 5 int	0.009	0.036	0.797
Age 7 mat on age 5 mat	0.127	0.042	**0.002***
Age 7 mat on age 5 pat	0.042	0.043	0.336
Age 7 mat on age 5 inc	-0.015	0.023	0.507
Age 7 pat on age 5 int	-0.008	0.035	0.826
Age 7 pat on age 5 mat	0.055	0.049	0.262

Age 7 pat on age 5 pat	0.187	0.072	**0.009***
Age 7 pat on age 5 inc	-0.071	0.031	**0.021***
Age 7 inc on age 5 int	0.039	0.019	**0.040***
Age 7 inc on age 5 mat	0.009	0.022	0.682
Age 7 inc on age 5 pat	-0.037	0.022	0.101
Age 7 inc on age 5 inc	0.500	0.036	**<.001***
Age 5 int on age 3 int	0.082	0.049	0.093
Age 5 int on age 3 mat	0.010	0.046	0.832
Age 5 int on age 3 pat	-0.057	0.052	0.272
Age 5 int on age 3 inc	0.015	0.029	0.602
Age 5 mat on age 3 int	-0.067	0.035	0.059
Age 5 mat on age 3 mat	0.073	0.052	0.157
Age 5 mat on age 3 pat	-0.030	0.053	0.569
Age 5 mat on age 3 inc	0.019	0.025	0.466
Age 5 pat on age 3 int	0.005	0.046	0.917
Age 5 pat on age 3 mat	0.031	0.044	0.481
Age 5 pat on age 3 pat	0.051	0.065	0.426
Age 5 pat on age 3 inc	-0.012	0.023	0.608
Age 5 inc on age 3 int	-0.001	0.021	0.953
Age 5 inc on age 3 mat	-0.018	0.018	0.328
Age 5 inc on age 3 pat	-0.046	0.021	**0.027***
Age 5 inc on age 3 inc	0.505	0.032	**<.001***

Notes: int, internalising problems; inc, family income; mat, maternal distress; pat, paternal distress; Correlations are significant at $p < .05$. Children's ages are in accordance with their respective median ages at the time of data collection.

Table B5. Standardised autoregressive and cross-lagged parameters from RICLMP for male's externalising outcome

Parameter	***Estimate***	***SE***	***P***
Age 14 ext on age 11 ext	0.495	0.024	**<.001***
Age 14 ext on age 11 mat	-0.018	0.026	0.505
Age 14 ext on age 11 pat	0.040	0.034	0.242
Age 14 ext on age 11 inc	-0.052	0.034	0.131
Age 14 ext on age 7 inc	0.003	0.024	0.909
Age 14 mat on age 11 ext	-0.001	0.031	0.964
Age 14 mat on age 11 mat	0.331	0.036	**<.001***
Age 14 mat on age 11 pat	0.007	0.047	0.875
Age 14 mat on age 11 inc	-0.006	0.027	0.820
Age 14 pat on age 11 ext	0.098	0.051	0.055
Age 14 pat on age 11 mat	0.025	0.043	0.559
Age 14 pat on age 11 pat	0.377	0.059	**<.001***
Age 14 pat on age 11 inc	0.030	0.039	0.439
Age 14 inc on age 11 ext	-0.014	0.026	0.596
Age 14 inc on age 11 mat	-0.051	0.023	**0.028***
Age 14 inc on age 11 pat	-0.055	0.031	0.071
Age 14 inc on age 11 inc	0.505	0.041	**<.001***
Age 11 ext on age 7 ext	0.357	0.033	**<.001***
Age 11 ext on age 7 mat	0.039	0.038	0.298
Age 11 ext on age 7 pat	0.027	0.033	0.406
Age 11 ext on age 7 inc	0.009	0.024	0.723
Age 11 ext on age 5 inc	-0.009	0.024	0.692
Age 11 mat on age 7 ext	0.072	0.033	**0.030***
Age 11 mat on age 7 mat	0.240	0.042	**<.001***
Age 11 mat on age 7 pat	-0.001	0.053	0.986
Age 11 mat on age 7 inc	-0.045	0.025	0.073
Age 11 pat on age 7 ext	0.020	0.042	0.638
Age 11 pat on age 7 mat	0.065	0.047	0.165
Age 11 pat on age 7 pat	0.250	0.057	**<.001***
Age 11 pat on age 7 inc	-0.079	0.026	**0.003***
Age 11 inc on age 7 ext	-0.042	0.045	0.349
Age 11 inc on age 7 mat	-0.046	0.030	0.131
Age 11 inc on age 7 pat	-0.105	0.050	**0.034***
Age 11 inc on age 7 inc	0.057	0.044	0.197
Age 7 ext on age 5 int	0.337	0.040	**<.001***
Age 7 ext on age 5 mat	0.049	0.038	0.199
Age 7 ext on age 5 pat	-0.025	0.041	0.541
Age 7 ext on age 5 inc	-0.014	0.027	0.597
Age 7 ext on age 3 inc	-0.016	0.025	0.523
Age 7 mat on age 5 ext	0.068	0.045	0.125
Age 7 mat on age 5 mat	0.101	0.040	**0.012***
Age 7 mat on age 5 pat	0.037	0.043	0.390
Age 7 mat on age 5 inc	-0.012	0.023	0.595
Age 7 pat on age 5 ext	0.067	0.037	0.070
Age 7 pat on age 5 mat	0.034	0.050	0.490

Age 7 pat on age 5 pat	0.182	0.073	**0.012***
Age 7 pat on age 5 inc	-0.066	0.030	**0.029***
Age 7 inc on age 5 ext	0.046	0.027	0.086
Age 7 inc on age 5 mat	0.006	0.022	0.776
Age 7 inc on age 5 pat	-0.037	0.023	0.104
Age 7 inc on age 5 inc	0.498	0.037	**<.001***
Age 5 ext on age 3 ext	0.200	0.037	**<.001***
Age 5 ext on age 3 mat	0.024	0.042	0.569
Age 5 ext on age 3 pat	0.001	0.045	0.981
Age 5 ext on age 3 inc	0.022	0.029	0.456
Age 5 mat on age 3 ext	0.062	0.034	0.071
Age 5 mat on age 3 mat	0.059	0.034	0.241
Age 5 mat on age 3 pat	-0.032	0.052	0.541
Age 5 mat on age 3 inc	0.018	0.025	0.454
Age 5 pat on age 3 ext	0.013	0.039	0.738
Age 5 pat on age 3 mat	0.026	0.043	0.541
Age 5 pat on age 3 pat	0.049	0.067	0.469
Age 5 pat on age 3 inc	-0.008	0.024	0.733
Age 5 inc on age 3 ext	0.013	0.039	0.181
Age 5 inc on age 3 mat	0.026	0.043	0.427
Age 5 inc on age 3 pat	0.049	0.067	**0.032***
Age 5 inc on age 3 inc	-0.008	0.024	**<.001***

Notes: ext, externalising problems; inc, family income; mat, maternal distress; pat, paternal distress; Correlations are significant at $p < .05$. Children's ages are in accordance with their respective median ages at the time of data collection.

Table B6. Indirect effects for female internalising problems

	β	*SE*	*p*	CI_{lower}	CI_{upper}
Age 3 family income to age 7 internalising					
Via maternal distress problems	0.000	0.001	0.733	-0.002	0.001
Via paternal distress problems	0.000	0.001	0.983	-0.002	0.002
Sum of indirect	0.000	0.001	0.859	-0.003	0.002
Age 5 family income to age 11 internalising					
Via maternal distress problems	0.001	0.001	0.610	-0.001	0.003
Via paternal distress problems	0.000	0.001	0.604	-0.003	0.001
Sum of indirect	0.000	0.001	0.955	-0.003	0.003
Age 7 family income to age 14 internalising					
Via maternal distress problems	-0.002	0.003	0.368	-0.006	0.002
Via paternal distress problems	-0.001	0.002	0.521	-0.005	0.001
Sum of indirect	-0.004	0.003	0.264	-0.009	0.001

Note: Based on standard maximum likelihood estimation, confidence intervals (CIs) are calculated from 1000 bootstrapped samples.

Table B7. Indirect effects for female externalising problems

	β	*SE*	*p*	CI_{lower}	CI_{upper}
Age 3 family income to age 7 externalising					
Via maternal distress problems	0.000	0.001	0.536	-0.003	0.004
Via paternal distress problems	0.000	0.002	0.918	-0.002	0.001
Sum of indirect	0.000	0.002	0.866	-0.003	0.004
Age 5 family income to age 11 externalising					
Via maternal distress problems	0.001	0.001	0.366	-0.001	0.004
Via paternal distress problems	0.000	0.001	0.998	-0.002	0.002
Sum of indirect	0.001	0.002	0.422	-0.002	0.005
Age 7 family income to age 14 externalising					
Via maternal distress problems	-0.007	0.003	0.036	-0.012	-0.002
Via paternal distress problems	0.001	0.002	0.635	-0.003	0.005
Sum of indirect	-0.006	0.004	0.122	-0.013	0.000

Note: Based on standard maximum likelihood estimation, confidence intervals (CIs) are calculated from 1000 bootstrapped samples.

Table B8. Indirect effects for male internalising problems

	β	*SE*	*p*	CI_{lower}	CI_{upper}
Age 3 family income to age 7 internalising					
Via maternal distress problems	0.001	0.001	0.543	-0.001	0.004
Via paternal distress problems	0.000	0.001	0.709	-0.002	0.001
Sum of indirect	0.000	0.001	0.738	-0.002	0.004
Age 5 family income to age 11 internalising					
Via maternal distress problems	0.000	0.001	0.596	-0.003	0.001
Via paternal distress problems	-0.002	0.003	0.441	-0.007	0.002
Sum of indirect	-0.003	0.003	0.365	-0.008	0.001
Age 7 family income to age 14 internalising					
Via maternal distress problems	0.001	0.001	0.694	-0.001	0.003
Via paternal distress problems	-0.003	0.004	-0.670	-0.010	0.004
Sum of indirect	-0.002	0.005	0.666	-0.010	0.006

Note: Based on standard maximum likelihood estimation, confidence intervals (CIs) are calculated from 1000 bootstrapped samples.

Table B9. Indirect effects for male externalising problems

	β	*SE*	*p*	CI_{lower}	CI_{upper}
Age 3 family income to age 7 externalising					
Via maternal distress problems	0.001	0.001	0.508	-0.001	0.004
Via paternal distress problems	0.000	0.001	0.760	-0.002	0.002
Sum of indirect	0.001	0.001	0.440	-0.002	0.005
Age 5 family income to age 11 externalising					
Via maternal distress problems	-0.002	0.003	0.471	-0.003	0.001
Via paternal distress problems	0.000	0.001	0.659	-0.007	0.001
Sum of indirect	-0.002	0.003	0.416	-0.008	0.001
Age 7 family income to age 14 externalising					
Via maternal distress problems	0.001	0.001	0.506	-0.002	0.003
Via paternal distress problems	-0.003	0.003	0.305	-0.009	0.001
Sum of indirect	-0.002	0.003	0.460	-0.009	0.003

Note: Based on standard maximum likelihood estimation, confidence intervals (CIs) are calculated from 1000 bootstrapped samples.

Appendix C

Table C1. Descriptive statistics.

	N	*%NA*	*Mean*	*SD*	*Min*	*Max*	*Skew*	*Kurtosis*	*Omega*
Age 3 internalising problems	9047	12.4	2.79	2.45	0	18	1.25	1.89	.61
Age 5 internalising problems	9465	8.4	2.44	2.48	0	18	1.56	3.16	.79
Age 7 internalising problems	9277	10.2	2.66	2.72	0	12	1.48	2.55	.67
Age 11 internalising problems	9380	9.2	3.12	3.09	0	19	1.44	2.29	.80
Age 14 internalising problems	9162	11.3	3.71	3.39	0	19	1.26	1.54	.71
Age 17 internalising problems	9031	12.6	3.80	3.48	0	18	1.20	1.15	.82
Age 3 externalising problems	9045	12.4	6.46	3.73	0	20	.58	-.01	.78
Age 5 externalising problems	9463	8.4	4.59	3.35	0	20	.89	.62	.82
Age 7 externalising problems	9275	10.2	4.54	3.52	0	19	.88	.46	.78
Age 11 externalising problems	9380	9.2	4.27	3.46	0	20	1.02	.90	.82
Age 14 externalising problems	9161	11.3	4.24	3.50	0	20	1.11	1.25	.79
Age 17 externalising problems	9035	12.5	3.65	3.31	0	20	1.24	1.67	.82
Age 3 maternal distress	8219	20.4	3.13	3.57	0	24	1.92	4.57	NA
Age 5 maternal distress	9090	12.0	3.05	3.64	0	24	1.94	4.62	NA
Age 7 maternal distress	8912	13.7	3.01	3.71	0	24	1.98	4.80	NA
Age 11 maternal distress	8944	13.4	3.89	4.26	0	24	1.71	3.35	NA
Age 14 maternal distress	8543	17.3	4.24	4.10	0	24	1.48	2.48	NA
Age 17 maternal distress	7098	31.3	4.19	4.38	0	24	1.46	2.03	NA
Age 3 paternal distress	6387	38.2	2.87	3.02	0	24	1.77	4.58	NA
Age 5 paternal distress	6715	35.0	2.93	3.24	0	24	1.89	5.01	NA
Age 7 paternal distress	6244	39.6	2.94	3.33	0	24	1.84	4.38	NA
Age 11 paternal distress	6000	41.9	3.77	3.79	0	24	1.62	3.31	NA
Age 14 paternal distress	5414	47.6	3.66	3.61	0	24	1.73	4.23	NA
Age 17 paternal distress	4424	57.2	3.55	3.83	0	24	1.69	3.66	NA

% NA, Percentage of missing data at each wave of data collection. Omega, McDonalds Omega. The parental distress value was derived from the sum-scores provided by the Millennium Cohort Study, thus a value of internal consistency for the parental mental health measure was not available. Kessler (K6) scale, however, has reported good reliability in Flouri et al (2019). Children's ages are in accordance with their respective median ages at the time of data collection.

Table C2 The correlation matrix of the study variables in the non-poverty group.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.
1. Age 3 Ext	1	.304**	.245**	.068**	.585**	.238**	.200**	.078**	.534**	.249**	.204**	.071**	.462**	.267**	.202**	.080**	.397**	.236**	.181**	.060**	.338**	.193**	.158**	.055**
2. Age 3 Int	.304**	1	.206**	.069**	.218**	.456**	.189**	.065**	.208**	.377**	.166**	.049**	.185**	.319**	.193**	.062**	.145**	.274**	.164**	.095**	.129**	.208**	.131**	.063**
3. Age 3 Mat	.245**	.206**	1	.149**	.213**	.198**	.554**	.122**	.185**	.204**	.517**	.104**	.199**	.232**	.488**	.096**	.169**	.194**	.476**	.112**	.135**	.176**	.444**	.090**
4. Age 3 Pat	.068**	.069**	.149**	1	.079**	.073**	.084**	.544**	.087**	.088**	.099**	.521**	.071**	.097**	.087**	.459**	.082**	.080**	.090**	.463**	.065**	.077**	.098**	.421**
5. Age 5 Ext	.585**	.218**	.213**	.079**	1	.328**	.256**	.088**	.703**	.302**	.220**	.076**	.609**	.311**	.222**	.072**	.523**	.284**	.191**	.080**	.447**	.240**	.180**	.063**
6. Age 5 Int	.238**	.456**	.198**	.073**	.328**	1	.269**	.064**	.268**	.565**	.203**	.057**	.247**	.440**	.231**	.059**	.210**	.389**	.222**	.095**	.197**	.322**	.197**	.077**
7. Age 5 Mat	.200**	.189**	.554**	.084**	.256**	.269**	1	.145**	.212**	.241**	.587**	.121**	.214**	.249**	.502**	.108**	.178**	.215**	.472**	.135**	.142**	.186**	.434**	.104**
8. Age 5 Pat	.078**	.065**	.122**	.544**	.088**	.064**	.145**	1	.073**	.072**	.126**	.580**	.071**	.077**	.083**	.493**	.066**	.090**	.100**	.484**	.074**	.098**	.128**	.402**
9. Age7 Ext	.534**	.208**	.185**	.087**	.703**	.268**	.212**	.073**	1	.381**	.254**	.084**	.694**	.342**	.224**	.080**	.595**	.304**	.185**	.080**	.497**	.267**	.176**	.063**
10. Age7 Int	.249**	.377**	.204**	.088**	.302**	.565**	.241**	.072**	.381**	1	.299**	.078**	.315**	.546**	.277**	.079**	.274**	.458**	.247**	.112**	.243**	.385**	.225**	.086**
11. Age7 Mat	.204**	.166**	.517**	.099**	.220**	.203**	.587**	.126**	.254**	.299**	1	.160**	.252**	.260**	.549**	.105**	.210**	.226**	.504**	.113**	.170**	.195**	.449**	.100**
12. Age7 Pat	.071**	.049**	.104**	.521**	.076**	.057**	.121**	.580**	.084**	.078**	.160**	1	.077**	.090**	.101**	.544**	.071**	.096**	.104**	.533**	.087**	.104**	.100**	.473**
13. Age 11 Ext	.462**	.185**	.199**	.071**	.609**	.247**	.214**	.071**	.694**	.315**	.252**	.077**	1	.447**	.285**	.112**	.712**	.365**	.239**	.107**	.596**	.330**	.223**	.075**
14. Age 11 Int	.267**	.319**	.232**	.097**	.311**	.440**	.249**	.077**	.342**	.546**	.260**	.090**	.447**	1	.347**	.121**	.348**	.606**	.291**	.132**	.298**	.497**	.273**	.100**
15. Age 11 Mat	.202**	.193**	.488**	.087**	.222**	.231**	.502**	.083**	.224**	.277**	.549**	.101**	.285**	.347**	1	.146**	.241**	.284**	.600**	.141**	.192**	.265**	.551**	.114**
16. Age 11 Pat	.080**	.062**	.096**	.459**	.072**	.059**	.108**	.493**	.080**	.079**	.105**	.544**	.112**	.121**	.146**	1	.106**	.111**	.093**	.622**	.103**	.112**	.129**	.518**
17. Age 14 Ext	.397**	.145**	.169**	.082**	.523**	.210**	.178**	.066**	.595**	.274**	.210**	.071**	.712**	.348**	.241**	.106**	1	.439**	.233**	.124**	.693**	.353**	.212**	.093**
18. Age 14 Int	.236**	.274**	.194**	.080**	.284**	.389**	.215**	.090**	.304**	.458**	.226**	.096**	.365**	.606**	.284**	.111**	.439**	1	.304**	.141**	.343**	.621**	.271**	.114**
19. Age 14 Mat	.181**	.164**	.476**	.090**	.191**	.222**	.472**	.100**	.185**	.247**	.504**	.104**	.239**	.291**	.600**	.093**	.233**	.304**	1	.153**	.183**	.262**	.616**	.117**
20. Age 14 Pat	.060**	.095**	.112**	.463**	.080**	.095**	.135**	.484**	.080**	.112**	.113**	.533**	.107**	.132**	.141**	.622**	.124**	.141**	.153**	1	.132**	.148**	.164**	.594**
21. Age 17 Ext	.338**	.129**	.135**	.065**	.447**	.197**	.142**	.074**	.497**	.243**	.170**	.087**	.596**	.298**	.192**	.103**	.693**	.343**	.183**	.132**	1	.452**	.238**	.110**
22. Age 17 Int	.193**	.208**	.176**	.077**	.240**	.322**	.186**	.098**	.267**	.385**	.195**	.104**	.330**	.497**	.265**	.112**	.353**	.621**	.262**	.148**	.452**	1	.326**	.147**
23. Age 17 Mat	.158**	.131**	.444**	.098**	.180**	.197**	.434**	.128**	.176**	.225**	.449**	.100**	.223**	.273**	.551**	.129**	.212**	.271**	.616**	.164**	.238**	.326**	1	.212**
24. Age 17 Pat	.055**	.063**	.090**	.421**	.063**	.077**	.104**	.402**	.063**	.086**	.100**	.473**	.075**	.100**	.114**	.518**	.093**	.114**	.117**	.594**	.110**	.147**	.212**	1

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress; **. Correlations are significant at $p < .01$.; *. Correlations are significant at $p < .05$.

Table C3 The correlation matrix of the study variables in the poverty group.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.
1. Age 3 Ext	1	.352**	.257**	.096**	.570**	.272**	.188**	.119**	.488**	.275**	.167**	.129**	.437**	.219**	.185**	.118**	.401**	.220**	.200**	.105**	.340**	.215**	.128**	.150**
2. Age 3 Int	.352**	1	.278**	.106**	.259**	.485**	.200**	.090**	.239**	.406**	.192**	.097**	.196**	.272**	.212**	.106**	.189**	.240**	.194**	.023	.137**	.195**	.115**	.023
3. Age 3 Mat	.257**	.278**	1	.188**	.195**	.228**	.534**	.126**	.223**	.266**	.494**	.145**	.206**	.227**	.469**	.127**	.183**	.238**	.424**	.125**	.173**	.195**	.358**	.168**
4. Age 3 Pat	.096**	.106**	.188**	1	.095**	.109**	.169**	.536**	.130**	.139**	.099**	.490**	.095**	.104**	.182**	.466**	.136**	.164**	.128**	.465**	.137**	.118**	.164**	.453**
5. Age 5 Ext	.570**	.259**	.195**	.095**	1	.400**	.228**	.114**	.664**	.335**	.223**	.121**	.548**	.316**	.181**	.126**	.491**	.289**	.207**	.120**	.419**	.248**	.171**	.119**
6. Age 5 Int	.272**	.485**	.228**	.109**	.400**	1	.299**	.101**	.312**	.531**	.235**	.102**	.242**	.378**	.246**	.102**	.233**	.327**	.209**	.101**	.221**	.282**	.187**	.105*
7. Age 5 Mat	.188**	.200**	.534**	.169**	.228**	.299**	1	.226**	.231**	.282**	.574**	.148**	.176**	.198**	.468**	.129**	.156**	.222**	.423**	.123**	.154**	.192**	.412**	.175**
8. Age 5 Pat	.119**	.090**	.126**	.536**	.114**	.101**	.226**	1	.116**	.133**	.090**	.614**	.084**	.074*	.186**	.539**	.122**	.137**	.166**	.500**	.124**	.120**	.101**	.438**
9. Age7 Ext	.488**	.239**	.223**	.130**	.664**	.312**	.231**	.116**	1	.460**	.289**	.164**	.649**	.379**	.221**	.152**	.571**	.336**	.211**	.164**	.490**	.283**	.181**	.141**
10. Age7 Int	.275**	.406**	.266**	.139**	.335**	.531**	.282**	.133**	.460**	1	.347**	.172**	.316**	.519**	.266**	.128**	.284**	.448**	.262**	.110**	.256**	.370**	.231**	.116**
11. Age7 Mat	.167**	.192**	.494**	.099**	.223**	.235**	.574**	.090**	.289**	.347**	1	.135**	.223**	.255**	.542**	.097**	.206**	.239**	.486**	.073*	.166**	.211**	.468**	.108*
12. Age7 Pat	.129**	.097**	.145**	.490**	.121**	.102**	.148**	.614**	.164**	.172**	.135**	1	.135**	.140**	.207**	.599**	.197**	.186**	.155**	.541**	.159**	.153**	.167**	.514**
13. Age 11 Ext	.437**	.196**	.206**	.095**	.548**	.242**	.176**	.084**	.649**	.316**	.223**	.135**	1	.492**	.298**	.218**	.673**	.371**	.223**	.187**	.586**	.330**	.232**	.102*
14. Age 11 Int	.219**	.272**	.227**	.104**	.316**	.378**	.198**	.074*	.379**	.519**	.255**	.140**	.492**	1	.342**	.198**	.353**	.600**	.269**	.165**	.353**	.520**	.278**	.141**
15. Age 11 Mat	.185**	.212**	.469**	.182**	.181**	.246**	.468**	.186**	.221**	.266**	.542**	.207**	.298**	.342**	1	.251**	.226**	.266**	.565**	.229**	.172**	.222**	.522**	.168**
16. Age 11 Pat	.118**	.106**	.127**	.466**	.126**	.102**	.129**	.539**	.152**	.128**	.097**	.599**	.218**	.198**	.251**	1	.191**	.200**	.149**	.610**	.180**	.186**	.199**	.516**
17. Age 14 Ext	.401**	.189**	.183**	.136**	.491**	.233**	.156**	.122**	.571**	.284**	.206**	.197**	.673**	.353**	.226**	.191**	1	.469**	.253**	.233**	.672**	.355**	.261**	.206**
18. Age 14 Int	.220**	.240**	.238**	.164**	.289**	.327**	.222**	.137**	.336**	.448**	.239**	.186**	.371**	.600**	.266**	.200**	.469**	1	.315**	.216**	.378**	.617**	.323**	.207**
19. Age 14 Mat	.200**	.194**	.424**	.128**	.207**	.209**	.423**	.166**	.211**	.262**	.486**	.155**	.223**	.269**	.565**	.149**	.253**	.315**	1	.233**	.227**	.258**	.637**	.190**
20. Age 14 Pat	.105**	.023	.125**	.465**	.120**	.101**	.123**	.500**	.164**	.110**	.073*	.541**	.187**	.165**	.229**	.610**	.233**	.216**	.233**	1	.176**	.143**	.186**	.674**
21. Age 17 Ext	.340**	.137**	.173**	.137**	.419**	.221**	.154**	.124**	.490**	.256**	.166**	.159**	.586**	.353**	.172**	.180**	.672**	.378**	.227**	.176**	1	.499**	.250**	.231**
22. Age 17 Int	.215**	.195**	.195**	.118**	.248**	.282**	.192**	.120**	.283**	.370**	.211**	.153**	.330**	.520**	.222**	.186**	.355**	.617**	.258**	.143**	.499**	1	.323**	.215**
23. Age 17 Mat	.128**	.115**	.358**	.164**	.171**	.187**	.412**	.101**	.181**	.231**	.468**	.167**	.232**	.278**	.522**	.199**	.261**	.323**	.637**	.186**	.250**	.323**	1	.294**
24. Age 17 Pat	.150**	.023	.168**	.453**	.119**	.105*	.175**	.438**	.141**	.116**	.108*	.514**	.102*	.141**	.168**	.516**	.206**	.207**	.190**	.674**	.231**	.215**	.294**	1

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress; **. Correlations are significant at $p < .01$.; *. Correlations are significant at $p < .05$.

Table C4. Multi-group ALT-SRs with unconstrained autoregressive and cross-lagged parameters for girls, non-poverty group.

Parameter	***Est.***	***SE***	***p***
Age 17 Int → Age 14 Int	0.442	0.020	<.001*
Age 17 Int → Age 14 Ext	0.087	0.022	<.001*
Age 17 Int → Age 14 Mat	0.055	0.022	0.014*
Age 17 Int → Age 14 Pat	0.053	0.026	0.043*
Age 17 Ext → Age 14 Int	0.085	0.024	<.001*
Age 17 Ext → Age 14 Ext	0.388	0.030	<.001*
Age 17 Ext → Age 14 Mat	-0.017	0.023	0.467
Age 17 Ext → Age 14 Pat	0.054	0.026	0.038*
Age 17 Mat → Age 14 Int	0.078	0.026	0.003*
Age 17 Mat → Age 14 Ext	0.042	0.026	0.111
Age 17 Mat → Age 14 Mat	0.355	0.033	<.001*
Age 17 Mat → Age 14 Pat	0.136	0.039	<.001*
Age 17 Pat → Age 14 Int	0.013	0.028	0.646
Age 17 Pat → Age 14 Ext	-0.001	0.036	0.986
Age 17 Pat → Age 14 Mat	0.004	0.030	0.894
Age 17 Pat → Age 14 Pat	0.403	0.042	<.001*
Age 14 Int → Age 11 Int	0.387	0.024	<.001*
Age 14 Int → Age 11Ext	0.102	0.024	<.001*
Age 14 Int → Age 11 Mat	0.021	0.024	0.393
Age 14 Int → Age 11 Pat	0.017	0.025	0.486
Age 14 Ext → Age 11 Int	0.050	0.026	0.052
Age 14 Ext → Age 11 Ext	0.413	0.027	<.001*
Age 14 Ext → Age 11 Mat	0.072	0.025	0.004*
Age 14 Ext → Age 11 Pat	0.037	0.028	0.183
Age 14 Mat → Age 11 Int	0.069	0.026	0.008*
Age 14 Mat → Age 11 Ext	0.055	0.028	0.047*
Age 14 Mat → Age 11 Mat	0.267	0.033	<.001*
Age 14 Mat → Age 11 Pat	-0.015	0.032	0.635
Age 14 Pat → Age 11 Int	0.039	0.026	0.132
Age 14 Pat → Age 11 Ext	0.019	0.033	0.570
Age 14 Pat → Age 11 Mat	0.078	0.034	0.022*
Age 14 Pat → Age 11 Pat	0.388	0.038	<.001*
Age 11 Int → Age 7 Int	0.271	0.025	<.001*
Age 11 Int → Age 7 Ext	0.116	0.026	<.001*
Age 11 Int → Age 7 Mat	0.038	0.024	0.112
Age 11 Int → Age 7 Pat	0.031	0.031	0.324
Age 11 Ext → Age 7 Int	0.032	0.024	0.186

Age 11 Ext → Age 7 Ext	0.410	0.026	<.001*
Age 11 Ext → Age 7 Mat	0.052	0.026	0.048*
Age 11 Ext → Age 7 Pat	0.015	0.028	0.608
Age 11 Mat → Age 7 Int	0.093	0.030	0.002*
Age 11 Mat → Age 7 Ext	0.099	0.025	<.001*
Age 11 Mat → Age 7 Mat	0.080	0.035	0.022*
Age 11 Mat → Age 7 Pat	0.068	0.032	0.031*
Age 11 Pat → Age 7 Int	0.033	0.030	0.265
Age 11 Pat → Age 7 Ext	0.032	0.029	0.260
Age 11 Pat → Age 7 Mat	-0.059	0.033	0.070
Age 11 Pat → Age 7 Pat	0.216	0.042	<.001*
Age 7 Int → Age 5 Int	0.215	0.032	<.001*
Age 7 Int → Age 5 Ext	0.056	0.027	0.036*
Age 7 Int → Age 5 Mat	-0.032	0.030	0.283
Age 7 Int → Age 5 Pat	-0.029	0.027	0.282
Age 7 Ext → Age 5 Int	-0.021	0.025	0.394
Age 7 Ext → Age 5 Ext	0.484	0.022	<.001*
Age 7 Ext → Age 5 Mat	0.032	0.023	0.169
Age 7 Ext → Age 5 Pat	-0.042	0.028	0.123
Age 7 Mat → Age 5 Int	-0.051	0.030	0.088
Age 7 Mat → Age 5 Ext	0.144	0.028	<.001*
Age 7 Mat → Age 5 Mat	0.165	0.034	<.001*
Age 7 Mat → Age 5 Pat	0.026	0.029	0.368
Age 7 Pat → Age 5 Int	-0.014	0.037	0.708
Age 7 Pat → Age 5 Ext	0.058	0.030	0.054
Age 7 Pat → Age 5 Mat	0.084	0.038	0.025*
Age 7 Pat → Age 5 Pat	0.054	0.048	0.257
Age 5 Int → Age 3 Int	0.076	0.036	0.036*
Age 5 Int → Age 3 Ext	-0.012	0.027	0.652
Age 5 Int → Age 3 Mat	-0.092	0.032	0.004*
Age 5 Int → Age 3 Pat	-0.010	0.037	0.783
Age 5 Ext → Age 3 Int	-0.083	0.026	0.001*
Age 5 Ext → Age 3 Ext	0.263	0.026	<.001*
Age 5 Ext → Age 3 Mat	-0.012	0.029	0.675
Age 5 Ext → Age 3 Pat	0.002	0.032	0.957
Age 5 Mat → Age 3 Int	-0.032	0.035	0.348
Age 5 Mat → Age 3 Ext	0.017	0.027	0.544
Age 5 Mat → Age 3 Mat	0.055	0.046	0.229
Age 5 Mat → Age 3 Pat	-0.087	0.036	0.016*
Age 5 Pat → Age 3 Int	-0.061	0.041	0.139

Age 5 Pat ⟶ Age 3 Ext	0.002	0.032	0.944
Age 5 Pat ⟶ Age 3 Mat	0.012	0.041	0.765
Age 5 Pat ⟶ Age 3 Pat	-0.112	0.061	0.068

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. * Significant based on p<.05. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C5. Multi-group ALT-SRs with unconstrained autoregressive and cross-lagged parameters for girls, poverty group.

Parameter	***Est.***	***SE***	***p***
Age 17 Int ⟶ Age 14 Int	0.473	0.036	<.001*
Age 17 Int ⟶ Age 14 Ext	0.082	0.036	0.023*
Age 17 Int ⟶ Age 14 Mat	0.019	0.038	0.623
Age 17 Int ⟶ Age 14 Pat	0.065	0.066	0.325
Age 17 Ext ⟶ Age 14 Int	0.102	0.036	0.005*
Age 17 Ext ⟶ Age 14 Ext	0.408	0.045	<.001*
Age 17 Ext ⟶ Age 14 Mat	0.076	0.040	0.056
Age 17 Ext ⟶ Age 14 Pat	0.033	0.066	0.617
Age 17 Mat ⟶ Age 14 Int	0.081	0.043	0.059
Age 17 Mat ⟶ Age 14 Ext	0.087	0.047	0.062
Age 17 Mat ⟶ Age 14 Mat	0.407	0.056	<.001*
Age 17 Mat ⟶ Age 14 Pat	0.015	0.093	0.875
Age 17 Pat ⟶ Age 14 Int	0.032	0.071	0.653
Age 17 Pat ⟶ Age 14 Ext	-0.014	0.071	0.843
Age 17 Pat ⟶ Age 14 Mat	-0.073	0.090	0.418
Age 17 Pat ⟶ Age 14 Pat	0.469	0.087	<.001*
Age 14 Int ⟶ Age 11 Int	0.387	0.039	<.001*
Age 14 Int ⟶ Age 11Ext	0.084	0.042	0.044*
Age 14 Int ⟶ Age 11 Mat	0.017	0.032	0.594
Age 14 Int ⟶ Age 11 Pat	0.077	0.039	0.047*
Age 14 Ext ⟶ Age 11 Int	0.016	0.036	0.657
Age 14 Ext ⟶ Age 11 Ext	0.373	0.055	<.001*
Age 14 Ext ⟶ Age 11 Mat	0.024	0.046	0.608
Age 14 Ext ⟶ Age 11 Pat	0.081	0.071	0.252
Age 14 Mat ⟶ Age 11 Int	0.060	0.045	0.188
Age 14 Mat ⟶ Age 11 Ext	0.038	0.054	0.474
Age 14 Mat ⟶ Age 11 Mat	0.304	0.054	<.001*
Age 14 Mat ⟶ Age 11 Pat	0.006	0.074	0.932
Age 14 Pat ⟶ Age 11 Int	-0.049	0.069	0.475

Age 14 Pat → Age 11 Ext	0.091	0.067	0.172
Age 14 Pat → Age 11 Mat	0.208	0.105	0.047*
Age 14 Pat → Age 11 Pat	0.180	0.085	0.035*
Age 11 Int → Age 7 Int	0.232	0.038	<.001*
Age 11 Int → Age 7 Ext	0.140	0.040	<.001*
Age 11 Int → Age 7 Mat	0.040	0.046	0.386
Age 11 Int → Age 7 Pat	-0.067	0.061	0.265
Age 11 Ext → Age 7 Int	-0.002	0.039	0.961
Age 11 Ext → Age 7 Ext	0.391	0.046	<.001*
Age 11 Ext → Age 7 Mat	-0.003	0.046	0.951
Age 11 Ext → Age 7 Pat	0.039	0.065	0.552
Age 11 Mat → Age 7 Int	0.051	0.038	0.183
Age 11 Mat → Age 7 Ext	0.080	0.045	0.072
Age 11 Mat → Age 7 Mat	0.135	0.061	0.027*
Age 11 Mat → Age 7 Pat	0.105	0.097	0.277
Age 11 Pat → Age 7 Int	0.010	0.069	0.883
Age 11 Pat → Age 7 Ext	0.179	0.073	0.014*
Age 11 Pat → Age 7 Mat	0.119	0.086	0.164
Age 11 Pat → Age 7 Pat	0.281	0.081	0.001*
Age 7 Int → Age 5 Int	0.227	0.042	<.001*
Age 7 Int → Age 5 Ext	0.051	0.051	0.312
Age 7 Int → Age 5 Mat	0.035	0.055	0.519
Age 7 Int → Age 5 Pat	0.180	0.086	0.036*
Age 7 Ext → Age 5 Int	0.028	0.036	0.437
Age 7 Ext → Age 5 Ext	0.392	0.047	<.001*
Age 7 Ext → Age 5 Mat	0.041	0.048	0.388
Age 7 Ext → Age 5 Pat	0.137	0.091	0.131
Age 7 Mat → Age 5 Int	0.046	0.058	0.429
Age 7 Mat → Age 5 Ext	0.032	0.046	0.494
Age 7 Mat → Age 5 Mat	0.284	0.062	<.001*
Age 7 Mat → Age 5 Pat	0.030	0.086	0.725
Age 7 Pat → Age 5 Int	-0.039	0.062	0.529
Age 7 Pat → Age 5 Ext	-0.029	0.064	0.654
Age 7 Pat → Age 5 Mat	0.100	0.079	0.208
Age 7 Pat → Age 5 Pat	0.435	0.095	<.001*
Age 5 Int → Age 3 Int	0.227	0.047	<.001*
Age 5 Int → Age 3 Ext	0.064	0.040	0.109
Age 5 Int → Age 3 Mat	0.015	0.051	0.772
Age 5 Int → Age 3 Pat	-0.037	0.065	0.573
Age 5 Ext → Age 3 Int	0.011	0.048	0.820

Age 5 Ext ⟶	Age 3 Ext	0.339	0.040	<.001*
Age 5 Ext ⟶	Age 3 Mat	-0.054	0.050	0.277
Age 5 Ext ⟶	Age 3 Pat	0.092	0.072	0.202
Age 5 Mat ⟶	Age 3 Int	-0.028	0.040	0.478
Age 5 Mat ⟶	Age 3 Ext	0.071	0.036	0.049*
Age 5 Mat ⟶	Age 3 Mat	0.196	0.065	0.002*
Age 5 Mat ⟶	Age 3 Pat	0.092	0.077	0.228
Age 5 Pat ⟶	Age 3 Int	0.071	0.068	0.294
Age 5 Pat ⟶	Age 3 Ext	0.127	0.071	0.077
Age 5 Pat ⟶	Age 3 Mat	0.130	0.091	0.156
Age 5 Pat ⟶	Age 3 Pat	0.151	0.119	0.203

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. * Significant based on p<.05. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C6. Confidence intervals of standardized model results for unconstrained non-poverty group of girls.

Non-Poverty Group	Lower 2.5%	Estimate	Upper 2.5%
Age 17 Int ON			
Age 14 Int	0.402	0.442	0.482
Age 14 Ext	0.044	0.087	0.130
Age 14 Mat	0.011	0.055	0.099
Age 14 Pat	0.002	0.053	0.104
Age 17 Ext ON			
Age 14 Int	0.037	0.085	0.133
Age 14 Ext	0.329	0.388	0.447
Age 14 Mat	-0.062	-0.017	0.028
Age 14 Pat	0.003	0.054	0.104
Age 17 Mat ON			
Age 14 Int	0.026	0.078	0.130
Age 14 Ext	-0.010	0.042	0.094
Age 14 Mat	0.290	0.355	0.420
Age 14 Pat	0.060	0.136	0.212
Age 17 Pat ON			
Age 14 Int	-0.043	0.013	0.069
Age 14 Ext	-0.072	-0.001	0.071
Age 14 Mat	-0.054	0.004	0.062
Age 14 Pat	0.321	0.403	0.484
Age 14 Int ON			
Age 11 Int	0.341	0.387	0.433
Age 11 Ext	0.054	0.102	0.149
Age 11 Mat	-0.027	0.021	0.069
Age 11 Pat	-0.032	0.017	0.066
Age 14 Ext ON			
Age 11 Int	0.000	0.050	0.101

Age 11 Ext	0.360	0.413	0.466
Age 11 Mat	0.022	0.072	0.121
Age 11 Pat	-0.017	0.037	0.091
Age 14 Mat ON			
Age 11 Int	0.018	0.069	0.120
Age 11 Ext	0.001	0.055	0.109
Age 11 Mat	0.201	0.267	0.332
Age 11 Pat	-0.078	-0.015	0.048
Age 14 Pat ON			
Age 11 Int	-0.012	0.039	0.090
Age 11 Ext	-0.046	0.019	0.083
Age 11 Mat	0.011	0.078	0.146
Age 11 Pat	0.313	0.388	0.463
Age 11 Int ON			
Age 7 Int	0.221	0.271	0.321
Age 7 Ext	0.065	0.116	0.166
Age 7 Mat	-0.009	0.038	0.084
Age 7 Pat	-0.030	0.031	0.092
Age 11 Ext ON			
Age 7 Int	-0.015	0.032	0.079
Age 7 Ext	0.359	0.410	0.460
Age 7 Mat	0.001	0.052	0.103
Age 7 Pat	-0.041	0.015	0.070
Age 11 Mat ON			
Age 7 Int	0.034	0.093	0.152
Age 7 Ext	0.049	0.099	0.148
Age 7 Mat	0.012	0.080	0.148
Age 7 Pat	0.006	0.068	0.130
Age 11 Pat ON			
Age 7 Int	-0.025	0.033	0.092
Age 7 Ext	-0.024	0.032	0.088
Age 7 Mat	-0.123	-0.059	0.005
Age 7 Pat	0.133	0.216	0.299
Age 7 Int ON			
Age 5 Int	0.153	0.215	0.278
Age 5 Ext	0.004	0.056	0.108
Age 5 Mat	-0.090	-0.032	0.026
Age 5 Pat	-0.083	-0.029	0.024
Age 7 Ext ON			
Age 5 Int	-0.069	-0.021	0.027
Age 5 Ext	0.440	0.484	0.527
Age 5 Mat	-0.013	0.032	0.077
Age 5 Pat	-0.096	-0.042	0.012
Age 7 Mat ON			
Age 5 Int	-0.109	-0.051	0.007
Age 5 Ext	0.089	0.144	0.198
Age 5 Mat	0.099	0.165	0.232

Age 5 Pat	-0.031	0.026	0.083
Age 7 Pat ON			
Age 5 Int	-0.086	-0.014	0.058
Age 5 Ext	0.001	0.058	0.118
Age 5 Mat	0.011	0.084	0.158
Age 5 Pat	-0.040	0.054	0.148
Age 5 Int ON			
Age 3 Int	0.005	0.076	0.147
Age 3 Ext	-0.064	-0.012	0.040
Age 3 Mat	-0.154	-0.092	-0.030
Age 3 Pat	-0.082	-0.010	0.062
Age 5 Ext ON			
Age 3 Int	-0.134	-0.083	-0.032
Age 3 Ext	0.213	0.263	0.314
Age 3 Mat	-0.068	-0.012	0.044
Age 3 Pat	-0.063	-0.002	0.060
Age 5 Mat ON			
Age 3 Int	-0.100	-0.032	0.035
Age 3 Ext	-0.037	0.17	0.070
Age 3 Mat	-0.035	0.055	0.145
Age 3 Pat	-0.157	-0.087	-0.016
Age 5 Pat ON			
Age 3 Int	-0.141	-0.061	0.020
Age 3 Ext	-0.061	0.002	0.066
Age 3 Mat	-0.069	0.012	0.094
Age 3 Pat	-0.232	-0.112	0.008

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C7. Confidence intervals of standardized model results for unconstrained poverty group of girls.

Poverty Group	Lower 2.5%	Estimate	Upper 2.5%
Age 17 Int ON			
Age 14 Int	0.403	0.473	0.543
Age 14 Ext	0.011	0.082	0.153
Age 14 Mat	-0.056	0.019	0.094
Age 14 Pat	-0.064	0.065	0.194
Age 17 Ext ON			
Age 14 Int	0.032	0.102	0.173
Age 14 Ext	0.320	0.408	0.495
Age 14 Mat	-0.002	0.076	0.154
Age 14 Pat	-0.096	0.033	0.161
Age 17 Mat ON			
Age 14 Int	-0.003	0.081	0.165
Age 14 Ext	-0.005	0.087	0.179

Age 14 Mat	0.297	0.407	0.517
Age 14 Pat	-0.168	0.015	0.198
Age 17 Pat ON			
Age 14 Int	-0.107	0.032	0.170
Age 14 Ext	-0.153	-0.014	0.125
Age 14 Mat	-0.250	-0.073	0.104
Age 14 Pat	0.298	0.469	0.641
Age 14 Int ON			
Age 11 Int	0.309	0.387	0.464
Age 11 Ext	0.002	0.084	0.166
Age 11 Mat	-0.046	0.017	0.079
Age 11 Pat	-0.001	0.077	0.153
Age 14 Ext ON			
Age 11 Int	-0.055	0.016	0.087
Age 11 Ext	0.265	0.373	0.482
Age 11 Mat	-0.067	0.024	0.115
Age 11 Pat	-0.058	0.081	0.219
Age 14 Mat ON			
Age 11 Int	-0.029	0.060	0.149
Age 11 Ext	-0.067	0.038	0.144
Age 11 Mat	0.198	0.304	0.411
Age 11 Pat	-0.139	0.006	0.151
Age 14 Pat ON			
Age 11 Int	-0.184	-0.049	0.086
Age 11 Ext	-0.040	0.091	0.222
Age 11 Mat	-0.003	0.208	0.414
Age 11 Pat	0.012	0.180	0.347
Age 11 Int ON			
Age 7 Int	0.157	0.232	0.306
Age 7 Ext	0.062	0.140	0.218
Age 7 Mat	-0.050	0.040	0.130
Age 7 Pat	-0.186	-0.067	0.051
Age 11 Ext ON			
Age 7 Int	-0.079	-0.002	0.075
Age 7 Ext	0.302	0.391	0.481
Age 7 Mat	-0.092	-0.003	0.087
Age 7 Pat	-0.089	0.039	0.166
Age 11 Mat ON			
Age 7 Int	-0.024	0.051	0.126
Age 7 Ext	-0.007	0.080	0.167
Age 7 Mat	0.015	0.135	0.256
Age 7 Pat	-0.084	0.105	0.295
Age 11 Pat ON			
Age 7 Int	-0.125	0.010	0.146
Age 7 Ext	-0.036	0.179	0.323
Age 7 Mat	-0.049	0.119	0.288
Age 7 Pat	0.122	0.281	0.440

Age 7 Int ON			
Age 5 Int	0.145	0.277	0.309
Age 5 Ext	-0.048	0.051	0.150
Age 5 Mat	-0.072	0.035	0.143
Age 5 Pat	0.011	0.180	0.349
Age 7 Ext ON			
Age 5 Int	-0.043	0.028	0.099
Age 5 Ext	0.300	0.392	0.484
Age 5 Mat	-0.053	0.041	0.135
Age 5 Pat	-0.041	0.137	0.315
Age 7 Mat ON			
Age 5 Int	-0.068	0.046	0.160
Age 5 Ext	-0.059	0.032	0.122
Age 5 Mat	0.162	0.284	0.406
Age 5 Pat	-0.139	0.030	0.199
Age 7 Pat ON			
Age 5 Int	-0.161	-0.039	0.083
Age 5 Ext	-0.154	-0.029	0.097
Age 5 Mat	-0.056	0.100	0.256
Age 5 Pat	0.249	0.435	0.622
Age 5 Int ON			
Age 3 Int	0.134	0.227	0.320
Age 3 Ext	-0.014	0.064	0.142
Age 3 Mat	-0.085	-0.015	0.114
Age 3 Pat	-0.165	-0.037	0.091
Age 5 Ext ON			
Age 3 Int	-0.083	0.011	0.105
Age 3 Ext	0.261	0.339	0.417
Age 3 Mat	-0.153	-0.054	0.044
Age 3 Pat	-0.049	0.092	0.233
Age 5 Mat ON			
Age 3 Int	-0.151	-0.028	0.050
Age 3 Ext	-0.037	0.071	0.142
Age 3 Mat	0.056	0.196	0.324
Age 3 Pat	-0.128	0.092	0.243
Age 5 Pat ON			
Age 3 Int	-0.131	0.071	0.203
Age 3 Ext	-0.128	0.127	0.267
Age 3 Mat	-0.160	0.130	0.309
Age 3 Pat	-0.144	0.151	0.383

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. Children's ages are in accordance with their respective median ages at the time of data collection. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C8. Multi-group ALT-SRs with unstandardized constrained autoregressive and cross-lagged parameters for girls.

Parameters	***Est.***	***SE***	***p***
Age 17 Int ⟶ Age 14 Int	0.488	0.020	<.001*
Age 17 Int ⟶ Age 14 Ext	0.103	0.024	<.001*
Age 17 Int ⟶ Age 14 Mat	0.037	0.018	0.041*
Age 17 Int ⟶ Age 14 Pat	0.066	0.029	0.023*
Age 17 Ext ⟶ Age 14 Int	0.070	0.017	<.001*
Age 17 Ext ⟶ Age 14 Ext	0.376	0.024	<.001*
Age 17 Ext ⟶ Age 14 Mat	0.007	0.014	0.631
Age 17 Ext ⟶ Age 14 Pat	0.041	0.021	0.052
Age 17 Mat ⟶ Age 14 Int	0.088	0.025	0.001*
Age 17 Mat ⟶ Age 14 Ext	0.071	0.032	0.026*
Age 17 Mat ⟶ Age 14 Mat	0.390	0.033	<.001*
Age 17 Mat ⟶ Age 14 Pat	0.138	0.040	0.001*
Age 17 Pat ⟶ Age 14 Int	0.016	0.028	0.557
Age 17 Pat ⟶ Age 14 Ext	-0.009	0.044	0.842
Age 17 Pat ⟶ Age 14 Mat	-0.008	0.028	0.768
Age 17 Pat ⟶ Age 14 Pat	0.464	0.046	<.001*
Age 14 Int ⟶ Age 11 Int	0.443	0.024	<.001*
Age 14 Int ⟶ Age 11Ext	0.121	0.026	<.001*
Age 14 Int ⟶ Age 11 Mat	0.018	0.018	0.311
Age 14 Int ⟶ Age 11 Pat	0.025	0.021	0.250
Age 14 Ext ⟶ Age 11 Int	0.038	0.021	0.070
Age 14 Ext ⟶ Age 11 Ext	0.421	0.027	<.001*
Age 14 Ext ⟶ Age 11 Mat	0.045	0.017	0.008*
Age 14 Ext ⟶ Age 11 Pat	0.026	0.022	0.226
Age 14 Mat ⟶ Age 11 Int	0.081	0.028	0.003*
Age 14 Mat ⟶ Age 11 Ext	0.073	0.034	0.031*
Age 14 Mat ⟶ Age 11 Mat	0.277	0.029	<.001*
Age 14 Mat ⟶ Age 11 Pat	-0.022	0.032	0.486
Age 14 Pat ⟶ Age 11 Int	0.035	0.026	0.186
Age 14 Pat ⟶ Age 11 Ext	0.030	0.036	0.406
Age 14 Pat ⟶ Age 11 Mat	0.081	0.031	0.009*
Age 14 Pat ⟶ Age 11 Pat	0.343	0.037	<.001*
Age 11 Int ⟶ Age 7 Int	0.330	0.027	<.001*
Age 11 Int ⟶ Age 7 Ext	0.120	0.022	<.001*
Age 11 Int ⟶ Age 7 Mat	0.035	0.022	0.108
Age 11 Int ⟶ Age 7 Pat	0.016	0.030	0.603
Age 11 Ext ⟶ Age 7 Int	0.025	0.026	0.337

Age 11 Ext → Age 7 Ext	0.378	0.023	<.001*
Age 11 Ext → Age 7 Mat	0.033	0.022	0.134
Age 11 Ext → Age 7 Pat	0.014	0.027	0.587
Age 11 Mat → Age 7 Int	0.130	0.039	0.001*
Age 11 Mat → Age 7 Ext	0.114	0.028	<.001*
Age 11 Mat → Age 7 Mat	0.104	0.038	0.006*
Age 11 Mat → Age 7 Pat	0.082	0.038	0.032*
Age 11 Pat → Age 7 Int	0.046	0.039	0.240
Age 11 Pat → Age 7 Ext	0.053	0.032	0.096
Age 11 Pat → Age 7 Mat	-0.045	0.036	0.206
Age 11 Pat → Age 7 Pat	0.272	0.046	<.001*
Age 7 Int → Age 5 Int	0.238	0.029	<.001*
Age 7 Int → Age 5 Ext	0.048	0.021	0.023*
Age 7 Int → Age 5 Mat	-0.003	0.020	0.885
Age 7 Int → Age 5 Pat	-0.002	0.026	0.943
Age 7 Ext → Age 5 Int	-0.017	0.031	0.583
Age 7 Ext → Age 5 Ext	0.474	0.024	<.001*
Age 7 Ext → Age 5 Mat	0.039	0.020	0.048*
Age 7 Ext → Age 5 Pat	-0.041	0.032	0.203
Age 7 Mat → Age 5 Int	-0.042	0.036	0.234
Age 7 Mat → Age 5 Ext	0.123	0.023	<.001*
Age 7 Mat → Age 5 Mat	0.193	0.032	<.001*
Age 7 Mat → Age 5 Pat	0.018	0.033	0.575
Age 7 Pat → Age 5 Int	-0.027	0.043	0.531
Age 7 Pat → Age 5 Ext	0.043	0.027	0.111
Age 7 Pat → Age 5 Mat	0.092	0.032	0.004*
Age 7 Pat → Age 5 Pat	0.133	0.052	0.010*
Age 5 Int → Age 3 Int	0.122	0.029	<.001*
Age 5 Int → Age 3 Ext	0.003	0.014	0.813
Age 5 Int → Age 3 Mat	-0.046	0.021	0.031*
Age 5 Int → Age 3 Pat	-0.013	0.028	0.647
Age 5 Ext → Age 3 Int	-0.067	0.030	0.029*
Age 5 Ext → Age 3 Ext	0.231	0.020	<.001*
Age 5 Ext → Age 3 Mat	-0.019	0.026	0.470
Age 5 Ext → Age 3 Pat	0.019	0.036	0.595
Age 5 Mat → Age 3 Int	-0.029	0.036	0.422
Age 5 Mat → Age 3 Ext	0.023	0.019	0.224
Age 5 Mat → Age 3 Mat	0.108	0.039	0.006*
Age 5 Mat → Age 3 Pat	-0.064	0.041	0.117
Age 5 Pat → Age 3 Int	-0.049	0.039	0.213

Age 5 Pat ⟶ Age 3 Ext	0.006	0.020	0.762
Age 5 Pat ⟶ Age 3 Mat	0.022	0.032	0.489
Age 5 Pat ⟶ Age 3 Pat	-0.076	0.056	0.173

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. * Significant based on p<.05. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C9. Multi-group ALT-SRs with unconstrained autoregressive and cross-lagged parameters for boys, non-poverty group.

Parameter	***Est.***	***SE***	***p***
Age 17 Int ⟶ Age 14 Int	0.458	0.023	<.001*
Age 17 Int ⟶ Age 14 Ext	0.103	0.025	<.001*
Age 17 Int ⟶ Age 14 Mat	0.023	0.022	0.287
Age 17 Int ⟶ Age 14 Pat	0.028	0.026	0.290
Age 17 Ext ⟶ Age 14 Int	0.035	0.026	0.178
Age 17 Ext ⟶ Age 14 Ext	0.478	0.026	<.001*
Age 17 Ext ⟶ Age 14 Mat	-0.012	0.021	0.589
Age 17 Ext ⟶ Age 14 Pat	0.039	0.026	0.143
Age 17 Mat ⟶ Age 14 Int	0.091	0.028	0.001*
Age 17 Mat ⟶ Age 14 Ext	0.037	0.029	0.201
Age 17 Mat ⟶ Age 14 Mat	0.349	0.034	<.001*
Age 17 Mat ⟶ Age 14 Pat	0.056	0.042	0.187
Age 17 Pat ⟶ Age 14 Int	0.056	0.033	0.093
Age 17 Pat ⟶ Age 14 Ext	0.026	0.035	0.465
Age 17 Pat ⟶ Age 14 Mat	0.037	0.035	0.288
Age 17 Pat ⟶ Age 14 Pat	0.323	0.040	<.001*
Age 14 Int ⟶ Age 11 Int	0.415	0.025	<.001*
Age 14 Int ⟶ Age 11Ext	0.131	0.027	<.001*
Age 14 Int ⟶ Age 11 Mat	0.058	0.024	0.016*
Age 14 Int ⟶ Age 11 Pat	0.044	0.026	0.091
Age 14 Ext ⟶ Age 11 Int	0.083	0.023	<.001*
Age 14 Ext ⟶ Age 11 Ext	0.463	0.028	<.001*
Age 14 Ext ⟶ Age 11 Mat	-0.002	0.022	0.926
Age 14 Ext ⟶ Age 11 Pat	0.058	0.026	0.023*
Age 14 Mat ⟶ Age 11 Int	0.079	0.026	0.002*
Age 14 Mat ⟶ Age 11 Ext	0.045	0.025	0.075
Age 14 Mat ⟶ Age 11 Mat	0.306	0.038	<.001*
Age 14 Mat ⟶ Age 11 Pat	-0.024	0.031	0.430
Age 14 Pat ⟶ Age 11 Int	0.045	0.036	0.201
Age 14 Pat ⟶ Age 11 Ext	0.024	0.039	0.582

Age 14 Pat → Age 11 Mat	0.028	0.037	0.461
Age 14 Pat → Age 11 Pat	0.338	0.043	<.001*
Age 11 Int → Age 7 Int	0.286	0.031	<.001*
Age 11 Int → Age 7 Ext	0.141	0.025	<.001*
Age 11 Int → Age 7 Mat	-0.001	0.029	0.972
Age 11 Int → Age 7 Pat	0.007	0.025	0.765
Age 11 Ext → Age 7 Int	0.064	0.029	0.026*
Age 11 Ext → Age 7 Ext	0.404	0.031	<.001*
Age 11 Ext → Age 7 Mat	0.039	0.030	0.200
Age 11 Ext → Age 7 Pat	0.004	0.026	0.884
Age 11 Mat → Age 7 Int	0.085	0.031	0.006*
Age 11 Mat → Age 7 Ext	0.021	0.028	0.451
Age 11 Mat → Age 7 Mat	0.175	0.039	<.001*
Age 11 Mat → Age 7 Pat	0.004	0.037	0.914
Age 11 Pat → Age 7 Int	0.001	0.027	0.968
Age 11 Pat → Age 7 Ext	0.005	0.033	0.868
Age 11 Pat → Age 7 Mat	0.006	0.035	0.868
Age 11 Pat → Age 7 Pat	0.164	0.045	<.001*
Age 7 Int → Age 5 Int	0.290	0.033	<.001*
Age 7 Int → Age 5 Ext	0.113	0.025	<.001*
Age 7 Int → Age 5 Mat	0.063	0.027	0.022*
Age 7 Int → Age 5 Pat	-0.006	0.027	0.828
Age 7 Ext → Age 5 Int	0.005	0.027	0.860
Age 7 Ext → Age 5 Ext	0.455	0.028	<.001*
Age 7 Ext → Age 5 Mat	0.023	0.025	0.367
Age 7 Ext → Age 5 Pat	-0.007	0.031	0.828
Age 7 Mat → Age 5 Int	-0.004	0.028	0.873
Age 7 Mat → Age 5 Ext	0.081	0.033	0.015*
Age 7 Mat → Age 5 Mat	0.167	0.043	<.001*
Age 7 Mat → Age 5 Pat	0.025	0.034	0.464
Age 7 Pat → Age 5 Int	-0.037	0.032	0.253
Age 7 Pat → Age 5 Ext	0.023	0.033	0.488
Age 7 Pat → Age 5 Mat	0.001	0.031	0.969
Age 7 Pat → Age 5 Pat	0.199	0.049	<.001*
Age 5 Int → Age 3 Int	0.085	0.042	0.042*
Age 5 Int → Age 3 Ext	0.024	0.029	0.400
Age 5 Int → Age 3 Mat	-0.043	0.037	0.244
Age 5 Int → Age 3 Pat	-0.049	0.040	0.218
Age 5 Ext → Age 3 Int	-0.044	0.029	0.134
Age 5 Ext → Age 3 Ext	0.247	0.028	<.001*

Age 5 Ext → Age 3 Mat	0.007	0.030	0.812
Age 5 Ext → Age 3 Pat	0.020	0.030	0.512
Age 5 Mat → Age 3 Int	-0.056	0.037	0.130
Age 5 Mat → Age 3 Ext	0.079	0.033	0.015*
Age 5 Mat → Age 3 Mat	0.052	0.045	0.146
Age 5 Mat → Age 3 Pat	-0.054	0.036	0.141
Age 5 Pat → Age 3 Int	-0.041	0.034	0.227
Age 5 Pat → Age 3 Ext	0.002	0.028	0.942
Age 5 Pat → Age 3 Mat	0.020	0.035	0.574
Age 5 Pat → Age 3 Pat	0.085	0.053	0.107

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. * Significant based on p<.05. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C10. Multi-group ALT-SRs with unconstrained autoregressive and cross-lagged parameters for boys, poverty group.

Parameter	***Est.***	***SE***	***p***
Age 17 Int → Age 14 Int	0.502	0.039	<.001*
Age 17 Int → Age 14 Ext	0.068	0.039	0.084
Age 17 Int → Age 14 Mat	0.005	0.041	0.910
Age 17 Int → Age 14 Pat	0.022	0.093	0.815
Age 17 Ext → Age 14 Int	0.136	0.042	0.001*
Age 17 Ext → Age 14 Ext	0.389	0.055	<.001*
Age 17 Ext → Age 14 Mat	-0.003	0.053	0.951
Age 17 Ext → Age 14 Pat	0.036	0.119	0.760
Age 17 Mat → Age 14 Int	0.135	0.050	0.007*
Age 17 Mat → Age 14 Ext	0.044	0.055	0.40
Age 17 Mat → Age 14 Mat	0.430	0.057	<.001*
Age 17 Mat → Age 14 Pat	0.010	0.131	0.940
Age 17 Pat → Age 14 Int	0.248	0.069	<.001*
Age 17 Pat → Age 14 Ext	-0.014	0.101	0.888
Age 17 Pat → Age 14 Mat	0.067	0.071	0.344
Age 17 Pat → Age 14 Pat	0.473	0.082	<.001*
Age 14 Int → Age 11 Int	0.482	0.036	<.001*
Age 14 Int → Age 11Ext	0.114	0.038	0.003*
Age 14 Int → Age 11 Mat	-0.022	0.036	0.548
Age 14 Int → Age 11 Pat	0.057	0.062	0.351
Age 14 Ext → Age 11 Int	0.122	0.036	0.001*
Age 14 Ext → Age 11 Ext	0.432	0.047	<.001*
Age 14 Ext → Age 11 Mat	0.021	0.035	0.555

Age 14 Ext → Age 11 Pat	-0.024	0.045	0.593
Age 14 Mat → Age 11 Int	0.077	0.039	0.050*
Age 14 Mat → Age 11 Ext	0.004	0.047	0.937
Age 14 Mat → Age 11 Mat	0.234	0.057	<.001*
Age 14 Mat → Age 11 Pat	0.036	0.082	0.660
Age 14 Pat → Age 11 Int	0.115	0.095	0.228
Age 14 Pat → Age 11 Ext	0.045	0.094	0.633
Age 14 Pat → Age 11 Mat	0.010	0.112	0.926
Age 14 Pat → Age 11 Pat	0.436	0.058	<.001*
Age 11 Int → Age 7 Int	0.282	0.045	<.001*
Age 11 Int → Age 7 Ext	0.225	0.041	<.001*
Age 11 Int → Age 7 Mat	0.012	0.034	0.734
Age 11 Int → Age 7 Pat	0.094	0.064	0.143
Age 11 Ext → Age 7 Int	0.024	0.043	0.581
Age 11 Ext → Age 7 Ext	0.372	0.048	<.001*
Age 11 Ext → Age 7 Mat	0.063	0.046	0.174
Age 11 Ext → Age 7 Pat	0.082	0.060	0.168
Age 11 Mat → Age 7 Int	0.060	0.053	0.253
Age 11 Mat → Age 7 Ext	0.059	0.054	0.274
Age 11 Mat → Age 7 Mat	0.175	0.061	0.004*
Age 11 Mat → Age 7 Pat	0.111	0.094	0.238
Age 11 Pat → Age 7 Int	0.100	0.063	0.111
Age 11 Pat → Age 7 Ext	-0.017	0.083	0.835
Age 11 Pat → Age 7 Mat	-0.102	0.063	0.107
Age 11 Pat → Age 7 Pat	0.370	0.109	0.001*
Age 7 Int → Age 5 Int	0.311	0.045	<.001*
Age 7 Int → Age 5 Ext	0.165	0.041	<.001*
Age 7 Int → Age 5 Mat	0.075	0.043	0.084
Age 7 Int → Age 5 Pat	0.066	0.066	0.314
Age 7 Ext → Age 5 Int	0.049	0.032	0.121
Age 7 Ext → Age 5 Ext	0.431	0.037	<.001*
Age 7 Ext → Age 5 Mat	0.056	0.039	0.149
Age 7 Ext → Age 5 Pat	-0.030	0.062	0.652
Age 7 Mat → Age 5 Int	-0.010	0.042	0.804
Age 7 Mat → Age 5 Ext	0.138	0.051	0.007*
Age 7 Mat → Age 5 Mat	0.138	0.056	0.014*
Age 7 Mat → Age 5 Pat	-0.089	0.074	0.229
Age 7 Pat → Age 5 Int	0.062	0.069	0.370
Age 7 Pat → Age 5 Ext	0.069	0.064	0.282
Age 7 Pat → Age 5 Mat	0.061	0.092	0.511

Age 7 Pat → Age 5 Pat	0.195	0.086	0.023*
Age 5 Int → Age 3 Int	0.273	0.048	<.001*
Age 5 Int → Age 3 Ext	0.030	0.034	0.390
Age 5 Int → Age 3 Mat	0.072	0.050	0.145
Age 5 Int → Age 3 Pat	0.024	0.068	0.719
Age 5 Ext → Age 3 Int	0.007	0.041	0.859
Age 5 Ext → Age 3 Ext	0.306	0.040	<.001*
Age 5 Ext → Age 3 Mat	0.110	0.046	0.016*
Age 5 Ext → Age 3 Pat	-0.131	0.059	0.028*
Age 5 Mat → Age 3 Int	0.044	0.038	0.247
Age 5 Mat → Age 3 Ext	0.051	0.042	0.230
Age 5 Mat → Age 3 Mat	0.104	0.056	0.064
Age 5 Mat → Age 3 Pat	0.147	0.075	0.051
Age 5 Pat → Age 3 Int	0.096	0.058	0.097
Age 5 Pat → Age 3 Ext	0.063	0.062	0.312
Age 5 Pat → Age 3 Mat	0.020	0.079	0.801
Age 5 Pat → Age 3 Pat	0.079	0.108	0.464

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. * Significant based on p<.05. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C11. Confidence intervals of standardized model results for unconstrained non-poverty group of boys.

Non-Poverty Group	Lower 2.5%	Estimate	Upper 2.5%
Age 17 Int ON			
Age 14 Int	0.412	0.458	0.503
Age 14 Ext	0.054	0.103	0.152
Age 14 Mat	-0.020	0.023	0.066
Age 14 Pat	-0.024	0.028	0.079
Age 17 Ext ON			
Age 14 Int	-0.016	0.035	0.085
Age 14 Ext	0.427	0.478	0.529
Age 14 Mat	-0.054	-0.012	0.030
Age 14 Pat	-0.013	0.039	0.090
Age 17 Mat ON			
Age 14 Int	0.037	0.091	0.145
Age 14 Ext	-0.020	0.037	0.095
Age 14 Mat	0.281	0.349	0.416
Age 14 Pat	-0.027	0.056	0.139
Age 17 Pat ON			
Age 14 Int	-0.009	0.056	0.121
Age 14 Ext	-0.043	0.026	0.095
Age 14 Mat	-0.031	0.037	0.106
Age 14 Pat	0.245	0.323	0.402

Age 14 Int ON			
Age 11 Int	0.366	0.415	0.463
Age 11 Ext	0.078	0.131	0.184
Age 11 Mat	0.011	0.058	0.105
Age 11 Pat	-0.007	0.044	0.094
Age 14 Ext ON			
Age 11 Int	0.038	0.083	0.128
Age 11 Ext	0.408	0.463	0.518
Age 11 Mat	-0.045	-0.002	0.041
Age 11 Pat	0.008	0.058	0.108
Age 14 Mat ON			
Age 11 Int	0.028	0.079	0.131
Age 11 Ext	-0.005	0.045	0.094
Age 11 Mat	0.231	0.306	0.381
Age 11 Pat	-0.084	-0.024	0.036
Age 14 Pat ON			
Age 11 Int	-0.024	0.045	0.115
Age 11 Ext	-0.055	0.024	0.098
Age 11 Mat	-0.046	0.028	0.101
Age 11 Pat	0.254	0.338	0.421
Age 11 Int ON			
Age 7 Int	0.225	0.286	0.346
Age 7 Ext	0.091	0.141	0.190
Age 7 Mat	-0.058	-0.001	0.056
Age 7 Pat	-0.041	0.007	0.056
Age 11 Ext ON			
Age 7 Int	0.008	0.064	0.121
Age 7 Ext	0.342	0.404	0.465
Age 7 Mat	-0.020	0.039	0.098
Age 7 Pat	-0.047	0.004	0.054
Age 11 Mat ON			
Age 7 Int	0.025	0.085	0.145
Age 7 Ext	-0.034	0.021	0.076
Age 7 Mat	0.099	0.175	0.250
Age 7 Pat	-0.068	0.004	0.076
Age 11 Pat ON			
Age 7 Int	-0.052	0.001	0.055
Age 7 Ext	-0.058	0.005	0.069
Age 7 Mat	-0.062	0.006	0.074
Age 7 Pat	0.077	0.164	0.252
Age 7 Int ON			
Age 5 Int	0.225	0.290	0.356
Age 5 Ext	0.063	0.113	0.163
Age 5 Mat	0.009	0.063	0.117
Age 5 Pat	-0.059	-0.006	0.047
Age 7 Ext ON			
Age 5 Int	-0.049	0.005	0.059

Age 5 Ext	0.400	0.455	0.510
Age 5 Mat	-0.026	0.023	0.072
Age 5 Pat	-0.068	-0.007	0.054
Age 7 Mat ON			
Age 5 Int	-0.059	-0.004	0.050
Age 5 Ext	0.016	0.081	0.145
Age 5 Mat	0.083	0.167	0.251
Age 5 Pat	-0.042	0.025	0.091
Age 7 Pat ON			
Age 5 Int	-0.100	-0.037	0.026
Age 5 Ext	-0.041	0.023	0.087
Age 5 Mat	-0.060	0.001	0.063
Age 5 Pat	0.102	0.199	0.296
Age 5 Int ON			
Age 3 Int	0.003	0.085	0.167
Age 3 Ext	-0.032	0.024	0.081
Age 3 Mat	-0.115	-0.043	0.029
Age 3 Pat	-0.128	-0.049	0.029
Age 5 Ext ON			
Age 3 Int	-0.102	-0.044	0.014
Age 3 Ext	0.192	0.247	0.302
Age 3 Mat	-0.052	0.007	0.066
Age 3 Pat	-0.039	0.020	0.079
Age 5 Mat ON			
Age 3 Int	-0.128	-0.056	0.016
Age 3 Ext	0.015	0.079	0.143
Age 3 Mat	-0.036	0.052	0.140
Age 3 Pat	-0.125	-0.054	0.018
Age 5 Pat ON			
Age 3 Int	-0.108	-0.041	0.026
Age 3 Ext	-0.054	0.002	0.058
Age 3 Mat	-0.049	0.020	0.088
Age 3 Pat	-0.018	0.085	0.188

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C12. Confidence intervals of standardized model results for unconstrained poverty group of boys.

Non-Poverty Group	Lower 2.5%	Estimate	Upper 2.5%
Age 17 Int ON			
Age 14 Int	0.425	0.502	0.578
Age 14 Ext	-0.009	0.068	0.146
Age 14 Mat	-0.076	0.005	0.086
Age 14 Pat	-0.161	0.022	0.204
Age 17 Ext ON			

Age 14 Int	0.055	0.136	0.218
Age 14 Ext	0.281	0.389	0.497
Age 14 Mat	-0.108	-0.003	0.101
Age 14 Pat	-0.197	0.036	0.270
Age 17 Mat ON			
Age 14 Int	0.038	0.135	0.233
Age 14 Ext	-0.063	0.044	0.152
Age 14 Mat	0.318	0.430	0.541
Age 14 Pat	-0.247	0.010	0.267
Age 17 Pat ON			
Age 14 Int	0.113	0.248	0.382
Age 14 Ext	-0.212	-0.014	0.184
Age 14 Mat	-0.071	0.067	0.205
Age 14 Pat	0.311	0.473	0.634
Age 14 Int ON			
Age 11 Int	0.411	0.482	0.553
Age 11 Ext	0.039	0.114	0.189
Age 11 Mat	-0.092	-0.022	0.049
Age 11 Pat	-0.063	0.057	0.178
Age 14 Ext ON			
Age 11 Int	0.051	0.122	0.194
Age 11 Ext	0.340	0.432	0.524
Age 11 Mat	-0.048	0.021	0.090
Age 11 Pat	-0.111	-0.024	0.064
Age 14 Mat ON			
Age 11 Int	0.000	0.077	0.153
Age 11 Ext	-0.088	0.004	0.096
Age 11 Mat	0.121	0.234	0.346
Age 11 Pat	-0.125	0.036	0.197
Age 14 Pat ON			
Age 11 Int	-0.072	0.115	0.301
Age 11 Ext	-0.140	0.045	0.229
Age 11 Mat	-0.209	0.010	0.230
Age 11 Pat	0.322	0.436	0.549
Age 11 Int ON			
Age 7 Int	0.193	0.282	0.371
Age 7 Ext	0.145	0.225	0.305
Age 7 Mat	-0.056	0.012	0.079
Age 7 Pat	-0.032	0.094	0.219
Age 11 Ext ON			
Age 7 Int	-0.061	0.024	0.108
Age 7 Ext	0.277	0.372	0.467
Age 7 Mat	-0.028	0.063	0.153
Age 7 Pat	-0.035	0.082	0.199
Age 11 Mat ON			
Age 7 Int	-0.043	0.060	0.163
Age 7 Ext	-0.047	0.059	0.165

Age 7 Mat	0.054	0.175	0.295
Age 7 Pat	-0.074	0.111	0.296
Age 11 Pat ON			
Age 7 Int	-0.023	0.100	0.223
Age 7 Ext	-0.180	-0.017	0.145
Age 7 Mat	-0.226	-0.102	0.022
Age 7 Pat	0.157	0.370	0.583
Age 7 Int ON			
Age 5 Int	0.222	0.311	0.400
Age 5 Ext	0.085	0.165	0.246
Age 5 Mat	-0.010	0.075	0.159
Age 5 Pat	-0.063	0.066	0.195
Age 7 Ext ON			
Age 5 Int	-0.013	0.049	0.112
Age 5 Ext	0.358	0.431	0.504
Age 5 Mat	-0.020	0.056	0.133
Age 5 Pat	-0.151	-0.030	0.091
Age 7 Mat ON			
Age 5 Int	-0.092	-0.010	0.072
Age 5 Ext	0.039	0.138	0.238
Age 5 Mat	0.027	0.138	0.249
Age 5 Pat	-0.233	-0.089	0.056
Age 7 Pat ON			
Age 5 Int	-0.073	0.062	0.197
Age 5 Ext	-0.057	0.069	0.194
Age 5 Mat	-0.120	0.061	0.241
Age 5 Pat	0.027	0.195	0.363
Age 5 Int ON			
Age 3 Int	0.179	0.273	0.367
Age 3 Ext	-0.038	0.030	0.097
Age 3 Mat	-0.025	0.072	0.170
Age 3 Pat	-0.108	0.024	0.157
Age 5 Ext ON			
Age 3 Int	-0.073	0.007	0.088
Age 3 Ext	0.228	0.306	0.385
Age 3 Mat	0.021	0.110	0.199
Age 3 Pat	-0.247	-0.131	-0.014
Age 5 Mat ON			
Age 3 Int	-0.031	0.044	0.119
Age 3 Ext	-0.032	0.051	0.134
Age 3 Mat	-0.006	0.104	0.215
Age 3 Pat	0.000	0.147	0.295
Age 5 Pat ON			
Age 3 Int	-0.018	0.096	0.210
Age 3 Ext	-0.059	0.063	0.185
Age 3 Mat	-0.134	0.020	0.174
Age 3 Pat	-0.133	0.079	0.292

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. Children's ages are in accordance with their respective median ages at the time of data collection.

Table C13. Multi-group ALT-SRs with unstandardized constrained autoregressive and cross-lagged parameters for boys.

Parameter	***Est.***	***SE***	***p***
Age 17 Int ⟶ Age 14 Int	0.470	0.019	<.001*
Age 17 Int ⟶ Age 14 Ext	0.095	0.021	<.001*
Age 17 Int ⟶ Age 14 Mat	0.016	0.018	0.391
Age 17 Int ⟶ Age 14 Pat	0.018	0.027	0.492
Age 17 Ext ⟶ Age 14 Int	0.057	0.020	0.005*
Age 17 Ext ⟶ Age 14 Ext	0.434	0.024	<.001*
Age 17 Ext ⟶ Age 14 Mat	-0.010	0.018	0.582
Age 17 Ext ⟶ Age 14 Pat	0.028	0.027	0.296
Age 17 Mat ⟶ Age 14 Int	0.121	0.030	<.001*
Age 17 Mat ⟶ Age 14 Ext	0.045	0.032	0.160
Age 17 Mat ⟶ Age 14 Mat	0.440	0.034	<.001*
Age 17 Mat ⟶ Age 14 Pat	0.064	0.050	0.200
Age 17 Pat ⟶ Age 14 Int	0.094	0.033	0.005*
Age 17 Pat ⟶ Age 14 Ext	0.027	0.040	0.502
Age 17 Pat ⟶ Age 14 Mat	0.048	0.035	0.175
Age 17 Pat ⟶ Age 14 Pat	0.414	0.045	<.001*
Age 14 Int ⟶ Age 11 Int	0.489	0.024	<.001*
Age 14 Int ⟶ Age 11Ext	0.139	0.024	<.001*
Age 14 Int ⟶ Age 11 Mat	0.031	0.018	0.091
Age 14 Int ⟶ Age 11 Pat	0.038	0.022	0.088
Age 14 Ext ⟶ Age 11 Int	0.099	0.021	<.001*
Age 14 Ext ⟶ Age 11 Ext	0.473	0.026	<.001*
Age 14 Ext ⟶ Age 11 Mat	0.001	0.017	0.960
Age 14 Ext ⟶ Age 11 Pat	0.032	0.021	0.119
Age 14 Mat ⟶ Age 11 Int	0.087	0.023	<.001*
Age 14 Mat ⟶ Age 11 Ext	0.040	0.025	0.101
Age 14 Mat ⟶ Age 11 Mat	0.275	0.032	<.001*
Age 14 Mat ⟶ Age 11 Pat	-0.028	0.028	0.331
Age 14 Pat ⟶ Age 11 Int	0.059	0.034	0.084
Age 14 Pat ⟶ Age 11 Ext	0.026	0.038	0.504
Age 14 Pat ⟶ Age 11 Mat	0.022	0.030	0.459
Age 14 Pat ⟶ Age 11 Pat	0.337	0.034	<.001*
Age 11 Int ⟶ Age 7 Int	0.328	0.030	<.001*

Age 11 Int ⟶ Age 7 Ext	0.159	0.021	<.001*
Age 11 Int ⟶ Age 7 Mat	0.004	0.021	0.863
Age 11 Int ⟶ Age 7 Pat	0.022	0.025	0.375
Age 11 Ext ⟶ Age 7 Int	0.062	0.028	0.027*
Age 11 Ext ⟶ Age 7 Ext	0.383	0.026	<.001*
Age 11 Ext ⟶ Age 7 Mat	0.044	0.027	0.103
Age 11 Ext ⟶ Age 7 Pat	0.009	0.026	0.725
Age 11 Mat ⟶ Age 7 Int	0.109	0.037	0.003*
Age 11 Mat ⟶ Age 7 Ext	0.037	0.028	0.196
Age 11 Mat ⟶ Age 7 Mat	0.202	0.037	<.001*
Age 11 Mat ⟶ Age 7 Pat	0.016	0.042	0.704
Age 11 Pat ⟶ Age 7 Int	0.017	0.031	0.589
Age 11 Pat ⟶ Age 7 Ext	0.002	0.031	0.959
Age 11 Pat ⟶ Age 7 Mat	-0.018	0.036	0.619
Age 11 Pat ⟶ Age 7 Pat	0.232	0.051	<.001*
Age 7 Int ⟶ Age 5 Int	0.349	0.032	<.001*
Age 7 Int ⟶ Age 5 Ext	0.105	0.018	<.001*
Age 7 Int ⟶ Age 5 Mat	0.063	0.020	0.002*
Age 7 Int ⟶ Age 5 Pat	0.004	0.025	0.887
Age 7 Ext ⟶ Age 5 Int	0.032	0.029	0.266
Age 7 Ext ⟶ Age 5 Ext	0.457	0.025	<.001*
Age 7 Ext ⟶ Age 5 Mat	0.035	0.023	0.121
Age 7 Ext ⟶ Age 5 Pat	-0.012	0.032	0.721
Age 7 Mat ⟶ Age 5 Int	0.002	0.033	0.954
Age 7 Mat ⟶ Age 5 Ext	0.095	0.028	0.001*
Age 7 Mat ⟶ Age 5 Mat	0.166	0.036	<.001*
Age 7 Mat ⟶ Age 5 Pat	0.007	0.034	0.841
Age 7 Pat ⟶ Age 5 Int	-0.023	0.036	0.534
Age 7 Pat ⟶ Age 5 Ext	0.019	0.027	0.468
Age 7 Pat ⟶ Age 5 Mat	0.009	0.032	0.773
Age 7 Pat ⟶ Age 5 Pat	0.194	0.047	<.001*
Age 5 Int ⟶ Age 3 Int	0.154	0.034	<.001*
Age 5 Int ⟶ Age 3 Ext	0.021	0.014	0.136
Age 5 Int ⟶ Age 3 Mat	0.006	0.023	0.791
Age 5 Int ⟶ Age 3 Pat	-0.040	0.035	0.250
Age 5 Ext ⟶ Age 3 Int	-0.029	0.032	0.362
Age 5 Ext ⟶ Age 3 Ext	0.230	0.021	<.001*
Age 5 Ext ⟶ Age 3 Mat	0.050	0.027	0.062
Age 5 Ext ⟶ Age 3 Pat	-0.026	0.037	0.482
Age 5 Mat ⟶ Age 3 Int	-0.025	0.036	0.497

Age 5 Mat ⟶	Age 3 Ext	0.062	0.021	0.004*
Age 5 Mat ⟶	Age 3 Mat	0.078	0.034	0.024*
Age 5 Mat ⟶	Age 3 Pat	-0.033	0.040	0.414
Age 5 Pat ⟶	Age 3 Int	-0.023	0.036	0.532
Age 5 Pat ⟶	Age 3 Ext	0.007	0.019	0.724
Age 5 Pat ⟶	Age 3 Mat	0.024	0.030	0.433
Age 5 Pat ⟶	Age 3 Pat	0.071	0.057	0.212

Int, internalising problems; Ext, externalising problems; Mat, maternal distress; Pat, paternal distress. * Significant based on $p<.05$. Children's ages are in accordance with their respective median ages at the time of data collection.