Childhood adversity, resilience, and internalising and externalising outcomes (Alternative Format Thesis)

Bauer, Andreas

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Table of Contents

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Abstract

Adverse and traumatic experiences in childhood are associated with multiple forms of child and adolescent psychopathology. However, the vast majority of research on this topic has been carried out in high-income and/or Western countries, and it remains unclear whether those findings are applicable across social and cultural contexts. Thus, the aim of the current thesis was to examine the relationship between childhood adversities and psychopathology, especially focusing on low- and middle-income and non-Western countries and aggressive and antisocial behaviours in young people. In line with previous research from high-income countries, study 1 provides evidence of childhood trauma as a transdiagnostic risk factor for psychopathology in childhood, using data from a large birth cohort study from Brazil, a middle-income country. As well as confirming the harmful effects of interpersonal trauma, study 1 further identifies non-interpersonal trauma as a significant contributor to child psychopathology. Using data from the same birth cohort, study 2 found reciprocal effects between harsh parenting and child conduct problems, and unidirectional effects of harsh parenting on child emotional problems, corroborating results from high-income countries. Extending existing developmental trajectories of conduct problems from ages 4-13 years up to age 17 years in a large UK birth cohort, study 3 found similar patterns of associations between child abuse and early-onset persistent and adolescence-onset conduct problems, suggesting quantitative as opposed to qualitative differences between these conduct problem trajectories. Study 4 used data from two nationally representative samples of school-aged children in South Korea. In line with research from Western countries, the findings highlight the potential protective effect of neighbourhood collective efficacy on family violence and youth antisocial behaviour. Finally, study 5 examined child resilience to maternal depression, again using longitudinal data from a Brazilian birth cohort study. The study found evidence of indirect effects from SES to resilience via early cognitive stimulation and IQ, suggesting that preventive

interventions focusing on promoting cognitive stimulation and cognitive development may foster positive outcomes in children exposed to maternal depression. Collectively, while these studies provide further evidence of the harmful effects of adverse and traumatic experiences in childhood across social and cultural contexts, they also highlight the role of potential protective individual-, family-, and neighbourhood-level factors.

Chapter 1 – Introduction

The current thesis examined the association between childhood adversities and child and adolescent psychopathology. To gain a better understanding of the individual components that make up this relationship, a brief overview of issues and concepts related to mental health problems in young people and exposure to adverse and/or traumatic experiences in childhood is provided. First, the prevalence, comorbidity, and consequences of experiencing mental health issues in childhood and/or adolescence are each discussed, with a particular focus on child conduct problems. Next, definitional issues of childhood adversities are examined, including consideration of their prevalence, comorbidity, timing, specificity across mental health outcomes, and major limitations in the existing literature on adverse childhood experiences and childhood trauma. Finally, research questions are presented alongside a brief outline of each empirical chapter included in the thesis.

Mental health problems in young people

Prevalence and co-occurrence

Using data from 41 studies across 27 countries, Polanczyk et al. (2015) estimated the worldwide prevalence of mental disorders in children and adolescents aged 4-18 years to be 13.4%.[1] Breaking this figure down by diagnostic group, global prevalence rates were 6.5% for anxiety disorders, 2.6% for mood disorders, 3.4% for ADHD/hyperactivity disorders, and 5.7% for conduct/oppositional disorders (Polanczyk et al., 2015). Importantly, these estimates did not vary across seven geographic locations, suggesting more similarities than differences in the prevalence of childhood-onset psychiatric disorders across the world. However, it should be noted that some of the world regions were poorly represented; for example, only two prevalence studies were included from Africa and the Middle East. This lack of evidence from

[1] Less frequent psychiatric diagnostic categories, such as eating and psychotic disorders, were not presented separately; thus, the number of those diagnosed with 'any mental disorder' exceeds the totals for the major diagnostic groups.

some world regions is further illustrated in a study on the global coverage of prevalence data of mental disorders in young people (Erskine, Baxter, et al., 2016). More specifically, there was no data available from 124 out of 187 countries, with a particularly poor coverage in Sub-Saharan Africa, Latin America, Asia, and Oceania (Erskine, Baxter, et al., 2016). Furthermore, as Costello (2015) points out, there may be variability within geographic locations, suggesting caution when grouping countries into broader geographical clusters. Finally, focusing on younger children, Vasileva et al. (2020) estimated the global prevalence of mental disorders in children aged 1-7 years to be 20.1%. The higher prevalence of mental disorders in younger compared to older children may be explained by methodological factors (e.g., number of included psychiatric diagnoses) and the fact that some disorders are typically diagnosed in younger children (e.g., separation anxiety). For specific diagnostic categories, prevalence rates were 8.5% for anxiety disorders, 1.1% for mood disorders, 4.3% for ADHD/hyperactivity disorders, and 4.9% for conduct/oppositional disorders (Vasileva et al., 2020). However, again, 7 out of the 10 included studies were conducted in the US and Western Europe. Collectively, both these syntheses of global prevalence studies contributed substantially to the field of developmental psychopathology. Nonetheless, they revealed major gaps in our understanding of the prevalence of childhood psychiatric disorders, especially in low- and middle-income and non-Western countries across all age groups.

Vasileva et al. (2020) also examined the global comorbidity of mental disorders in young children, which was estimated at 6.4%, representing the proportion of children aged 1-7 years that are diagnosed with two or more co-occurring psychiatric disorders. Thus, almost one in three children with a mental disorder is diagnosed with another mental disorder. In a nationally representative sample of US adolescents aged 13-18 years, the *lifetime* prevalence of mental disorders was estimated to be 49.5%, with a comorbidity rate of 40% (Merikangas

et al., 2010). Put differently, almost half of adolescents with a psychiatric disorder fulfilled the diagnostic criteria for at least one additional psychiatric disorder in their lifetime.

Adverse consequences of childhood psychiatric disorders

Childhood mental disorders are the leading cause of disability in young people worldwide (Baranne & Falissard, 2018; Erskine et al., 2015). In high-income countries, they account for the largest proportion of disease burden (Baranne & Falissard, 2018; Erskine et al., 2015). Given the recent improvements in health service provision, especially for infectious diseases and neonatal disorders, the proportion of disease burden attributable to childhood mental disorders is likely to increase in low- and middle-income countries (Baranne & Falissard, 2018; Erskine et al., 2015). Furthermore, the detrimental effects of childhood-onset psychiatric disorders often continue into adulthood. For example, Copeland et al. (2015) reported that those with childhood-onset psychiatric disorders were substantially more likely to show negative outcomes across health, legal, financial, and social domains in young adulthood compared to those with no history of mental illness, even in the case of subthreshold diagnoses and when the mental disorder itself did not persist into adulthood. Similarly, Costello and Maughan (2015) reported that although half of adults with a history of mental illness in childhood were disorder-free in adulthood, they still showed impaired adult functioning compared to their peers without a history of mental illness, probably due to a lack of protective personal characteristics (e.g., impulse control in the case of conduct/oppositional disorders) and/or supportive home and school environments. Finally, beyond the personal costs of childhood mental health problems, these disorders lead to major societal costs. As an example of this, Snell et al. (2013) used data from children aged 5-10 years in the UK who were diagnosed with a psychiatric disorder and found that costs related to increased use of health, education, and social care services amounted to £1.47 billion in one year. This highlights the substantial economic impact of childhood psychiatric disorders on the public sector and society more generally.

In focus: conduct problems

Although childhood adversities have been proposed as transdiagnostic risk factors for multiple forms of psychopathology (McLaughlin, 2016; McLaughlin, Colich, Rodman, & Weissman, 2020), given the focus on aggressive and antisocial behaviour in the current thesis, some of their key characteristics are summarised here.

Conduct problems refer to antisocial behaviours displayed in childhood and/or adolescence that are symptomatic of conduct disorder and oppositional defiant disorder (American Psychiatric Association, 2013). As mentioned above, the global prevalence of conduct/oppositional disorders was estimated to be 4.9% in children aged 1-7 years and 5.7% in those aged 4-18 years (Polanczyk et al., 2015; Vasileva et al., 2020). Erskine et al. (2013) estimated the global prevalence of conduct disorder to be 3.6% in boys and 1.5% in girls, with little variation across the world. However, similar to the study by Polanczyk et al. (2015), some world regions were poorly represented. For example, in contrast to North America and Europe, there was no data available for large parts of Africa and Latin America. In another systematic review focusing on Brazil, Murray et al. (2013) reported a prevalence of 4.1% for conduct/oppositional disorders. Interestingly, the prevalence of child conduct problems was higher in Brazil compared to other countries in studies using screening questionnaires but not when diagnostic instruments were used. The authors proposed that Brazilian children may show higher levels of sub-clinical behaviour problems to adapt to harsher environments and/or as a result of peer pressure among antisocial groups. Alternatively, this finding might be the result of informant bias, as lower literacy levels in Brazilian parents may mean that they do not fully understand the nuances in questionnaire items (Murray et al., 2013). However, it should be noted that different cut-off scores were used across included studies. For example, while some studies required *high* (formerly called ‘abnormal’) scores on the SDQ conduct problems subscale, sometimes with and sometimes without impairment symptoms, one included study

defined conduct problems as experiencing *slightly raised* (formerly called 'borderline') symptoms. Thus, previous findings showing a higher prevalence of conduct problems in Brazil may also stem from different methodological approaches.

Conduct disorder often co-occurs with other psychiatric disorders, particularly with ADHD/hyperactivity disorders (see Figure 1) (Angold, Costello, & Erkanli, 1999). Thus, these disorders are often referred to jointly as *externalising disorders* or *externalising problems*. However, it should be noted that there is some evidence suggesting that children who only experience either conduct problems or hyperactive-impulsive-attention problems differ from those who show both types of behavioural problems (Waschbusch, 2002). For example, children in the latter group may be more likely to show early-onset persistent disruptive behaviours (Waschbusch, 2002).

Conduct disorder places a substantial burden on young people, ranking as the 30th leading cause of impairment and reduced quality of life worldwide (Erskine et al., 2014). Furthermore, conduct disorder is associated with negative outcomes in multiple domains, such as academic achievement, criminality, and substance abuse (Erskine, Norman, et al., 2016). Comparing Brazilian and British birth cohorts, Hammerton et al. (2019) reported that child conduct problems in both cohorts were associated with increased risk of criminal behaviour, emotional disorders, and unemployment in young adulthood. However, while associations with alcohol and substance abuse were stronger in Brazil compared to the UK, associations for criminal behaviour and unemployment were stronger in the British cohort. The fact that children in the low-risk British cohort had stronger associations for some outcomes compared to children in the high-risk Brazilian cohort may be explained with reference to the *gender paradox* (Loeber & Keenan, 1994). More precisely, previous research has shown that the sex with the lower prevalence rate of a psychiatric disorder are at higher risk of poor outcomes (Diamantopoulou, Verhulst, & van der Ende, 2011). Thus, according to this logic, while British

children showed lower levels of conduct problems compared to Brazilian children, their conduct problems may be more pervasive and the risk of developing poor outcomes may be higher. Tiet and colleagues (2001) proposed a number of explanations for the gender paradox, including (i) different risk thresholds for each sex; (ii) greater genetic variability in boys; and (iii) the possibility that sex may affect outcomes differently at different levels of risk. Following this line of thought, conduct problems may be more socially acceptable in Brazil, which would make it less likely for children showing conduct problems to be excluded. Alternatively, conduct problems in lower prevalence settings may have different underpinnings, such as higher genetic load in contexts with fewer environmental risk factors.

Finally, child conduct problems place a major burden on the public sector and society at large, especially conduct problems that emerge in childhood and continue into adulthood, which account for disproportionally high costs of criminal justice, health, and social welfare services (Rivenbark et al., 2018). The societal costs are particularly high for those diagnosed with conduct disorder, but are still substantial for children showing sub-clinical conduct problems compared to those showing no conduct problems (Scott, Knapp, Henderson, & Maughan, 2001).

Figure 1 Associations between psychiatric diagnoses (reproduced from Angold et al., 1999)

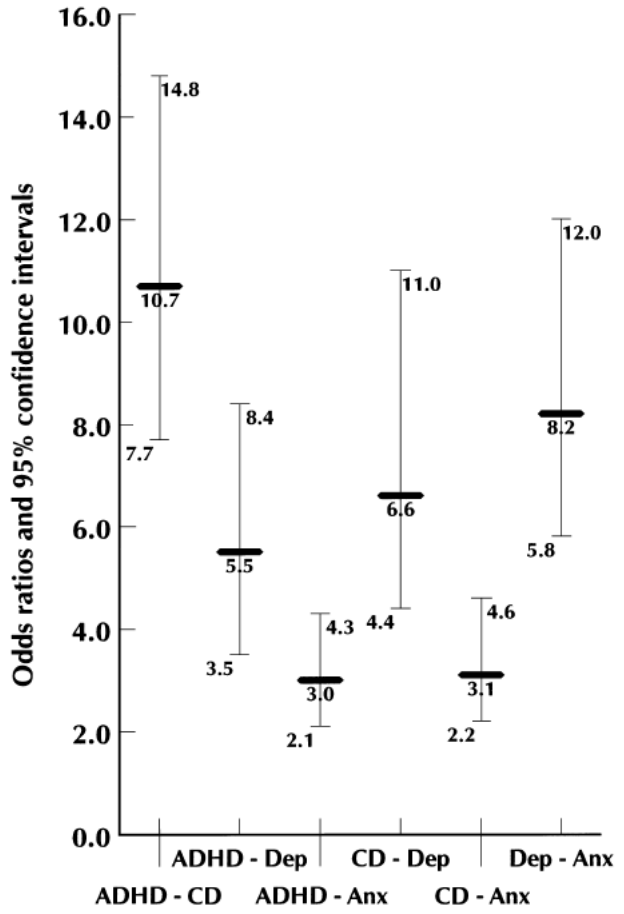


Figure 1. Median odds ratios and 95% confidence intervals from the meta-analysis of community sample based studies of comorbidity.

Note. ADHD = Attention deficit hyperactivity disorder; Anx = Anxiety disorders; CD = Conduct disorder; Dep = Depression.

The developmental taxonomy of antisocial behaviour

Moffitt's (1993) developmental taxonomic theory proposes two subtypes of antisocial behaviour: *life-course persistent* (also called '*early-onset persistent'*) and *adolescence-limited* (see Figure 2). Life-course persistent antisocial behaviours are proposed to emerge in childhood, originating from genetic, congenital, or acquired neuropsychological deficits, and persist throughout the lifespan, as a result of accumulating interactions with high-risk environments, such as harsh and abusive parenting or poverty. Thus, neurocognitive

impairments, difficult child temperament, and adverse family environments are proposed to be the main determinants of life-course persistent antisocial behaviour. By contrast, adolescence-limited antisocial behaviours are proposed to emerge and remit in adolescence, and are caused by an extended period of dependence, labelled the *maturity gap*, in which the biological maturity of the young person stands in contrast to their societal treatment (as a child). Individuals showing adolescence-limited antisocial behaviour are not exposed to the same risk factors as their life-course persistent peers, such as family adversity, but are rather the result of delinquent peer relationships and mimicry of antisocial peers. In sum, Moffitt (1993) proposed two developmental trajectories of antisocial behaviour that are distinct in terms of their emergence, course, and proposed risk factors, including: (i) life-course persistent antisocial behaviour, which is considered a neurodevelopmental disorder, and (ii) adolescence-limited antisocial behaviour, which is viewed as normative and transient – an exaggerated form of normal teenage rebellion.

The revised developmental taxonomy of antisocial behaviour

Based on the empirical evidence, including Moffitt's own research using the data from the Dunedin Study (New Zealand), Fairchild et al. (2013) proposed three potential revisions to the original model: (i) the addition of a second *adolescence-onset* group, in which antisocial behaviour persists into adulthood, and (ii) childhood-onset class, in which antisocial behaviour remits in the transition to adolescence (*childhood-limited*), and (iii) the assumption of common versus distinct individual and environmental risk factors across these groups (see Figure 3). Thus, exposure to adversity or trauma (i.e., environmental risks) and individual risk factors may also contribute to the aetiology of adolescence-onset antisocial behaviours. In sum, while Moffitt's (1993) model proposes qualitative differences between two developmental trajectories of antisocial behaviour, Fairchild et al. (2013) propose a greater variability with

respect to the emergence, desistance, and persistence of antisocial behaviour, and differences between these trajectories that are quantitative versus qualitative in nature.

Figure 2 Moffitt's (2018) developmental taxonomy of antisocial behaviour

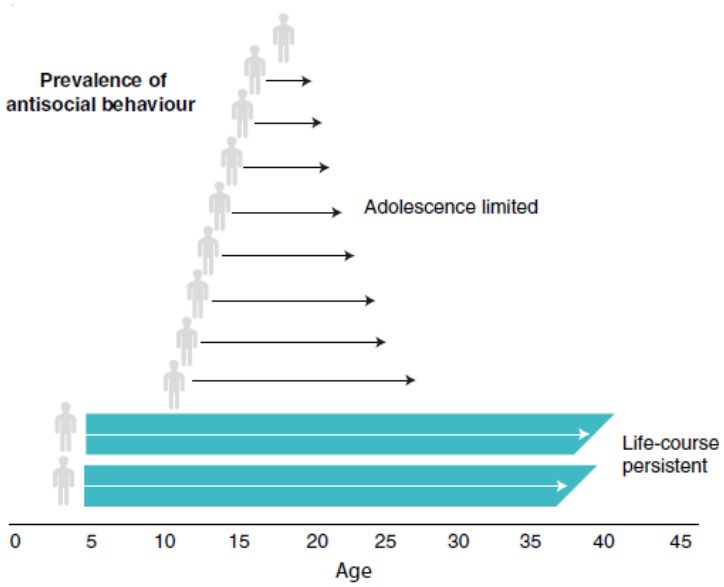
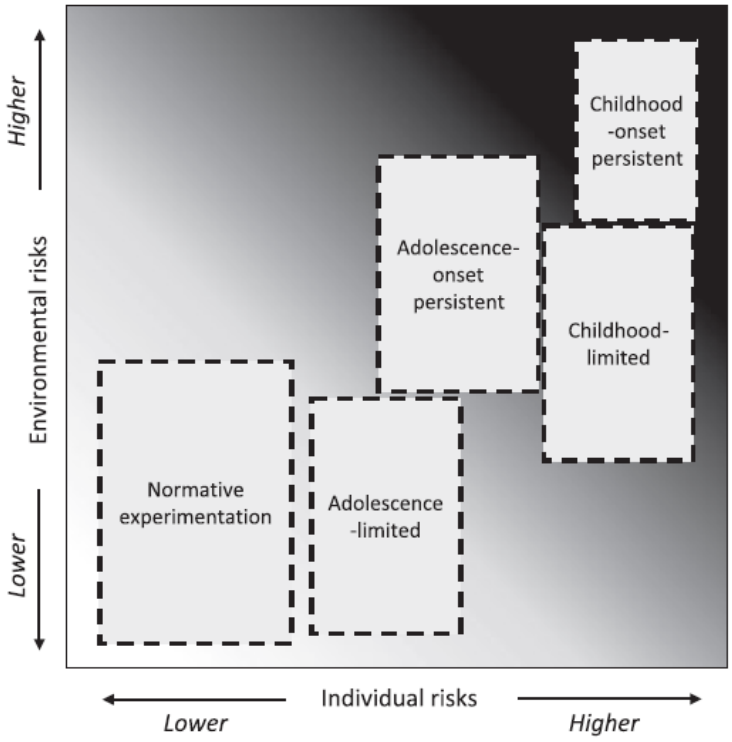


Figure 3 Fairchild et al.'s (2013) revised developmental taxonomy of antisocial behaviour



Risk factors

Given the focus on childhood adversities in the current thesis, the following section provides a brief overview of individual, family, and environmental risk factors of child and adolescent psychopathology, with a particular focus on aggressive and antisocial behaviour. There is evidence suggesting that internalising and externalising dimensions of psychopathology share common genetic factors, which may explain their co-occurrence (Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011). Antisocial behaviour in particular has been shown to be highly heritable and polygenic (Tielbeek et al., 2017), with genetic factors explaining over 50% of its variance (Burt, 2009). Furthermore, child neurocognitive risk factors, such as low IQ, have been particularly linked to early-onset persistent, as opposed to adolescence-onset, antisocial behaviour, as described above (Moffitt, 2018). Related to this, there are several prenatal risk factors for child mental health problems, which may cause or exacerbate child neurocognitive risk factors. For example, prenatal smoking and alcohol consumption have been linked to both cognitive deficits and conduct problems in children (Flak et al., 2014; Gaysina et al., 2013; Moore et al., 2020; Schoeps et al., 2018). A broad range of parenting strategies have been linked to child mental health outcomes. For example, while parental warmth and behavioural control have been associated with a decrease in child internalising and externalising problems over time, harsh and psychologically controlling parenting is linked to an increase in such problems (Pinquart, 2016, 2017). Finally, a wide range of neighbourhood-level risk factors for common mental disorders in young people have been identified (Curtis et al., 2013). For example, community violence has been strongly linked to internalising and externalising problems, particularly when it occurs in close proximity (Fowler, Tompsett, Braciszewski, Jacques-Tiura, & Baltes, 2009). Given this emerging evidence for environmental effects on child conduct problems and other forms of psychopathology, in the next section the

classification and measurement of a particularly damaging environmental risk factor – childhood adversity and trauma – will be considered.

Childhood adversities

The following section will consider concepts and measurement issues of childhood adversities.

Definitions

Over two decades ago, Felitti et al. (1998) developed the adverse childhood experiences (ACEs) scale, which included items on child abuse (i.e., physical, emotional, and sexual); child neglect (i.e., physical and emotional); and household dysfunction, including substance abuse, mental illness, domestic violence, incarceration, and parental separation. Subsequently, some researchers have proposed a revised inventory of ACEs, including, for example, the addition of items on exposure to community violence and peer victimisation, to improve predictive modelling of physical and mental health outcomes (Finkelhor, Shattuck, Turner, & Hamby, 2013, 2015). However, to date, there is no agreed definition of ACEs (Lacey & Minnis, 2020). Thus, other studies have included additional items, such as parental death and poverty (Green et al., 2010)

By contrast, the DSM-5 provides a definition of trauma as part of the diagnostic criteria of post-traumatic stress disorder (American Psychiatric Association, 2013). More precisely, trauma is defined as exposure to actual or threatened death, serious injury, or sexual violence, and can be directly and indirectly experienced, such as through an actual physical assault and learning that a violent personal assault happened to a close family member or friend, respectively, and personally witnessed (e.g., observing a violent assault of another person) (American Psychiatric Association, 2013).

In sum, although most, if not all, traumatic events occurring in childhood can be classified as ACEs (e.g., sexual violence), the opposite is not necessarily true (e.g., parental separation). Thus, while ACEs represent a more *inclusive* conceptualisation of childhood

adversity, as they cover both traumatic and non-traumatic experiences, they are also more *restrictive*, as they primarily focus on adverse family environments, as opposed to non-interpersonal/single-event trauma, such as severe motor vehicle accidents and disasters, which are consistent with the DSM-5 definition of trauma (American Psychiatric Association, 2013). Furthermore, although ACEs include personally witnessed events (e.g., domestic violence), indirect exposure (i.e., through learning about an event), as in the DSM-5, is not covered (American Psychiatric Association, 2013). Finally, while childhood trauma is mainly limited to experiences of *threat* (i.e., exposure to events involving actual or threatened harm), ACEs also include the lack of normative experiences (i.e., *deprivation*), such as the absence of exposure to environmental stimuli (McLaughlin, 2016; McLaughlin et al., 2020). These dimensions – threat and deprivation – may involve shared and distinct transdiagnostic mechanisms linking childhood adversities to psychopathology (McLaughlin, 2016; McLaughlin et al., 2020)

Prevalence and co-occurrence

In the original ACEs study, Felitti et al. (1998) assessed ACEs in adults aged 19-92 years, of which more than half (52%) reported having experienced at least one ACE. By comparison, Kessler et al. (2010) reported a lower figure of around 40% across 22 representative samples from 21 countries. Equally, in the US, a majority of children (61.8%) are reported to have been exposed to a traumatic event before reaching adulthood (McLaughlin et al., 2013). In the UK, this figure was much lower, with a prevalence of 31.3% (Lewis et al., 2019). These discrepancies may be explained by true differences in exposure to childhood adversities, updated diagnostic criteria (e.g., DSM-IV versus DSM-5), or different measures covering a diverse range of trauma indices and ACEs items.

Studies have consistently shown that ACEs co-occur, i.e., a majority of individuals who were exposed to one ACE reported at least one additional ACE (Dong et al., 2004). This

interrelatedness or clustering of adverse experiences is also demonstrated in studies that examined multiple ACEs jointly as a maladaptive family functioning *factor*, as opposed to *single items* (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2010; McLaughlin et al., 2012). Furthermore, using person-centred approaches, such as latent class analysis, researchers identified specific patterns of childhood adversities, which may carry different levels of risk. For example, Lanier et al. (2018) found seven *classes* of ACEs, with those being exposed to parental mental illness and poverty being at particularly high risk of mental and physical health problems in childhood. Similarly, in a systematic review including 17 studies that examined patterns of trauma exposure, O'Donnell et al. (2017) identified three main classes, including those that mainly experience either 'sexual violence' or 'physical violence', and a 'high trauma class' that is exposed to a wide range of traumatic experiences.

Specificity

In the original ACEs study, Felitti et al. (1998) demonstrated an association between ACEs and negative outcomes across multiple domains, including mental and physical health problems. Over the past two decades, these findings have been consistently replicated, as shown in a large number of studies (Petruccelli, Davis, & Berman, 2019; Sahle et al., 2021). Similarly, population-based studies found largely non-specific effects of childhood trauma across psychiatric disorders, i.e., trauma exposure was associated with multiple forms of psychopathology (Copeland, Keeler, Angold, & Costello, 2006; Lewis et al., 2019; Park et al., 2014; Zlotnick et al., 2008). These findings provide empirical support for transdiagnostic models proposing general pathways linking childhood adversity to psychopathology (McLaughlin, 2016; McLaughlin et al., 2020). More precisely, McLaughlin (2016) proposes two pathways in which ACEs may be linked to increased risk of all forms of psychopathology. While experiences of threat primarily disrupt emotional processing, which, for example, may cause heightened emotional reactivity, experiences of deprivation are linked to deficits in

executive functioning, which may affect working memory, inhibitory control, and cognitive flexibility. In a similar model focusing on childhood trauma, McLaughlin et al. (2020) present two additional mechanisms of threat, including disruptions in social information processing (e.g., hostile attribution bias) and accelerated biological aging (i.e., earlier pubertal timing and cellular aging), which are again proposed to be linked to transdiagnostic psychopathology.

Sensitive periods

Similar to the original ACEs study that asked participants about experiences while *growing up* (i.e., at any point up to age 18 years) (Felitti et al., 1998), the developmental timing of exposure has been largely ignored in the existing literature (Lacey & Minnis, 2020). Testing life course models of child psychopathology, Dunn et al. (2018) found that the *accumulation* and *recency* of exposure to child abuse, rather than sensitive periods, predicted levels of child emotional and behavioural problems in a UK birth cohort; by comparison, family financial difficulties and parental criminality were particularly harmful at age 8 years, suggesting a sensitive period in early childhood for these ACEs. In a second study, Dunn et al. (2017) found that exposure to childhood interpersonal violence at any age was associated with increased risk for psychiatric disorders, albeit particularly exposure in adolescence increased the risk for conduct/oppositional disorders, indicating a sensitive period for this disorder class. However, in a third study examining interpersonal violence, Dunn et al. (2020) reported that exposure up to age 3 years was associated with increased levels of child externalizing problems, indicating another sensitive period in very early childhood. In sum, these findings suggest that the impact of timing may vary according to the type of exposure and outcome under investigation. To further complicate this issue, exposure to specific childhood adversities may vary according to the age of the child (Ziobrowski et al., 2020). For example, using latent class analysis, Ziobrowski et al. (2020) found a ‘child physical abuse’ class and an ‘adolescent emotional abuse’ class, indicating that some forms of abuse are particularly linked to different

developmental stages. Thus, more research is needed to determine whether developmental stages exist in which exposure to childhood adversities is particularly harmful, and whether these sensitive periods depend on the type of adversity or trauma and form of psychopathology.

Limitations

Major limitations of the current evidence base relating to childhood adversities include the use of: (i) retrospective self-reports in adulthood to assess childhood trauma, with the assumption that these measures are equivalent to prospective measures; (ii) small, selected samples of children, with the assumption that results from studies using these samples will generalise to a broader population; (iii) measures of childhood adversity covering a large age range (e.g., from birth to age 18 years), with the assumption that negative experiences will have the same impact regardless of their timing (see also Danese (2020) for a more detailed overview); and (iv) the serious lack of evidence from low- and middle-income and non-Western countries, with the assumption that results from studies based on samples from high-income and/or Western countries will be universally applicable irrespective of the cultural context. The current thesis particularly focuses on the latter, with the global mental health community challenging the notion that findings from high-income countries can be assumed to translate to low- and middle-income countries (Summerfield, 2008). This lack of evidence is particularly concerning considering that almost 90% of the world's children live in low- and middle-income countries (UNICEF, 2005). For example, in a recently published systematic review from 2019 on the association of ACEs with mental and physical health outcomes, including 96 studies, just one study was conducted in a low- and middle-income country (the Philippines), with the overwhelming majority of studies using samples from the US (86%) (Petruccelli et al., 2019). This lack of evidence is further illustrated in an umbrella review from 2021 on the association between ACEs and common mental disorders, including 68 systematic reviews and/or meta-analyses, with the authors concluding that most associations were based on samples from high-

income countries, and highlighting the need for further research from low- and middle-income countries (Sahle et al., 2021).

Outline of studies

The aim of the current thesis was to examine the relationship between childhood adversities and child and adolescent psychopathology, with a particular focus on conduct problems, using representative population-based samples of children and adolescents, especially from low- and middle-income countries and non-Western countries. Each of the included studies approached this issue from a different angle, addressing specific research gaps identified in the existing literature. A more detailed overview of each study is provided in the following section.

The following research questions were addressed:

- What is the relationship between environmental risk factors, such as childhood trauma and adverse family environments, and child externalising and internalising psychopathology? Are associations specific to certain forms of psychopathology or more general in nature?
- Are findings from high-income and/or Western countries applicable to low- and middle-income and non-Western countries?
- Are there time-dependent effects of trauma exposure on child psychopathology, such that trauma is particularly damaging if experienced within certain developmental stages in childhood?
- Which individual, family, and social factors protect against the negative consequences of childhood adversities on risk for psychopathology?

Study 1 – Associations between childhood trauma and childhood psychiatric disorders: Evidence from the 2004 Pelotas (Brazil) Birth Cohort

Childhood trauma has been proposed as a key transdiagnostic risk factor for psychopathology (McLaughlin et al., 2020), which may account for almost half of all childhood-onset psychiatric disorders (Green et al., 2010). However, major gaps in our understanding of the prevalence of childhood trauma and the epidemiology of trauma-related disorders remain. More precisely, a substantial proportion of population-based studies used retrospective self-report in adulthood to assess trauma exposure and psychiatric disorders in childhood. However, retrospective and prospective measures of childhood trauma show limited agreement and may identify two distinct groups of trauma-exposed individuals (Baldwin, Reuben, Newbury, & Danese, 2019). Furthermore, most studies focused on maladaptive family functioning, as opposed to the full range of traumatic events in childhood, and were conducted in high-income countries, mostly the US. Therefore, the first study examined associations between childhood trauma and childhood psychiatric disorders, using data from a population-based, prospective birth cohort from Brazil, a middle-income country. We used binary logistic regression analyses to examine the specificity of trauma exposure across psychiatric disorders, in addition to distinguishing between the effects of interpersonal and non-interpersonal trauma in the first study to do this outside of the post-traumatic stress disorder literature.

Study 2 – Harsh parenting and child conduct and emotional problems: Parent- and child-effects in the 2004 Pelotas (Brazil) Birth Cohort

The first study focused on childhood trauma as a risk factor for poor mental health, but child psychopathology may, in some cases, lead to a greater risk of trauma exposure. Transactional models of developmental psychopathology, such as the coercive processes model (Patterson, 1982), propose parent- and child-effects in the development of child psychopathology. Indeed, harsh parenting has been found to be reciprocally related to child conduct problems (Pinquart,

2017), whereas only a unidirectional relationship from harsh parenting to child emotional problems has been observed (Pinquart, 2016). However, once again the vast majority of studies examining these effects were conducted in high-income countries. Importantly, the effects of harsh parenting on child conduct and emotional problems may depend partly on cultural norms (Gershoff et al., 2010; Lansford et al., 2005), and it remains unclear if the findings obtained in high-income countries translate to low- and middle-income countries. Therefore, in this second study, we used cross-lagged path analysis to examine whether there are unidirectional or reciprocal relationships between harsh and abusive parenting and child conduct and emotional problems, using data from a birth cohort comprised of children from Brazil, a middle-income country.

Study 3 – Associations between developmental timing of child abuse and conduct problem trajectories in a UK birth cohort

In the third study, we continued examining the effects of harsh and abusive family environments, however, in this case we focused on child conduct problems and, more specifically, their developmental course and degree of persistence over time. Developmental taxonomic theories have been crucial in shifting focus from considering child conduct problems as a unitary phenomenon to understanding different developmental trajectories (Fairchild et al., 2013; Moffitt, 1993). Although child abuse has been identified as a key environmental risk factor for child conduct problems (Braga, Gonçalves, Basto-Pereira, & Maia, 2017; Wilson, Stover, & Berkowitz, 2009), little is known about how child abuse relates to conduct problem trajectories, especially with respect to its timing and persistence. Therefore, using data from a UK birth cohort, we used latent class growth analysis to extend existing developmental trajectories of conduct problems from ages 4-13 to age 17 years. We then examined associations between these trajectories and child abuse occurring in childhood, adolescence, or persistently across childhood and adolescence.

Study 4 – The protective effect of neighbourhood collective efficacy on family violence and youth antisocial behaviour in two South Korean prospective longitudinal cohorts

We further examined the association between adverse family environments and child conduct problems in the next study, focusing on a new construct that may aid our understanding of their relationship. Neighbourhood collective efficacy, a concept including informal social control (i.e., the residents' willingness to intervene) and social cohesion (i.e., mutual trust among neighbours), has been proposed to exert protective effects against child externalising problems (Leventhal & Brooks-Gunn, 2000). However, there is limited evidence on the proximal mechanisms, such as the family environment, through which collective efficacy influences behavioural problems in young people. Furthermore, the vast majority of studies on neighbourhood collective efficacy have been conducted in the US, and little is known about the generalisability of these findings to non-Western countries, where cultural differences may influence neighbourhood relationships and shared expectations of informal social control. Therefore, we used data from two nationally representative cohorts from South Korea, including primary school students aged 10-12 years and secondary school students aged 15-17 years. We examined the interplay between neighbourhood collective efficacy, family violence, and youth antisocial behaviour, using fully latent structural regression models. More precisely, we examined whether higher levels of collective efficacy are associated with decreases in both family violence and youth antisocial behaviour, and whether there is an indirect effect of collective efficacy on youth antisocial behaviour through family violence.

Study 5 – Pathways of child resilience to maternal depression: Individual, family, and socioeconomic factors in the 2004 Pelotas (Brazil) Birth Cohort

In the final study, we continued examining protective factors for adverse family environments. However, in this instance we focused on parental mental illness as an environmental risk factor. Maternal depression is a well-established risk factor for child psychopathology (Goodman et

al., 2011). However, some children show positive mental health outcomes even when exposed to persistent maternal depression – this is often referred to as *resilience*. Previous studies have identified individual, family, and socioeconomic factors that predict resilience in children exposed to maternal depression, but these *protective factors* have mainly been examined individually, as opposed to jointly. Furthermore, most studies were conducted in high-income countries, using cross-sectional designs. To address these gaps in the literature, we examined pathways of child resilience to maternal depression using data from a Brazilian birth cohort. More precisely, we examined individual (IQ; at age 6 years), family (child cognitive stimulation; at ages 24 and 48 months), and socioeconomic factors (SES; at birth) that may distinguish resilient from non-resilient children (at age 11 years) exposed to persistent maternal depression. Resilience was defined based on scoring in the normative range on measures of child emotional and behavioural problems.

Summary

We sought to use a range of existing, prospective longitudinal studies to answer questions about associations between childhood adversity/trauma and psychopathology, with a particular focus on conduct problems. We also considered a range of positive outcomes, including resilience to maternal depression, and protective factors, such as neighbourhood collective efficacy. In the next chapter, we will describe the first of these studies examining associations between a broad range of traumatic experiences and childhood psychiatric diagnoses in the 2004 Pelotas Birth Cohort Study.

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Chapter 2

Associations between childhood trauma and childhood psychiatric disorders: Evidence from the 2004 Pelotas (Brazil) Birth Cohort

Study formatted for *JAMA Psychiatry*

Chapter rationale

Childhood trauma has been proposed as a key transdiagnostic risk factor for psychopathology. However, previous studies have been limited by their use of retrospective self-reports in adulthood, focus on family adversity, and high-income country setting. Therefore, study 1 aimed to examine the specificity of trauma exposure across psychiatric disorders, including interpersonal and non-interpersonal trauma, in a birth cohort study from Brazil, a middle-income country.

This declaration concerns the article entitled:

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Candidate's contribution to the paper (provide details, and also indicate as a percentage)	The candidate contributed to / considerably contributed to / predominantly executed the… **Formulation of ideas:** Andreas Bauer contributed considerably to the formulation of the ideas for the current study (80%). **Design of methodology:** The data used in the current study was part of a larger data set. Andreas Bauer contributed considerably to the design of the methodology in this current chapter (70%). **Experimental work:** Andreas Bauer predominantly executed the analysis and interpretation of results for this study (90%). **Presentation of data in journal format:** The presentation of the data into journal format was predominantly carried out by Andreas Bauer (95%).
Statement from Candidate	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature.

Signed	[redacted]	**Date**	27.06.2021

Associations between childhood trauma and childhood psychiatric disorders: Evidence from the 2004 Pelotas (Brazil) Birth Cohort

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Abstract

IMPORTANCE Childhood trauma has been proposed as a transdiagnostic risk factor for psychopathology, but most studies have been limited by their use of adult samples from high-income countries, focusing on intrafamilial adversities.

OBJECTIVE To examine associations between childhood trauma and childhood psychiatric disorders in children from Brazil, a middle-income country.

DESIGN, SETTING, AND PARTICIPANTS This study used data from the 2004 Pelotas Birth Cohort, a population-based, prospective longitudinal study from Brazil. The sample (*N*=4231) included all hospital births occurring in 2004 in the city of Pelotas.

OUTCOME AND MEASURES Lifetime childhood trauma and current childhood psychiatric disorders were assessed via clinical interviews with parents or caregivers at ages 6 and 11 years.

RESULTS 34.4% of children were exposed to trauma by age 11 years. In adjusted analysis, childhood trauma was associated with increased odds of 'any psychiatric disorder' at ages 6 (OR 1.75, 95% CI 1.36-2.27) and 11 (OR 1.50, 95% CI 1.12-2.00), and additionally of anxiety disorders at age 6 (OR 2.05, 95% CI 1.49-2.83) and conduct/oppositional disorders at age 11 (OR 1.95, 95% CI 1.06-3.58). Exposure to interpersonal trauma was associated with increased risk for all disorder categories at age 11 (ORs 1.51-2.51, *p*s < .05), except for ADHD/hyperactivity disorders, even when adjusting for co-occurring non-interpersonal trauma. In addition, non-interpersonal trauma was associated with increased odds of 'any psychiatric disorder' (OR 1.41, 95% CI 1.06-1.88) and anxiety disorders (OR 1.64, 95% CI 1.07-2.49), even when adjusting for co-occurring interpersonal trauma.

CONCLUSIONS AND RELEVANCE We provide robust, longitudinal evidence that trauma exposure is a transdiagnostic risk factor for psychopathology in middle and late

childhood, in a middle-income country sample in which trauma exposure is high. We further identify significant contributions of both interpersonal and non-interpersonal trauma to child psychiatric disorders, highlighting the importance of assessing both domains.

INTRODUCTION

The majority of children living in the US will experience a traumatic event before reaching adulthood (McLaughlin et al., 2013). This *childhood* trauma has been proposed as a key transdiagnostic risk factor for psychopathology (McLaughlin, Colich, Rodman, & Weissman, 2020), and may account for almost half of all childhood-onset psychiatric disorders (Green et al., 2010). Over the past two decades, there has been an increasing number of population-based studies examining the association between childhood trauma and childhood psychiatric disorders. However, major gaps in our understanding of the epidemiology of trauma-related disorders remain.

A substantial proportion of population-based studies have assessed childhood trauma and childhood-onset psychiatric disorders via retrospective self-report in adulthood (Benjet, Borges, & Medina-Mora, 2010; Green et al., 2010; Kessler et al., 2010; Slopen et al., 2010). However, prospective and retrospective measures of childhood trauma, such as child abuse, show poor agreement, and may identify two distinct groups of trauma-exposed individuals, with potentially different risk pathways to psychopathology (Baldwin, Reuben, Newbury, & Danese, 2019). Thus, those identified retrospectively as having experienced trauma may need different preventive and treatment strategies than those identified prospectively (Baldwin et al., 2019). Furthermore, there is some evidence suggesting that effect sizes for associations between trauma and mental health outcomes increase with the length of the recall period, suggesting recall bias (Green et al., 2010). Additionally, those with concurrent mental health problems may be more likely to recall traumatic childhood experiences (Colman et al., 2016). Consequently, studies relying on retrospective data may have overestimated associations between childhood trauma and childhood psychiatric disorders.

In contrast to the extensive body of knowledge related to childhood trauma in adults, only a small number of studies have examined the association between trauma and mental

disorders in population-based samples of children and adolescents (Benjet, Borges, Méndez, Fleiz, & Medina-Mora, 2011; Copeland, Keeler, Angold, & Costello, 2006; Dunn et al., 2017; Lewis et al., 2019; McLaughlin et al., 2012). Interestingly, while there is strong empirical support for non-specific effects of childhood adversity and trauma across psychiatric disorders in studies using retrospective self-report in adulthood (i.e., general pathways linking childhood trauma to psychopathology) (Green et al., 2010; Kessler et al., 2010), studies including children and adolescents have identified more specific patterns of effects, although not consistently. Specifically, some studies have reported particularly strong associations for conduct/oppositional disorders (McLaughlin et al., 2012), or alternatively for anxiety and mood disorders (Copeland et al., 2006), while others have shown no specificity (Benjet et al., 2011; Lewis et al., 2019). More research from population-based studies of children is needed to determine whether associations between trauma and childhood psychiatric disorders are specific or general.

In addition to the limited study of childhood trauma in young people, the existing evidence base has several other limitations. First, the majority of studies, irrespective of when childhood trauma and psychiatric disorders were assessed, have examined childhood *adversities*, with a particular focus on maladaptive family functioning (Benjet et al., 2010; Benjet et al., 2011; Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012; Slopen et al., 2010). Although most, if not all, traumatic events occurring in childhood can be classified as childhood adversities (e.g., sexual abuse), the converse is not necessarily true (e.g., parental divorce). Furthermore, while childhood trauma is focused on experiences of *threat* (i.e., exposure to events involving actual or threatened harm), childhood adversities also include the lack of experiences (i.e., *deprivation*), such as the failure to provide adequate care (McLaughlin, 2016). Finally, childhood adversities do not capture the full range of traumatic events in childhood, such as indirect trauma exposure (i.e., witnessing or learning about an

event) or non-interpersonal trauma (e.g., severe motor vehicle accidents or natural disasters). Notably, the WHO developed the Adverse Childhood Experiences (ACEs) International Questionnaire, expanding the definition of ACEs to also cover experiences more commonly found in low- and middle-income countries, such as exposure to collective violence and witnessing community violence (WHO, 2020). Nonetheless, the vast majority of studies to date have been conducted in high-income countries (HICs) such as the US. To our knowledge, there is only one population-based study on childhood adversities in young people that has been conducted in a low- and middle-income country (LMIC). This study examined the impact of maladaptive family functioning across psychiatric disorders, using a representative sample of Mexican adolescents aged 12-17 years (Benjet et al., 2011). Although Benjet et al. found largely non-specific effects across major diagnostic groups (Benjet et al., 2011), which is consistent with adult studies from HICs, the focus on family functioning provides limited insight into the effects of childhood trauma exposure. Hence, there is a serious lack of evidence from LMICs where almost 90% of the world's children live (UNICEF, 2005), and where experiences of potentially traumatic events are disproportionally higher compared to HICs (WHO, 2002).

Third, as mentioned earlier, non-interpersonal trauma has been largely ignored in this area, possibly because it was not part of the original adverse childhood experiences (ACEs) concept (Felitti et al., 1998). Furthermore, comparisons between interpersonal and non-interpersonal trauma have been mainly limited to the post-traumatic stress disorder (PTSD) literature. For example, a meta-analysis demonstrated that children exposed to interpersonal trauma are particularly at risk for PTSD compared to those exposed to non-interpersonal trauma (Alisic et al., 2014). Just one population-based study of US adults examined non-interpersonal trauma types when assessing childhood adversity. The findings suggested more robust associations with mood and anxiety disorders, rather than conduct/oppositional disorders

(Kessler, Davis, & Kendler, 1997), indicating specific, as opposed to general, effects for this trauma category. However, more child mental health research is needed that is inclusive of childhood trauma types beyond those experienced within the family.

The current study aims to address these research gaps by using data from a population-based, prospective birth cohort study from Brazil, a middle-income country. We examined associations between trauma exposure and risk for psychiatric disorders at ages 6 and 11 years, investigating whether there was any specificity in such effects for particular classes of disorders (e.g., conduct/oppositional disorders). Furthermore, we contrasted the effects of interpersonal versus non-interpersonal trauma whilst accounting for potential clustering of these distinct exposure types in our analyses (i.e., controlling for non-interpersonal trauma when assessing the effects of interpersonal trauma, and vice versa).

METHODS

Study design and sample

The 2004 Pelotas Birth Cohort is an ongoing population-based, prospective longitudinal study, including all hospital births occurring in 2004 in the city of Pelotas, Rio Grande do Sul, Brazil. Out of 4263 live births, 4231 (99.2%; 51.9% boys) were included, and assessed at birth and when the child was aged 3, 12, 24, and 48 months, and 6 and 11 years. Further details on the cohort can be found elsewhere (Santos et al., 2011; Santos et al., 2014). All 2004 Pelotas Birth Cohort follow-up waves were approved by the Federal University of Pelotas Medical School Research Ethics Committee. All guardians of the participating children signed an informed consent form before data collection. At the 11-year follow-up, children also provided written informed consent. Cases of severe mental health problems, as identified by the psychologists, were evaluated and, when necessary, were referred to the psychiatric or psychological care facilities of the city.

Measures

Childhood trauma

Lifetime trauma exposure was assessed at ages 6 and 11 years, using the PTSD section of the Development and Well-Being Assessment (DAWBA) (Goodman, Ford, Richards, Gatward, & Meltzer, 2000). The DAWBA has been cross-culturally adapted and validated for use in Brazil (Fleitlich-Bilyk & Goodman, 2004). A trained psychologist asked caregivers, the majority of whom were mothers at ages 6 (89.0%) and 11 years (92.5%), whether their child was exposed to "*events or situations that are exceptionally stressful, and that would really upset almost anyone*" during their lifetime. If answered affirmatively, this screening question was followed by a checklist of 11 (at age 6 years) and 13 (at age 11 years) distinct traumatic events (including an 'other trauma' category). We used responses to the screening question to create the following indices capturing trauma: i) exposure *up to age 6 years* (present/absent); ii) exposure *up to age 11 years* (present/absent; i.e., being exposed at age 6 and/or age 11 years); iii) exposure to *interpersonal trauma up to age 11 years* (present/absent; events included exposure to attack or threat, physical abuse, sexual abuse, rape; witnessing domestic violence; and witnessing or learning about an attack or threat towards a family member or friend); and iv) exposure to *non-interpersonal trauma up to age 11 years* (present/absent; events included exposure to accident, fire, or other disaster; witnessing a sudden death; and witnessing or learning about an accident of a family member or friend). The 'other trauma' category was not included in interpersonal and non-interpersonal trauma codes due to limited event information, and these codes were not examined based on exposure up to age 6 years due to low frequencies (see Table 1).

Childhood psychiatric disorders

Current psychiatric disorders were assessed at ages 6 and 11 years using the parent-reported DAWBA for children aged 5-17 years (Goodman et al., 2000). A trained psychologist

interviewed mothers or caregivers based on diagnostic criteria from DSM-IV (at age 6 years) and DSM-5 (at age 11 years), assessing: anxiety disorder (separation anxiety disorder; specific phobia; social phobia; generalized anxiety disorder; PTSD; panic disorder; agoraphobia; obsessive-compulsive disorder), mood disorder (depression, and at age 11 only, DSM-5-based disruptive mood dysregulation disorder), attention-deficit/hyperactivity disorder, and oppositional defiant/conduct disorder. A second psychologist independently assessed 10% of clinical assessments. Inter-rater agreements were 91.2% (any psychiatric disorder), 75.9% (anxiety disorder), 73.5% (mood disorder), 72.7% (ADHD/hyperactivity disorder), and 72.9% (conduct/oppositional disorder).

Covariates

All covariates were assessed at birth, unless otherwise stated. Child sex was coded as *male* or *female*. Maternal relationship status was coded as *married/living with partner* or *single/divorced/widowed*. Maternal skin colour was coded as *White* or *Black/Mixed race*. Maternal smoking and alcohol consumption were coded as *absent* or *present*, and defined as at least one cigarette daily and any amount of alcohol intake, respectively, during any trimester of pregnancy. Maternal education was coded continuously as completed school years. Monthly family income was coded continuously in Brazilian Real (R$). Maternal depression was measured at 12 months postpartum using the self-reported Edinburgh Postnatal Depression Scale (EPDS) (Cox, Holden, & Sagovsky, 1987), which asks about symptoms of depression in the last week. The 10 items are each rated on a 4-point scale (0-3). The EPDS has been validated for use in Brazil (Santos et al., 2007).

Analysis strategy

We used binary logistic regression analysis to examine associations between childhood trauma indices and presence/absence of any psychiatric diagnosis, as well as diagnostic classes of: anxiety disorder; mood disorder; ADHD/hyperactivity disorder; and conduct/oppositional

disorder. All analyses were adjusted for child sex, maternal relationship status, maternal skin colour, maternal smoking and alcohol consumption during pregnancy, maternal education, family income, and maternal depression. The regression models examining interpersonal and non-interpersonal trauma up to age 11 years were additionally adjusted for clustering of these trauma categories. Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs) and *p*-values.

Missingness ranged from 15.4-20.5% for childhood trauma and from 15.3-15.8% for childhood psychiatric disorders. For covariates, missingness was 1.0% for maternal education, 1.1% for maternal skin colour, and 9.3% for maternal depression; there was no missing data for all other covariates.

We addressed missing data using multivariate imputation by chained equation with 100 imputed data sets. The following auxiliary variables were included to predict missing values: birth weight; the parent-reported Child Behaviour Checklist (assessed at 48 months), including the following subscales: withdrawn, somatic complaints, anxious/depressed, social problems, thought problems, attention problems, rule breaking behaviour, and aggressive behaviour (Achenbach, 1991); and the parent-rated parent-to-child version of the Conflict Tactics Scale (assessed at ages 6 and 11 years), including the following subscales: psychological aggression, corporal punishment, and physical maltreatment (Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). The imputation algorithm was assessed for convergence, and imputed values were checked for plausibility. Primary analyses are based on imputed data. Results based on complete cases for trauma exposure up to age 6 years (N = 3348) and trauma exposure up to age 11 years (N = 3317) are presented in Appendices 1 and 2. Multiple imputation and statistical analyses were performed using RStudio, Version 1.3.1056 (RStudio Team, 2016).

RESULTS

Prevalence of childhood psychiatric diagnoses and childhood trauma

Table 1 presents the prevalence of psychiatric diagnoses at ages 6 and 11 years for the overall sample and for those exposed versus unexposed to trauma. The prevalence of any psychiatric disorder at age 6 years (16.2%) was higher than at age 11 years (13.1%). While the prevalence of anxiety disorders decreased from 8.8% to 4.5% between ages 6 and 11 years, the prevalence of mood disorders and ADHD/hyperactivity disorders increased from 1.3% to 3.3% and 2.5% to 4.0%, respectively. Anxiety disorder was the most common diagnostic class at both ages. Overall, the prevalence of psychiatric disorders was approximately 1.5 to 2 times higher in those reporting trauma exposure compared to those reporting no trauma exposure at both ages.

Up to age 6 years, 12.4% of children had experienced a traumatic event, with 5.1% and 4.9% exposed to interpersonal and non-interpersonal trauma, respectively.[2] More precisely, while 4.4% and 4.2% were exposed to either interpersonal or non-interpersonal trauma, 0.7% were exposed to both trauma types. By age 11 years, these prevalence rates increased almost threefold, with 34.4% of children having experienced a traumatic event, and 14.3% and 15.8% experiencing interpersonal and non-interpersonal trauma, respectively.[2] Of these, 8.5% and 10.0% were exposed to either interpersonal or non-interpersonal trauma, and 5.8% were exposed to both trauma types.

[Insert Table 1 here]

Associations between childhood trauma and childhood psychiatric diagnoses

Table 2 shows associations between trauma exposure and psychiatric diagnoses at ages 6 and 11 years. Children exposed to trauma up to age 6 years had a two-fold higher risk for 'any psychiatric disorder', and more than twice the odds of anxiety disorders and mood disorders

[2] As 'other trauma' could not be coded under the interpersonal/non-interpersonal trauma categories, the number of those exposed to any trauma exceeds the totals of interpersonal and non-interpersonal trauma.

specifically, compared to unexposed children, with ORs of 2.01-2.56 (all *p*s < .01). When adjusting for covariates, the strength of the associations for 'any psychiatric disorder' and anxiety disorders were attenuated (ORs of 1.75-2.05, all *p*s < .01), and the association with mood disorders was only marginally significant (*p* = .05).

Trauma exposure up to age 11 years was associated with a significant increase in the odds of 'any psychiatric disorder' and all four specific diagnostic classes at 11 years, with ORs of 1.66-2.44 (all *p*s < .05). After adjusting for covariates, associations between trauma exposure and 'any psychiatric disorder' and conduct/oppositional disorders remained significant (ORs of 1.50-1.95, all *p*s < .05), albeit weaker, but the other associations did not.

[Insert Table 2 here]

Associations between interpersonal and non-interpersonal childhood trauma and childhood psychiatric diagnoses

Table 3 presents associations between interpersonal and non-interpersonal trauma experienced and psychiatric diagnoses at age 11 years only (equivalent age 6 analyses could not be conducted due to low frequencies of trauma subtypes). First, we examined interpersonal and non-interpersonal trauma exposure separately. Interpersonal trauma was associated with a significant increase in the odds of 'any psychiatric disorder' and all four diagnostic categories, ORs of 1.84-3.31 (all *p*s < .01) and associations were retained after adjusting for covariates, with ORs of 1.61-2.64 (all *p*s < .05). Non-interpersonal trauma was similarly associated with increased odds of 'any psychiatric disorder' and all four diagnostic categories, with ORs of 1.77-2.11 (all *p*s < .05), in unadjusted models. In models adjusted for covariates, associations with 'any psychiatric disorder', and with anxiety and mood disorders were retained (ORs of 1.60-1.95, all *p*s < .05), albeit weaker, whereas associations with ADHD/hyperactivity and conduct/oppositional disorders were no longer significant (see Table 3).

Second, we additionally adjusted for clustering of these trauma categories by including both exposure types in the same model (i.e., controlling for non-interpersonal trauma when assessing the effects of interpersonal trauma, and vice versa; see Table 3). For interpersonal trauma, effects for 'any psychiatric disorder' and the diagnostic categories of anxiety disorders, mood disorders, and conduct/oppositional disorders were retained (ORs of 1.51-2.51, all *p*s < .05), but effects for ADHD/hyperactivity disorders were not. For exposure to non-interpersonal trauma, increased risk of 'any psychiatric disorder' and anxiety disorders (ORs of 1.41-1.64, all *p*s < .05) were identified after adjusting for interpersonal trauma exposure, but there was no longer evidence of effects for the other diagnostic categories.

[Insert Table 3 here]

DISCUSSION

We examined associations between childhood trauma and childhood psychiatric disorders, using data from a birth cohort from Brazil, a middle-income country. More than one third of children had experienced a traumatic event before reaching adolescence (i.e., up to age 11 years). After adjusting for covariates, childhood trauma was associated with a significant increase in the odds of 'any psychiatric disorder' and anxiety disorders and conduct/oppositional disorders specifically. Exposure to interpersonal trauma was associated with increased odds of all four diagnostic categories and 'any psychiatric disorder', whereas non-interpersonal trauma showed a less robust pattern of effects but was still associated with increased odds of 'any psychiatric disorder', and anxiety and mood disorders specifically. These patterns of effects were largely maintained when adjusting for clustering of these two trauma categories.

There was an almost threefold increase in the prevalence of trauma exposure from age 6 to 11 years, with more than one third (34.4%) of children in the sample having experienced a traumatic event by the age of 11 years. Comparisons with other population-based studies are

difficult, as most have focused on ACEs, as opposed to childhood trauma, or included older children. However, using data from a UK birth cohort, Haag et al. reported a prevalence of 15.9% in children aged 10 years (Haag et al., 2020), using the same measure as in the current study (parent-reported DAWBA). In another UK birth cohort, Lewis et al. reported a similar prevalence rate as reported in the current study (31.1%); however, this figure represents lifetime trauma exposure *up to age 18 years* (Lewis et al., 2019). By contrast, Copeland et al. reported that two-thirds (67.8%) of children experienced a traumatic event by the age of 16 years in the Great Smoky Mountains Study (US), a representative sample of children from rural counties of North Carolina (Copeland et al., 2006). Thus, overall, comparison with other birth cohort studies from HICs suggest that children in Southern Brazil are at relatively high risk for trauma exposure.

Trauma exposure in childhood was associated with elevated levels of psychiatric disorders already by age 6 years – an effect that was maintained at age 11. There was little evidence that associations between trauma and psychopathology varied by age, although, in adjusted analysis, the only disorder class that was elevated in trauma-exposed children at age 6 was anxiety disorders, whereas at age 11 effects were present only for conduct/oppositional disorders. These observations are in line with some other studies using population-based samples of children that found particularly strong associations between childhood trauma and anxiety and conduct/oppositional disorders (Copeland et al., 2006; McLaughlin et al., 2012). However, most studies found little specificity of trauma exposure across psychiatric disorders, and in the current study confidence intervals for estimated effects by diagnostic categories were all overlapping, consistent with this conclusion (Benjet et al., 2011; Lewis et al., 2019). Overall, our findings extend those of population-studies conducted in HICs to a middle-income country birth cohort, and additionally shift the focus from examining broader ACEs to investigating trauma exposure specifically in children, including experiences occurring outside of the family

context. In comparison to the only other population-based study that assessed trauma exposure, as opposed to ACEs, in children using data from the Great Smoky Mountains Study (US) which found ORs from 2.0-4.5 (Copeland et al., 2006), effect sizes in the current study were slightly smaller, with ORs from 1.5-2.0.

While research in the PTSD field has consistently examined the consequences of both interpersonal and non-interpersonal trauma (Alisic et al., 2014), the latter has received much less attention in the broader mental health research field where the focus has been on ACEs and on child maltreatment within the family. Although these types of exposure are undoubtedly important and damaging, non-interpersonal trauma is also relatively common among children. As such, we examined both interpersonal versus non-interpersonal trauma as predictors of psychiatric disorders at age 11 years. We found more robust evidence that interpersonal trauma exposure was predictive of psychiatric diagnoses, with elevated odds of anxiety, mood, and conduct/oppositional disorders, even when adjusting for co-occurring non-interpersonal trauma. Importantly, we also found that non-interpersonal trauma exposure was predictive of psychopathology, and uniquely associated with the presence of anxiety disorders, even when we adjusted for the co-occurrence of interpersonal trauma.[3] Collectively, these findings suggest that whether the effects of trauma lead to a general increase in risk for psychopathology or heightened risk for specific disorder classes could depend on the types of trauma that are being studied. In the current study we found that the type of trauma that is widely assumed to be most damaging – interpersonal – led to an increased risk for 'any psychiatric disorder' and all four specific classes of psychiatric disorders, whereas children exposed to non-interpersonal trauma

[3] The more consistent patterns of effects for interpersonal and non-interpersonal trauma compared to our main analysis of 'any trauma' exposure may be explained by the fact that 'other trauma' was not included in the coding of the two trauma categories. More precisely, while individuals who only experienced 'other trauma' were allocated to the *unexposed* group for interpersonal and non-interpersonal trauma, they were assigned to the *exposed* group in our main analysis of 'any trauma' exposure. As 'other trauma' may have involved less severe traumatic experiences, associations with psychiatric disorders were attenuated for any trauma exposure.

may be particularly vulnerable to anxiety disorders only. Overall, our observations suggest that although interpersonal and non-interpersonal trauma can co-occur, they may each make important independent contributions to psychiatric disorders emerging during development.

Key strengths of the current study include the use of a large, population-based sample of children from Brazil, and the fact that a wide range of traumatic events and psychiatric diagnoses were assessed by trained psychologists using well-validated and reliable measures. Trauma exposure was assessed at two separate time points in development and the impact of trauma was investigated across two developmental stages. Importantly, when examining specific types of trauma, we controlled for clustering effects, preventing inflated associations for interpersonal and non-interpersonal trauma.

There are also some limitations that should be considered when interpreting the findings: First, results may have been subject to shared rater bias, as all measures were completed by a single rater, mostly the mother. At the same time, using caregiver reports may have avoided the issue that the child's current mental health status may influence the likelihood of reporting traumatic experiences, which might have been the case with child self-reports. Second, clinical interviews were conducted only with caregivers and prior research has shown a low concordance between parent and child reports of psychopathology, with parents tending to report lower levels of internalizing problems when compared to children (Cantwell, Lewinsohn, Rohde, & Seeley, 1997). Third, childhood trauma and childhood psychiatric diagnoses were assessed at the same time point, and we were not able to determine at what age trauma exposure occurred. Thus, causal relationships cannot be assumed, especially for conduct/oppositional and ADHD/hyperactivity disorders, which have been shown to increase risk for exposure to traumatic events (Carliner, Gary, McLaughlin, & Keyes, 2017), suggesting bidirectional effects. Fourth, due to a highly skewed distribution, it was necessary to dichotomize our measure of childhood trauma as either present or absent. Thus, we were not able to consider

the frequency, duration, and severity of traumatic experiences and these factors merit investigation in future research. Finally, we performed a large number of tests, which increased the probability of type 1 error. Therefore, we mainly focused on effect sizes, as opposed to *p*-values, and interpreted associations cautiously, particularly when there was weak evidence of an effect. In conclusion, we found stronger evidence for general, as opposed to specific, effects of childhood trauma exposure on child psychopathology, supporting transdiagnostic models of childhood adversity and trauma. The findings further highlight the importance of assessing traumatic experiences beyond the family environment, as we were able to demonstrate the damaging impact of non-interpersonal trauma. Finally, more research is needed from LMICs, as prevalence rates of childhood trauma and patterns of effects may vary across cultural contexts – and more than 90% of the world's children live in LMICs.

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Table 1 Prevalence of psychiatric diagnoses at ages 6 and 11 years for the overall sample and those exposed and unexposed to any, interpersonal, and non-interpersonal trauma up to ages 6 and 11 years

	Overall sample	**Any trauma[a]**		**Interpersonal trauma**		**Non-interpersonal trauma**	
		No	Yes	No	Yes	No	Yes
Up to age 6 years							
	N = 3348	*n* = 2932	*n* = 416	*n* = 3178	*n* = 170	*n* = 3183	*n* = 165
Any psychiatric disorder	542 (16.2%)	431 (14.7%)	111 (26.7%)	493 (15.5%)	49 (28.8%)	501 (15.7%)	41 (24.8%)
Anxiety disorder	293 (8.8%)	225 (7.7%)	68 (16.3%)	259 (8.2%)	34 (20.0%)	272 (8.5%)	21 (12.7%)
Mood disorder	43 (1.3%)	31 (1.1%)	12 (2.9%)	39 (1.2%)	4 (2.4%)	41 (1.3%)	2 (1.2%)
ADHD/hyperactivity disorder	83 (2.5%)	71 (2.4%)	12 (2.9%)	76 (2.4%)	7 (4.1%)	77 (2.4%)	6 (3.6%)
Conduct/oppositional disorder	84 (2.5%)	70 (2.4%)	14 (3.4%)	73 (2.3%)	11 (6.5%)	80 (2.5%)	4 (2.4%)
Up to age 11 years							
	N = 3317	*n* = 2178	*n* = 1139	*n* = 2843	*n* = 474	*n* = 2793	*n* = 542
Any psychiatric disorder	433 (13.1%)	236 (10.8%)	197 (17.3%)	338 (11.9%)	95 (20.0%)	333 (11.9%)	100 (19.1%)
Anxiety disorder	148 (4.5%)	76 (3.5%)	72 (6.3%)	109 (3.8%)	39 (8.2%)	107 (3.8%)	41 (7.8%)
Mood disorder	108 (3.3%)	54 (2.5%)	54 (4.7%)	79 (2.8%)	29 (6.1%)	80 (2.9%)	28 (5.3%)
ADHD/hyperactivity disorder	133 (4.0%)	71 (3.3%)	62 (5.4%)	104 (3.7%)	29 (6.1%)	101 (3.6%)	32 (6.1%)
Conduct/oppositional disorder	84 (2.5%)	37 (1.7%)	47 (4.1%)	57 (2.0%)	27 (5.7%)	63 (2.3%)	21 (4.0%)

Note. Based on complete data. [a] Any Trauma also includes 'other trauma' which was not coded for interpersonal or non-interpersonal trauma categories.

Table 2 Associations between trauma exposure up to age 6 years and psychiatric diagnoses at age 6 years, and trauma exposure up to age 11 years and psychiatric diagnoses at age 11 years

	Any trauma					
	Up to age 6 years			Up to age 11 years		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value
Unadjusted						
Any psychiatric disorder	2.01	1.57-2.58	< .001	1.71	1.29-2.25	< .001
Anxiety disorder	2.29	1.68-3.12	< .001	1.66	1.08-2.54	= .02
Mood disorder	2.56	1.26-5.23	< .01	1.92	1.11-3.31	= .02
ADHD/hyperactivity disorder	1.19	0.64-2.21	= .58	1.78	1.09-2.90	= .02
Conduct/oppositional disorder	1.42	0.80-2.51	= .23	2.44	1.36-4.36	< .01
Adjusted						
Any psychiatric disorder	1.75	1.36-2.27	< .001	1.50	1.12-2.00	< .01
Anxiety disorder	2.05	1.49-2.83	< .001	1.47	0.94-2.29	= .09
Mood disorder	2.08	1.00-4.33	= .05	1.69	0.96-2.98	= .07
ADHD/hyperactivity disorder	1.01	0.53-1.91	= .98	1.58	0.95-2.64	= .08
Conduct/oppositional disorder	1.08	0.60-1.95	= .80	1.95	1.06-3.58	= .03

Note. Based on imputed data. Confounders include child sex, maternal smoking, maternal relationship status, maternal alcohol consumption, maternal skin colour, maternal education, maternal depression, and family income. Key: OR = Odds ratio; CI = Confidence interval.

Table 3 Interpersonal and non-interpersonal trauma up to age 11 years and psychiatric diagnoses at age 11 years

	Trauma up to age 11 years					
	Interpersonal			**Non-interpersonal**		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value
Univariable (unadjusted)						
Any psychiatric disorder	1.94	1.49-2.54	< .001	1.77	1.35-2.33	< .001
Anxiety disorder	2.32	1.51-3.57	< .001	2.11	1.41-3.18	< .001
Mood disorder	2.53	1.58-4.06	< .001	1.94	1.18-3.19	< .01
ADHD/hyperactivity disorder	1.84	1.17-2.90	< .01	1.77	1.10-2.86	= .02
Conduct/oppositional disorder	3.31	2.03-5.40	< .001	1.94	1.14-3.31	= .02
Multivariable (adjusting for confounders)						
Any psychiatric disorder	1.68	1.28-2.21	< .001	1.60	1.20-2.12	< .01
Anxiety disorder	2.06	1.33-3.19	< .01	1.95	1.29-2.95	< .01
Mood disorder	2.17	1.34-3.53	< .01	1.70	1.01-2.84	= .04
ADHD/hyperactivity disorder	1.61	1.01-2.55	= .04	1.62	0.99-2.64	= .05
Conduct/oppositional disorder	2.64	1.59-4.40	< .001	1.61	0.92-2.80	= .09
Multivariate (adjusting for confounders and other type of trauma)						
Any psychiatric disorder	1.51	1.15-1.99	< .01	1.41	1.06-1.88	= .02
Anxiety disorder	1.75	1.12-2.75	= .01	1.64	1.07-2.49	= .02
Mood disorder	1.97	1.20-3.21	< .01	1.36	0.81-2.30	= .25
ADHD/hyperactivity disorder	1.43	0.90-2.26	= .13	1.45	0.89-2.38	= .14
Conduct/oppositional disorder	2.51	1.51-4.16	< .001	1.17	0.67-2.05	= .57

Note. Based on imputed data. Confounders include child sex, maternal smoking, maternal relationship status, maternal alcohol consumption, maternal skin colour, maternal education, maternal depression, and family income. Key: OR = Odds ratio; CI = Confidence interval.

Appendix 1 Associations between trauma exposure up to age 6 years and psychiatric diagnoses at age 6 years, and trauma exposure up to age 11 years and psychiatric diagnoses at age 11 years

	Any trauma					
	Up to age 6 years			Up to age 11 years		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value
Unadjusted						
Any psychiatric disorder	2.11	1.66-2.68	< .001	1.72	1.40-2.11	< .001
Anxiety disorder	2.35	1.74-3.14	< .001	1.87	1.34-2.60	< .001
Mood disorder	2.78	1.36-5.32	< .01	1.96	1.33-2.88	< .001
ADHD/hyperactivity disorder	1.20	0.61-2.14	= .57	1.71	1.20-2.42	< .01
Conduct/oppositional disorder	1.42	0.76-2.47	= .24	2.49	1.61-3.88	< .001
Adjusted						
Any psychiatric disorder	1.85	1.44-2.36	< .001	1.52	1.23-1.87	< .001
Anxiety disorder	2.12	1.56-2.84	< .001	1.67	1.19-2.34	< .01
Mood disorder	2.20	1.06-4.28	= .03	1.75	1.18-2.59	< .01
ADHD/hyperactivity disorder	1.02	0.52-1.85	= .95	1.53	1.07-2.19	= .02
Conduct/oppositional disorder	1.06	0.56-1.87	= .85	1.95	1.24-3.07	< .01

Note. Based on complete data. Confounders include child sex, maternal smoking, maternal relationship status, maternal alcohol consumption, maternal skin colour, maternal education, maternal depression, and family income. Key: OR = Odds ratio; CI = Confidence interval.

Appendix 2 Interpersonal and non-interpersonal trauma up to age 11 years and psychiatric diagnoses at age 11 years

	Trauma up to age 11 years					
	Interpersonal			Non-interpersonal		
	OR	95% CI	*P*-value	OR	95% CI	*P*-value
Univariable (unadjusted)						
Any psychiatric disorder	1.86	1.44-2.38	< .001	1.74	1.36-2.22	< .001
Anxiety disorder	2.25	1.52-3.26	< .001	2.13	1.45-3.07	< .001
Mood disorder	2.28	1.45-3.49	< .001	1.91	1.21-2.94	< .01
ADHD/hyperactivity disorder	1.72	1.11-2.59	= .01	1.73	1.14-2.58	< .01
Conduct/oppositional disorder	2.95	1.82-4.67	< .001	1.81	1.07-2.94	= .02
Multivariable (adjusting for confounders)						
Any psychiatric disorder	1.63	1.25-2.10	< .001	1.58	1.22-2.02	< .001
Anxiety disorder	2.01	1.35-2.93	< .001	1.99	1.35-2.88	< .001
Mood disorder	1.98	1.25-3.07	< .01	1.71	1.07-2.65	= .02
ADHD/hyperactivity disorder	1.50	0.96-2.29	= .07	1.60	1.04-2.40	= .03
Conduct/oppositional disorder	2.31	1.41-3.71	< .001	1.47	0.86-2.43	= .15
Multivariate (adjusting for confounders and other type of trauma)						
Any psychiatric disorder	1.47	1.11-1.92	< .01	1.41	1.08-1.84	= .01
Anxiety disorder	1.71	1.12-2.55	= .01	1.70	1.13-2.52	< .01
Mood disorder	1.78	1.09-2.82	= .02	1.44	0.88-2.29	= .13
ADHD/hyperactivity disorder	1.34	0.83-2.08	= .21	1.47	0.93-2.26	= .09
Conduct/oppositional disorder	2.23	1.32-3.68	< .01	1.13	0.64-1.94	= .66

Note. Based on complete data. Confounders include child sex, maternal smoking, maternal relationship status, maternal alcohol consumption, maternal skin colour, maternal education, maternal depression, and family income. Key: OR = Odds ratio; CI = Confidence interval.

Chapter 3

Reciprocal relations between aggressive parenting and child conduct, but not emotional, problems in the 2004 Pelotas Birth Cohort

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Chapter rationale

Transactional models of developmental psychopathology propose parent- and child-effects in the development of child psychopathology. However, previous research has been mostly limited to high-income countries, and it remains unclear if those findings apply across cultural contexts. Therefore, this second study examined whether there are unidirectional or reciprocal relationships between harsh parenting and child conduct and emotional problems, using data from a Brazilian birth cohort study.

This declaration concerns the article entitled:

Reciprocal relations between aggressive parenting and child conduct, but not emotional, problems in the 2004 Pelotas Birth Cohort

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Candidate's contribution to the paper (provide details, and also indicate as a percentage)	The candidate contributed to / considerably contributed to / predominantly executed the… **Formulation of ideas:** Andreas Bauer contributed considerably to the formulation of ideas for the current study (80%). **Design of methodology:** The data used in the current study was part of a larger data set. Andreas Bauer contributed considerably to the design of the methodology in this current chapter (70%). **Experimental work:** Andreas Bauer predominantly executed the analysis and interpretation of results for this study (90%). **Presentation of data in journal format:** The presentation of the data into journal format was predominantly carried out by Andreas Bauer (95%).
Statement from Candidate	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature.
Signed	[redacted] **Date** 27.06.2021

Harsh parenting and child conduct and emotional problems: parent- and child-effects in the 2004 Pelotas Birth Cohort

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Conflict of interest:

The authors declare that they have no conflicts of interest.

Author contributions:

Andreas Bauer, Graeme Fairchild, Sarah L. Halligan, Gemma Hammerton, Joseph Murray, and Alicia Matijasevich contributed to the study design and wrote the manuscript. All co-authors revised and edited the manuscript draft. Alicia Matijasevich, Ina S. Santos, Tiago N. Munhoz, Aluísio J. D. Barros, and Fernando C. Barros contributed to the setup and management of the 2004 Pelotas Birth Cohort. All authors approved the manuscript.

Abstract

In high-income countries, links between harsh and abusive parenting and child conduct and emotional problems are well-documented. However, less is known about these relationships in low- and middle-income countries, where harsh parenting may be more widely accepted and higher rates of conduct or emotional problems may exist which could influence the strength of these associations. We sought to investigate these relationships in a large population-based, prospective longitudinal study from Brazil, which also allowed us to test for sex differences. Using data from the 2004 Pelotas Birth Cohort Study (*N*=4231) at ages 6 and 11 years, we applied cross-lagged path analysis to examine the relationships between harsh parenting (Conflict Tactics Scale Parent-Child version), and child conduct and emotional problems (Strengths and Difficulties Questionnaire). We found reciprocal relationships between harsh parenting and child conduct problems, with harsh parenting at age 6 predicting child conduct problems at age 11, and vice versa, even after adjusting for initial levels of conduct problems and harsh parenting, respectively. For child emotional problems, only unidirectional effects were found, with harsh parenting at age 6 predicting child emotional problems at age 11, after adjusting for initial levels of emotional problems, but not vice versa. No significant sex differences were observed in these relationships. These observations based on a middle-income country birth cohort highlight the potential universality of detrimental effects of harsh parenting on child conduct and emotional problems, and affirm the importance of addressing parent- and child-effects in preventive and treatment interventions, especially those targeting conduct problems.

Key words: harsh parenting, child abuse, conduct problems, emotional problems, cross-lagged panel design, transactional model

Introduction

For decades, the role of parenting behaviors in the development of child psychopathology has been a major focus of research. Early *parent-effect* models, which proposed a unidirectional relationship from parenting to child outcomes (Chess, 1964), evolved to take into account *child-effects*, in which child characteristics and behaviors modify parental behaviors (Bell, 1968). Thus, the coercive processes model proposes that the parents' failure to maintain child compliance in their early interactions initiates a continuing cycle of dysfunctional exchanges. More precisely, ineffective parental demands in response to a child's problem behavior (e.g., aggression or anger outbursts) are followed by the child's refusal to comply, which, in turn, elicits further ineffective parenting (e.g., withdrawal) (Patterson, 1982, 1986). Consequently, over time, the child's aggressive behavior increases and the parents' capacity to regulate the child's problematic behavior decreases.

Repetti et al.'s (2002) model places a major emphasis on such neglectful and harsh family environments, which are proposed to result in emotional dysregulation in children, which, in turn, is thought to be implicated in the development of both externalizing and internalizing psychopathology. With respect to the latter, internalizing symptoms in children and adolescents are particularly linked to coping strategies involving disengagement, such as emotion suppression, avoidance, and denial (Compas et al., 2017). Thus, unlike children showing externalizing problems who become ensnared in coercive exchanges with parents, siblings, and peers (Patterson, 1986), those with internalizing symptoms may prevent or disrupt these vicious cycles by withdrawing from the hostile situation.

To date, two meta-analyses have examined the relationships between harsh parenting and child externalizing and internalizing problems, respectively (Pinquart, 2016, 2017). In the first, Pinquart examined whether later externalizing symptoms are predicted by harsh parenting at earlier stages of development, after adjusting for initial levels of externalizing symptoms, and

vice versa (Pinquart, 2017). Consistent with transactional models of developmental psychopathology, Pinquart found bidirectional effects between harsh parenting and child externalizing symptoms, i.e., harsh parenting led to higher rates of externalizing symptoms in the child, while externalizing problems in the child elicited more harsh parenting over time (Pinquart, 2017). In contrast, in a second meta-analysis of cross-lagged associations between harsh parenting and child internalizing problems, Pinquart (2016) found only a unidirectional effect, whereby harsh parenting predicted internalizing problems, but *not* vice versa. In sum, harsh parenting appears to be reciprocally related to child externalizing symptoms, whereas only a unidirectional relationship from harsh parenting to child internalizing symptoms has been observed.

However, it should be noted that the vast majority (96%) of the studies included in the meta-analysis on cross-lagged associations between harsh parenting and child externalizing symptoms were from high-income countries (HICs) (Pinquart, 2017) (the relevant information could not be extracted from the meta-analysis on internalizing symptoms (Pinquart, 2016)). Just four studies were conducted in low- and middle-income countries (LMICs), and only one of these was population-based (Akcinar & Baydar, 2016). This lack of evidence from LMICs is also reflected in a third meta-analysis by Pinquart and Kauser (Pinquart & Kauser, 2018), in which country-level differences in cross-lagged associations between harsh parenting and child externalizing and internalizing problems could not be estimated due to the small number of studies from non-Western countries.

This gap in the evidence base is concerning, given that almost 90% of all children and adolescents worldwide live in LMICs (UNICEF, 2005). Importantly, effects of harsh parenting on child externalizing and internalizing problems may depend partly on cultural norms. In particular, it is proposed that the effects may be attenuated in countries in which harsh punishment is more common and widely accepted (Gershoff et al., 2010; Lansford et al., 2005).

For example, Lansford et al. found that corporal punishment led to increased levels of child externalizing and internalizing problems across low-, middle-, and high-income countries (Lansford et al., 2005). However, effect sizes were smaller in countries in which corporal punishment was perceived as more normative (Lansford et al., 2005). Although the use of physical and verbal punishment is common worldwide, there is considerable between- and within-country variability (Runyan et al., 2010), especially for more severe forms of harsh parental discipline (Lansford & Deater-Deckard, 2012). Consequently, the effects of harsh parenting on child externalizing and internalizing problems may differ across countries, and it remains unclear if the findings obtained in HICs translate to LMICs.

To address these gaps in the literature, we examined the association between harsh, aggressive, or abusive parenting (hereafter referred to as *harsh parenting*), defined as physical and psychological aggression towards the child, and child externalizing and internalizing symptoms, in the 2004 Pelotas Birth Cohort study. This is a large population-based sample based in Brazil, a middle-income country with high levels of crime and violence, especially amongst adolescents (Murray, Cerqueira, & Kahn, 2013; Murray et al., 2015). The main objectives of the present study were: (i) to test whether harsh parenting is associated with child conduct and emotional problems in a LMIC context; and (ii) to examine whether there are unidirectional or reciprocal relationships between harsh parenting and child conduct and emotional problems, using autoregressive path models to test for cross-lagged associations. In line with previous research (Pinquart, 2016, 2017), we hypothesized that harsh parenting would be reciprocally related to child conduct problems, whereas only a unidirectional relationship would be observed between harsh parenting and child emotional problems. Given that examination of sex differences has been limited in previous research – even when considering HICs (Pinquart, 2016, 2017) – we tested whether the effects of harsh parenting vary according

to the sex of the child, and also whether the stability of child externalizing and internalizing symptoms differs by sex.

Methods

Participants

The 2004 Pelotas Birth Cohort is a population-based, prospective longitudinal study, investigating the impact of early life exposure to a wide range of risk factors on maternal and child health outcomes (Santos et al., 2011; Santos et al., 2014). Pelotas, Rio Grande do Sul (South Brazil) has a population of approximately 340,000 people, predominantly residing in urban areas (93%), with 98% of births occurring in hospitals. Out of the 4263 live births in 2004 identified through daily hospital visits, 4231 (99.2%; 51.9% boys) were included and mothers were interviewed within 24 hours postpartum. Mother-child dyads were assessed again at ages 3 (99.2%), 12 (95.7%), 24 (93.5%), and 48 (92.0%) months, and when the child was 6 (90.2%) and 11 (86.6%) years of age. Data were collected during home visits up to when the children were aged 48 months, and in the study clinic at ages 6 and 11 years. The current sample was restricted to singletons (N = 4145; 52.0% boys). Further details about the cohort and the assessments undertaken can be found in Santos et al. (Santos et al., 2011; Santos et al., 2014).

Measures

Harsh parenting

Caregivers, the majority of whom were mothers at ages 6 (89.0%) and 11 (92.5%) years, were asked about harsh parenting strategies using the parent-to-child version of the Conflict Tactics Scale (CTSPC) (Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). The CTSPC comprises 22 items across three subscales measuring parental behaviors towards the child over the past 12 months related to non-violent discipline (4 items); psychological aggression (5 items); and

physical assault, including corporal punishment (5 items), physical maltreatment (4 items), and severe physical maltreatment (4 items; not administered in this study). In line with two previously published meta-analyses (Pinquart, 2016, 2017), we defined harsh parenting as comprising the sum scores of the psychological aggression (e.g., "Shouted, yelled, or screamed at him/her"), corporal punishment (e.g., "Spanked him/her on the bottom with our bare hands"), and physical maltreatment (e.g., "Slapped him/her on the face or head or ears") subscales. All items were rated on a 3-point scale (0-2), from *never* to *once* and *more than once*, yielding overall scores ranging from 0-28. The Portuguese version of the CTSPC has been cross-culturally adapted and validated for use in Brazil (Reichenheim & Moraes, 2003, 2006).

Conduct and emotional problems

Child conduct and emotional problems were measured at ages 6 and 11 years using the parent-rated conduct problems (e.g., "Often fights with other children or bullies them") and emotional problems (e.g., "Many worries, often seems worried") subscales of the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 2001). We used individual subscales, as opposed to the externalizing problems subscale (which is an aggregate of the conduct problems and hyperactivity subscales), as research has indicated meaningful differences between these symptom clusters (Waschbusch, 2002). Each subscale comprises five items, which are rated on a 3-point scale (0-2), from *not true* to *somewhat true* and *certainly true*, yielding overall scores ranging from 0-10. The Portuguese version of the SDQ has been validated for use in Brazil (Saur & Loureiro, 2012; Woerner et al., 2004). The scales showed modest internal reliability with Cronbach's alphas of 0.59-0.65 and 0.52-0.59 for the conduct and emotional problems subscales, respectively.

Covariates

We included sociodemographic characteristics, prenatal environmental factors, and maternal psychopathology, which have been identified as risk factors for negative parenting and child conduct and emotional problems. Maternal depression was measured at 12 months after delivery using the self-reported Edinburgh Postnatal Depression Scale (EPDS) (Cox, Holden, & Sagovsky, 1987). The 10 items are rated on a 4-point scale (0-3), with scores ranging from 0-30. The Portuguese version of the EPDS has been validated for use in Brazil (Santos et al., 2007). Information on all other covariates was collected within 24 hours postpartum by maternal self-report, unless otherwise stated. Mothers who smoked 1+ cigarettes daily during any trimester of pregnancy were classified as smoking during pregnancy. Any amount of alcohol intake during any trimester of pregnancy was considered as prenatal alcohol exposure. Maternal skin color was determined by the interviewer and, for the purposes of this study, classified as White versus Black/Mixed race. Mothers who were single, widowed, divorced, or who lived without a partner were classified as single mothers. Maternal education was coded as complete school years of formal education. Income was coded as the weekly family income in the month prior to the child's birth.

Analysis strategy

We used observed-variable autoregressive path models to examine the reciprocal associations between harsh parenting and child conduct problems and emotional problems, respectively (Kline, 2016). These models estimate the effect of one variable on another, temporally succeeding variable (i.e., cross-lagged effects), while also adjusting for the stability of each variable over time (i.e., autoregressive effects). Figure 1 presents a schematic diagram of reciprocal change between harsh parenting and child conduct and emotional problems, respectively.

[Insert Figure 1 here]

The amount of missing data for the harsh parenting and child conduct and emotional problems variables ranged from 15.3% to 15.9%, apart from harsh parenting at age 6 years, which had a higher rate of missingness (33.8%). There were no differences between complete cases and those with missing data in harsh parenting at ages 6 and 11 years. However, compared to complete cases, those with missing data showed higher conduct problems at age 6 years, and lower levels of conduct and emotional problems at age 11 years. For the covariates, maternal education and maternal skin color had small amounts of missing data (<2%), whereas maternal depression showed a higher rate of missingness (8.4%). Compared to complete cases, those with missing data at ages 6 and/or 11 years on either the CTSPC and/or SDQ reported higher incomes. Even for variables where there was a significant difference between those with versus without data, the respective effect sizes for the comparisons were small (*d*s ranging from -0.09 to 0.13) (see Appendix 1 for all pairwise comparisons).

We addressed missing data for all CTSPC and SDQ variables using multiple imputation. In Mplus, multiple imputation uses Bayesian analysis based on the Markov Chain Monte Carlo method, which simulates random draws from the posterior distribution of the missing scores (Kline, 2016; Muthén & Muthén, 2017). These scores were generated under the missing at random data loss mechanism, using all harsh parenting and child conduct and emotional problems variables, in addition to all covariates (Kline, 2016). As we were interested in examining moderation effects by sex, we imputed data separately for boys and girls, which has been shown to preserve the multiple group data structure (Enders & Gottschall, 2011). We used 40 imputed datasets, which has been shown to improve power, even for larger amounts of missing data (Graham, Olchowski, & Gilreath, 2007). The resulting analysis sample consisted of 3718 participants. Subsequently, we ran all models again using listwise deletion, resulting in a sample size of 2447 participants. The results were largely identical to the model based on multiple imputation (i.e., path coefficients were of similar magnitude; see Appendix 2 for all

model estimates based on listwise deletion). Due to moderate positive skew on all CTSPC and SDQ variables, all models were estimated using the Mplus MLR estimator, which produces standard errors which are robust to non-normality (Lai, 2018).

To evaluate the direction of associations between harsh parenting and child conduct and emotional problems, we assessed the importance of the parent-to-child and child-to-parent paths based on the strength of associations in the reciprocal models for the total sample. Wald's test was used for determining whether path coefficients differed between boys and girls. All models were adjusted for maternal depression, smoking, alcohol consumption, relationship status, income, education, and skin color. Model estimates and the correlation matrix are based on imputed data, and descriptive statistics on complete cases. Multiple imputation and path models were performed in Mplus, Version 8.1 (Muthén & Muthén, 2017). All other data analyses were performed in RStudio, Version 1.1.447 (RStudio Team, 2016).

Results

Descriptive statistics

At age 6 years, 14.7% (16.1% boys; 13.1% girls) of the sample showed high levels of conduct problems (i.e., a score of 4 or above) and 13.5% (13.0% boys; 14.0% girls) showed high levels of emotional problems (i.e., a score of 5 or above). At age 11 years, 13.0% (14.3% boys; 11.6% girls) and 20.0% (19.1% boys; 21.0% girls) of the sample showed high levels of conduct and emotional problems, respectively.

Compared to boys, girls were exposed to lower levels of harsh parenting and showed lower levels of conduct problems at ages 6 and 11 years, although effect sizes were small (*d*s ranging between -0.10 and -0.16, all *p*s < .01). No sex differences were observed for emotional problems at ages 6 and 11 years (see Table 1 for all pairwise comparisons).

Figure 2 shows the correlation matrix for all variables in the study. Harsh parenting, conduct problems, and emotional problems were moderately correlated both within and between time points (*r*s ranging between 0.09-0.52, all *p*s < .001). Harsh parenting showed higher concurrent and longitudinal associations with conduct problems (*r*s ranging between 0.22-0.37, all *p*s < .001) than emotional problems (*r*s ranging between 0.09-0.17, all *p*s < .001).

[Insert Table 1 here]

[Insert Figure 2 here]

Harsh parenting and child conduct problems

Table 2 shows standardized coefficients from path models of the relationships between harsh parenting and child conduct problems (unstandardized path coefficients are presented in Appendix 3). There was a moderate degree of stability from ages 6 to 11 years for both harsh parenting and child conduct problems for the total sample, as well as for males and females separately, as indicated by significant autoregressive effects (βs ranging between 0.32-0.48, all *p*s < .001). In addition, a significant proportion of change over time in each variable was explained by temporally preceding parent-to-child and child-to-parent effects. For the total sample, as well as for males and females separately, harsh parenting at age 6 years predicted conduct problems at age 11, even after controlling for prior levels of conduct problems (βs ranging between 0.07-0.12, all *p*s < .01). Similarly, conduct problems at age 6 years predicted harsh parenting at age 11, even after controlling for prior levels of harsh parenting and independently of sex (βs ranging between 0.06-0.09, all *p*s < .05). There were no significant sex differences in autoregressive effects for harsh parenting ($\chi(1) = 0.590$, $p = .44$) or conduct problems ($\chi(1) = 1.508$, $p = .22$), indicating that the degree of stability over time did not differ between boys and girls. Similarly, there were no significant sex differences in cross-lagged effects from harsh parenting at age 6 years to conduct problems at age 11 ($\chi(1) = 1.528$, $p =$

.22), and from conduct problems at age 6 years to harsh parenting at age 11 ($\chi(1) = 0.346$, $p = .56$), suggesting that the magnitude of parent- and child-effects did not differ between boys and girls.

Harsh parenting and child emotional problems

Table 2 shows standardized coefficients from path models of harsh parenting and child emotional problems (unstandardized path coefficients are presented in Appendix 3). Similar to the model examining relationships between harsh parenting and conduct problems, there was a moderate degree of stability over time for both harsh parenting and emotional problems for the total sample, as well as for each sex separately, as indicated by significant autoregressive effects (βs ranging between 0.33-0.49, all ps < .001). However, in contrast to the reciprocal relationship between harsh parenting and child conduct problems, only temporally preceding parent-to-child, but not child-to-parent, effects predicted change over time. More specifically, harsh parenting at age 6 years predicted emotional problems at age 11, even after controlling for prior levels of emotional problems ($\beta = .04$, $p = .03$). In contrast, emotional problems at age 6 years were not predictive of harsh parenting at age 11, after adjusting for prior levels of harsh parenting ($\beta = .00$, $p = .86$) and independently of sex. The observed parent-to-child effect was significant in females ($\beta = .07$, $p = .02$), but not in males ($\beta = .03$, $p = .32$). However, when directly comparing boys and girls, no significant sex differences were found ($\chi(1) = 1.098$, $p = .30$). In addition, there were no sex differences in autoregressive effects for harsh parenting ($\chi(1) = 0.539$, $p = .46$) or emotional problems ($\chi(1) = 0.040$, $p = .84$), suggesting similar degrees of stability over time in boys and girls.

[Insert Table 2 here]

Discussion

To our knowledge, this is the first study to use a prospective longitudinal design and a population-based sample to examine cross-lagged associations between harsh parenting and child conduct and emotional problems in a low- and middle-income country (LMIC). We found bidirectional effects between harsh parenting and child conduct problems (i.e., harsh parenting at age 6 years predicted conduct problems at age 11, even after adjusting for initial levels of conduct problems, and vice versa), but only a unidirectional relationship between harsh parenting and child emotional problems (i.e., harsh parenting at age 6 years predicted emotional problems at age 11, even after adjusting for baseline emotional problems, but not vice versa). We also examined whether sex moderated the strength or nature of the cross-lagged and autoregressive effects, but found no robust evidence for sex differences in these associations.

Previous studies have indicated that the effects of harsh parenting on child externalizing and internalizing problems may depend partly on cultural norms related to harsh parenting practices (Lansford et al., 2005), suggesting heterogeneous effects across different cultural contexts. However, our findings from Brazil are in line with two meta-analyses of cross-lagged associations that almost exclusively included studies from high-income countries (HICs), which showed bidirectional effects for externalizing problems and unidirectional effects for internalizing problems (Pinquart, 2016, 2017). Effect sizes were small, but in line with those reported in previous meta-analyses (Pinquart, 2016, 2017). On the basis of small effect sizes for parenting effects, some researchers have argued that there is insufficient evidence to categorically oppose physical punishment (Larzelere, Gunnoe, Ferguson, & Roberts, 2019). Others, however, have disputed this idea, stating the lack of evidence in support of physical punishment (Gershoff et al., 2019). Some researchers have argued for a continuum of violence against children (Straus, 2001), with spanking and physical abuse both involving expression of harsh parenting and negative child outcomes, just to different degrees (Brown, Holden, &

Ashraf, 2018). Thus, it should be noted that harsh parenting in the current study may better be described as harsh, aggressive, and abusive parenting. Nevertheless, there are substantial differences between countries in the prevalence of harsh parenting, and future research across cultural contexts is warranted (Lansford & Deater-Deckard, 2012).

The present findings provide support for transactional models between negative parental discipline and child conduct problems. According to Patterson's coercive processes model of antisocial behavior (1982, 1986), dysfunctional parent-child interactions in early development lead to an incremental decline in the quality of the parent-child relationship. These coercive cycles may continue into middle and late childhood as well as adolescence and extend beyond the family context to affect behavior in school or within the peer group. According to social information processing theory and social learning theory (Bandura, 1977; Crick & Dodge, 1994), children may internalize their parents' harsh and abusive behavior, and, as a consequence, are unable to generate appropriate responses to situations of conflict and distress. Consequently, harsh parenting may play an important role in initiating child conduct problems. However, as Patterson notes, child characteristics, including, for example, difficult temperament, may negatively impact parenting practices (Patterson, 1986).

The findings also provide evidence for a unidirectional parent-effects model of the association between negative parental discipline and child internalizing problems. Serbin et al. (2015) found a negative feedback loop between parenting and child internalizing outcomes measured in the context of a longitudinal design, i.e., child internalizing problems at wave 1 led to an increase in positive parenting behaviors at wave 2, which, in turn, led to a decrease in internalizing problems at wave 3. In contrast, a recent meta-analysis found child internalizing symptoms led to reduced parental warmth and authoritative parenting, and increases in psychologically controlling and permissive parenting behaviors (Pinquart, 2016). This implies that similar vicious cycles to those proposed by Patterson (Patterson, 1982, 1986) may apply

to child internalizing problems, but with different expressions of ineffective parenting strategies. For example, cold, unsupportive, and neglectful parenting may lead to an increase in child internalizing problems and, similarly, a withdrawn child may evoke less parental engagement and fewer stimulating interactions. However, the current study was not designed to examine whether such effects exist in our sample.

In line with previous research, we found higher levels of conduct problems in boys compared to girls (Moffitt, Caspi, Rutter, & Silva, 2001). Furthermore, boys were exposed to higher levels of harsh parenting than girls, which may have contributed to them developing higher rates of conduct problems, and vice versa. However, despite these sex differences, the reciprocal relationship between harsh parenting and child conduct problems did not differ by sex. In contrast, the association between harsh parenting and child emotional problems was only significant for girls, but not boys. However, when we directly compared boys and girls, there was no significant sex difference in the strength of this effect. Unlike in previous studies, we did not find higher levels of emotional problems in girls compared to boys, which may partly explain the non-significant sex difference (Silverman & Carter, 2006). As studies on sex differences in these relationships have mostly been limited to HICs, with just two small-scale studies testing for sex differences in cross-lagged associations between harsh parenting and child externalizing problems in LMICs (Skinner, Oburu, Lansford, & Bacchini, 2014; Xing, Wang, Zhang, He, & Zhang, 2011), further research on this topic is needed in LMICs.

Key strengths of the current study include the use of a large, birth cohort sample from Brazil, with very high retention rates, and the availability of prospective longitudinal data. Furthermore, the majority of studies testing for cross-lagged associations between parenting dimensions and child externalizing and internalizing problems have been conducted in HICs (Pinquart, 2016, 2017; Pinquart & Kauser, 2018). Thus, we were able to examine whether the

findings obtained in HICs extend to LMICs, including the direction of effects in the parent-child relationship and sex differences.

However, our study also had a number of limitations which should be considered when interpreting the findings: First, all measures were completed by a single rater, usually the mother, and therefore may have been subject to shared rater bias, which may have inflated associations between variables. Second, parents may under-report child emotional problems (Lagattuta, Sayfan, & Bamford, 2012), especially in the case of girls (Michels et al., 2013). Thus, future studies should attempt to mitigate against these issues by using both parent- and self-reports of child psychopathology. Third, there was selective attrition over time. These effects, however, were small and addressed through the use of multiple imputation, using an adequate number of imputed data sets and taking the child's sex into account (Enders & Gottschall, 2011; Graham et al., 2007). Furthermore, the findings were similar when listwise deletion was used to deal with missing data rather than multiple imputation. Fourth, the SDQ subscales showed modest internal consistency. Although, the SDQ is a widely used measure, the current results require replication, using a measure of child conduct and emotional problems with better psychometric properties. Fifth, with data available from only two time points, we were not able to examine a sequence of change (i.e., a feedback loop) between harsh parenting and child conduct and emotional problems, respectively, which would require data from a minimum of three time points. Data collection for the age 15 time point is currently underway, which will allow researchers to investigate these issues, in addition to modelling developmental trajectories of child conduct and emotional problems, respectively. Finally, the relationship between harsh parenting and child conduct problems may be in part explained by genetically mediated child-effects, especially in the case of less severe forms of harsh and abusive parenting (Jaffee et al., 2004). However, the current study was not designed to investigate this possibility.

Given the bidirectional effects between harsh parenting and child conduct problems reported here and in other studies conducted in HICs (Pinquart, 2017), future interventions aimed at targeting harsh and abusive parenting to reduce conduct problems should also include child-focused components, directly targeting child behavior problems. In contrast, preventive interventions to address child internalizing problems may primarily focus on parent-focused components. In HICs, there is strong evidence for the effectiveness of parent training programs for child conduct problems, focusing on reducing harsh parenting and promoting positive parenting (Piquero et al., 2016), with only preliminary evidence available from LMICs (Knerr, Gardner, & Cluver, 2013). Upcoming trials will further elucidate the effectiveness of such parenting programs in Brazil (Murray et al., 2019).

In conclusion, we found reciprocal relationships between harsh parenting and child conduct problems, and unidirectional effects of harsh parenting on child emotional problems, with no significant sex differences observed in either model. Our findings highlight the detrimental impact of harsh parenting on child psychopathology and demonstrate the importance of targeting both parent- and child-effects in preventive interventions aiming to reduce harsh parenting and promote positive parenting.

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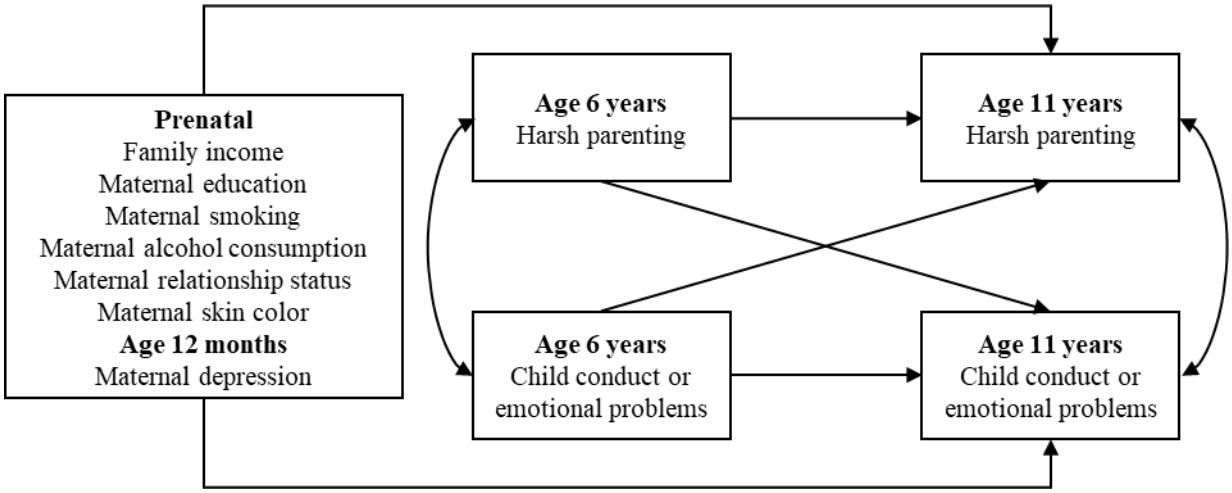
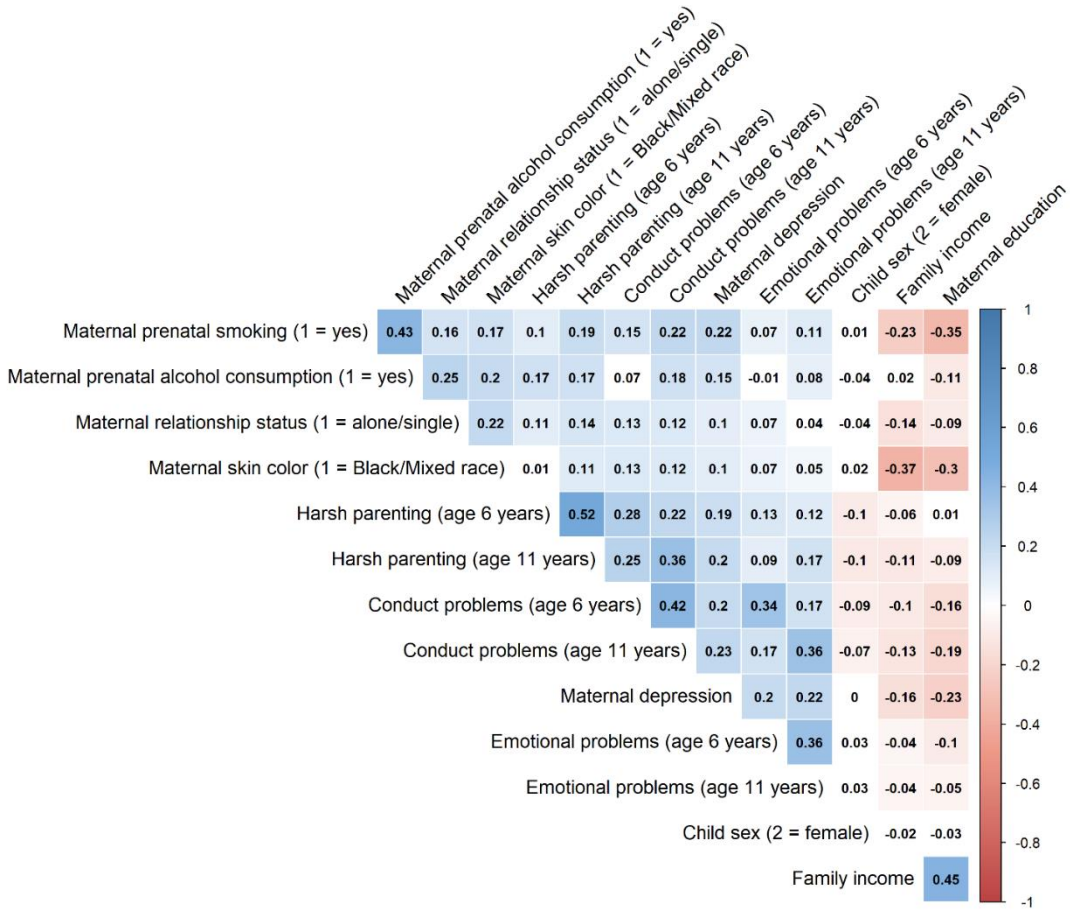
Figure 1. Schematic representation of an observed-variable autoregressive path model examining reciprocal interactions between harsh parenting and child conduct or emotional problems, after adjusting for covariates



Note. Lines with single arrowheads represent hypothesised direct effects. Curved lines with two arrowheads represent correlations. Analyses were conducted separately for child conduct and emotional problems.

Figure 2. Correlation matrix of all variables used in the cross-lagged models



Note. Imputed, rather than observed, values are presented. The color bar represents correlation coefficients from -1 (red) to +1 (blue). Blue squares represent significant positive correlations. Red squares represent significant negative correlations. Darker color tones represent larger correlation coefficients. White squares represent non-significant correlation coefficients at $p < .05$.

Table 1. Descriptive statistics for the total sample and separated by sex

Variables (ranges in parentheses)	**Total** Mean (SD) or %	**Male** Mean (SD) or %	**Female** Mean (SD) or %	**Gender comparison** *t*(df) or $\chi 2$(df)	**Effect size** *d* (95% CI) or OR (95% CI)
Harsh parenting (0-28)					*d*
Age 6	6.74 (4.26)	7.03 (4.35)	6.42 (4.13)	$t(2737.8) = 3.81$, $p < .001$	-0.14 (-0.22 to -0.07)
Age 11	6.52 (4.49)	6.87 (4.64)	6.16 (4.29)	$t(3484.5) = 4.71$, $p < .001$	-0.16 (-0.23 to -0.09)
Conduct problems (0-10)					
Age 6	1.53 (1.82)	1.65 (1.87)	1.40 (1.76)	$t(3505.1) = 3.97$, $p < .001$	-0.14 (-0.20 to -0.07)
Age 11	1.39 (1.84)	1.48 (1.89)	1.29 (1.78)	$t(3487.9) = 3.11$, $p = .002$	-0.10 (-0.17 to -0.04)
Emotional problems (0-10)					
Age 6	2.20 (2.05)	2.16 (2.02)	2.25 (2.09)	$t(3459.2) = -1.32$, $p = .19$	0.04 (-0.02 to 0.11)
Age 11	2.69 (2.33)	2.64 (2.34)	2.74 (2.33)	$t(3475.2) = -1.20$, $p = .23$	0.04 (-0.02 to 0.11)
Covariates					
Continuous					
Weekly family income (BRL)	200.87 (277.10)	205.13 (295.22)	196.24 (256.04)	$t(4126.9) = 1.04$, $p = .30$	-0.03 (-0.09 to 0.03)
Maternal education (years)	8.11 (3.47)	8.19 (3.49)	8.02 (3.45)	$t(4079.4) = 1.58$, $p = .11$	-0.05 (-0.11 to 0.01)
Maternal depression (0-30)	7.21 (5.04)	7.23 (5.00)	7.19 (5.08)	$t(3765/3) = 0.25$, $p = .80$	-0.01 (-0.07 to 0.06)
Binary					OR
Maternal prenatal smoking (yes)	27.6	27.2	27.9	$\chi^2(1) = 0.29$, $p = .59$	1.04 (0.90-1.19)
Maternal prenatal alcohol consumption (yes)	3.4	3.6	3.1	$\chi^2(1) = 0.81$, $p = .37$	0.86 (0.60-1.22)
Maternal relationship status (single)	16.3	17.1	15.5	$\chi^2(1) = 1.82$, $p = .18$	0.89 (0.75-1.06)
Maternal skin color (Black/Mixed race)	37.9	37.8	38.9	$\chi^2(1) = 0.60$, $p = .44$	1.05 (0.92-1.19)

Note. Observed, rather than imputed values are presented. BRL = Brazilian real (2.89 BRL = 1 USD in January 2004 when recruitment of the families commenced); CI = Confidence interval; *d* = Cohen's *d*; df = Degrees of freedom; OR = Odds ratio; SD = Standard deviation.

Table 2. Path estimates using *multiple imputation* for the total sample and separated by sex

	Total sample (*N* = 3718)		**Males** (*N* = 1931)		**Females** (*N* = 1787)	
	β (SE)	*P*	*β* (SE)	*P*	*β* (SE)	*P*
Harsh parenting and conduct problems						
Autoregressive effects						
Conduct problems (age 6) → conduct problems (age 11)	0.351 (0.020)	< .001	0.374 (0.028)	< .001	0.321 (0.027)	< .001
Harsh parenting (age 6) → harsh parenting (age 11)	0.471 (0.016)	< .001	0.476 (0.022)	< .001	0.462 (0.024)	< .001
Cross-lagged effects						
Conduct problems (age 6) → harsh parenting (age 11)	0.076 (0.017)	< .001	0.063 (0.025)	= .010	0.085 (0.026)	= .001
Harsh parenting (age 6) → conduct problems (age 11)	0.093 (0.019)	< .001	0.073 (0.026)	= .005	0.117 (0.026)	< .001
Harsh parenting and emotional problems						
Autoregressive effects						
Emotional problems (age 6) → emotional problems (age 11)	0.332 (0.017)	< .001	0.326 (0.024)	< .001	0.335 (0.025)	< .001
Harsh parenting (age 6) → harsh parenting (age 11)	0.490 (0.015)	< .001	0.493 (0.021)	< .001	0.481 (0.023)	< .001
Cross-lagged effects						
Emotional problems (age 6) → harsh parenting (age 11)	0.003 (0.016)	= .859	0.004 (0.023)	= .857	0.004 (0.023)	= .865
Harsh parenting (age 6) → emotional problems (age 11)	0.043 (0.019)	= .026	0.027 (0.027)	= .315	0.066 (0.028)	= .017

Note. All models were adjusted for maternal depression, smoking, alcohol consumption, relationship status, income, education, and skin color.
β = standardized regression coefficient; SE = standard error; *P* = *p*-value.

Appendix 1 Attrition between complete cases (n = 2576) and those with at least one missing score (n = 1569) for the harsh parenting and/or child conduct and emotional problems variables

Variables (ranges in parentheses)	**Complete cases** Mean (SD) or %	**Missing** Mean (SD) or %	**Comparison** t(df) or χ^2(df)	**Effect size** d (95% CI) or OR (95% CI)
Harsh parenting (0-28)				*d*
Age 6	6.75 (4.26)	6.50 (4.19)	$t(188.98) = -0.75$, $p = .46$	-0.06 (-0.22 to 0.10)
Age 11	6.50 (4.45)	6.59 (4.60)	$t(1552.5) = 0.54$, $p = .59$	0.02 (-0.06 to 0.10)
Conduct problems (0-10)				
Age 6	1.47 (1.78)	1.71 (1.92)	$t(1551.5) = 3.38$, $p < .001$	0.13 (0.06 to 0.21)
Age 11	1.43 (1.87)	1.27 (1.73)	$t(1722.5) = -2.26$, $p = .02$	-0.09 (-0.16 to -0.01)
Emotional problems (0-10)				
Age 6	2.18 (2.04)	2.27 (2.08)	$t(1619) = 1.13$, $p = .26$	0.05 (-0.02 to 0.13)
Age 11	2.74 (2.36)	2.53 (2.25)	$t(1674.5) = -2.41$, $p = .02$	-0.09 (-0.17 to -0.02)
Covariates				
Continuous				
Weekly family income (BRL)	191.2 (257.56)	216.74 (305.92)	$t(2886.6) = 2.76$, $p = .006$	0.09 (0.03 to 0.12)
Maternal education (years)	8.03 (3.39)	8.24 (3.60)	$t(3125.2) = 1.88$, $p = .06$	0.06 (-0.00 to 0.12)
Maternal depression (0-30)	7.27 (5.07)	7.09 (4.99)	$t(2655.2) = -1.08$, $p = .28$	-0.04 (-0.10 to 0.03)
Binary				OR
Maternal prenatal smoking (yes)	26.7	28.9	$\chi^2(1) = 2.42$, $p = .12$	0.89 (0.78-1.03)
Maternal prenatal alcohol consumption (yes)	3.2	3.7	$\chi^2(1) = 0.79$, $p = .37$	0.86 (0.60-1.23)
Maternal relationship status (alone/single)	16.0	16.8	$\chi^2(1) = 0.45$, $p = .50$	0.94 (0.79-1.12)
Maternal skin color (Black/Mixed race)	38.4	38.2	$\chi^2(1) = 0.02$, $p = .88$	1.01 (0.89-1.15)
Child's sex (female)	48.3	47.6	$\chi^2(1) = 0.16$, $p = .69$	1.03 (0.90-1.17)

Note. Observed, rather than imputed values are presented. BRL = Brazilian real (1 USD in January 2004 when recruitment of the families commenced); CI = Confidence interval; *d* = Cohen's *d*; df = Degrees of freedom; OR = Odds ratio; SD = Standard deviation.

Appendix 2 Path estimates using *listwise deletion* for the total sample and separated by sex

	Total sample (*N* = 2447)			**Males** (*N* = 1261)			**Females** (*N* = 1186)		
	B (SE)	*β* (SE)	*P*	*B* (SE)	*β* (SE)	*P*	*B* (SE)	*β* (SE)	*P*
Harsh parenting and conduct problems									
Autoregressive effects									
Conduct problems (age 6) → conduct problems (age 11)	0.350 (0.025)	0.333 (0.023)	< .001	0.379 (0.035)	0.367 (0.032)	< .001	0.312 (0.037)	0.291 (0.033)	< .001
Harsh parenting (age 6) → harsh parenting (age 11)	0.486 (0.020)	0.469 (0.018)	< .001	0.502 (0.029)	0.476 (0.026)	< .001	0.464 (0.027)	0.458 (0.027)	< .001
Cross-lagged effects									
Conduct problems (age 6) → harsh parenting (age 11)	0.148 (0.050)	0.059 (0.020)	= .003	0.118 (0.071)	0.047 (0.028)	= .098	0.161 (0.068)	0.064 (0.027)	= .018
Harsh parenting (age 6) → conduct problems (age 11)	0.042 (0.009)	0.097 (0.020)	< .001	0.034 (0.012)	0.079 (0.028)	= .005	0.051 (0.013)	0.117 (0.029)	< .001
Harsh parenting and emotional problems									
Autoregressive effects									
Emotional problems (age 6) → emotional problems (age 11)	0.396 (0.024)	0.338 (0.020)	< .001	0.385 (0.029)	0.326 (0.024)	< .001	0.376 (0.029)	0.335 (0.025)	< .001
Harsh parenting (age 6) → harsh parenting (age 11)	0.501 (0.019)	0.483 (0.018)	< .001	0.524 (0.025)	0.493 (0.021)	< .001	0.498 (0.025)	0.481 (0.023)	< .001
Cross-lagged effects									
Emotional problems (age 6) → harsh parenting (age 11)	0.009 (0.038)	0.004 (0.017)	= .809	0.010 (0.053)	0.004 (0.023)	= .857	0.008 (0.047)	0.004 (0.023)	= .865
Harsh parenting (age 6) → emotional problems (age 11)	0.025 (0.011)	0.046 (0.019)	= .019	0.015 (0.015)	0.027 (0.027)	= .315	0.037 (0.016)	0.066 (0.028)	= .039

Note. All models were adjusted for maternal depression, smoking, alcohol consumption, relationship status, income, education, and skin color.
B = unstandardized regression coefficient; *β* = standardized regression coefficient; SE = standard error; *P* = *p*-value.

Appendix 3 Unstandardized regression coefficients using multiple imputation for the total sample and separated by sex

	Total sample (*N* = 3718)		**Males** (*N* = 1931)		**Females** (*N* = 1787)	
	B (SE)	*P*	*B* (SE)	*P*	*B* (SE)	*P*
Harsh parenting and conduct problems						
Autoregressive effects						
Conduct problems (age 6) → conduct problems (age 11)	0.354 (0.021)	< .001	0.376 (0.030)	< .001	0.326 (0.029)	< .001
Harsh parenting (age 6) → harsh parenting (age 11)	0.494 (0.018)	< .001	0.506 (0.026)	< .001	0.478 (0.025)	< .001
Cross-lagged effects						
Conduct problems (age 6) → harsh parenting (age 11)	0.189 (0.044)	< .001	0.159 (0.062)	= .010	0.211 (0.063)	= .001
Harsh parenting (age 6) → conduct problems (age 11)	0.040 (0.008)	< .001	0.031 (0.011)	= .005	0.050 (0.011)	< .001
Harsh parenting and emotional problems						
Autoregressive effects						
Emotional problems (age 6) → emotional problems (age 11)	0.382 (0.020)	< .001	0.385 (0.029)	< .001	0.376 (0.029)	< .001
Harsh parenting (age 6) → harsh parenting (age 11)	0.514 (0.017)	< .001	0.524 (0.025)	< .001	0.498 (0.025)	< .001
Cross-lagged effects						
Emotional problems (age 6) → harsh parenting (age 11)	0.006 (0.035)	= .859	0.010 (0.053)	= .857	0.008 (0.047)	= .865
Harsh parenting (age 6) → emotional problems (age 11)	0.023 (0.011)	= .026	0.015 (0.015)	= .315	0.037 (0.016)	= .017

Note. All models were adjusted for maternal depression, smoking, alcohol consumption, relationship status, income, education, and skin color.
B = unstandardized regression coefficient; SE = standard error; *P* = *p*-value.

Chapter 4

Associations between developmental timing of child abuse and conduct problem trajectories in a UK birth cohort

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Chapter rationale

Child abuse is a key environmental risk factor for child conduct problems. However, little is known about its relationship with developmental trajectories of conduct problems, especially with respect to timing and persistence. Therefore, this third study examined associations between conduct problem trajectories from ages 4-17 years and child abuse occurring in childhood, adolescence, or persistently across both developmental periods.

This declaration concerns the article entitled:

Associations between developmental timing of child abuse and conduct problem trajectories in a UK birth cohort

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Candidate's contribution to the paper (provide details, and also indicate as a percentage)	The candidate contributed to / considerably contributed to / predominantly executed the… **Formulation of ideas:** Andreas Bauer contributed considerably to the formulation of ideas for the current study (80%). **Design of methodology:** The data used in the current study was part of a larger data set. Andreas Bauer considerably contributed to the design of the methodology in this current chapter (80%). **Experimental work:** Andreas Bauer predominantly executed the analysis and interpretation of results for this study (90%). **Presentation of data in journal format:** The presentation of the data into journal format was predominantly carried out by Andreas Bauer (95%).
Statement from Candidate	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature.

Signed	[redacted]	**Date**	27.06.2021

Associations between developmental timing of child abuse and conduct problem trajectories in a UK birth cohort

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Abstract

Background Although there is strong evidence for a relationship between child abuse and neglect and conduct problems, associations between child abuse experienced at different developmental stages and developmental trajectories of conduct problems have not been examined. We sought to investigate effects of timing of child abuse on conduct problem trajectories in a large UK birth cohort study.

Methods We applied latent class growth analysis to identify conduct problem trajectories in the Avon Longitudinal Study of Parents and Children, using parent-rated conduct problems from ages 4-17 years (*N*=10648). Childhood-only and adolescence-only abuse, in addition to abuse in both developmental periods ('persistent' abuse), were assessed by retrospective self-report at age 22 years (*N*=3172).

Results We identified four developmental trajectories: early-onset persistent (4.8%), adolescence-onset (4.5%), childhood-limited (15.4%), and low (75.3%) conduct problems. Childhood-only abuse and 'persistent' abuse were associated with increased odds of being on

the early-onset persistent and adolescence-onset conduct problem trajectories compared to the low conduct problems trajectory. Adolescence-only abuse was not predictive of trajectory membership. There were no associations between abuse and childhood-limited trajectory membership.

Conclusions Early-onset persistent and adolescence-onset conduct problems showed similar patterns of association with abuse exposure, challenging developmental theories that propose qualitative, as opposed to quantitative, differences in environmental risk factors between these trajectories. The results also highlight that childhood-only and 'persistent' abuse were more strongly linked to elevated conduct problem trajectories than adolescence-only abuse, and that 'persistent' abuse is particularly detrimental.

Key words

Child abuse, child maltreatment, antisocial behavior, conduct problems, developmental trajectories, latent class growth analysis, ALSPAC

Background

Conduct problems refer to antisocial behaviors displayed in childhood and/or adolescence that are symptomatic of conduct disorder and oppositional defiant disorder (American Psychiatric Association, 2013). They account for a substantial proportion of personal (Erskine et al., 2014), familial (Roberts, McCrory, Joffe, De Lima, & Viding, 2018), and societal burden (Moffitt, Caspi, Harrington, & Milne, 2002; Rivenbark et al., 2018; Scott, Knapp, Henderson, & Maughan, 2001), and are associated with negative outcomes across multiple domains, including mental and physical health problems (Colman et al., 2009; Fairchild et al., 2019). It is therefore crucial to thoroughly understand the etiology of such difficulties and to develop effective prevention and intervention programs.

According to Moffitt's developmental taxonomic theory (1993, 2018), individuals with elevated conduct problems can be classified into two subtypes: *early-onset persistent* (also called *'life-course persistent'*) and *adolescence-limited*. Early-onset persistent conduct problems are proposed to emerge in childhood, originating from genetic, congenital, or acquired neuropsychological deficits. Accumulating interactions with high-risk environments culminate in antisocial and aggressive behavior that persists throughout the lifespan. Thus, neurocognitive impairments, difficult child temperament, and adverse family environments have been proposed as the main risk factors for early-onset persistent conduct problems (Moffitt, 2018). By contrast, adolescence-limited conduct problems are considered to be caused by an extended period of dependence, labeled the *maturity gap*, in which the individual is treated as a child despite being biologically mature (Moffitt, 1993, 2018). This leads them to imitate the behavior of their peers showing early-onset persistent conduct problems. Thus, delinquent peer relationships are proposed to be the main determinant of adolescence-limited conduct problems (Moffitt, 2018). Accordingly, while early-onset persistent conduct problems are considered a neurodevelopmental disorder, adolescence-limited conduct problems are

viewed as normative and transient – an exaggerated form of normal teenage rebellion (Moffitt, 1993, 2018).

The developmental taxonomic theory has been crucial in shifting focus from considering adolescent conduct problems as a unitary phenomenon to understanding different trajectories of conduct problems that may result from distinct risk factors. Nonetheless, accumulating empirical evidence from a range of disciplines suggests three potential revisions to this model: (i) the addition of a second *adolescence-onset* subtype that emerges in adolescence but persists into adulthood; (ii) the inclusion of a second childhood-onset subtype, in which antisocial behavior remits in the transition from childhood to adolescence (*childhood-limited*); and (iii) the reformulation of the assumption of distinct etiological causes to a model of common individual and environmental risks across subtypes, albeit with different timings and magnitudes of exposure (Fairchild, van Goozen, Calder, & Goodyer, 2013). In sum, whereas the original developmental taxonomic theory proposes a *qualitative* distinction between early-onset persistent and adolescence-limited conduct problems in terms of etiology and developmental course, there is accumulating evidence for additional conduct problem trajectories, including adolescence-onset and childhood-limited, and *quantitative* differences across all subtypes – with children with early-onset persistent conduct problems being exposed to the highest levels of individual and environmental risk and those with adolescence-limited conduct problems exposed to the lowest. More precisely, the magnitude, number, and range of risk factors may be more influential in differentiating between early-onset persistent and adolescence-limited conduct problems than any individual risk factor (Jiang et al., 2015; Jolliffe, Farrington, Piquero, Loeber, & Hill, 2017; Roisman et al., 2010).

A key environmental risk factor implicated in the development of conduct problems is child abuse (i.e., physical, psychological, or sexual) and neglect (i.e., physical or psychological), with evidence from prospective longitudinal studies showing that those

exposed to abuse and neglect in childhood and/or adolescence are at increased risk of developing conduct problems compared to those who are not exposed (Braga, Gonçalves, Basto-Pereira, & Maia, 2017; Wilson, Stover, & Berkowitz, 2009). With respect to conduct problem trajectories, several studies have reported that child abuse and/or neglect are associated with childhood-onset conduct problems (i.e., early-onset persistent or childhood-limited), but not those that develop in adolescence (i.e., adolescence-limited or adolescence-onset), which is broadly consistent with the hypothesis of distinct risk factors across these groups (Aguilar, Sroufe, Egeland, & Carson, 2000; Moore & Mezuk, 2014; Raine et al., 2005). By contrast, Odgers et al. (2008) reported higher rates of child abuse and neglect in individuals with early-onset persistent, childhood-limited, *and* adolescence-onset conduct problems. Collectively, these studies all provide evidence that child abuse and neglect is associated with the early-onset persistent conduct problems trajectory, but the findings for conduct problems emerging in adolescence are less conclusive. Furthermore, child abuse and neglect was exclusively measured in childhood in these studies, rather than in adolescence or both developmental periods; consequently, existing evidence is limited in terms of understanding the relationship between developmental timing of abuse and different trajectories of conduct problems, especially considering that exposure to abuse may be more common in adolescence than in childhood (Radford et al., 2011).

Determining how the developmental timing of abuse or its persistence maps onto trajectories of conduct problems may provide new insights into the mechanisms underlying risk for conduct problems. For example, while some have proposed that childhood may be a period of particular sensitivity to adverse rearing environments, due to their potential impacts on neural, cognitive, and social development (McLaughlin, Fox, Zeanah, & Nelson, 2011), others have argued that adolescence may be a sensitive period, as it is a key stage of maturation of specific brain regions, such as the medial prefrontal cortex (Andersen & Teicher, 2008;

Blakemore & Mills, 2014). Alternatively, the *accumulation* of negative experiences may be most relevant in determining outcomes, irrespective of their timing (Evans, Li, & Whipple, 2013). However, evidence relating to timing or persistence of exposure to abuse in relation to conduct problems is limited to studies examining outcomes in adolescence and adulthood only, and these have yielded mixed findings. Thornberry and colleagues found adolescence-only and persistent abuse to be consistently predictive of adolescent and adult antisocial behavior, whereas childhood-only abuse showed weaker or null associations (Ireland, Smith, & Thornberry, 2002; Thornberry, Henry, Ireland, & Smith, 2010; Thornberry, Ireland, & Smith, 2001). By contrast, Mersky et al. (2012) found childhood-only, adolescence-only, and persistent abuse to be equally predictive of antisocial behavior in adolescents. However, these studies did not investigate conduct problem *trajectories*, meaning that our understanding of the impact of timing of abuse on the longitudinal development and course of conduct problems remains limited.

To address these gaps in the literature, we examined developmental trajectories of conduct problems in a large population-based sample and differentiated between childhood-only and adolescence-only abuse, in addition to abuse occurring in both developmental periods (hereafter referred to as 'persistent' abuse). The main objectives of the current study were: (i) to estimate developmental trajectories of conduct problems from ages 4-17 years in a longitudinal population-based sample, extending existing trajectories from the same sample which only covered the period from 4-13 years (Barker & Maughan, 2009); and (ii) to examine associations between exposure to abuse across childhood and/or adolescence and our derived conduct problem trajectories. We expected *temporal ordering effects*; while exposure to abuse in childhood may predict the subsequent development of conduct problems in adolescence, the converse relationship would not apply. According to this logic, exposure to adolescence-only abuse would be associated with adolescence-onset, but not childhood-limited, conduct

problems. Consistent with a *dose-response* or *accumulative effect*, we further hypothesized that 'persistent', as opposed to time-limited, abuse would yield the strongest effects for all elevated conduct problem trajectories, especially for the early-onset persistent trajectory, as it may cause the emergence of conduct problems in childhood and contribute to their maintenance in adolescence. For our primary analyses, we used an aggregate measure of abuse, encompassing physical, psychological, and sexual abuse, as the base rates of individual abuse subtypes for some conduct problem trajectories were low in our sample. However, in a set of exploratory analyses, we also investigated whether particular abuse subtypes were more strongly associated with the elevated conduct problem trajectories than others.

Methods

Participants

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a prospective birth cohort study, investigating genetic and environmental influences on health and development across the lifespan. All pregnant women residing in the Avon catchment area in South-West England, with an estimated delivery date between April 1991 and December 1992, were eligible for inclusion. Individuals were recruited through media information, community outreach, and promotional material supplied at routine antenatal and maternity health services. Out of 20248 eligible pregnancies, 14541 (71.8%) were initially recruited. Of those, 68 had no known birth outcome. The remaining 14472 pregnancies consisted of 14676 fetuses, with 14062 live births, of whom 13988 were alive at age 12 months. The current sample was restricted to singletons or first-born twins, resulting in an overall sample size of 13793 participants (51.6% boys). Prior to 2014, questionnaires were sent out to parents/carers by post. If a response was not received within 7 days, two reminder letters were sent and eventually participants were called or visited at their homes. Questionnaires from 2014 onwards were available online or in paper format,

and collected and managed using REDCap electronic data capture tools hosted at the University of Bristol (Harris et al., 2009). Participants were sent four reminders at three-week intervals. Further details on the cohort can be found elsewhere (Boyd et al., 2013; Fraser et al., 2013).

Measures: conduct problems

Conduct problems were measured at ages 4, 7, 8, 10, 12, 13, and 17 years, using the parent-rated conduct problems subscale of the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997, 2001). This widely used scale consists of five items asking about the child's behavior over the last six months: (1) "often has temper tantrums or hot tempers"; (2) "generally obedient, usually does what adults request" (reverse coded); (3) "often fights with other children or bullies them"; (4) "often lies or cheats"; and (5) "steals from home, school or elsewhere". All items are rated on a 3-point scale (0-2), from *not true* to *somewhat true* and c*ertainly true*, yielding overall scores ranging from 0-10. Previously reported developmental trajectories of conduct problems from ages 4-13 years in ALSPAC dichotomized the conduct problems subscale as 'high risk' versus 'not high risk' (Barker & Maughan, 2009). In order to maximize variability in conduct problems, but also account for the highly skewed distribution, we used the updated 4-band categorization that has been validated for ages 4-17 years (see www.SDQinfo.org), with scores of 0-2 classified as 'close to average', 3 as 'slightly raised', 4-5 as 'high', and 6-10 as 'very high'. The mean internal consistency was modest (α = 0.54, range = 0.50-0.59), which may be attributed in part to the scale's efforts to cover a wide range of problem behaviors across childhood and adolescence. Nonetheless, in their review, Stone et al. (2010) reported a similar value of α = 0.58, and demonstrated acceptable reliability and validity of the SDQ conduct problems subscale on the basis of a more rigorous psychometric assessment.

Validation of derived conduct problem trajectories

We used the Edinburgh Study of Youth Transitions and Crime (ESYTC) questionnaire to validate the derived conduct problem trajectories (Smith & McVie, 2003). The ESYTC was administered via self-report at ages 14 (N = 5604) and 18 (N = 3743) years, and included six items, asking, for example, whether the participant "deliberately damaged or destroyed property" or had "broken into a car or van with intention of stealing something out of it". Items are rated on a 4-point scale, from *not at all*, to *just once*, *2-5 times*, and *6 or more times*. Cronbach's alphas were 0.52 and 0.45 at ages 14 and 18 years, respectively. We chose to dichotomize this measure – antisocial behavior was either considered 'present' (at least *just once* for one or more items) or 'absent' (*not at all* for all items) – due to a highly skewed distribution.

Measures: child abuse

We measured physical, psychological, and sexual abuse occurring in childhood (defined as before age 11 years) and adolescence (defined as between ages 11-17 years) at age 22 years by retrospective self-report. The measure has been used previously in the Growing Up Today Study, a US population-based cohort (see www.nhs2survey.org). Since we were interested in *time-dependent* associations between child abuse and conduct problem trajectories, continuous scales had to be converted into binary variables. Similar to prior research examining the developmental timing of abuse in relation to conduct problems, which distinguished between abuse occurring up to age 11 years and between ages 12-17 years (Ireland et al., 2002; Mersky et al., 2012; Thornberry et al., 2010; Thornberry et al., 2001), we created three abuse exposure categories. These included *childhood-only* (i.e., only before the age of 11 years), *adolescence-only* (i.e., only between ages 11-17 years), and *'persistent'* abuse (i.e., abuse in both developmental periods). For our primary analysis, we computed an aggregate measure of *any abuse* (i.e., either physical, psychological, or sexual abuse) as preliminary analyses indicated

high correlations between abuse subtypes (see Appendix 1 for the correlation matrix), in addition to low frequencies of some abuse subtypes. Nonetheless, we also performed exploratory analyses testing for associations between abuse subtypes and conduct problem trajectories to examine whether certain subtypes were more influential than others.

Physical abuse

We used two items to assess physical abuse, asking whether an adult in the family "hit you so hard it left you with bruises or marks?" or "actually kicked, punched, or hit you with something that could hurt you, or physically attacked you in another way?". Items were rated on a 5-point scale from *never* to *rarely*, *sometimes*, *often*, and *very often*. In line with previous studies (Roberts, Galea, et al., 2012; Roberts, Rosario, Corliss, Koenen, & Austin, 2012), physical abuse was coded as 'present' or 'absent'.

Psychological abuse

Four items were used to assess psychological abuse, asking participants whether an adult in the family "shouted at you?"; "said hurtful or insulting things to you?"; "punished you in a way that seemed cruel?"; and "threatened to kick, punch, or hit you with something that could hurt you or physically attack you in another way?". Again, items were rated on a 5-point scale (0-4), from *never* to *very often*. Considering the complex nature of psychological abuse, we followed Roberts et al. (2012) and computed a sum score ranging from 0-16, with participants scoring in the top decile (i.e., scores of ≥ 7 in our sample) being classified as having experienced psychological abuse.

Sexual abuse

We used two items to assess sexual abuse, including "Were you touched in a sexual way by an adult or an older child or were you forced to touch an adult or older child in a sexual way when you did not want to?" and "Did an adult or an older child force you or attempt to force you into

any sexual activity by threatening you or holding you down or hurting you in some way when you did not want to?". In line with previous work (Roberts, Galea, et al., 2012), sexual abuse was coded as 'present' or 'absent'.

Covariates

Information on all covariates was collected by maternal self-report during pregnancy, except for child sex, which was obtained from the birth certificate. Housing tenure was assessed at 8 weeks gestation. Participants were asked whether their house was *bought/mortgaged*, *owned*, *rented*, or *other*. We dichotomized this variable into 'mortgaged/owned' or 'other'. Maternal severe depression was assessed at 12 weeks gestation. Participants were asked whether they had ever had severe depression. *Yes, had it recently* and *Yes, in the past, not now* was coded as 'yes' and *No, never* was coded as 'no'. At 18 weeks gestation, mothers were asked whether they had smoked tobacco in the first three months of pregnancy. *Cigarettes*, *Cigars*, *Pipe*, and *Other* were coded as 'yes' and *No* was coded as 'no'. Maternal education was assessed at 32 weeks gestation using educational qualifications in common use at the time in the UK. Considering different school systems across countries, we coded this variable as 'no high school' (*CSE/none* or *vocational*), 'high school' (*O-level*), or 'beyond high school' (*A-level* or *degree*).

Data analysis plan

We applied latent class growth analysis (LCGA) to identify developmental trajectories of conduct problems, using a bias-adjusted 3-step approach (Vermunt, 2010; Wickrama, Lee, O'Neal, & Lorenz, 2016). This method accounts for misclassification error rates in latent class membership when estimating the effect of covariates (Vermunt, 2010; Wickrama et al., 2016).

First, an *unconditional* latent class model was estimated (i.e., the meaning of classes was exclusively based on the SDQ conduct problems subscale, without being influenced by covariates). We addressed missing data in this model using a full information maximum

likelihood estimator with robust standard errors (i.e., parameters were estimated using all available data). This missing data method has been shown to produce unbiased parameter estimates compared to listwise deletion, especially under the missing at random data loss mechanism and where there are higher rates of missing data (Enders & Bandalos, 2001). We modeled linear, quadratic, and cubic patterns of change, each with between one and six class solutions. The following model fit indices were used to select the optimal class model: Bayesian Information Criterion (BIC) and sample size adjusted BIC (SSABIC), which are used to reduce the risk of overfitting the model to a single sample (lower values indicate a better model fit), and the Lo-Mendell-Rubin Likelihood Ratio Test (LMR-LRT), adjusted LMR-LRT, and Bootstrapped Likelihood Ratio Test (BLRT), which compare two adjacent class models (significant *p*-values indicate a better fit of the *k* class model compared to the *k*-1 class model). We further considered entropy values (0.40, 0.60, and 0.80 represent low, medium, and high class separation, respectively), sample size of the smallest class, and interpretability of each class trajectory (Wickrama et al., 2016).

Second, after the best-fitting model was identified, the class membership information (i.e., most likely class) of each participant and misclassification error rates of each latent class were retrieved.

Third, to preserve the class membership information of the unconditional latent class model (step 1), we used the misclassification error rates obtained in step 2 when examining associations between child abuse and conduct problems trajectory membership. We addressed missing data in this *conditional* model using inverse probability weighting (IPW). Complete-case analysis may produce biased estimates if excluded cases are systematically different from those which were included. IPW can minimize this bias by allocating sampling weights to complete cases and thereby restoring total sample estimates (Seaman & White, 2013). IPW has been recommended over other techniques for handling missing data (e.g., multiple imputation)

when participants have missing data on entire assessment waves, as opposed to single items, which is especially common in longitudinal research (Seaman & White, 2013) (see Appendix 2 for information on how weights were derived). We used multinomial logistic regression to estimate the association between childhood-only, adolescence-only, and 'persistent' abuse and latent classes of conduct problems. Multinomial logistic regression estimates multinomial odds ratios (or relative risk ratios); however, we refer to effects as odds ratios (usually used for two exhaustive categories) throughout the results section for clarity. We primarily focused on the 'any abuse' category, but subsequently tested for associations between abuse subtypes and conduct problem trajectories. All analyses were adjusted for child sex, housing tenure, maternal severe depression, maternal smoking, and maternal education.

Missing data

The conduct problems trajectory model was based on 10648 participants (77.2% of the total ALSPAC sample; 51.4% boys), with missing data addressed using full information maximum likelihood. Complete data for physical, psychological, and sexual abuse and all covariates was available for 3127 participants (29.4% of those included in the conduct problems trajectory model; 35.9% boys). Those with versus without missing data on child abuse and/or covariates showed higher rates of conduct problems across all time points, albeit with small effect sizes (*r*s ranging between 0.08-0.09, all *p*s < .001). Furthermore, participants with missing data were more likely to be male (OR 2.47) and more likely to be classified as early-onset persistent (OR 1.56) or childhood-limited (OR 1.24), and less likely to be classified in the low conduct problems trajectory (OR 0.78) than participants without missing data (all *p*s < .01; see Appendix 3 for all pairwise comparisons). The sample sizes in adjusted analyses for any, physical, psychological, and sexual abuse were 3172, 3275, 3295, and 3279, respectively. See Appendix 4 for the retention flow chart across measures/analyses.

Results

Conduct problem trajectories

Models with cubic patterns of change yielded the best combination of model fit indices, interpretability of class trajectories, class sample sizes, and consistency with previous longitudinal research (Bevilacqua, Hale, Barker, & Viner, 2018), including prior modeling of conduct problem trajectories in the ALSPAC sample (Barker & Maughan, 2009). Most of the model fit indices suggested that the 5- or 6-class models were the optimal models (e.g., lower BIC and SSABIC values, and statistically significant *p*-values for the LMR-LRT and BLRT). However, the results of the 5- and 6-class models were questionable because of two early-onset persistent class variants (low vs. high), with small sample sizes (< 2%). The existence of such classes at the population level is doubtful, as they have not been reported in previous longitudinal research (Bevilacqua et al., 2018), including prior latent growth modeling in the ALSPAC sample (Barker & Maughan, 2009). Additionally, such small class sizes are unlikely to be useful in subsequent analysis. The 4-class model (BIC = 57164; SSABIC = 57097) had better fit indices compared to the 3-class model (BIC = 57311; SSABIC = 57260) and each class was an acceptable size. We therefore rejected the 5- and 6-class models in favor of the 4-class model. The four classes and their respective proportions of the overall sample were: early-onset persistent (4.8%), adolescence-onset (4.5%), childhood-limited (15.4%), and low (75.3%) conduct problems (see Table 1 for model fit statistics). Figure 1 presents the plots of predicted SDQ category proportions of the 4-class model. In sum, the early-onset persistent class showed particularly high rates of 'high' conduct problems across all assessment waves, while the childhood-limited class showed a sharp and persistent decline in elevated conduct problems. The adolescence-onset class showed 'slightly raised' conduct problems in childhood and a continuous increase of 'high' conduct problems in adolescence. Finally, the low class showed predominantly 'close to average' conduct problems across all assessment waves.

[Insert Table 1 here]

[Insert Figure 1 here]

Validation of derived conduct problem trajectories

Those with early-onset persistent, adolescence-onset, and childhood-limited conduct problems had significantly increased odds of showing antisocial behavior at age 14 years (2.6, 2.9, and 1.6 times greater odds, respectively) and 18 years (1.9, 1.9, and 1.6 times greater odds, respectively) compared to those with low conduct problems (all *p*s < .05), as measured using the ESYTC self-report measure. Additionally, at age 14 years, those with early-onset persistent and adolescence-onset conduct problems had 1.8 and 1.7 times greater odds, respectively, of showing antisocial behavior compared to those with childhood-limited conduct problems (all *p*s < .05; see Appendix 5 for all pairwise comparisons).

Descriptive statistics

Overall, across abuse exposure categories, 19.6% of the sample reported experiencing at least some form of abuse (i.e., 'any abuse'), with 11.3%, 8.9%, and 8.1% of the sample reporting physical, psychological, and sexual abuse, respectively. 40.9% of participants in the early-onset persistent and 37.5% in the adolescence-onset conduct problem classes reported experiencing some form of abuse, compared with 23.8% of the childhood-limited and 16.8% of the low classes. For specific types of abuse, the proportions for the early-onset persistent, adolescence-onset, childhood-limited, and low conduct problem classes were: 31.0%, 25.9%, 13.1%, and 9.3% for physical abuse; 28.5%, 21.3%, 11.1%, and 6.9% for psychological abuse; and 11.7%, 10.2%, 10.7%, and 7.3% for sexual abuse, respectively. Frequencies of exposure in each of the developmental phases (i.e., childhood-only, adolescence-only, and 'persistent' abuse) for the four conduct problem classes are presented in Table 2. Descriptive statistics of sociodemographic variables in the analysis sample can be found in Appendix 2.

[Insert Table 2 here]

Associations between child abuse and conduct problem trajectories

Weighted analyses are presented in Table 3, with all abuse comparisons being relative to those not exposed to any abuse. The strongest effects were observed for abuse that was reported in both childhood and adolescence. This 'persistent' abuse was associated with an 8- to 10-fold increase in the odds of being in the early-onset persistent and adolescence-onset classes compared to the low conduct problems class. In addition, 'persistent' abuse was associated with a 6- to 8-fold increased odds of being in the early-onset persistent and adolescence-onset classes compared to the childhood-limited conduct problems class. There was no evidence that exposure to 'persistent' abuse differentiated between the early-onset persistent and adolescence-onset conduct problem trajectories, or was associated with increased odds of being in the childhood-limited conduct problems class (versus the low class).

The effects for childhood-only abuse were similar, albeit slightly weaker. Childhood-only versus no abuse was associated with a 4- to 6-fold increased odds of being in the early-onset persistent and adolescence-onset classes relative to the low conduct problems class. However, it did not distinguish these classes from each other or from the childhood-limited conduct problems class, nor was it associated with increased odds of being in the childhood-limited compared to the low conduct problems class. Lastly, we found no evidence that adolescence-only abuse was associated with conduct problems trajectory membership – which may partly reflect the fact that adolescence-only abuse was rarer than childhood-only or 'persistent' abuse.

In sum, we found relatively robust associations between abuse occurring either in childhood alone or in both childhood and adolescence and the early-onset persistent and adolescence-onset conduct problem trajectories. We found no evidence that abuse occurring in either childhood and/or adolescence was associated with childhood-limited conduct problems,

and abuse occurring only in adolescence was not associated with any elevated conduct problems trajectory. *Unweighted* analyses, which showed the same pattern of associations albeit with slightly weaker effects, are also provided in Table 3 for comparison purposes. We re-ran these analyses additionally adjusting for child IQ, which is a well-established risk factor for conduct problems (Murray & Farrington, 2010). The results were identical when comparing the low conduct problems trajectory to the elevated conduct problem trajectories, confirming the significant associations observed for the early-onset and adolescence-onset trajectories. However, it should be noted that adding IQ to the model reduced the sample size (*N*=2586), which resulted in small cell sizes and consequently very large confidence intervals. Thus, these findings need to be interpreted with extreme caution and require replication. See Appendix 6 for full details.

[Insert Table 3 here]

Exploratory analyses assessing abuse subtypes

Weighted analyses are presented in Table 4 (*unweighted* results were similar and are available on request), with those not exposed to the respective abuse subtype serving as the reference group in each case. In contrast to the pattern of effects observed for the any abuse category, physical and psychological abuse showed strong effects across all three developmental periods studied. More precisely, childhood-only, adolescence-only, and 'persistent' physical abuse was associated with a 4- to 8-fold increase in the odds of being in the early-onset persistent and adolescence-onset classes compared to the low conduct problems class. Similarly, childhood-only, adolescence-only, and 'persistent' psychological abuse was associated with a 5- to 11-fold increase in the odds of being in the early-onset persistent and adolescence-onset classes versus the low conduct problems class (although the association between childhood-only psychological abuse and adolescence-onset trajectory membership was not significant). Similar to the findings for any abuse, there was no evidence that exposure to physical or psychological

abuse across childhood and/or adolescence differentiated between the early-onset persistent and adolescence-onset conduct problem trajectories, or was associated with the childhood-limited conduct problem trajectory. For sexual abuse, the early-onset persistent and adolescence-onset classes showed cell counts of less than 5 for some developmental periods. Therefore, meaningful analyses of associations between sexual abuse and conduct problem classes could not be performed.

[Insert Table 4 here]

Discussion

Using data from a prospective longitudinal study with a large, population-based sample, we identified developmental trajectories of conduct problems from ages 4-17 years, and investigated links between abuse experienced at different times during development and the derived conduct problem trajectories. In contrast to previous research using developmental trajectories of conduct problems that focused on abuse experienced during childhood (Aguilar et al., 2000; Moore & Mezuk, 2014; Odgers et al., 2008; Raine et al., 2005), we used measures covering both childhood and adolescence, which enabled us to explore the impact of abuse timing and persistence. We found that abuse exposure was associated with substantially greater odds of being in the early-onset persistent and adolescence-onset conduct problem classes, particularly when it was present across both childhood and adolescence. We did not find stronger associations between child abuse and membership of the early-onset persistent compared to the adolescence-onset class, which is in contrast to some previous findings (Aguilar et al., 2000; Moore & Mezuk, 2014; Raine et al., 2005). However, it has to be noted that the adolescence-onset class showed slightly raised conduct problems already in childhood, a pattern that has also been observed in prior modeling of conduct problem trajectories (Barker

& Maughan, 2009; Odgers et al., 2008; Raine et al., 2005). We also did not replicate previous findings showing an association between abuse exposure and increased odds of being in the childhood-limited class (compared to the low conduct problems class) (Odgers et al., 2008; Raine et al., 2005). Overall, our findings suggest that conduct problems with an onset in adolescence show similar associations with abuse to conduct problems that emerge in childhood and persist, with any differences between these trajectories being quantitative (i.e., implying common risk factors) rather than qualitative (i.e., distinct risk factors) in nature.

We extended previously published conduct problem trajectories from ages 4-13 years up to age 17 years in a large UK birth cohort (Barker & Maughan, 2009). Using a full information maximum likelihood estimator and the updated 4-band categorization of the SDQ conduct problems subscale, we were able to increase the sample size (N = 10648) and capture more variability in conduct problems, compared to the sample size previously used to estimate developmental trajectories (N = 7218), which also used a dichotomous approach, classifying individuals as either 'high risk' or 'not high risk' in terms of conduct problems (Barker & Maughan, 2009). This has the potential to enable other researchers to examine associations between other environmental or genetic risk factors and conduct problem trajectories covering both childhood and adolescence. Furthermore, the current study brings together two areas of developmental psychopathology, namely: (i) studies using conduct problem trajectories, which, however, measured child abuse exclusively during childhood, rather than in adolescence or in both developmental periods (Aguilar et al., 2000; Moore & Mezuk, 2014; Odgers et al., 2008; Raine et al., 2005); and (ii) studies examining the impact of timing of child abuse, which have been limited to adolescent and adult antisocial behavior, rather than developmental trajectories (Ireland et al., 2002; Mersky et al., 2012; Thornberry et al., 2010; Thornberry et al., 2001).

In line with official UK government statistics from 2020 on child abuse in England and Wales (Office of National Statistics, 2020, March), we found that one in five participants (19.6%) reported at least one form of child abuse (i.e., 'any abuse'). Prevalence rates for specific types of abuse were also broadly comparable with official statistics, ranging from 8-11%. The current study was limited to child abuse, rather than child neglect. Thus, comparisons with official statistics on the prevalence of neglect are not possible.

The current study builds on previous research by examining timing of exposure to child abuse in relation to developmental trajectories of conduct problems. Importantly, our findings support the hypothesis that persistent abuse has a more detrimental effect than time-limited abuse (Evans et al., 2013). Thus, in line with the cumulative risk hypothesis, abuse exposure in both childhood and adolescence was associated with greater odds of being in the early-onset persistent and adolescence-onset classes, with effect sizes twice the size of those observed for childhood-only abuse. In addition, different patterns were observed for childhood-only versus adolescence-only exposure when using the aggregate measure of abuse ('any abuse'). Specifically, whereas childhood-only abuse was associated with increased odds of being in the early-onset persistent and adolescence-onset conduct problem classes, adolescence-only abuse was not associated with membership of any of the elevated conduct problem trajectories. The latter observation runs counter to previous research suggesting that adolescence-only abuse has more detrimental effects than childhood-only abuse (Ireland et al., 2002; Thornberry et al., 2010; Thornberry et al., 2001). On the contrary, the current results indicate that abuse occurring in childhood may be more influential than that occurring in adolescence (at least in terms of increasing risk for conduct problems), suggesting there may be a sensitive period in which abuse is particularly likely to lead to persistent conduct problems. Alternatively, it may be that abuse occurring specifically in adolescence, versus in childhood or in both developmental periods, is experienced differently by the individual or arises for different reasons, given that

significant conflict in the parent-child relationship is relatively common (and possibly normative) during adolescence (Laursen, Coy, & Collins, 1998).

These findings for childhood-only versus adolescence-only abuse were not replicated in an exploratory analysis that examined the impacts of physical and psychological abuse separately. More precisely, adolescence-only abuse also emerged as predictive of these trajectories, alongside the positive associations already identified for childhood-only abuse. In line with many studies published in this field, small cell sizes mean that caution is essential in interpreting these findings. They also prevented us from investigating the specific impact of sexual abuse, which has been consistently linked to adolescent conduct problems (Braga et al., 2017; Wilson et al., 2009), because of particularly low frequencies in our sample when split across conduct problem classes. As shown in the correlation matrix (see Appendix 1), physical and psychological abuse were highly correlated, whereas correlations between these forms of abuse and sexual abuse were much weaker. This may indicate shared risk environments in which both physical and psychological abuse occur, which may explain the similar pattern of effects for these abuse subtypes. Although sexual abuse was most commonly reported in adolescence, it might be less likely to result in conduct problems if experienced within this developmental period. Consequently, the inclusion of sexual abuse in our aggregate measure of abuse may have suppressed associations with adolescence-only abuse. Future studies with larger and/or high-risk samples with a higher prevalence of sexual abuse are needed to further investigate the association between sexual abuse and conduct problem trajectories.

In contrast to the effects observed for the early-onset persistent and adolescence-onset classes, we did not find any evidence of associations between abuse and childhood-limited conduct problems, which contradicts some previous findings in this area (Odgers et al., 2008; Raine et al., 2005). These studies, however, also included child neglect, a form of child maltreatment not investigated in the current study, which may have influenced associations.

Alternatively, individual risk factors, such as neurodevelopmental problems, may be particularly pronounced in these individuals (Fairchild et al., 2013), and, thus, more relevant in the etiology of this trajectory compared to environmental risk factors such as child abuse. For example, Raine et al. (2005) found a range of neurocognitive impairments related to intelligence and memory especially in children with childhood-limited conduct problems compared to those on the low trajectory (Raine et al., 2005).

The relationship between child abuse and the early-onset persistent and adolescence-onset conduct problem trajectories may be explained with recourse to social information processing theory (Crick & Dodge, 1994). Children with aggressive behavior show biases in social information processing (e.g., hostile attributional biases) (Orobio de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002). These biases have been shown to mediate the relationship between harsh and abusive parenting and conduct problems (Dodge, Pettit, Bates, & Valente, 1995; Weiss, Dodge, Bates, & Pettit, 1992). Children may internalize their parents' aggressive and threatening behaviors, and, as a result, rely on these aggressive schemata in future social interactions. Equipped with this limited repertoire of behaviors, children may struggle to generate non-aggressive responses to situations of conflict and may also evaluate physically and verbally aggressive responses more positively than their non-abused peers (Crick & Dodge, 1994). Furthermore, there are well-established bidirectional effects in the relationship between harsh and abusive parenting and child conduct problems (Pinquart, 2017). Consequently, children showing conduct problems may become ensnared in coercive exchanges with their parents (Patterson, 1982). By contrast, abuse experienced in adolescence might be less likely to be internalized and viewed as a behavior to emulate, which may explain the null findings for adolescence-only abuse when using the aggregate measure of abuse. Alternatively, adolescence-only abuse may be more relevant for other types of antisocial behavior, which are not assessed by the SDQ conduct problems subscale, and other forms of

psychopathology. For example, Mersky et al. (2012) found that adolescence-only abuse was linked to juvenile offending (i.e., arrests, court petitions, and various types of offenses) and particularly drug-related convictions in adulthood (Mersky et al., 2012). Finally, the non-significant associations for adolescence-only abuse may partly reflect the relative rarity of abuse only occurring in this developmental period.

Limitations

First, the findings should be interpreted in the context of limitations relating to our measures of abuse in the current study. A highly varied set of experiences could lead to an individual being classified as having experienced child abuse. This problem is inherent in any measure that attempts to capture something as complex as exposure to adversity in a scale score, but is compounded in cohort studies where low prevalence of child abuse necessitates the use of categorical variables. In addition, child abuse was assessed using retrospective self-report at age 22 years, which may have been subject to recall bias. Prospective and retrospective measures of child abuse often show poor agreement, representing two constructs with limited overlap (Baldwin, Reuben, Newbury, & Danese, 2019). However, despite this discrepancy, false positives of retrospective reports of child abuse in adulthood have been shown to be rare (Hardt & Rutter, 2004). Furthermore, most instances of child abuse are not reported to authorities (MacMillan, Jamieson, & Walsh, 2003), which compromises the representativeness of officially documented child abuse cases – the main alternative to self-report. Therefore, while retrospective self-report measures have limitations, it is difficult to develop feasible and ethically acceptable alternatives, particularly in large prospective cohort studies. We further used a brief measure of child abuse which has not been fully validated, although the included items are extremely similar to those included in well-established measures (e.g., Childhood Trauma Questionnaire; 58) . Future studies need to replicate our findings using a larger number of items from a measure with established psychometric properties. Finally, our measure of

'persistent' abuse, defined as exposure to abuse occurring in both childhood and adolescence, may have captured two isolated instances of abuse, rather than a repeated and ongoing pattern of abuse that spans childhood and adolescence. Unfortunately, the available data did not permit a more detailed approach but this issue merits investigation in future research. Second, as already noted, despite this study deriving from a large, representative birth cohort, frequencies of some forms of abuse were low across the different conduct problem trajectories. The findings relating to physical and psychological abuse particularly require replication, and we were not able to examine sexual abuse as a separate category due to very small cell counts for some classes. Third, the present study suffered from high attrition rates, possibly due to the high assessment burden and/or participants' unwillingness to answer questions about highly intrusive experiences, potentially resulting in systematic differences between the sample included in the analyses estimating conduct problem trajectories (N = 10648) and the sample for whom retrospective data on child abuse were available (n = 3172). More precisely, those with missing data were more likely to be male and in the early-onset persistent and childhood-limited conduct problem classes. This may have led to an underestimation of the effects of abuse and compromised the generalizability of our findings, particularly given that conduct problem trajectories where associated with missingness. However, we employed IPW to minimize the impact of this bias by allocating sample weights to complete cases (Seaman & White, 2013), and the findings of weighted and unweighted analyses yielded almost identical results. Fourth, the SDQ conduct problems subscale showed modest internal consistency, similar to previous research modeling developmental trajectories of behavior and emotional problems in the ALSPAC sample (Barker, Oliver, & Maughan, 2010). Although, the SDQ is an extremely widely used measure, our findings require replication, using a measure of conduct problems with better psychometric properties. Similarly, the ESYTC, which we used to validate the derived conduct problem trajectories, showed poor reliability. Collectively, these

limitations highlight the need for more reliable measures of behavior problems in young people. Fifth, relying on parent-reported conduct problems in adolescence may have underestimated the level of behavioral problems, as parents may be unaware of their child's antisocial behavior in this developmental phase (De Los Reyes et al., 2015). However, the use of different informants for conduct problems versus abuse experiences minimizes potential for inflation of effects by informant bias. Moreover, considering the age range of our sample from ages 4-17 years, neither parent- nor self-report would have perfectly captured conduct problems occurring in both childhood and adolescence. Crucially, we were able to validate our conduct problem trajectories using self-reported measures of antisocial behavior during adolescence, which showed higher rates of antisocial behavior in the elevated conduct problems classes. This information, which is not typically available for studies of this type, supports the validity of our derived trajectories. Nevertheless, an important area of future research will be to compare associations between child abuse and developmental trajectories of conduct problems based on self- versus parent-report. Finally, the temporal overlap between our derived conduct problem trajectories and measures of child abuse precludes causal inferences. Thus, child abuse may be a risk factor for conduct problems or conduct problems may elicit more harsh and abusive parenting, or both factors may interact with each other in a transactional way.

Conclusions

Our findings demonstrate a particularly strong association between 'persistent' abuse – i.e., that occurring in both childhood and adolescence – and the early-onset persistent and adolescence-onset conduct problem trajectories. The findings are consistent with the view that the differences between the early-onset persistent and adolescence-onset conduct problem trajectories are more quantitative than qualitative in nature. In other words, common risk factors are involved in both subtypes but to different degrees, rather than early-onset persistent conduct problems stemming from entirely different risk factors compared to adolescence-onset

conduct problems. For example, levels of exposure to environmental risk factors, such as child abuse, may be more similar than previously thought, as direct comparisons between these conduct problems trajectories revealed no significant differences in abuse exposure. Consequently, child services may want to screen for a history of child abuse and provide additional support to young people showing adolescence-onset conduct problems, as these may not be as developmentally normative as previously suggested (Moffitt, 1993). Thus, psychosocial interventions focusing on ameliorating adverse family environments may be also effective in reducing adolescence-onset conduct problems. Furthermore, studies of the effectiveness of interventions aimed at targeting harsh and abusive parenting should assess outcomes in adolescence, as well as outcomes that are concurrent with the delivery of the intervention – as the full benefits may not be apparent until many years later. Our findings also demonstrate the importance of adopting measures covering both childhood and adolescence when investigating the timing and persistence of child abuse, as harsh and abusive parenting may persist up to emerging adulthood.

List of abbreviations

ALSPAC: Avon Longitudinal Study of Parents and Children; BIC: Bayesian Information Criterion; BLRT: Bootstrapped Likelihood Ratio Test; ESYTC: Edinburgh Study of Youth Transitions and Crime; IPW: Inverse probability weighting; LCGA: Latent class growth analysis; LMR-LRT: Lo-Mendell-Rubin Likelihood Ratio Test; SDQ: Strengths and Difficulties Questionnaire; SSABIC: Sample size adjusted Bayesian Information Criterion

Declarations

Ethics approval and consent to participate

Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. Informed written consent for the use of data

collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time.

Consent for publication

Not applicable

Availability of data and materials

The study website contains details of all the data that are available through a fully searchable data dictionary and variable search tool (http://www.bristol.ac.uk/alspac/researchers/our-data/).

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

AB, SH, GF, and AF contributed to the conceptualisation of the study. AB and GH performed the analysis. AB, SH, GF, and GH wrote the manuscript. All authors revised, edited, and approved the manuscript.

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Table 1 Model fit statistics for cubic latent class growth analysis one to six class solutions

Fit statistics	**1 class**	**2 classes**	**3 classes**	**4 classes**	**5 classes**	**6 classes**
LL (No. of Para.)	-32500.791 (6)	-29037.350 (11)	-28581.588 (16)	-28484.751 (21)	-28388.525 (26)	-28354.757 (31)
BIC	65057.220	58176.705	57311.546	57164.237	57018.152	56996.981
SSABIC	65038.153	58141.749	57260.700	57097.502	56935.527	56898.467
Entropy		0.763	0.725	0.709	0.688	0.697
LMR-LRT *p*-value		0.0000	0.0000	0.0403	0.0016	0.1998
Adj. LMR-LRT *p*-value		0.0000	0.0000	0.0426	0.0018	0.2061
BLRT *p*-value		0.0000	0.0000	0.0000	0.0000	0.0000
Group size (%)[a]						
C1	10648	8598 (80.7%)	447 (4.2%)	480 (4.5%)	135 (1.3%)	1566 (14.7%)
C2		2050 (19.3%)	2343 (22.0%)	1643 (15.4%)	465 (4.3%)	7691 (72.2%)
C3			7858 (73.8%)	8019 (75.3%)	1616 (15.2%)	84 (0.8%)
C4				506 (4.8%)	7720 (72.5%)	167 (1.6%)
C5					712 (6.7%)	597 (5.6%)
C6						543 (5.1%)

Note. Based on *N* = 10648. LL = Log-Likelihood value; No. of Para. = Number of estimated (freed) parameters; BIC = Bayesian Information Criterion; SSABIC = Sample Size Adjusted BIC; LMR-LRT = Lo-Mendell-Rubin Likelihood Ratio Test; Adj. LMR-LRT = Adjusted LMRT-LRT; BLRT = Bootstrapped Likelihood Ratio Test; C = Class. [a] Based on most likely latent class membership.

Figure 1 Predicted category proportions for each class in the conduct problems trajectory model (*N* = 10648)

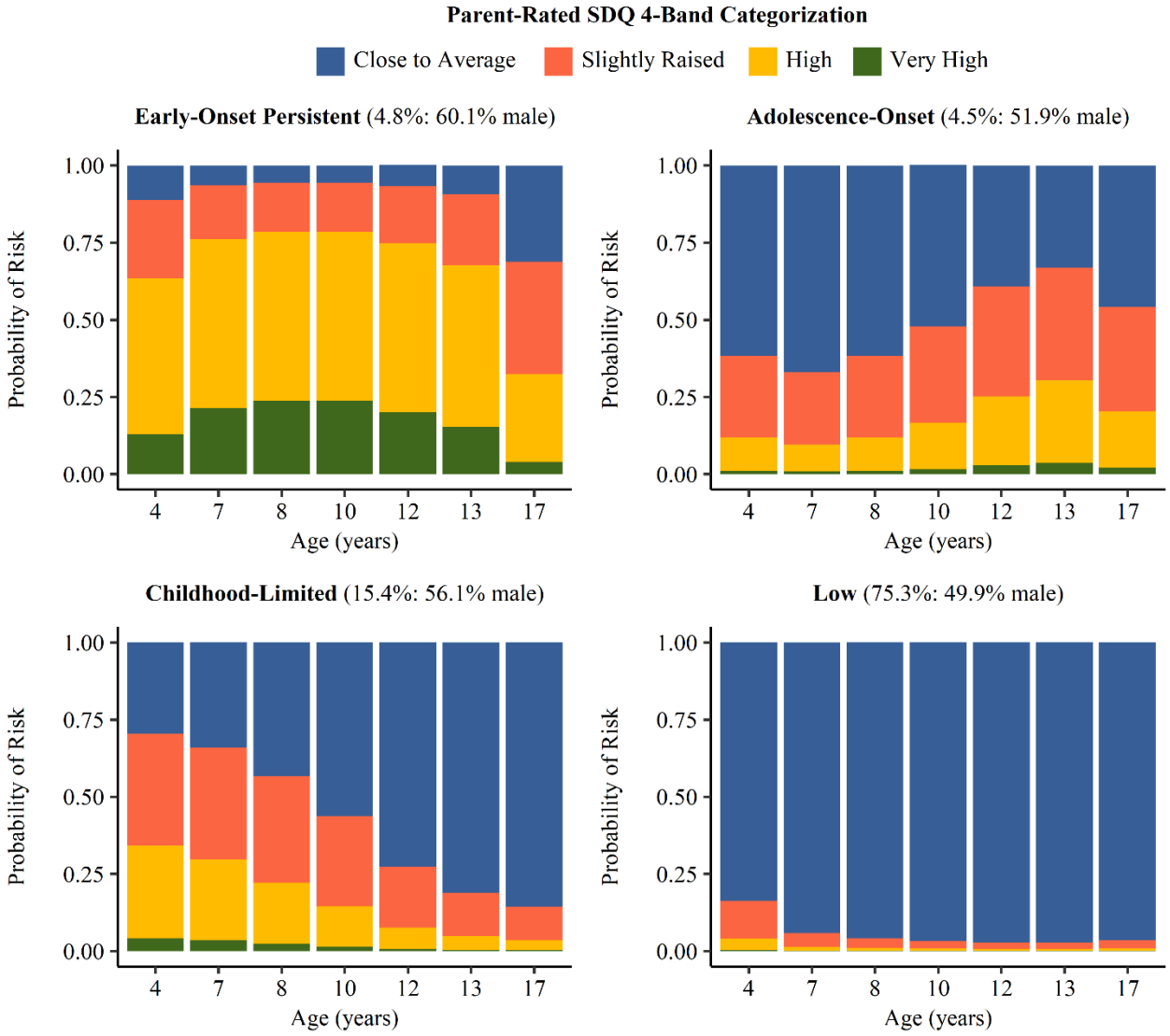


Table 2 Frequencies of abuse exposure in each of the developmental phases for the four conduct problem trajectories

	Low *n* (%)	**CL** *n* (%)	**AO** *n* (%)	**EOP** *n* (%)
Any abuse (*N* = 3172)				
Childhood-only	118 (4.7)	33 (7.7)	13 (9.0)	12 (10.9)
Adolescence-only	140 (5.6)	26 (6.1)	11 (7.6)	8 (7.3)
'Persistent'	161 (6.5)	43 (10.0)	30 (20.8)	25 (22.7)
Physical abuse (*N* = 3275)				
Childhood-only	88 (3.4)	21 (4.7)	11 (7.5)	11 (9.5)
Adolescence-only	55 (2.1)	10 (2.2)	7 (4.8)	9 (7.8)
'Persistent'	95 (3.7)	28 (6.2)	20 (13.6)	16 (13.8)
Psychological abuse (*N* = 3295)				
Childhood-only	50 (1.9)	18 (4.0)	7 (4.7)	10 (8.6)
Adolescence-only	48 (1.9)	12 (2.7)	8 (5.3)	7 (6.0)
'Persistent'	80 (3.1)	20 (4.5)	17 (11.3)	16 (13.8)
Sexual abuse (*N* = 3279)				
Childhood-only	49 (1.9)	14 (3.1)	5 (3.4)	< 5
Adolescence-only	111 (4.3)	24 (5.4)	8 (5.4)	5 (4.5)
'Persistent'	29 (1.1)	10 (2.2)	< 5	< 5

Note. Sample sizes based on complete data. Cells with a count of < 5 were not included in subsequent analyses. Childhood-only = Before age 11 years; Adolescence-only = Between ages 11-17 years; 'Persistent' = Before age 11 years AND between ages 11-17 years. AO = Adolescence-onset; CL = Childhood-limited; EOP = Early-onset persistent.

Table 3 Associations between developmental timing of abuse (collapsing across abuse subtypes to form an 'any abuse' category) and conduct problems trajectory membership

	Timing of abuse					
	Childhood-only		**Adolescence-only**		**'Persistent'**	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Weighted						
Low[a]						
CL	1.44	0.49-4.18	0.70	0.21-2.35	1.24	0.41-3.75
AO	**3.98**	**1.40-11.34**	2.72	0.97-7.57	**7.51**	**3.42-16.48**
EOP	**6.17**	**2.39-15.94**	2.89	0.81-10.32	**9.80**	**4.45-21.58**
CL[a]						
AO	2.77	0.48-15.98	3.87	0.62-24.28	**6.04**	**1.30-28.15**
EOP	4.29	0.97-18.97	4.12	0.64-26.60	**7.89**	**1.89-32.98**
AO[a]						
EOP	1.55	0.39-6.13	1.06	0.20-5.57	1.31	0.42-4.02
Unweighted						
Low[a]						
CL	1.65	0.76-3.60	0.92	0.34-2.44	0.98	0.33-2.97
AO	**3.24**	**1.27-8.26**	2.50	0.97-6.45	**6.48**	**3.31-12.68**
EOP	**3.94**	**1.71-9.02**	1.99	0.67-5.86	**6.75**	**3.49-13.06**
CL[a]						
AO	1.96	0.50-7.69	2.73	0.56-13.34	**6.59**	**1.53-28.34**
EOP	2.38	0.75-7.56	2.17	0.49-9.66	**6.87**	**1.77-26.72**
AO[a]						
EOP	1.21	0.35-4.17	0.79	0.18-3.49	1.04	0.42-2.61

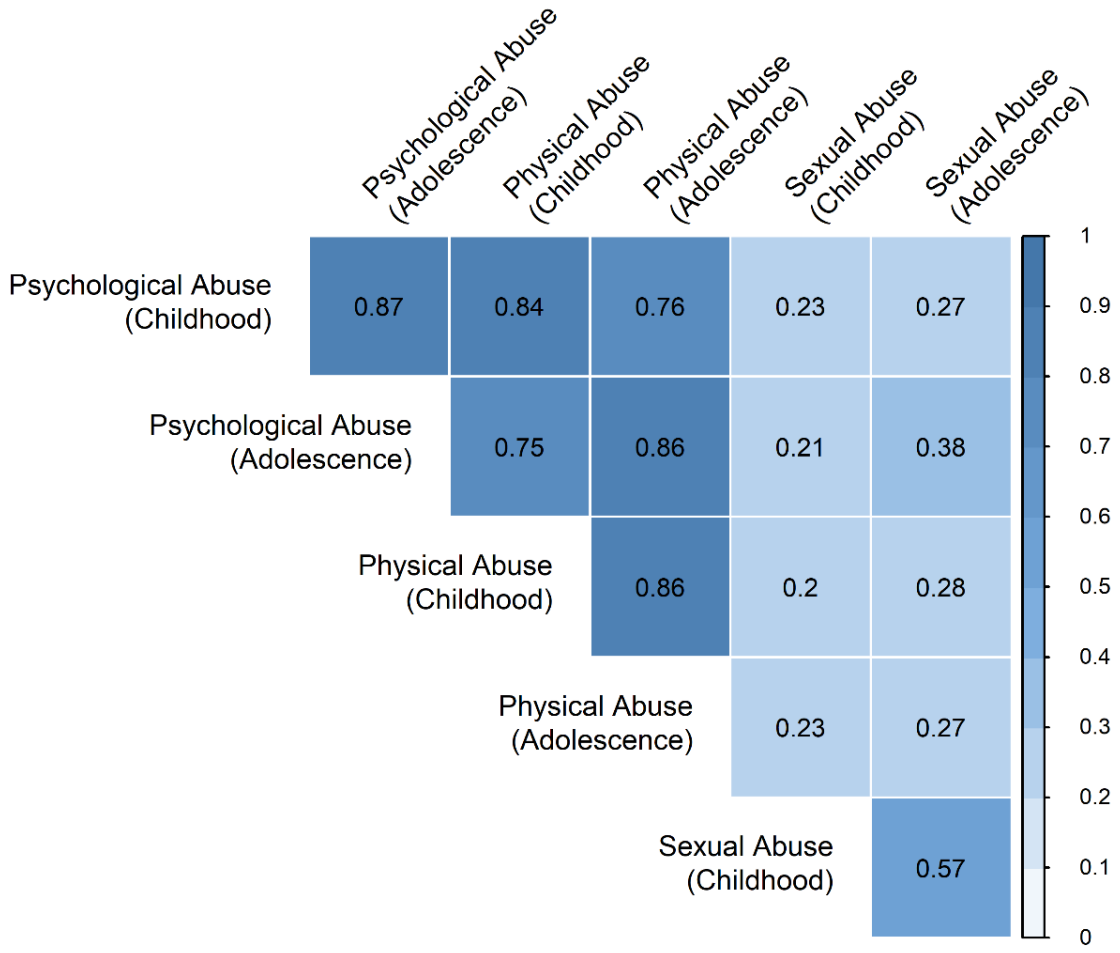
Note. Based on *N* = 3192. Inverse probability weighting was used to allocate sampling weights to complete cases in weighted analysis. In unweighted analysis, each case carries the same weight. All pairwise comparisons are adjusted for child sex, housing tenure, maternal severe depression, maternal smoking, and maternal educational level. Bold values indicate statistically significant associations. [a] Reference group. Key: Childhood-only = Before age 11 years; Adolescence-only = Between ages 11-17 years; 'Persistent' = Before age 11 years AND between ages 11-17 years; AO = Adolescence-onset; CL = Childhood-limited; EOP = Early-onset persistent; OR = Multinomial odds ratio.

Table 4 *Weighted* associations between physical (versus no physical) and psychological (versus no psychological) abuse and conduct problems trajectory membership

	Timing of abuse subtypes					
	Childhood-only		**Adolescence-only**		**'Persistent'**	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Physical abuse (*N* = 3275)						
Low[a]						
CL	0.93	0.21-4.14	N/A	N/A	1.73	0.57-5.20
AO	**4.50**	**1.63-12.43**	**3.86**	**1.43-10.38**	**7.76**	**3.20-18.82**
EOP	**4.99**	**1.91-13.03**	**6.00**	**2.29-15.74**	**7.60**	**3.24-17.82**
CL[a]						
AO	4.85	0.63-37.24	N/A	N/A	4.49	0.97-20.73
EOP	5.37	0.93-31.10	N/A	N/A	**4.39**	**1.04-18.59**
AO[a]						
EOP	1.11	0.29-4.27	1.56	0.37-6.46	0.98	0.30-3.17
Psychological abuse (*N* = 3295)						
Low[a]						
CL	1.52	0.28-8.22	1.58	0.32-7.79	0.80	0.11-5.66
AO	4.16	0.87-19.85	**4.96**	**1.59-15.45**	**5.48**	**2.43-12.33**
EOP	**10.48**	**4.04-27.15**	**10.83**	**3.45-34.01**	**11.22**	**4.97-25.36**
CL[a]						
AO	2.74	0.18-42.32	3.14	0.39-25.09	6.84	0.72-64.79
EOP	**6.90**	**1.06-44.78**	6.86	0.83-56.99	N/A	N/A
AO[a]						
EOP	2.52	0.44-14.29	2.18	0.47-10.15	2.05	0.70-6.02

Note. Inverse probability weighting was used to allocate sampling weights to complete cases. N/A = Not available due to fixed parameters. All pairwise comparisons are adjusted for child sex, housing tenure, maternal severe depression, maternal smoking, and maternal educational level. Bold values indicate statistically significant associations. [a] Reference group. Key: Childhood-only = Before age 11 years; Adolescence-only = Between ages 11-17 years; 'Persistent' = Before age 11 years AND between ages 11-17 years; AO = Adolescence-onset; CL = Childhood-limited; EOP = Early-onset persistent; OR = Multinomial odds ratio.

Appendix 1 Tetrachoric correlation matrix of physical, psychological, and sexual abuse occurring in either childhood or adolescence



Note. Based on $N = 3497$. All $ps < .001$. Darker colors represent larger correlation coefficients.

Appendix 2 Associations between indicators used to derive the inverse probability weights and inclusion in the analysis sample of 'any abuse'

Sociodemographic and parental factors in pregnancy	**In analysis sample** % (*n*)	**Not in analysis sample** % (*n*)	**OR (95% CI)**	***p*-value**
	(*N* = 3172)	(*N* = 7476)		
Child sex				
Female	64.1% (2034)	42.0% (3140)	0.41 (0.37-0.44)	< .001
Maternal education				
No high school	16.5% (522)	28.7% (2143)	Reference	
High school	33.7% (1070)	34.2% (2556)	0.58 (0.52-0.66)	< .001
Beyond high school	49.8% (1580)	37.1% (2777)	0.43 (0.38-0.48)	
Maternal smoking				
Yes	15.1% (479)	24.2% (1807)	1.79 (1.60-2.01)	< .001
Housing tenure				
Other[a]	13.8% (438)	23.9% (1787)	1.96 (1.75-2.20)	< .001

Note. [a] Reference is 'mortgaged/owned'. Outcome coded as 0 'in analysis sample' and 1 'not in analysis sample'.

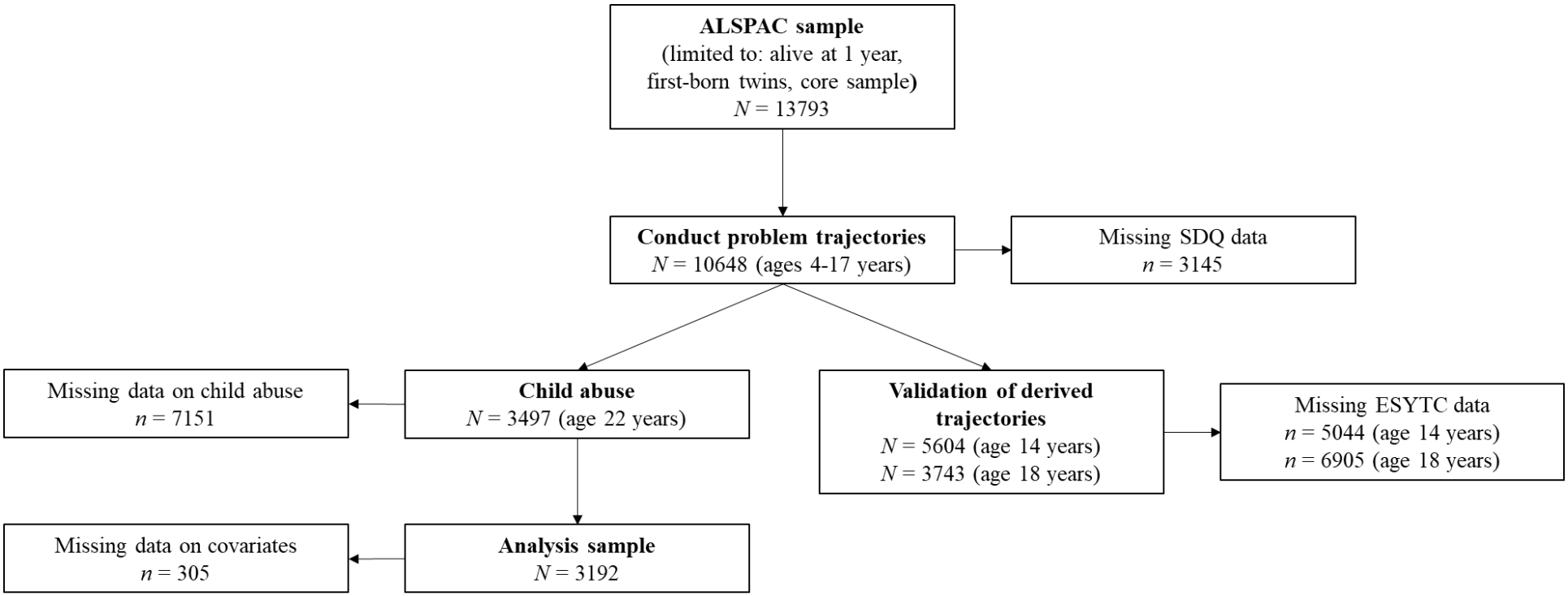
Measures assessed in pregnancy (child sex, maternal education, maternal smoking, and housing tenure) that were independently predictive of child abuse exposure, conduct problem trajectories, or missingness in the analysis sample were selected as inverse probability weighting (IPW) indicators. Due to slightly varying attrition rates across child abuse variables, we looked at missingness in each group separately, resulting in four missingness models (i.e., different weights for any, physical, psychological, and sexual abuse). Missing data on IPW indicators were singly imputed as the mode value (all indicators had < 7% of values missing, except maternal education with 11.1% of values missing). The Hosmer-Lemeshow test was used to assess model fit of the missingness models, with results showing no indication of poor fit (*p*-values ranging between .19 and .36). Weights ranged from 1.9 to 13.3. Due to almost identical values across missingness models, only associations between IPW indicators and inclusion in the analysis sample for 'any abuse' are shown in Table 1.

Appendix 3 Descriptive statistics and group comparisons between participants with class membership information (N = 10648) and with and without complete data on measures of child abuse and covariates (N = 3172)

	In analysis sample (N = 3172)		**Not in analysis sample** (N = 7476)		**Group comparison**	**Effect size**
	Mean (SD) or **%**	***N***	**Mean (SD)** or **%**	***N***	***t*(df)** or **χ^2(df)**	***r*** or **(95% CI)**
Conduct problems[a]						
Age 4	0.37 (0.27)	2919	0.40 (0.29)	6355	$t(5920.2) = 5.91$, $p < .001$	0.08
Age 7	0.29 (0.28)	2802	0.33 (0.29)	5287	$t(6020.9) = 6.33$, $p < .001$	0.08
Age 8	0.27 (0.27)	2721	0.31 (0.30)	4637	$t(6026.5) = 6.53$, $p < .001$	0.08
Age 10	0.22 (0.26)	2701	0.26 (0.28)	4531	$t(6051.1) = 6.04$, $p < .001$	0.08
Age 12	0.20 (0.25)	2631	0.25 (0.29)	3996	$t(6125.7) = 7.27$, $p < .001$	0.09
Age 13	0.21 (0.25)	2608	0.26 (0.28)	3799	$t(6006.2) = 6.69$, $p < .001$	0.09
Age 17	0.16 (0.23)	2378	0.21 (0.26)	2687	$t(5061.9) = 6.54$, $p < .001$	0.09
Class proportions[b]						
EOP	3.5	110	5.3	396	$\chi^2(1) = 16.46$, $p < .001$	1.56 (1.25 – 1.95)
AO	4.5	144	4.5	336	$\chi^2(1) = 0.01$, $p = .92$	0.99 (0.81 – 1.22)
CL	13.5	428	16.3	1215	$\chi^2(1) = 12.99$, $p < .01$	1.24 (1.10 – 1.40)
Low	78.5	2490	74.0	5529	$\chi^2(1) = 24.72$, $p < .001$	0.78 (0.70 – 0.86)
Male	35.9	1138	58.0	4336	$\chi^2(1) = 436.32$, $p < .001$	2.47 (2.26 – 2.69)

Note. [a] Assessed with the Strengths and Difficulties Questionnaire conduct problems subscale. [b] Based on most likely latent class membership. SD = Standard deviation; df = Degrees of freedom; r = Correlation coefficient; OR = Odds ratio; CI = Confidence interval; EOP = Early-onset persistent; AO = Adolescence-onset; CL = Childhood-limited.

Appendix 4 Retention flow chart across measures/analyses



Note. SDQ = Strengths and Difficulties Questionnaire; ESYTC = Edinburgh Study of Youth Transition and Crime.

Appendix 5 Validation of conduct problem classes against an independent measure of self-reported antisocial behavior

	ESYTC			
	Age 14 (*N* = 5604)		**Age 18** (*N* = 3743)	
	OR	**95% CI**	**OR**	**95% CI**
Low[a]				
CL	**1.55**	**1.19-2.01**	**1.58**	**1.03-2.44**
AO	**2.85**	**1.87-4.33**	**1.85**	**1.00-3.42**
EOP	**2.56**	**1.77-3.69**	**1.89**	**1.11-3.21**
CL[a]				
AO	**1.65**	**1.03-2.66**	1.17	0.51-2.67
EOP	**1.84**	**1.07-3.16**	1.19	0.59-2.43

Note. Bold values indicate statistically significant associations. [a] Reference group. Key: ESYTC = Edinburgh Study of Youth Transitions and Crime; AO = Adolescence-onset; CI = Confidence interval; CL = Childhood-limited; EOP = Early-onset persistent; OR = Multinomial odds ratio.

Appendix 6 Associations between developmental timing of abuse (collapsing across abuse subtypes to form an 'any abuse' category) and conduct problems trajectory membership, additionally adjusting for child IQ

	Timing of abuse					
	Childhood-only		**Adolescence-only**		**'Persistent'**	
	OR	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Weighted						
Low[a]						
CL	0.85	0.16-4.47	0.95	0.34-2.65	0.86	0.09-8.18
AO	**5.03**	**1.63-15.56**	2.82	0.86-9.27	**12.06**	**4.43-32.84**
EOP	**6.81**	**2.68-17.30**	2.58	0.58-11.54	**9.50**	**3.89-23.17**
CL[a]						
AO	5.93	0.55-64.41	2.98	0.52-16.99	14.10	0.77-258.4
EOP	**8.02**	**1.14-56.38**	2.73	0.39-19.05	11.10	0.93-133.0
AO[a]						
EOP	1.35	0.32-5.65	0.91	0.13-6.65	0.79	0.20-3.08
Unweighted						
Low[a]						
CL	1.01	0.30-3.43	0.89	0.32-2.47	0.68	0.08-5.79
AO	**4.46**	**1.64-12.09**	**2.84**	0.97-8.27	**9.74**	**4.32-21.96**
EOP	**4.44**	**1.89-10.44**	**2.19**	0.63-7.58	**6.71**	**3.15-14.29**
CL[a]						
AO	4.43	0.70-28.11	3.18	0.60-16.76	**14.27**	**1.03-196.9**
EOP	4.41	0.97-20.15	2.46	0.46-13.04	9.83	0.92-104.9
AO[a]						
EOP	1.00	0.27-3.70	0.77	0.14-4.25	0.69	0.24-2.00

Note. Based on $N = 2586$. Inverse probability weighting was used to allocate sampling weights to complete cases in weighted analysis. In unweighted analysis, each case carries the same weight. All pairwise comparisons are adjusted for child sex, IQ, housing tenure, maternal severe depression, maternal smoking, and maternal educational level. Bold values indicate statistically significant associations. [a] Reference group. Key: Childhood-only = Before age 11 years; Adolescence-only = Between ages 11-17 years; 'Persistent' = Before age 11 years AND between ages 11-17 years; AO = Adolescence-onset; CL = Childhood-limited; EOP = Early-onset persistent; OR = Multinomial odds ratio.

Chapter 5

The protective effect of neighbourhood collective efficacy on family violence and youth antisocial behaviour in two South Korean prospective longitudinal cohorts

Manuscript is published in *Research on Child and Adolescent Psychopathology*

Chapter rationale

Neighbourhood collective efficacy, a concept including informal social control and social cohesion, has been proposed to exert protective effects against child externalising problems. However, little is known about its proximal mechanisms, such as the family environment, and the generalisability of previous findings, which are mostly based on studies from Western countries. Therefore, this fourth study will examine the interplay between neighbourhood collective efficacy, family violence, and youth antisocial behaviour in two South Korean cohorts including school-aged children.

This declaration concerns the article entitled:

The protective effect of neighbourhood collective efficacy on family violence and youth antisocial behaviour in two South Korean prospective longitudinal cohorts

Publication status (tick one)

Draft manuscript		Submitted		In review		Accepted		Published	x

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Candidate's contribution to the paper (provide details, and also indicate as a percentage)	The candidate contributed to / considerably contributed to / predominantly executed the… **Formulation of ideas:** Andreas Bauer predominantly contributed to the formulation of ideas for the current study (85%). **Design of methodology:** The data used in the current study was part of a larger data set. Andreas Bauer contributed considerably to the design of the methodology in this current chapter (80%). **Experimental work:** Andreas Bauer predominantly executed the analysis and interpretation of results for this study (90%). **Presentation of data in journal format:** The presentation of the data into journal format was predominantly carried out by Andreas Bauer (95%).
Statement from Candidate	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature.
Signed	[redacted] **Date** 27.06.2021

The protective effect of neighbourhood collective efficacy on family violence and youth antisocial behaviour in two South Korean prospective longitudinal cohorts

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Abstract

Neighbourhood collective efficacy has been proposed as a protective factor against family violence and youth antisocial behaviour. However, little is known about its impact on parent and child behaviour in non-Western countries. Using data from two population-based prospective cohorts from South Korea, including primary school students aged 10-12 years (*N*=2844) and secondary school students aged 15-17 years (*N*=3449), we examined the interplay between collective efficacy, family violence, and youth antisocial behaviour, and whether effects vary by SES. In a first series of models, in both samples, higher levels of collective efficacy were associated with lower levels of family violence, whereas higher levels of family violence were associated with higher levels of youth antisocial behaviour. There was no direct effect of collective efficacy on youth antisocial behaviour; however, there was an indirect effect via family violence. Although these effects were more pronounced in low SES children, there was no evidence of moderation by SES. In a second series of models, in primary school students, collective efficacy was not associated with youth antisocial behaviour. However, there was a direct effect of collective efficacy on family violence, even after adjusting for youth antisocial behaviour. Again, there was no evidence of moderation by SES. In secondary school students, the pattern of results was less consistent, however, again, suggesting more pronounced effects of collective efficacy in low SES children. The findings suggest that collective efficacy may influence family violence more directly, whereas youth antisocial behaviour may be affected more indirectly through the family environment.

Keywords: neighbourhood collective efficacy; family violence; child abuse; domestic violence; antisocial behaviour; mediation

Introduction

There are well-documented effects of neighbourhood structural characteristics on child and adolescent behavioural outcomes (Leventhal & Brooks-Gunn, 2000). Neighbourhood social processes have been proposed as mechanisms linking structural factors to behaviour problems among children and adolescents (Sampson, Morenoff, & Gannon-Rowley, 2002). Thus, according to the *social disorganisation* theory, neighbourhood structural disadvantage, such as poverty and residential instability, weakens social bonds among residents, which, in turn, impedes collective neighbourhood action directed towards community problems (Shaw & McKay, 1942). As a result, residents in structurally disadvantaged neighbourhoods are less able to monitor and deter youth problem behaviour than residents of neighbourhoods with more favourable structural conditions.

Sampson et al. (1997) extended this work by developing the concept of neighbourhood collective efficacy, a measure of *social organisation*, including informal social control (i.e., the residents' willingness to intervene) and social cohesion (i.e., mutual trust among neighbours). In a landmark study, Sampson et al. (1997) demonstrated that collective efficacy is a key factor in explaining the association between neighbourhood structural factors and community violence. More specifically, collective efficacy largely mediated the associations of concentrated disadvantage and residential instability with violent crime. Furthermore, a meta-analysis identified low collective efficacy as one of the strongest neighbourhood-level predictors of crime (Pratt & Cullen, 2005). Thus, collective efficacy has been proposed as a mechanism through which neighbourhood structural characteristics influence aggressive and antisocial behaviour in young people (Leventhal & Brooks-Gunn, 2000). For example, using data from the Environmental Risk Longitudinal Twin study, a nationally representative cohort from the UK, Odgers et al. (2009) examined the association between neighbourhood collective efficacy and developmental trajectories of antisocial behaviour from ages 5-10 years. In

deprived, but not affluent, neighbourhoods, collective efficacy was negatively associated with children's antisocial behaviour at school entry, even after adjusting for adverse family characteristics, such as family violence.

Similar to aggressive and antisocial behaviour in young people, collective efficacy has been proposed as a mechanism linking neighbourhood structural characteristics to child maltreatment (Coulton, Crampton, Irwin, Spilsbury, & Korbin, 2007; Zielinski & Bradshaw, 2006). Thus, collective efficacy may provide community and social support to families, especially in structurally disadvantaged neighbourhoods, which, in turn, may decrease the use of harsh and abusive parenting strategies (Coulton et al., 2007; Molnar et al., 2016; Zielinski & Bradshaw, 2006). Furthermore, collective efficacy has been shown to be associated with a decrease in domestic violence (Beyer, Wallis, & Hamberger, 2015; Jackson, 2016; Pinchevsky & Wright, 2012; Wright & Benson, 2010), which often co-occurs with child abuse and neglect (Hamby, Finkelhor, Turner, & Ormrod, 2010), indicating the clustering of different forms of family violence.

Although parent characteristics have been proposed as pathways through which neighbourhood effects are transferred to children and adolescents (Leventhal & Brooks-Gunn, 2000; Sampson & Laub, 1993), given the well-documented bidirectional effects between parent and child behaviour (Pinquart, 2017), the reverse may equally apply – child characteristics as pathways through which neighbourhood effects are transferred to parents. For example, while child abuse is considered a key risk factor for aggressive and antisocial behaviour in children (Braga, Gonçalves, Basto-Pereira, & Maia, 2017; Wilson, Stover, & Berkowitz, 2009), child externalising problems have been shown to elicit more harsh and abusive parenting (Pinquart, 2017), indicating a reciprocal relationship. According to this logic, decreases in child externalising symptoms, as a result of, for example, neighbourhood intervention, would be associated with decreases in harsh parenting.

In sum, collectively efficacy has been proposed to exert protective effects on both youth antisocial behaviour and family violence, which, in turn, show bidirectional associations. However, these potential mediating pathways have not been investigated systematically. In the Fragile Families and Child Well-Being Study, a nationally representative US birth cohort, low levels of neighbourhood collective efficacy and high levels of corporal punishment were independently associated with externalising problems among children aged 3-5 years (Ma, 2016; Ma & Grogan-Kaylor, 2017). However, corporal punishment did not mediate the association between collective efficacy and child externalising problems. Thus, while harsh parenting as a proximal mechanism through which neighbourhood collective efficacy may influence child externalising problems has received some attention, the alternative pathway of child behavioural problems as a mediator between collective efficacy and family violence has been largely ignored.

The effects of neighbourhood- and family-level factors may vary across development. For example, harsh and abusive parenting may have more detrimental effects on aggressive and antisocial behaviour for younger compared to older children, possibly due to the relative rarity of corporal punishment in adolescence or the greater influence of factors outside the family environment, such as peers, for older children (Gershoff, 2002). The latter is particularly important when considering developmental differences in neighbourhood effects. As parents increase the level of autonomy and the time to engage in activities outside the home environment for children with increasing age (Veitch, Bagley, Ball, & Salmon, 2006), older children may have more interactions with residents from the neighbourhood. Thus, it has been proposed that direct neighbourhood influences may be stronger in adolescence when time spent outside increases, whereas, in childhood, effects may be more indirect, i.e., mainly mediated through the family environment (Leventhal & Brooks-Gunn, 2000). According to this logic, we may expect the indirect pathway of collective efficacy on youth antisocial behaviour

through family violence to be stronger in younger children, and the direct effect of collective efficacy on youth antisocial behaviour to be stronger in older children. Similarly, the indirect effect of collective efficacy on family violence via youth antisocial behaviour would be stronger in older children.

To date, studies examining the interplay between neighbourhood collective efficacy, family violence, and antisocial behaviour in children and adolescents have mainly been conducted in Western countries (Jaffee, Caspi, Moffitt, Polo-Tomás, & Taylor, 2007; Ma, 2016; Ma & Grogan-Kaylor, 2017; Odgers et al., 2009; Wilkinson, Lantos, McDaniel, & Winslow, 2019; Yonas et al., 2010). Little is known about the generalisability of findings to non-Western countries, where cultural differences may influence neighbourhood relationships and shared expectations of informal social control towards community problems. For example, Yoshizawa et al. (2020) found no effects of neighbourhood collective efficacy on youth antisocial behaviour across three different Asian countries.

To summarise, there is evidence for protective effects of neighbourhood collective efficacy on youth antisocial behaviour and family violence. However, the pathways through which collective efficacy influences parent and child behaviour remain poorly understood. More specifically, although there are well-established bidirectional effects between harsh and abusive parenting and child externalising problems (Pinquart, 2017), studies have been limited to family violence as a mediator of the association between collective efficacy and antisocial behaviour, as opposed to the reverse association of antisocial behaviour as a mediator between collective efficacy and family violence. Furthermore, these studies have been limited to early childhood, as opposed to other developmental periods, and focused on corporal punishment, rather than more severe or other forms of family violence (Ma, 2016; Ma & Grogan-Kaylor, 2017). In addition, most studies have been limited to high-risk (i.e., low SES) samples (e.g., the Project on Human Development in Chicago Neighbourhoods), such as inner-city

neighbourhoods in the US, with some studies suggesting that the protective effect of collective efficacy may be limited to these settings (Odgers et al., 2009). Thus, it is particularly important to investigate whether effects of collective efficacy vary by SES. Finally, the vast majority of studies examining the protective effects of collective efficacy have been conducted in Western countries, and the generalisability of these findings to other cultural contexts is unknown.

To address these gaps in the literature, we examined longitudinal associations between neighbourhood collective efficacy, family violence, and youth antisocial behaviour, using two nationally representative cohorts from South Korea. These included primary school students followed from age 10 to 12 years and secondary school students followed through ages 15 to 17 years, which enabled us to examine whether direct and indirect effects would be replicated across age groups. The main objectives of the present study were: (i) to examine whether higher levels of collective efficacy are associated with decreases in both youth antisocial behaviour and family violence over time; (ii) to test whether there are indirect effects of collective efficacy on youth antisocial behaviour through family violence and on family violence via youth antisocial behaviour; (iii) to examine whether there is evidence of remaining direct effects of collective efficacy on youth antisocial behaviour (after adjusting for family violence) and family violence (after adjusting for youth antisocial behaviour); (iv) to examine whether these associations are evident for both younger and older children; and (v) to investigate whether these effects vary by SES. Based on previous research (Odgers et al., 2009), we predicted that the effects of collective efficacy would be more pronounced in children from low, as compared to medium-high, SES backgrounds.

Methods

Participants

The present study used data from the Korean Youth Panel Survey collected by the National Youth Policy Institute (NYPI), including two population-based prospective cohorts. The first survey was conducted from 2003 to 2008, including six annual waves from the 2nd year of secondary school to one year after graduation. All second-year junior high school students (N = 618,100) nationwide (except Jeju Island) were eligible for inclusion. A total of 3697 students were selected based on stratified multi-stage cluster sampling at the regional, school, and classroom levels. More precisely, at baseline, one classroom in each school was randomly selected based on 15 administrative districts in South Korea. The survey data comprised 3449 (93%; 50% boys) students and their parents at age 14 years (i.e., baseline). Children and their parents were assessed again at ages 15 (92%), 16 (91%), 17 (91%), 18 (86.0%), and 19 (82%) years. This cohort is hereafter referred to as the *secondary school* sample. The second survey was conducted from 2004 to 2008, including five annual waves from the 4th grade of primary school to the 2nd year of secondary school. All fourth-year elementary school students (N = 630,694) nationwide (except Jeju Island) were eligible for inclusion. A total of 2949 students were selected using the same sampling method as for the secondary school sample. The survey data comprised 2844 (96%; 54% boys) students and their parents at age 10 years (i.e., baseline). Children and their parents were assessed again at ages 11 (95%), 12 (94%), 13 (88%), and 14 (86%) years. This cohort is hereafter referred to as the *primary school* sample. We used waves 1-3 (i.e., ages 10-12 years) in the primary school sample, whereas, in the secondary school sample, we used waves 2-4 (i.e., ages 15-17 years), as measures of neighbourhood collective efficacy were not available at baseline. Written informed consent was obtained from children and their parents in both cohorts. More precisely, interviewers sent consent forms to schools prior to the survey, and they collected self-report data from children who agreed to participate.

Next, parents were invited by mail to participate, and were subsequently interviewed by telephone. Children's data were excluded when their parents refused to participate in the study. In subsequent waves, children were individually contacted to conduct face-to-face interviews and parents were interviewed by telephone. Further details on the two cohorts are available in English on the NYPI website (www.nypi.re.kr).

Measures

Neighbourhood collective efficacy

Collective efficacy was measured at wave 1 (age 10 years) in the primary school sample and wave 2 (age 15 years) in the secondary school sample. Children were asked whether neighbours: (1) have close relationships with each other, (2) trust each other, (3) scold them if they smoke or drink in the neighbourhood, and (4) intervene or report to the police if they are assaulted in the neighbourhood, and whether they (5) let neighbours know if friends smoke or drink in the neighbourhood and (6) intervene or report to the police if friends are assaulted in the neighbourhood. The six items were rated on a 5-point scale, from 0 '*very untrue*' *to* 4 '*very true*'. Internal reliabilities were acceptable with $\omega = 0.67$ and $\omega = 0.80$ for the primary and secondary school samples, respectively.

Family violence

Domestic violence and child abuse were measured at waves 2 (age 11 years) and 3 (age 12 years) in the primary school sample and waves 3 (age 16 years) and 4 (age 17 years) in the secondary school sample. Children were asked whether they frequently see: (1) their parents verbally abuse each other or (2) one parent beat the other one, and whether they are often (3) verbally abused or (4) severely beaten by parents. The first two items were used to assess domestic violence, while the latter two items were used to assess child abuse. The four items were rated on a 5-point scale, from 0 '*very untrue*' to 4 '*very true*', and were used to create a composite measure of family violence. The scale showed good internal reliability in the

primary school sample at waves 2 (ω = 0.78) and 3 (ω = 0.82) and in the secondary school sample at waves 3 (ω = 0.80) and 4 (ω = 0.86).

Youth antisocial behaviour

Children were asked about antisocial and aggressive behaviours at waves 2 (age 11 years) and 3 (age 12 years) in the primary school sample and waves 3 (age 16 years) and 4 (age 17 years) in the secondary school sample. Overall, 14 items were used in the primary school sample and 11 items were used in the secondary school sample, of which 10 were identical across cohorts, asking about behaviour problems in the past year, including unauthorised school absence, group bullying, severe teasing or banter, threatening, drinking[4], smoking[5], severely beating others, robbing, stealing, and running away. In addition, children in the primary school sample were asked whether they engaged in the following four problem behaviours: fare evasion, shouting at their teacher, cheating on an exam, and misappropriating expenses for school supplies. Children in the secondary school sample were additionally asked whether they had engaged in a gang fight. All items were coded as either 0 '*no*' or 1 '*yes*'. Analyses were based on 14 and 11 items for the primary and secondary school samples, respectively. The scales showed excellent internal reliability in the primary school sample at waves 2 (ω = 0.94) and 3 (ω = 0.94) and in the secondary school sample at waves 3 (ω = 0.95) and 4 (ω = 0.95).

Covariates

We included child sex, family composition, and indicators of income and education, each of which have been identified as risk factors for family violence and child antisocial behaviour (Piotrowska, Stride, Croft, & Rowe, 2015; Stith et al., 2009). Information on all covariates except child sex was collected by parent report at wave 1 in both samples. Child sex was coded as 0 '*female*' or 1 '*male*'. Family composition was coded as 0 '*living with biological father and*

[4] The legal drinking age in South Korea is 19 years.
[5] The legal smoking age in South Korea is 19 years.

mother' or 1 '*other*'. House ownership was coded as 0 '*own house*' or 1 '*other*'. Maternal and paternal education were coded as 0 '*no schooling*', 1 '*elementary school*', 2 '*middle school*', 3 '*high school*', 4 '*junior college*', 5 '*college/university*', 6 '*master's degree*', and 7 '*PhD*'. Average monthly family income was used as a continuous variable, measured in Korean Won (₩), with ₩1035 equating approximately to USD 1 in 2004 when the studies commenced.

Analysis strategy

For both samples, we specified two fully latent structural regression models to examine the associations between neighbourhood collective efficacy, family violence, and youth antisocial behaviour.

In a first series of models, the *structural* parts represented the hypotheses that: (i) collective efficacy has an effect on family violence; (ii) collective efficacy and family violence each have effects on youth antisocial behaviour; and (iii) collective efficacy also affects youth antisocial behaviour indirectly through family violence (i.e., family violence is a mediator in the association between collective efficacy and youth antisocial behaviour; see Figure 1 for a schematic diagram of hypothesised relations). In a second series of models, we tested for reversed associations, specifically whether youth antisocial behaviour might influence family violence. Here, the *structural* parts represented the hypotheses that: (i) collective efficacy has an effect on youth antisocial behaviour; (ii) collective efficacy and youth antisocial behaviour each have effects on family violence; and (iii) collective efficacy also affects family violence indirectly through youth antisocial behaviour (see Figure 2 for a schematic diagram of hypothesised relations).

The *measurement* part of each model featured three factors, including collective efficacy with six indicators, family violence with four indicators, and youth antisocial behaviour with 14 and 11 indicators for the primary and secondary school samples, respectively.

First, each measurement model was re-specified as a confirmatory factor analysis (CFA) model with correlated factors. The following indices were used to evaluate model fit: Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI), Root Mean Square Error of Approximation (RMSEA), and Standardised Root Mean Residual (SRMR), with values of ≥ .95, ≤ .06, and ≤ .08, respectively, indicating good model fit (Hu & Bentler, 1999). The chi-square statistic was not used to evaluate model fit as models based on large samples are too readily rejected.

Second, we examined hypotheses about direct, indirect, and total effects among latent variables in each structural model. In a first set of models, we tested whether collective efficacy is negatively associated with family violence (i.e., *path a*); whether family violence is positively associated with youth antisocial behaviour after adjusting for collective efficacy (i.e., *path b*); and whether collective efficacy is negatively associated with youth antisocial behaviour (i.e., *path c* or *total effect*). We also tested whether the association between collective efficacy and youth antisocial behaviour holds when adjusting for family violence (i.e., *path c'* or *direct effect*), and whether family violence mediates the association between collective efficacy and youth antisocial behaviour (i.e., *indirect effect*; see Figure 1). In a second set of models, we switched the mediator and the outcome, to test the alternative hypothesis that higher levels of collective efficacy are associated with decreased levels of youth antisocial behaviour, which, in turn, are associated with a decrease in family violence (see Figure 2).

We followed Hayes' (2018) approach to mediation, and estimated indirect effects even if *paths a* and *b* were non-significant, as well as in the absence of a significant total effect (*path c*). Indirect effects were estimated using the product of coefficient strategy with 1000 bootstrap samples and bias-corrected 95% confidence intervals (Williams & MacKinnon, 2008). Wald's test was used for determining whether path coefficients differed between low and medium-high SES. Children from families with a monthly income in the lowest quintile were classified

as low SES (primary school sample: ≤ ₩2,000,000, approximately USD 1,932; secondary school sample: ≤ ₩1,800,000, approximately USD 1,740).[6] All models were adjusted for child sex, family composition, house ownership, maternal and paternal education, and family income. CFA and mediation analysis were performed in Mplus, Version 8.1 (Muthén & Muthén, 2017). All other analyses were performed in RStudio, Version 1.1.447 (RStudio Team, 2016).

[Insert Figure 1 here]

[Insert Figure 2 here]

Missing data

Using full information maximum likelihood, in the primary school sample, the CFAs were based on 2844 participants (i.e., full baseline sample) and the mediation analyses after adjusting for covariates were based on 2667 (94%) participants. In the secondary school sample, the CFAs were based on 3346 (97%) participants and the mediation analyses after adjusting for covariates were based on 3059 (89%) participants, using full information maximum likelihood (Appendix 1 presents a flow chart of retention for each cohort). Those included in the mediation analyses were less likely to live with both biological parents compared to those from the baseline samples (primary school sample: OR 0.73, 95% CI 0.55-0.97; secondary school sample: OR 0.75, 95% CI 0.61-0.93) (see Appendix 2 for all comparisons between the baseline samples and those included in the mediation analyses).

[6] In the primary school sample, we noticed large increases in proportions for every ₩50,000, most likely as a result of rounding errors in the database. Thus, while ₩190,000 comprised 17.7% of the sample, the used cut-point of ₩200,000 already comprised 35.5% of the sample. Unfortunately, sensitivity analyses with the next lower value (₩190,000) were not feasible, as models did not converge. Thus, despite similar cut-points across both cohorts, low SES in the primary school sample might be better described as the lowest tercile as opposed to the lowest quintile.

Results

Descriptive statistics

In both samples, over 90% of children lived with both biological parents, around two thirds of families owned their own homes, and about 40% of children had at least one parent with a university degree. The average monthly income was approximately ₩3,000,000 (around USD 2,899 at the time of data collection) in both samples. Table 1 shows the full sample characteristics for each cohort and all sample comparisons. Compared to the secondary school sample, the primary school sample had higher levels of parental education, lower levels of home ownership, children were more likely to live with both biological parents, and children reported higher levels of neighbourhood collective efficacy, and lower levels of family violence and antisocial behaviour (see Table 1 for full details and Table 2 for the correlation matrices for the primary school sample and secondary school sample; sample proportions for all antisocial behaviour items are presented in Appendix 3).

[Insert Table 1 here]

[Insert Table 2 here]

Family violence as a mediator of the association between neighbourhood collective efficacy and youth antisocial behaviour

The measurement models for both the primary school (CFI = .91; TLI = .90; RMSEA = .03; SRMR = .06) and secondary school (CFI = .94; TLI = .93; RMSEA = .03; SRMR = .07) samples indicated acceptable model fit (Appendix 4 presents the factor loadings across samples).

Table 3 shows standardised path estimates after adjusting for covariates for the total sample and separated by SES across cohorts (see Figure 1 for a schematic diagram). In the primary school sample, for the total sample, higher levels of collective efficacy were associated

with decreases in family violence, and higher levels of family violence were associated with increases in youth antisocial behaviour. There was no evidence of a direct or total effect of collective efficacy on youth antisocial behaviour. However, there was a small indirect effect of collective efficacy on youth antisocial behaviour via family violence (see Table 3). There was no evidence that the pattern of effects differed across SES groups (Wald's test *p*-values ranging between .19 and .99). However, overall, findings were somewhat more pronounced in the low SES group with the total effect of collective efficacy on youth antisocial behaviour and the indirect pathway via family violence being statistically significant in the low SES, but not the medium-high SES, group (see Table 3).

Similar to the primary school sample, in the secondary school sample, higher levels of collective efficacy were associated with decreases in family violence, and higher levels of family violence were associated with increases in youth antisocial behaviour. Again, there was no evidence of a direct or total effect, but there was evidence of a small indirect effect of collective efficacy on youth antisocial behaviour via family violence (see Table 3). When analyses were re-run for SES categories, there was no evidence that low and medium-high SES groups differed in terms of any direct (Wald's test *p*-values ranging between .12 and .14) or indirect pathways (as evidenced by overlapping 95% confidence intervals; see Table 3).

[Insert Table 3 here]

Youth antisocial behaviour as a mediator of the association between neighbourhood collective efficacy and family violence

The measurement models for both the primary school (CFI = .91; TLI = .90; RMSEA = .03; SRMR = .06) and secondary school (CFI = .93; TLI = .92; RMSEA = .03; SRMR = .07) samples indicated acceptable model fit (Appendix 4 presents the factor loadings across samples).

Table 4 shows standardised path estimates after adjusting for covariates for the total sample and separated by SES across cohorts (see Figure 2 for a schematic diagram). In the primary school sample, while collective efficacy was not associated with youth antisocial behaviour, higher levels of youth antisocial behaviour predicted increases in family violence. Furthermore, higher levels of collective efficacy were associated with decreases in family violence, even after adjusting for the effect of youth antisocial behaviour. However, there was no evidence of an indirect effect of collective efficacy on family violence via youth antisocial behaviour (see Table 4). This pattern of results were replicated when comparing low and medium-high SES groups, with no evidence of group differences (Wald's test *p*-values ranging between .20 and .92; see Table 4).

In contrast to the primary school sample, in the secondary school sample, higher levels of collective efficacy were associated with decreases in youth antisocial behaviour, which, in turn, were associated with increases in family violence. While there was no evidence of a direct or total effect, there was evidence of a small indirect effect of collective efficacy on family violence via youth antisocial behaviour (see Table 4). When comparing SES categories, higher levels of collective efficacy were associated with lower levels of youth antisocial behaviour in low, but not medium-high, SES children ($\chi(1) = 4.63$, $p = 0.03$). However, there was no evidence that SES groups differed in terms of any direct (Wald's test *p*-values ranging between .20 and .83) or indirect pathways (as evidenced by overlapping 95% confidence intervals; see Table 4).

[Insert Table 4 here]

Discussion

The current study examined the effects of neighbourhood collective efficacy on family violence and youth antisocial behaviour, using two nationally representative, prospective longitudinal

cohorts from South Korea. In a first series of models, we examined family violence as a mediator of the association between neighbourhood collective efficacy and youth antisocial behaviour. In both the primary and secondary school samples, higher levels of collective efficacy predicted lower levels of family violence, and higher levels of family violence predicted higher levels of youth antisocial behaviour. In contrast to previous research, there was no direct effect of collective efficacy on youth antisocial behaviour. However, there was evidence of an indirect effect from collective efficacy to youth antisocial behaviour through family violence. Although there was no evidence that these effects varied according to SES, the pattern of results was more pronounced in the low SES group, with a significant total effect of collective efficacy and indirect effect via family violence in the primary school sample, which were not observed in the medium-high SES group. In a second series of models, we examined youth antisocial behaviour as a mediator of the association between neighbourhood collective efficacy and family violence. In the primary school sample, higher levels of collective efficacy were not associated with a decrease in youth antisocial behaviour. However, higher levels of collective efficacy predicted a decrease in family violence, even after adjusting for youth antisocial behaviour (i.e., collective efficacy had a direct effect on youth antisocial behaviour). There was no evidence of an indirect effect through youth antisocial behaviour or moderation by SES. Conversely, in the secondary school sample, higher levels of collective efficacy predicted a decrease in youth antisocial behaviour in low, but not medium-high, SES children, which, in turn, predicted an increase in family violence. Furthermore, there was a total effect of collective efficacy on family violence in low SES children, which, however, did not differ to medium-high SES children in direct comparison.

In contrast to previous studies (Ma, 2016; Ma & Grogan-Kaylor, 2017; Odgers et al., 2009), we found no evidence for a direct effect of collective efficacy on youth antisocial behaviour. Odgers et al. (2009) found that collective efficacy was associated with child

antisocial behaviour in deprived, but not affluent, neighbourhoods. Furthermore, in the Fragile Families and Child Well-Being Study, a cohort focusing on urban children from socioeconomically disadvantaged backgrounds, there was evidence for a direct effect of collective efficacy on child externalising problems (Ma, 2016; Ma & Grogan-Kaylor, 2017). In the primary school sample, there was a total effect of collective efficacy on youth antisocial behaviour as well as an indirect effect through family violence in children from low, but not medium-high, SES backgrounds. However, when directly comparing these groups, there was no evidence of moderation by SES. Thus, while the current study may indicate a more consistent pattern of effects for low SES children, it can only provide tentative evidence for more pronounced effects in children from deprived neighbourhoods in a South Korean context.

In line with previous studies (Beyer et al., 2015; Jaffee et al., 2007; Molnar et al., 2016; Pinchevsky & Wright, 2012; Wright & Benson, 2010), higher levels of neighbourhood collective efficacy were associated with lower levels of family violence. In contrast to the pattern of effects observed for youth antisocial behaviour, there was evidence of a direct effect of collective efficacy on family violence in the primary school sample. These effects remained even after adjusting for youth antisocial behaviour, which was positively associated with family violence. While some previous studies have focused on high-risk samples, such as the Project on Human Development in Chicago Neighbourhoods (Jackson, 2016; Wright & Benson, 2010), the current study found similar results in low and medium-high SES families, which supports previous studies on the protective effect of collective efficacy on child maltreatment irrespective of structural factors (Molnar et al., 2016). In the secondary school sample, there was a less consistent pattern of results, again, merely with tentative evidence for more pronounced effects of collective efficacy in low SES families.

Previous studies have focused on cohorts based in the US and UK, and findings may not translate to other cultural contexts. The current study used two nationally representative

South Korean cohorts, which included a mixture of disadvantaged and affluent families. For example, in both cohorts, around 40% of participants had at least one parent with a university degree, and over 90% of participants across samples lived with both biological parents. Thus, the current samples included a large proportion of youth from highly educated and intact families. This may explain why our findings are not in complete agreement with those obtained in samples residing in high-risk, inner-city neighbourhoods in the US. When we re-run analyses for low and medium-high SES children separately, the pattern of results were more in line with previous studies, showing larger effects for children from deprived neighbourhoods (Odgers et al., 2009). Alternatively, the absence of a direct effect of collective efficacy on youth antisocial behaviour may be explained by cultural differences. Asian cultures are viewed as more interdependent (i.e., seeing oneself as part of a greater whole), whereas American and Western European cultures are considered as more independent (i.e., seeing oneself as a distinct individual) (Markus & Kitayama, 1991). Thus, the effect of neighbourhood collective efficacy may be stronger in urban America, where social cohesion and informal social control may be considered more the exception than the rule, and where collective efficacy may provide community and social support to families that are not available elsewhere. Conversely, effects may be smaller in South Korea, where community supports are more accessible and/or already integrated into the more collectivist culture. Future research, needs to examine the constructs of collective efficacy across cultural contexts, and whether levels of collective efficacy differ across countries.

There is strong evidence for the effectiveness of parenting programmes targeting harsh and abusive parenting (Piquero et al., 2016) and perpetrators of intimate partner violence (Karakurt, Koc, Cetinsaya, Ayluctarhan, & Bolen, 2019). The current findings suggest that increasing levels of neighbourhood collective efficacy may have direct effects on family violence and indirect effects on youth antisocial behaviour by reducing levels of family

violence. In the US, there are promising community-based interventions, such as the Strong Communities for Children programme, which have been shown to decrease substantiated cases of child maltreatment (McDonell, Ben-Arieh, & Melton, 2015). Future research needs to ascertain whether such programmes can be translated into other cultural contexts.

According to previous research, neighbourhood influences affect adolescents more directly, whereas in childhood, these effects may operate more indirectly through proximal mechanisms, such as the family environment (Leventhal & Brooks-Gunn, 2000). However, the findings in the current study were largely comparable across younger (aged 10-12 years) and older (aged 15-17 years) children. Considering that our findings were replicated across two samples of different ages, more research is needed to examine direct and indirect pathways of neighbourhood influences, ideally using a wide age range from early childhood to late adolescence.

Key strengths of the current study include the use of two prospective, population-based cohorts from South Korea, with very high retention rates, spanning the age range of 10-17 years. Furthermore, few studies have examined the interplay between neighbourhood collective efficacy, family violence, and youth antisocial behaviour, and whether these relations vary by SES. In contrast to the vast majority of previous studies, which have used US-based samples, the current study provides novel prospective longitudinal data from a non-Western, more collectivist culture. Finally, the current study used a measure of family violence that included direct exposure to child abuse and indirect exposure through witnessing intimate partner violence, whereas previous research in this area has focused on more limited or normative forms of violence against children (e.g., corporal punishment).

The findings need to be interpreted in the context of the following limitations. First, all measures were self-report, and thus may have been subject to shared rater bias. For example, being exposed to family violence may influence young people's perceptions of how they are

treated by neighbours and the broader community. More precisely, the measurement error from using children as informants of neighbourhood influences may be correlated with the measurement error of family violence (Duncan & Raudenbush, 1999). Related to this, the current study used measures developed by the National Youth Policy Institute (South Korea), which have not been fully validated. Although our measure to assess neighbourhood collective efficacy tapped similar constructs as the scale developed by Sampson (1997) (i.e., social cohesion and informal social control), which has been widely used and is considered to be the gold standard, it was briefer and focused on alcohol use and smoking in the neighbourhood. Particularly in the primary school sample, the items related to social cohesion showed low factor loadings (see Appendix 4). However, using a latent variable approach, we were able to minimise measurement error, and internal reliability of each latent factor and model fit of measurement models were acceptable, which should strengthen confidence in our findings. Nevertheless, future studies should use multiple sources to assess neighbourhood collective efficacy, including, for example, reports from multiple residents living in the same neighbourhood as the index child (see e.g., Odgers et al., 2009). Third, the current study included a limited number of covariates. More precisely, the association between family violence and youth antisocial behaviour may be confounded by parental mental illness and parental history of antisocial behaviour. Similarly, the association of neighbourhood collective efficacy with family violence and youth antisocial behaviour may be confounded by other neighbourhood-level variables, such as community violence, which could both reduce collective efficacy and increase family violence and youth antisocial behaviour. Fourth, we were unable to compare the results directly across school contexts due to slight differences in outcome measures. Nonetheless, in the absence of formal statistical comparisons, it is notable that effects were broadly similar across samples – with overlapping confidence intervals.

In conclusion, neighbourhood collective efficacy may affect youth antisocial behaviour more indirectly through mitigating family violence. Although, these effects were more pronounced in low SES children, there was no evidence of moderation by SES. Furthermore, neighbourhood collective efficacy may affect family violence more directly, particularly in younger children and even after adjusting for youth antisocial behaviour. Again, there was a more pronounced pattern of effects for low SES children, which, however, did not differ from the effects observed for medium-high SES children. The findings highlight the potential protective effects of collective efficacy on family violence and youth antisocial behaviour, and demonstrates the importance of proximal mechanism, such as violence in the family environment, through which neighbourhood characteristics can influence child outcomes.

Declarations

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Conflict of interest

The authors have no conflicts of interest to declare in relation to this article.

Availability of data and material

All data are available in English on the South Korean National Youth Policy Institute's website (https://www.nypi.re.kr/archive/contents/siteMain.do?srch_mu_lang=ENG).

Code availability

Not applicable

Author contributions

Andreas Bauer, Jisu Park, and Yoonsun Han contributed to data acquisition. Andreas Bauer, Gemma Hammerton, Jisu Park, Joseph Murray, Sarah L. Halligan, and Graeme Fairchild contributed to the conceptualisation of the study. Andreas Bauer and Gemma Hammerton performed the analysis. Andreas Bauer, Gemma Hammerton, Joseph Murray, Alicia Matijasevich, Sarah L. Halligan, and Graeme Fairchild wrote the manuscript. All authors revised, edited, and approved the manuscript.

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Fig. 2 Hypothesised model with family violence as a mediator of the association between neighbourhood collective efficacy and youth antisocial behaviour

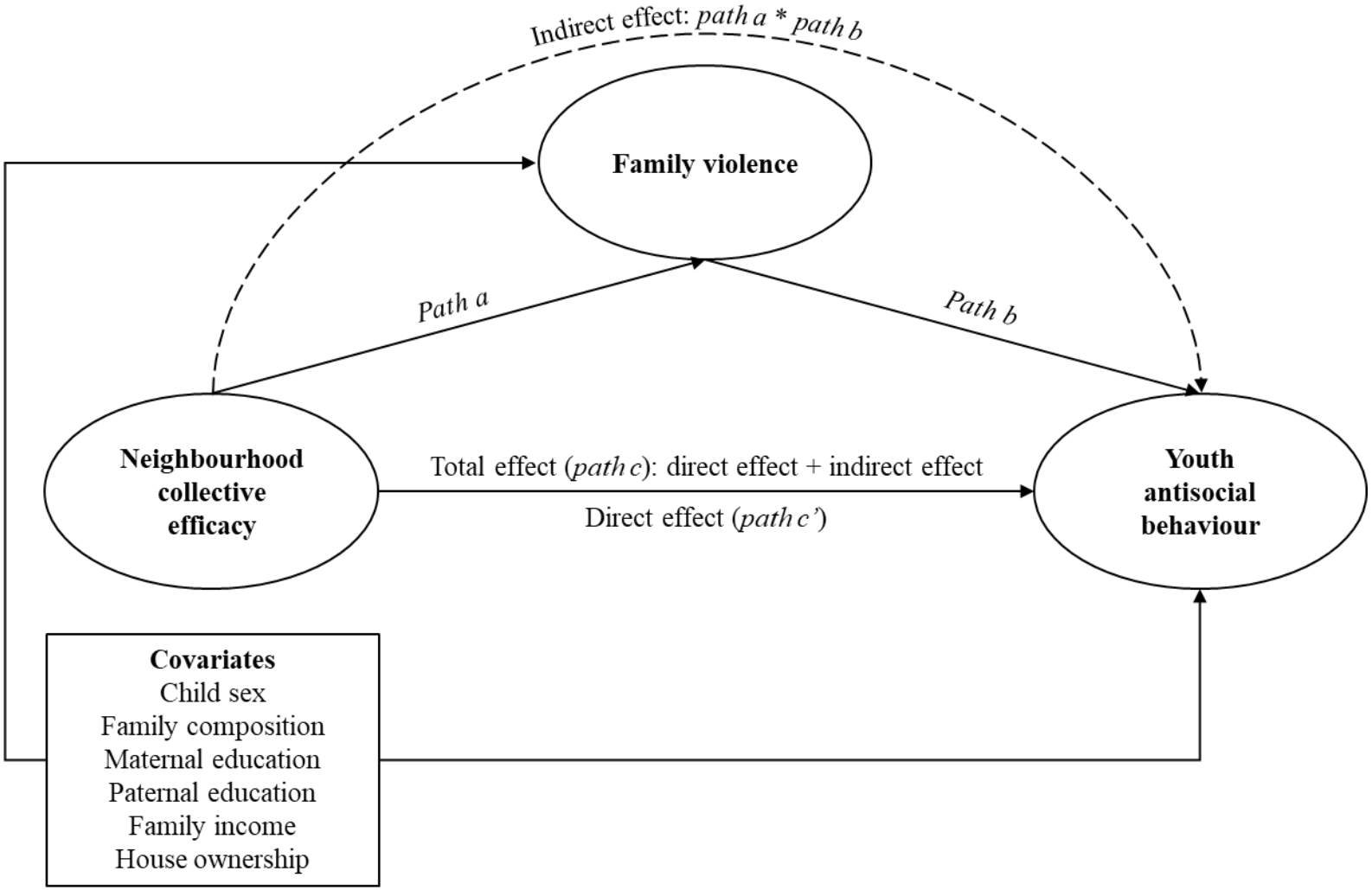


Fig. 2 Hypothesised model with youth antisocial behaviour as a mediator of the association between neighbourhood collective efficacy and family violence

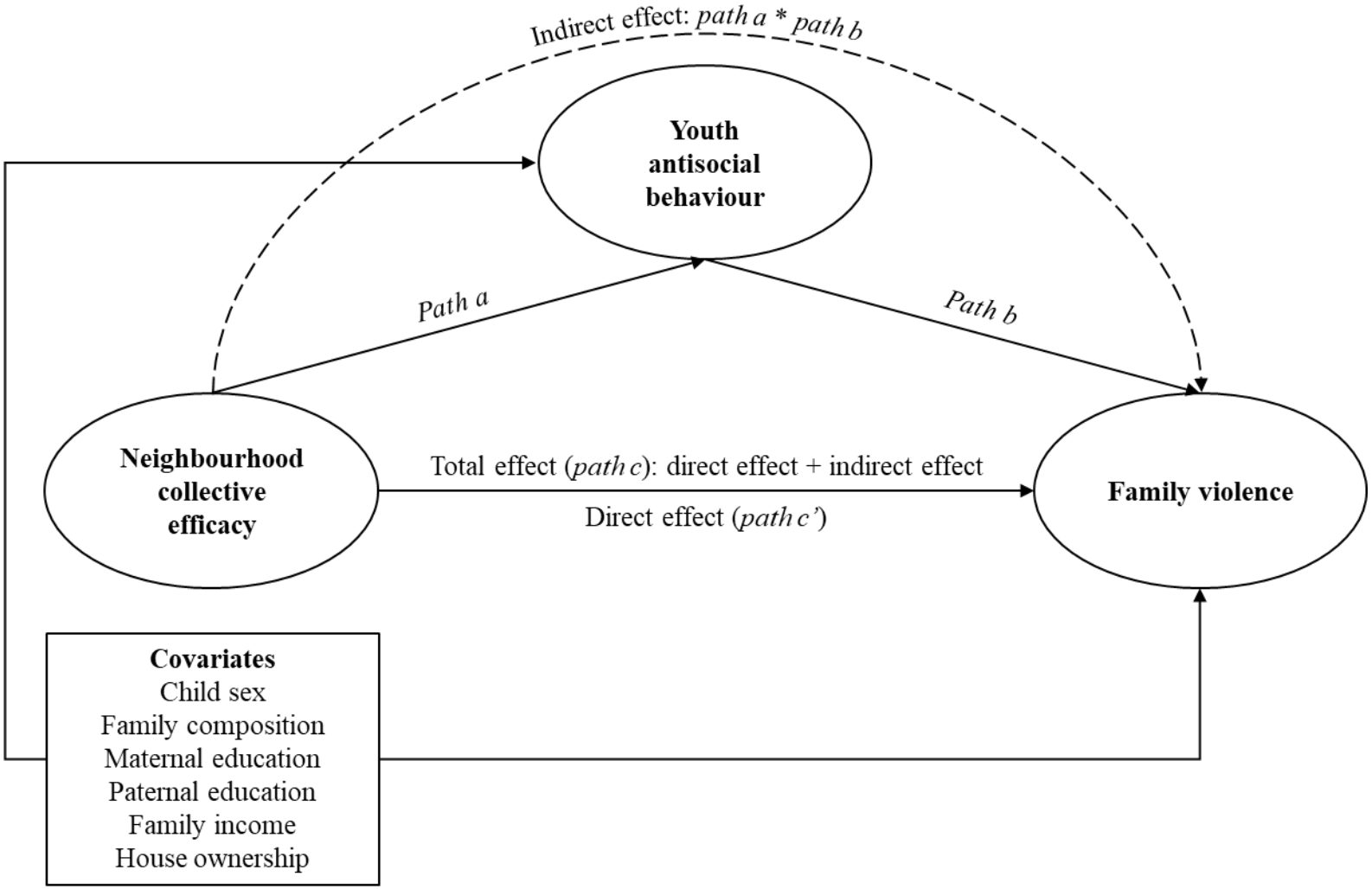


Table 1 Sample characteristics and comparisons between the primary school sample and secondary school sample

	Primary school	**Secondary school**	**Comparison**
	Mean (SD) or %	Mean (SD) or %	*r* (*p*) or OR (95% CI)
Collective efficacy (0-24)	16.51 (4.42)	12.16 (4.58)	.43 (< .001)
Family violence (0-16)			
Time 2	2.22 (2.96)	2.75 (2.76)	.09 (< .001)
Time 3	2.25 (2.97)	2.86 (3.03)	.10 (< .001)
Youth antisocial behaviour[a] (0-10)			
Time 2	0.41 (0.91)	0.70 (1.13)	.14 (< .001)
Time 3	0.35 (0.89)	0.77 (1.09)	.21 (< .001)
Maternal education (0-7)	3.50 (1.02)	3.25 (1.11)	.08 (< .001)
Paternal education (0-7)	3.93 (1.19)	3.74 (1.31)	.08 (< .001)
Monthly income (0-3000)[b]	302.14 (176.52)	299.73 (216.90)	.01 (= .63)
Child sex (male)	54	50	0.87 (0.78-0.96)
House ownership (other)[c]	38	31	0.73 (0.66-0.82)
Family composition (other)[d]	5	7	1.57 (1.25-1.97)

Note. Observed, rather than latent, variables are presented. [a] Limited to the 10 items that were identical across cohorts. [b] In units of ₩10,000 (approximately USD 10). [c] Reference is 'own house'. [d] Reference is 'living with biological father and mother'.

Table 2 Correlation matrix of all study variables in the primary school sample (*upper* triangular matrix) and the secondary school sample (*lower* triangular matrix)

		1	2	3	4	5	6	7	8	9	10	11
1	**Collective efficacy**		-.08**	-.03	-.11**	-.03	-.09**	.08**	.08**	.07**	-.11**	.01
2	**Family violence** (T2)	-.06**		.19**	.34**	.11**	.17**	-.09**	-.09**	-.05**	.04	.01
3	**Antisocial behaviour** (T2)	-.05**	.17**		.12**	.29**	.18**	-.05*	-.06**	-.02	.03	.03
4	**Family violence** (T3)	-.04*	.49**	.12**		.16**	.11**	-.10**	-.09**	-.04*	.03	-.03
5	**Antisocial behaviour** (T3)	-.04*	.12**	.55**	.13**		.08**	-.03	-.07**	-.04	.03	.09**
6	**Child sex** (male)	.05*	-.04	-.08**	-.01	-.14**		.00	.01	.02	.00	.05
7	**Maternal education**	-.04*	-.08**	-.05**	-.11**	-.05*	-.02		.68**	.38**	-.18**	-.27**
8	**Paternal education**	-.05*	-.08**	-.05**	-.09**	-.05**	-.02	.70**		.37**	-.20**	-.36**
9	**Family income**	-.02	-.08**	-.04	-.06**	-.04	-.01	.36**	.35**		-.27**	-.60**
10	**House ownership** (other)[a]	-.07**	.08**	.06**	.08**	.04	.04	-.14**	-.13**	-.32**		.29**
11	**Family composition** (other)[b]	-.08*	.09*	.08**	.09*	.07*	.03	-.30**	-.31**	-.61**	.33**	

Note. Observed, rather than latent, variables are presented. [a] Reference is 'own house'. [b] Reference is 'living with biological father and mother'. * $p < .05$. ** $p < .01$.

Table 3 Path estimates after adjusting for covariates for the total sample and separated by SES for the model examining family violence as a mediator of the association between neighbourhood collective efficacy and youth antisocial behaviour.

	Total sample		**Medium-high SES**		**Low SES**	
	β (SE)	*P* or 95% CI	*β* (SE)	*P* or 95% CI	*β* (SE)	*P* or 95% CI
Primary school						
Collective efficacy → Family violence	**-0.11 (0.02)**	< .001	**-0.10 (0.03)**	< .01	**-0.10 (0.04)**	< .01
Family violence → Antisocial behaviour	**0.15 (0.03)**	< .001	**0.08 (0.04)**	= .04	**0.26 (0.05)**	< .001
Direct effect	-0.02 (0.03)	= .49	0.01 (0.04)	= .77	-0.09 (0.05)	= .08
Total effect	-0.04 (0.03)	= .24	0.01 (0.04)	= .91	**-0.12 (0.05)**	= .02
Indirect effect	**-0.02 (0.01)**	-0.03, -0.01	-0.01 (0.01)	-0.02, 0.00	**-0.03 (0.01)**	-0.05, -0.01
Secondary school						
Collective efficacy → Family violence	**-0.07 (0.02)**	< .01	**-0.06 (0.03)**	= .02	**-0.13 (0.05)**	< .01
Family violence → Antisocial behaviour	**0.18 (0.03)**	< .001	**0.17 (0.03)**	< .001	**0.24 (0.03)**	< .001
Direct effect	-0.04 (0.03)	= .15	-0.04 (0.03)	= .19	0.06 (0.03)	= .06
Total effect	-0.05 (0.03)	= .06	-0.05 (0.03)	= .12	0.03 (0.03)	= .36
Indirect effect	**-0.01 (0.01)**	-0.03, -0.01	**-0.01 (0.01)**	-0.02, -0.00	**-0.03 (0.02)**	-0.08, -0.01

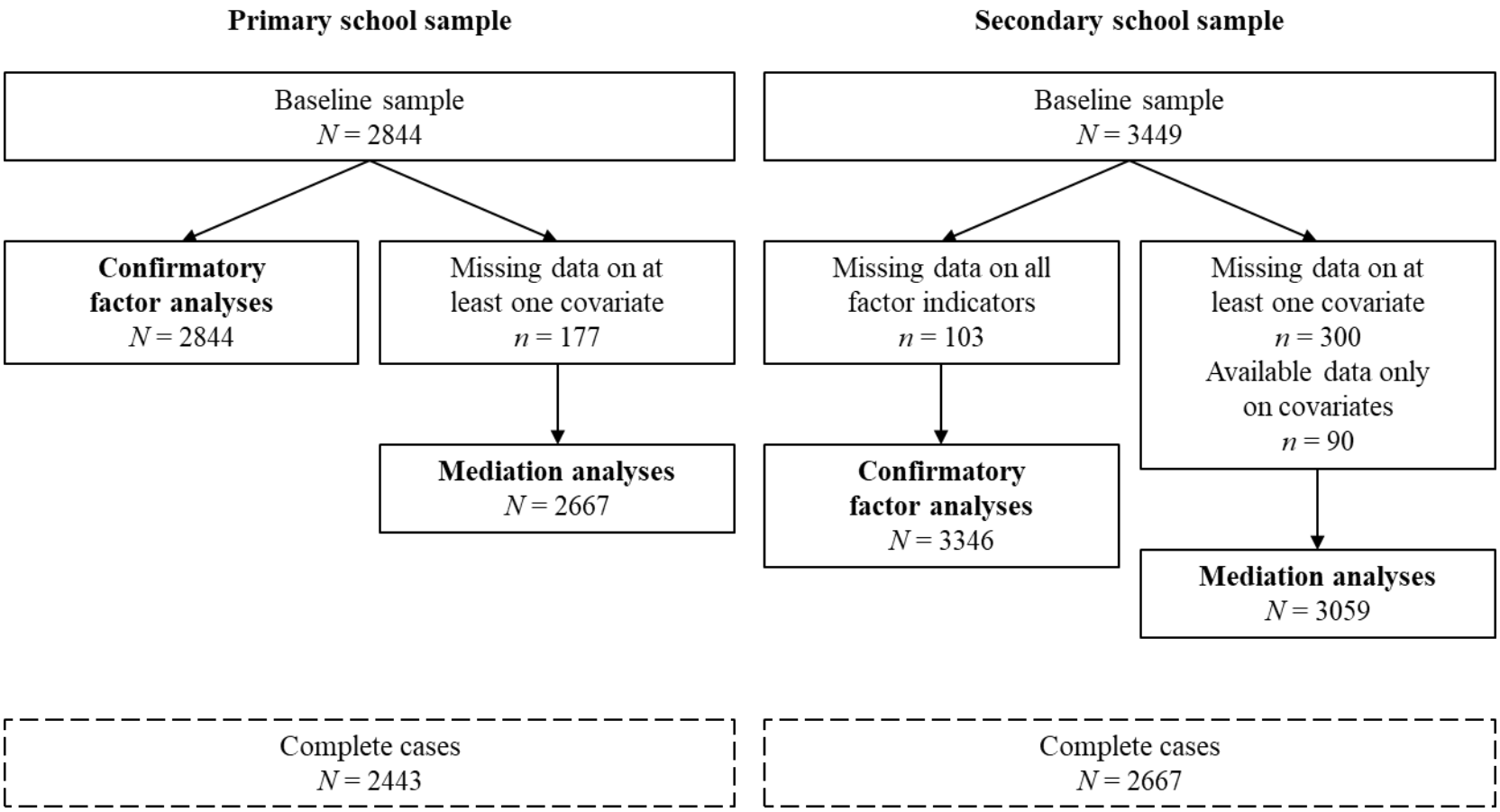
Note. All models were adjusted for child sex, family composition, house ownership, and maternal and paternal education, in addition to family income for the model using the total sample. Bold values indicate statistically significant associations at $p < .05$. *β* = Standardized regression coefficient; SE = Standard error; *P* = *P*-value; 95% CI = 95% confidence interval.

Table 4 Path estimates after adjusting for covariates for the total sample and separated by SES for the model examining youth antisocial behaviour as a mediator of the association between neighbourhood collective efficacy and family violence.

	Total sample		**Medium-high SES**		**Low SES**	
	β (SE)	*P* or 95% CI	*β* (SE)	*P* or 95% CI	*β* (SE)	*P* or 95% CI
Primary school						
Collective efficacy → Antisocial behaviour	-0.01 (0.03)	= .70	-0.04 (0.04)	= .32	0.04 (0.05)	= .42
Antisocial behaviour → family violence	**0.15 (0.03)**	< .001	**0.13 (0.04)**	< .001	**0.17 (0.04)**	< .001
Direct effect	**-0.14 (0.02)**	< .001	**-0.11 (0.03)**	< .001	**-0.17 (0.04)**	< .001
Total effect	**-0.14 (0.02)**	< .001	**-0.12 (0.03)**	< .001	**-0.17 (0.04)**	< .001
Indirect effect	-0.00 (0.01)	-0.01, 0.01	-0.01 (0.01)	-0.02, 0.00	0.01 (0.02)	-0.03, 0.03
Secondary school						
Collective efficacy → Antisocial behaviour	**-0.07 (0.03)**	= .02	-0.02 (0.03)	= .55	**-0.17 (0.05)**	< .01
Antisocial behaviour → family violence	**0.17 (0.03)**	< .001	**0.16 (0.03)**	< .001	**0.23 (0.04)**	< .001
Direct effect	-0.02 (0.02)	= .34	-0.02 (0.02)	= .36	-0.06 (0.05)	= .27
Total effect	-0.03 (0.02)	= .14	-0.03 (0.02)	= .30	**-0.10 (0.05)**	= .05
Indirect effect	**-0.01 (0.01)**	-0.03, -0.00	-0.00 (0.01)	-0.02, 0.01	-0.04 (0.030)	-0.11, 0.00

Note. All models were adjusted for child sex, family composition, house ownership, and maternal and paternal education, in addition to family income for the model using the total sample. Bold values indicate statistically significant associations at $p < .05$. *β* = Standardized regression coefficient; SE = Standard error; *P* = *P*-value; 95% CI = 95% confidence interval.

Appendix 1 Flow charts of included participants in the confirmatory factor analyses and mediation analyses after adjusting for covariates across samples using full information maximum likelihood



Appendix 2 Comparison between the baseline sample and those included in the mediation analyses adjusted for covariates, separated by cohort

Variables	**Baseline** Mean (SD) or %	*versus* *r* (*p*) or OR (95% CI)	**Mediation** Mean (SD) or %
Primary school sample			
Child's sex (male)	54	0.99 (0.89-1.10)	53
House ownership (other)[a]	38	0.98 (0.88-1.10)	37
Family composition (other)[b]	5	**0.73 (0.55-0.97)**	3
Paternal education (0-7)	3.93 (1.19)	.00 (= .78)	3.94 (1.19)
Maternal education (0-7)	3.50 (1.02)	.00 (= .91)	3.50 (1.01)
Monthly income (0-3000)[c]	302.14 (176.52)	.01 (= .49)	305.48 (176.82)
Secondary school sample			
Child's sex (male)	50	0.99 (0.89-1.09)	50
House ownership (other)[a]	31	0.96 (0.86-1.07)	30
Family composition (other)[b]	7	**0.75 (0.61-0.93)**	5
Paternal education (0-7)	4.74 (1.31)	.00 (= .91)	4.74 (1.29)
Maternal education (0-7)	4.25 (1.11)	.01 (= .66)	4.23 (1.09)
Monthly income (0-3000)[c]	299.73 (216.90)	.00 (= .92)	299.19 (214.46)

Note. Bold values indicate statistically significant group differences at $p < .05$. Pearson's correlation coefficient *r* and *p*-values were derived from independent *t*-tests. [a] Reference is 'own house'. [b] Reference is 'living with biological father and mother'. [c] In units of ₩10,000.

Appendix 3 Sample proportions for each item used to measure youth antisocial behaviour, separated by cohort

	Primary school (*N* = 2844)		**Secondary school** (*N* = 3449)	
	Age 11 %	**Age 12** %	**Age 16** %	**Age 17** %
Unauthorised school absence	8	5	6	6
Group bullying	10	9	2	1
Severe teasing or banter	10	7	4	3
Threatening	2	2	1	1
Drinking	5	6	37	45
Smoking	1	2	12	14
Severely beating others	2	2	3	2
Robbing	1	1	1	1
Stealing	2	2	2	2
Running away	2	1	3	3
Fare evasion	2	3	N/A	
Shouting at their teacher	6	6	N/A	
Cheating on exam	7	9	N/A	
Misappropriating expenses for school supplies	10	10	N/A	
Gang fight	N/A		1	1

Note. N/A = Item not available in cohort.

Appendix 4 Standardised factor loadings of the models examining family violence as a mediator of the relationship between neighbourhood collective efficacy and youth antisocial behaviour (model 1) and the model examining youth antisocial behaviour as a mediator of the relationship between neighbourhood collective efficacy and family violence (model 2), separated by cohort

	Primary school sample		**Secondary school sample**	
	Model 1	Model 2	Model 1	Model 2
Neighbourhood collective efficacy				
My neighbours have close relationships with each other	.32	.32	.57	.58
My neighbours trust each other	.39	.39	.64	.64
Elderly neighbours will scold me if I smoke or drink in the neighbourhood	.44	.43	.72	.72
My neighbours will intervene or report to the police if I am assaulted by other kids in the neighbourhood	.62	.61	.75	.74
I will let elderly neighbours know if my friends smoke or drink in the neighbourhood	.62	.64	.53	.53
I will intervene or report to the police if my friends are assaulted in the neighbourhood	.65	.65	.54	.54
Family violence				
I frequently see my parents verbally abuse each other	.58	.62	.59	.69
I frequently see one of my parents beat the other one	.70	.76	.71	.79
I am often verbally abused by parents	.79	.83	.82	.83
I am often severely beaten by parents	.72	.75	.73	.78
Youth antisocial behaviour				
Unauthorised school absence	.56	.54	.76	.73
Group bullying	.64	.73	.65	.70
Severe teasing or banter	.76	.78	.72	.76
Threatening	.91	.74	.88	.86
Drinking	.58	.53	.74	.70
Smoking	.77	.79	.82	.78
Severely beating others	.81	.68	.81	.80
Robbing	.88	.78	.71	.89
Stealing	.76	.78	.64	.58
Running away	.67	.59	.79	.78
Fare evasion	.61	.61	N/A	N/A
Shouting at teacher	.61	.64	N/A	N/A
Cheating on exam	.66	.55	N/A	N/A
Misappropriating expenses for school supplies	.63	.53	N/A	N/A
Gang fight	N/A	N/A	.76	.78

Note. All factor loadings show $p < .001$. N/A = Item not available in cohort.

Chapter 6

Pathways of child resilience to maternal depression: Individual, family, and socioeconomic factors in the 2004 Pelotas (Brazil) Birth Cohort

Chapter rationale

Maternal depression is a well-established risk factor for child psychology. Although previous studies have identified individual, family, and social factors that may contribute to positive mental health outcomes in children exposed to maternal depression, these have been mainly examined individually, as opposed to jointly, and are based on data from high-income countries. Therefore, this final study examined pathways of child resilience to maternal depression using data from a Brazilian birth cohort.

This declaration concerns the article entitled:

Pathways of child resilience to maternal depression: Individual, family, and socioeconomic factors in the 2004 Pelotas (Brazil) Birth Cohort

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Candidate's contribution to the paper (provide details, and also indicate as a percentage)	The candidate contributed to / considerably contributed to / predominantly executed the… **Formulation of ideas:** Andreas Bauer contributed to the formulation of ideas for the current study (50%). **Design of methodology:** The data used in the current study was part of a larger data set. Andreas Bauer contributed to the design of the methodology in the current chapter (50%). **Experimental work:** Andreas Bauer contributed considerably to the analysis and interpretation of results for this study (70%). **Presentation of data in journal format:** Andreas Bauer contributed considerably to the presentation of data in journal format (70%).
Statement from Candidate	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature.

Signed	[redacted]	**Date**	27.06.2021

Pathways of child resilience to maternal depression: Individual, family, and socioeconomic factors in the 2004 Pelotas (Brazil) Birth Cohort

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Abstract

Background

The negative impacts of maternal depression on child mental health are now well-documented. However, many children exposed to maternal depression show positive mental health outcomes, demonstrating psychological resilience. In a large birth cohort from Brazil, a middle-income country, we examined individual, family, and socioeconomic factors and their pathways that may differentiate resilient from non-resilient children exposed to persistent maternal depression.

Methods

Using data from the 2004 Pelotas Birth Cohort (*N*=4231), we limited the sample to those exposed to persistent maternal depression up to age 6 years (depression present at ≥2 out of 5 assessment waves; *n*=1132; 50% boys). Resilience at age 11 years was defined as having scores lower than or equal to those of unexposed children on the parent-reported Strengths and Difficulties Questionnaire. First, we examined univariable associations of SES (assessed at birth) and child cognitive stimulation (assessed at 24 and 48 months) with resilience status. Then, we examined pathways from SES to resilience via cognitive stimulation and IQ (assessed at 6 years), and from cognitive stimulation to resilience via IQ, using counterfactual mediation.

Results

In univariable analyses, SES and cognitive stimulation were not associated with resilience status, after adjusting for covariates. However, using counterfactual mediation analyses, there was evidence of indirect pathways from SES to resilience via cognitive stimulation and IQ. Furthermore, there was evidence of a total effect of cognitive stimulation on resilience via IQ.

Conclusions

These findings suggest that the promotion of cognitive stimulation and cognitive development in early childhood has the potential to protect children from the harmful effects of maternal depression.

Keywords: Resilience, Maternal depression, Socioeconomic status, Cognitive stimulation, IQ, Mediation

Introduction

Maternal depression is a highly prevalent psychiatric disorder, affecting approximately 20% of women globally, and represents a major public health concern (Hahn-Holbrook, Cornwell-Hinrichs, & Anaya, 2017). There is strong evidence for an association between maternal depressive symptoms and offspring negative outcomes in several domains, including mental and physical health, cognitive function, and socioemotional development (Meaney, 2018; Slomian, Honvo, Emonts, Reginster, & Bruyere, 2019). Negative mental health outcomes, including internalizing and externalizing problems, are particularly well-documented for this group (Goodman et al., 2011). However, many at-risk children, even when exposed to persistent and severe maternal depression, present positive mental health outcomes, thereby demonstrating psychological resilience, i.e., an ability to cope with and adapt to adversity (Collishaw et al., 2016; Khambati, Mahedy, Heron, & Emond, 2018; Mahedy et al., 2018; Savage-McGlynn et al., 2015).

Resilience has been conceptualized in numerous ways in the literature, and can be broadly defined as a dynamic ability to maintain psychological health in the face of adversity or trauma (Masten & Powell, 2003; Sisto et al., 2019). Resilience is a complex construct and can be thought of as a capacity which can be developed over time, rather than an inherent trait of the individual (Rutter, 2012). Since resilience cannot be measured directly but must instead be inferred on the basis of other measures (e.g., good mental health despite experiences of adversity), the conceptualization, assessment and operationalization of resilience vary widely across studies (Cosco et al., 2017). However, one common approach is the person-centred method, which compares individuals exposed to similar types or levels of adversity to ascertain what differentiates those who remain well from those with less optimal outcomes according to predefined criteria (Cosco et al., 2017; Masten & Powell, 2003). A thorough understanding of

how resilience develops is relevant for the identification of intervention targets to prevent maternal depression from leading to negative child outcomes.

Previous studies examining predictors of resilience in children exposed to maternal depressive symptoms identified the importance of factors on the (i) *individual level*: for example, high levels of child self-esteem, self-efficacy, and IQ (Collishaw et al., 2016; Lewandowski et al., 2014; Pargas, Brennan, Hammen, & Le Brocque, 2010; Savage-McGlynn et al., 2015); (ii) *family level*: for example, paternal emotional support, maternal positive parenting and warmth, and low marital conflict (Collishaw et al., 2016; Mahedy et al., 2018; Savage-McGlynn et al., 2015; Silk et al., 2007); and (iii) *socioeconomic level*: for example, higher SES, maternal educational level, and neighbourhood quality (Giallo et al., 2017; Masten & Powell, 2003). These factors are likely to be interrelated, and the accumulation of a range of protective factors may be more effective in mitigating against childhood adversity than any individual factor alone (Collishaw et al., 2016; Hammen, 2003).

Child cognitive development is one factor that may contribute to child resilience through its association with a number of other factors, indicating the multiple levels of influence. Thus, inadequate early child cognitive stimulation has been found to be a key determinant that prevents children from attaining their developmental potential in the context of maternal depression and exposure to violence (Walker et al., 2007). In previous research using the same birth cohort as used in the current study, a brief measure of child cognitive stimulation was found to be a strong predictor of optimal child development in several domains, including motor, communication and cognitive development, with stronger effects in children of less-educated mothers (Barros, Matijasevich, Santos, & Halpern, 2010). Furthermore, higher levels of early child stimulation at age 48 months were associated with better executive functioning at age 11 years (Matijasevich et al., 2019). Therefore, we may expect that early

cognitive stimulation protects against childhood adversity, such as maternal depression, and may be involved in several pathways linked to resilience development.

To date, the majority of studies examining child resilience in the context of maternal depression have used cross-sectional designs (Cosco et al., 2017; Hammen, 2003). However, longitudinal studies have the potential to provide a better understanding of the underlying dynamic processes, as well as ensuring the temporal order between exposure, outcome, and potential mediating variables (Cosco et al., 2017). Furthermore, previous research has mostly been limited to identifying resilience factors at either the individual, family, or socioeconomic level, rather than examining each of these levels simultaneously. The majority of studies on resilience have been conducted in high-income countries and evidence from low- or middle-income countries is lacking (Herba, Glover, Ramchandani, & Rondon, 2016). Differences between developing and developed countries regarding, for instance, social and economic inequalities, cultural influences, and access to healthcare may play an important role in how children experience life adversities and develop coping strategies as a response (Herba et al., 2016). Although we would expect that children from any part of the world would benefit from early cognitive stimulation, evidence from low- and middle-income countries is imperative to the development of context-specific interventions that reflect the wide variability among nations and cultures (Barry, Clarke, Jenkins, & Patel, 2013; Herba et al., 2016).

Using data from a population-based, prospective birth cohort from Brazil, a middle-income country, we aimed to examine the pathways of individual (child IQ), family (child cognitive stimulation), and socioeconomic (family SES) factors contributing to the development of resilience in 11-year-old children exposed to persistent maternal depressive symptoms in early life. More specifically, we examined whether SES and cognitive stimulation would emerge as predictors of resilience status. Furthermore, we investigated direct and indirect effects using counterfactual mediation analysis to examine specific pathways of these

variables. We hypothesized that higher SES will exert: i) direct positive effects on child resilience status, and ii) indirect effects operating through higher levels of cognitive stimulation and higher child IQ scores. Furthermore, we also hypothesized that iii) early cognitive stimulation will predict increased resilience via direct and IQ-mediated indirect pathways, even after adjusting for the effects of SES.

Methods

Participants

The 2004 Pelotas Birth Cohort is an ongoing population-based, prospective longitudinal study, including all hospital births (> 98% of all deliveries) in 2004 occurring in the city of Pelotas, Rio Grande do Sul (South Brazil). Out of 4263 live births, 4231 children (99.2%; 51.9% boys) were included. Mothers and their children were assessed at birth, 3, 12, 24, and 48 months, and 6 and 11 years. The current study focused on a subsample of children who were exposed to persistent maternal depression as defined below (exposed group, n = 1132; 50.0% boys) with the remaining unexposed group (n = 2430; 52.2% boys) being excluded. Further details about the cohort and data collection process can be found elsewhere (Santos et al., 2011; Santos et al., 2014).

Measures

Socioeconomic status

SES was assessed at birth by maternal self-report, using three continuous variables: (1) family income in the month prior to the child's birth, measured in Brazilian Real; and (2) maternal and (3) paternal education, coded as complete school years of formal education.

Cognitive stimulation

At ages 24 and 48 months, mothers were asked four questions related to child cognitive stimulation in the past week (Barros et al., 2010). The four items asked whether the child: (1) was read to or told a story; (2) went to a park or playground; (3) visited another person's home; and (4) had a children's book at home. All items were coded dichotomously.

IQ

At age 6 years, children completed a short form of the Wechsler Intelligence Scale for Children-III (WISC-III), which has been validated for use in Brazil (Wechsler, 2002). The WISC-III short form was composed of two verbal (*similarities* and *arithmetic*) and two performance (*block design* and *picture completion*) subtests, and has been shown to correlate highly (r = .94) with full-scale IQ in children aged 6 years (Kaufman, Kaufman, Balgopal, & McLean, 1996).

Resilience

Resilience status was determined based on exposure to persistent maternal depression and levels of child emotional and behavioural problems.

Maternal depression was assessed at 3, 12, 24, and 48 months, and 6 years, using the self-reported Edinburgh Postnatal Depression Scale (EPDS) (Cox, Holden, & Sagovsky, 1987). The 10 items are each rated on a 4-point scale (0-3), yielding total scores ranging from 0-30. Following a validation study in the same sample (Santos et al., 2007), we used a score of ≥ 10 as an indicator of maternal depression.

As described above, the current sample was limited to those exposed to persistent maternal depression as defined by depression being present at ≥ 2 assessment waves (*exposed* group, n = 1132; 50.0% boys). By contrast, children who were exposed to maternal depression at ≤ 1 time point were assigned to the *unexposed* group and excluded.

The parent-reported Strengths and Difficulties Questionnaire (SDQ) was used to assess child emotional and behavioural problems at age 11 years (Goodman, 2001). We used four subscales, including emotional problems (e.g., "Many worries, often seems worried"), conduct problems (e.g., "Often fights with other children or bullies them"), peer problems (e.g., "Picked on or bullied by other children"), and hyperactivity (e.g., "Restless, overactive, cannot stay still for long"). Each subscale consists of five items, which are rated on a 3-point scale (0-2), yielding overall scores from 0-10. The sum of these four subscales generates a total difficulties score, ranging from 0-40. The Portuguese version of the SDQ has been validated for use in Brazil (Saur & Loureiro, 2012; Woerner et al., 2004).

For our primary resilience outcome, children in the exposed group who scored below or equal to the mean of the unexposed group on all SDQ subscales (i.e., scores for emotional problems ≤ 2.3, conduct problems ≤ 1.1, peer problems ≤ 1.1, and hyperactivity ≤ 2.8) were classified as 'resilient' (n = 139; 12.3% of the exposed group), while n = 993 were classified as 'non-resilient'. However, as there is no predefined way of capturing resilience on the SDQ, we also constructed a second, more inclusive resilience category based on the total difficulties score. By this definition, children in the exposed group who scored below or equal to the unexposed group's mean SDQ total difficulties score (i.e., a score of 7.3) were classified as 'resilient' (n = 394, 34.8% of the exposed group), while those who scored above the mean score (n = 738) were classified as 'non-resilient'. We re-ran all analyses for our more stringent resilience categorisation with this more inclusive secondary outcome in order to assess the robustness of our findings.

Covariates

All models were adjusted for child sex (*male* or *female*) and parental marital status (*married/living with partner* or *single/living alone*).

Analysis strategy

All analyses were performed in Mplus, Version 8.1 (Muthén & Muthén, 2017).

Measurement model

Items contributing to the SES and cognitive stimulation measures were either on different scales (SES) or collected at more than one time point (cognitive stimulation) meaning that simple sum scores may not be appropriate and/or reliable. We therefore derived two latent factors, including SES with 3 indicators and cognitive stimulation with 8 indicators, which were specified as a confirmatory factor analysis (CFA) model with correlated factors to assess model fit with the following indices: Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI), Root Mean Square Error of Approximation (RMSEA), and Standardised Root Mean Residual (SRMR), with values of ≥ .95, ≤ .06, and ≤ .08, respectively, indicating good model fit (Hu & Bentler, 1999).

Univariable analyses and counterfactual mediation

First, we examined univariable associations between exposure variables (SES, cognitive stimulation) and the outcome variable (resilience), using binary logistic regression analyses. Appendix 1 presents schematic diagrams of the two models tested.

Second, to examine pathways linking SES and cognitive stimulation to resilience, we performed a series of counterfactual mediation models. Counterfactual mediation is the recommended approach when using common (> 10%) binary outcomes (VanderWeele, 2015). However, this meant that it was not possible to examine two mediators sequentially, as this method is not yet available for counterfactual mediation with latent variables. More specifically, the indirect effect from SES to resilience via both cognitive stimulation and IQ could not be examined. Thus, we constructed a series of single-mediator models representing the hypotheses that: (1) cognitive stimulation is a mediator of the association between SES and resilience; (2) IQ is a mediator of the association between SES and resilience; and (3) IQ is a

mediator of the association between cognitive stimulation and resilience (Figures 1-3 present schematic diagrams for each mediation model). We assessed the *natural direct effect*, *natural indirect effect*, and *total effect* (see Appendix 2 for effect definitions).

All models were adjusted for child sex and parental marital status. Additionally, the models examining cognitive stimulation as an exposure variable were adjusted for SES. There were small amounts of missing data for family income, maternal education, and cognitive stimulation (≤ 2%), whereas IQ (5.9%) and paternal education (22.2%) showed higher rates of missingness. To use all available data but maintain a consistent sample size across all models, we used a maximum likelihood (ML) estimator and restricted analyses to those with complete data on IQ, which resulted in a sample size of 1065 participants. Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs), which are expressed in standardised units (i.e., per increase in standard deviation). ORs for mediation effects were calculated based on predicted probabilities from probit regression analyses using the 'model indirect' command in Mplus (Muthén & Asparouhov, 2014). Results for counterfactual mediation are based on 1000 bootstrap samples.

Our main analyses focused on resilience as defined by equal to or below average scores on all four SDQ subscales. However, in exploratory analyses, we also examined whether the results replicate when using a less strict definition based on the SDQ total difficulties score, which are presented in Appendix 3.

Results

Descriptive statistics

Table 1 presents descriptive statistics for those included in the analysis (exposed group) and those excluded (unexposed group). Mothers in the exposed group reported lower family income and parental education than mothers in the unexposed group, albeit with small effect sizes (*r*s

ranging between .18-.28, all *p*s < .001). Similarly, mothers in the exposed group reported that their child received lower levels of cognitive stimulation than those in the unexposed group; again, these differences were small (both *r*s = .15, all *p*s < .001). Finally, children in the exposed group had a mean IQ 5.2 points lower than children in the unexposed group ($r = .19$, $p < .001$).

Measurement model

The two-factor CFA model, including SES with 3 indicators and cognitive stimulation with 8 indicators, showed acceptable model fit (CFI = .88; TLI = .85; RMSEA = .10; SRMR = .10). Proportions of explained variance ranged between .18-.76 for SES and .01-.87 for cognitive stimulation. Proportions of explained variance for each indicator and the correlations among latent factors are presented in Appendix 4.

Univariable analyses

To examine relationships between exposure and outcome variables, we conducted univariable analyses on the association between SES and resilience, as well as cognitive stimulation and resilience, after adjusting for child sex and parental marital status (see Appendix 2 for schematic diagrams of the two models).

Using our primary conceptualisation of resilience (i.e., low scores on all four SDQ subscales), there was no evidence of an association between SES and resilience (OR 1.04, 95% CI 0.84-1.29) or cognitive stimulation and resilience (OR 1.80, 95% CI 0.98-3.30).

Counterfactual mediation

Next, in a series of single-mediator models adjusted for child sex and parental marital status, we examined pathways linking SES, cognitive stimulation, and IQ to resilience.

First, we examined cognitive stimulation as a mediator of the association between SES and resilience (see Figure 1). There was no evidence of a direct effect from SES to resilience, when accounting for the effect of cognitive stimulation (OR 0.60, 95% CI 0.28-1.12). However,

there was evidence of an indirect effect of SES on resilience via cognitive stimulation (OR 1.74, 95% CI 1.04-3.34). Finally, there was no evidence of a total effect (i.e., direct plus indirect effect) from SES to resilience (OR 1.05, 95% CI 0.79-1.32).

Second, we examined IQ as a mediator of the association between SES and resilience (see Figure 2). Again, there was no evidence of a direct effect from SES to resilience, when accounting for the effect of IQ (OR 0.87, 95% CI 0.61-1.22). However, there was evidence of an IQ-mediated indirect effect from SES to resilience (OR 1.19, 95% CI 1.01-1.42). Again, there was no evidence of a total effect (OR 1.03, 95% CI 0.77-1.30).

Finally, we examined IQ as a mediator of the association between cognitive stimulation and resilience (see Figure 3). We found no evidence of a direct effect (OR 1.04, 95% CI 0.99-1.15) or indirect effect via IQ (OR 1.83, 95% CI 0.99-3.64) from cognitive stimulation to resilience. However, when combining both the direct and indirect effects, there was evidence of a total effect from cognitive stimulation to resilience (OR 1.90, 95% CI 1.03-3.68).

In sum, while in univariable analysis, there was no evidence of an association between SES and resilience, when using counterfactual mediation there was evidence of indirect pathways from SES to resilience via cognitive stimulation and IQ. Furthermore, while cognitive stimulation was not significantly associated with resilience in univariable analysis, there was evidence of a total effect via IQ (i.e., direct + indirect effect).

Sensitivity analyses

We re-ran the above analyses using our secondary, less stringent definition of resilience (i.e., a low SDQ total difficulties score) in order to evaluate the robustness of our findings. First, in univariable analyses, while SES emerged as a positive predictor of resilience (OR 1.32, 95% CI 1.13-1.54), there was again no evidence of an association between cognitive stimulation and resilience (OR 1.22, 95% CI 0.82-1.83).

Second, using counterfactual mediation, indirect effects from SES to resilience via cognitive stimulation and IQ observed when using the strict definition of resilience were not replicated when using the more inclusive definition of resilience. Instead, there was only evidence of a total effect from SES to resilience via either cognitive stimulation and IQ. Similarly, while there was evidence of a total effect from cognitive stimulation to resilience via IQ in our main analysis, cognitive stimulation was not associated with resilience when using the inclusive definition of resilience (see Appendix 3 for full reporting and effect sizes).

Discussion

We investigated pathways that may contribute to the development of resilience in children exposed to persistent maternal depression. We found indirect pathways from SES to resilience via cognitive stimulation and IQ, such that higher SES was associated with higher levels of cognitive stimulation and higher IQ, which, in turn, were each positively associated with resilience. Additionally, children who received higher levels of cognitive stimulation were more likely to be in the resilient group (total effect), even after adjusting for SES. However, contrary to our initial hypothesis, there was no evidence that the effects of early stimulation on resilience status operate through child IQ (indirect effects). In sum, while SES may mainly promote resilience via indirect pathways (such as child cognitive stimulation and IQ), cognitive stimulation may additionally influence resilience status in conjunction with other variables (total effect with child IQ).

SES in early life is a key determinant of both positive and negative mental health outcomes across the life course, especially low family income and parental education, which are strong predictors of child mental health problems (Reiss, 2013; WHO, 2014). However, as shown in the current study, SES may not directly influence resilience to maternal depression, but instead operates through several indirect pathways during an individual's life. For example,

higher SES households may provide more resources, and, consequently, a potentially more enriching and stimulating environment for the child, which may also positively influence IQ development (Giallo et al., 2017). Indeed, as reported in previous studies and observed in the current study, early cognitive stimulation, such as reading stories to children or having children's books at home, is associated with improved cognitive functioning in children and adolescents (Byford, Kuh, & Richards, 2012; Gartland et al., 2019). The high factor loading of 'had a children's book at home' item in our cognitive stimulation factor further indicates that such material resources related to cognitive development may play a major role in buffering against adverse childhood experiences.

Higher cognitive abilities, commonly assessed by IQ, are also linked to higher resilient outcomes in children facing adversities, including maternal depression (Khambati et al., 2018; Lewandowski et al., 2014; Pargas et al., 2010). Pargas et al. (2010) found that high IQ was the most consistently observed protective factor against maternal depression and a marker of *persistent resilience*, i.e., resilience over the individual's lifespan. A possible explanation could be related to an enhanced cognitive capability to self-regulate (e.g., through verbal communication) or to engage in other coping strategies that buffer against maternal depressive symptoms. Although IQ is a well-established resilience factor, the mechanisms that underpin its protective effects are still under-investigated and should be considered in future studies.

Low SES usually co-occurs with other adverse conditions (e.g., material hardship, inadequate housing, and neighbourhood deprivation) that can have cumulative and interactive effects on child development (Bradley & Corwyn, 2002; WHO, 2014). Maternal mental health status is an important factor involved in the complex pathways linking family SES and child mental health outcomes, with maternal depression acting as one more stressor in an unfavourable environment (Reiss, 2013). Material hardship (food insecurity, poor-housing quality or unmet medical needs), which is much more likely to occur in low SES families, can

be another explanatory pathway linking SES to resilience (Ashiabi & O'Neal, 2007; Gershoff, Aber, Raver, & Lennon, 2007). Gershoff et al. (2007) found that low family income and material hardship together impacted child behaviour through parent-mediated variables, and a reduction in material hardship was found to be associated with lower parental stress and positive parenting abilities, such as warmth and cognitive stimulation to the child. Positive parenting behaviour, in turn, had a protective effect on children exposed to maternal depressive symptoms, and was linked to higher levels of offspring resilience (Brennan, Le Brocque, & Hammen, 2003; Kirby, Wright, & Allgar, 2020). Additionally, Ashiabi and O'Neal (2007) showed that although family income is independently associated with child health, it also exerts strong indirect effects through material hardship and parental factors. Taken together, assessing family SES (e.g., family income and parental education as defined in the current study) in isolation may be an insufficient way of measuring a family's lack of adequate material resources and basic needs that impact children's health (Neckerman, Garfinkel, Teitler, Waldfogel, & Wimer, 2016). Material hardship is a multi-dimensional construct that captures a wide range of family deprivation and its assessment may add more information regarding the pathways of child resilience development (Neckerman et al., 2016).

A further potential pathway that explains why early cognitive stimulation contributes to resilience development could be that it is related more broadly to the quality and/or quantity of parent-child interactions. The cognitive stimulation scale used in our study comprises four markers of material resources and opportunities/attention given to the child, with a particularly high proportion of explained variance for the item 'had a children's book at home'. However, it is possible that higher scores on this scale are a proxy for a broader dimension of positive parenting practices and nurturing behaviours that may lead to better than expected mental health outcomes even in the presence of maternal depression (Zimmerman, Glew, Christakis, & Katon, 2005). In fact, Giallo et al. (2017) found that maternal involvement in cognitively

stimulating activities such as reading books or playing with their child was associated with offspring emotional and behavioural resilience even in the presence of maternal depression. The authors highlight that the interaction between a caregiver and the child during playing, talking or telling stories activities can provide opportunities to create a responsive environment and also strengthen the child-mother connection (Giallo et al., 2017). These findings suggest that interventions targeting depressive mothers, especially those from low SES families, offering cognitively stimulating materials or activities may result in benefits to the child through three possible pathways: (1) improvement of child cognitive functioning, which are directly associated with resilience outcomes; (2) promotion of positive child-caregiver experiences that may develop other non-cognitive skills related to positive mental health; and (3) support for depressive mothers to engage in and maintain activities that promote parental warmth and a sensitive environment. As cognitive stimulation is also related to material resources, low-income families would benefit the most from these interventions as they are more vulnerable to stress caused by economic strain and are also often financially unable to provide enriching materials to their child (Crosnoe et al., 2010; Gershoff et al., 2007).

As the development of resilience may be influenced by a multitude of factors, the rationale for its definition needs to be comprehensive and integrative to capture the variations observed in the population (Masten & Powell, 2003; Southwick, Bonanno, Masten, Panter-Brick, & Yehuda, 2014). We primarily focused on a relatively strict definition of resilience, with children having below average scores on all SDQ subscales. Using this strict definition we can be relatively confident of having identified a group of children free of current behavioural and emotional problems. However, the inclusion of more than one resilience operationalization may be a more informative approach to studying its determinants and underlying mechanisms (Southwick et al., 2014). Therefore, in exploratory analyses we used a more inclusive definition of resilience (defined as a low SDQ total difficulties score), SES was

more influential in determining resilience status than cognitive stimulation, as children from higher SES backgrounds were more likely to be in the resilient group (total effects). This more inclusive definition of resilience may include children with elevated levels of problems in one or more domains, but still below average SDQ scores overall. Nonetheless, the discrepant findings across our two definitions of resilience mean that the current findings require replication. Future consensus in terms of how mental health resilience is defined is needed to support robust investigations in this area.

The current study has a number of strengths. We used data from a prospective longitudinal study based on a birth cohort with a very large sample size and high retention rates, spanning from birth to age 11 years. Additionally, in contrast to previous cross-sectional studies, exposure, mediating and outcome variables were measured at consecutive time points, which is important when interpreting specific developmental pathways as it ensures the temporal order between the variables. In addition, maternal depression was measured repeatedly across the child's life in a way that allowed us to identify families in which the mother suffered from persistent depression. Moreover, previous research mainly focused on one definition of resilience, rather than considering other conceptualisations, such as defining resilience strictly or inclusively as in the current study. Finally, to the best of our knowledge, this is one of the very few studies that examines pathways of child resilience to maternal depression in a middle-income country. To date, most evidence regarding the impacts of maternal depression on child mental health outcomes has been generated in high-income countries, although the vast majority of the world's children live in low- and middle-income countries (Parsons, Young, Rochat, Kringelbach, & Stein, 2012). Further studies conducted in developing countries are needed to address this critical gap in the literature, especially considering that maternal depression and known risk factors for both maternal depression and

poor child mental health (e.g., lack of material resources, socioeconomic disadvantages, and limited access to healthcare services) are more frequent in these settings (Herba et al., 2016).

There are also some limitations that need to be considered. First, all variables assessed in our study were based on maternal self-report, including child mental health, which increases the chance of shared rater bias. Measures of cognitive stimulation and child mental health may be influenced by reporting bias, including due to the presence of maternal depressive symptoms (Breslau, Davis, & Prabucki, 1988; Najman et al., 2000; Najman et al., 2001). However, as our sample is entirely composed of women who experienced persistent depressive symptoms, informant bias in our study may be less of an issue than in other contexts, e.g., if non-depressed mothers had also been included in the analysis. Second, the estimates of the indirect effects from SES to resilience through IQ may be overestimated as we could not adjust for the effects of cognitive stimulation, which represents an intermediate confounder (i.e., a confounder of the mediator-outcome association which is on the causal pathway between the exposure and outcome). Methods for accounting for intermediate confounders are not yet available using counterfactual mediation with latent variables. Similarly, the absence of a total effect from SES to resilience in the counterfactual mediation models may be explained by the occurrence of inconsistent mediation. Inconsistent mediation, also known as a suppressor effect, is present when the direct and mediated effects of an independent variable on a dependent variable have opposite signs, resulting in an absence of the total effect (Loeys, Moerkerke, & Vansteelandt, 2014; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Oher possible explanations for the lack of a total effect in the presence of indirect effects include lack of power to test the total effects and the presence of unmeasured mediator-outcome confounding (Loeys et al., 2014; MacKinnon et al., 2002). Third, data regarding paternal depression was not available and therefore our findings only apply to mothers. However, there is an increasing awareness of the

role that paternal mental health can have in child development and wellbeing (Gutierrez-Galve et al., 2019; Lewis, Neary, Polek, Flouri, & Lewis, 2017; Pearson et al., 2013).

In conclusion, the findings from the current study show that despite the deleterious effects of persistent maternal depression on child mental health, around 10% of children present positive mental health outcomes. The study provides evidence that children of higher SES families were more likely to be resilient not only due to socioeconomic advantages, but also as a function of higher early cognitive stimulation and IQ levels. However, children who experienced more cognitive stimulation in early childhood were more likely to show positive mental health outcomes despite exposure to persistent maternal depression, regardless of their socioeconomic background. Although family socioeconomic disparities depend on a multitude of contextual factors, early cognitive stimulation represents a modifiable protective factor against the deleterious effects of maternal depression. Preventive interventions focusing on cognitive stimulation activities in early childhood, especially targeting at-risk families, may be effective in promoting child mental health resilience.

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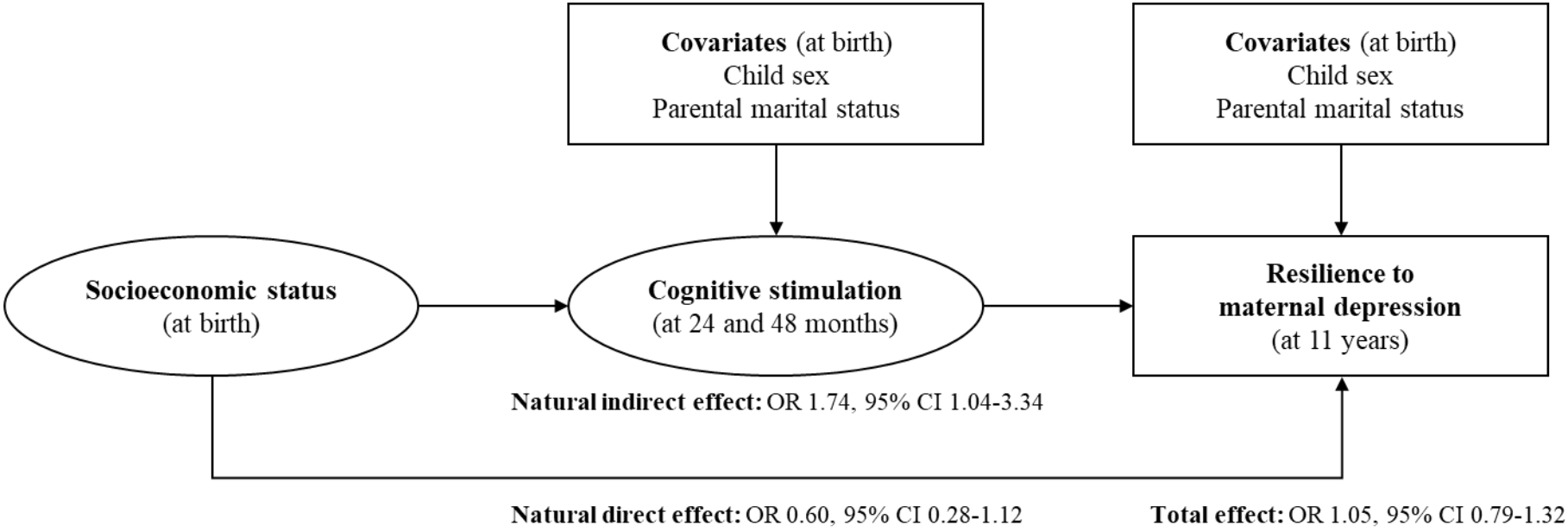
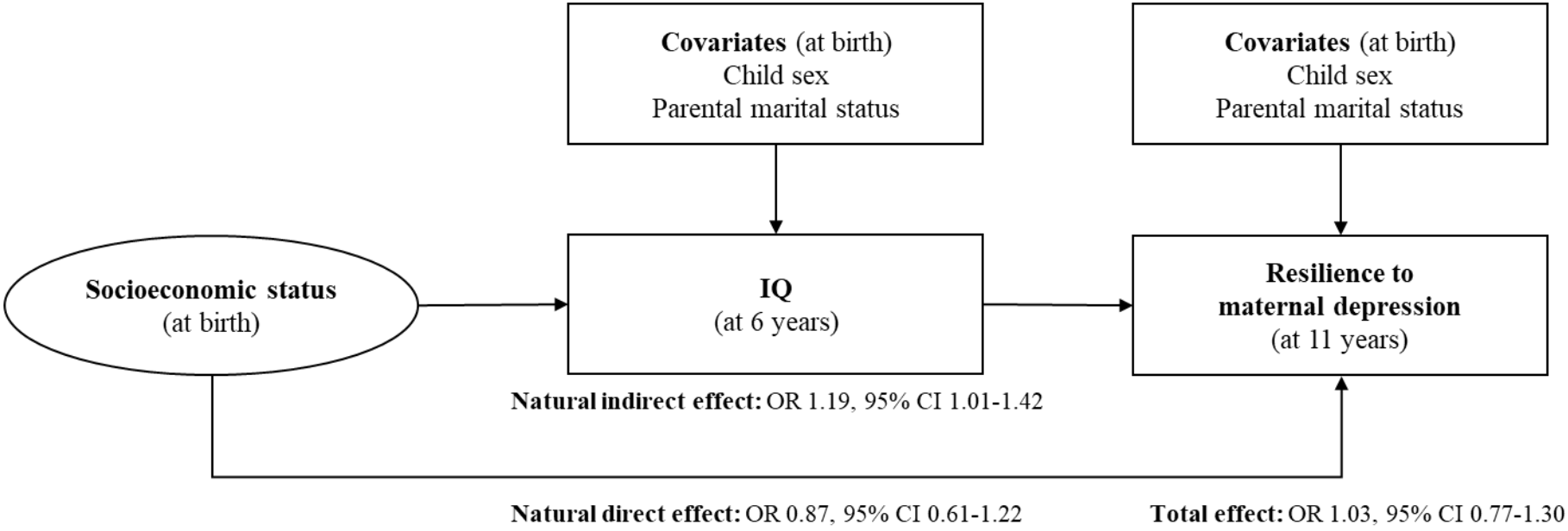
Figure 3 Single-mediator model representing the hypothesis that cognitive stimulation is a mediator of the association between socioeconomic status and resilience to maternal depression, using counterfactual mediation and after adjusting for covariates



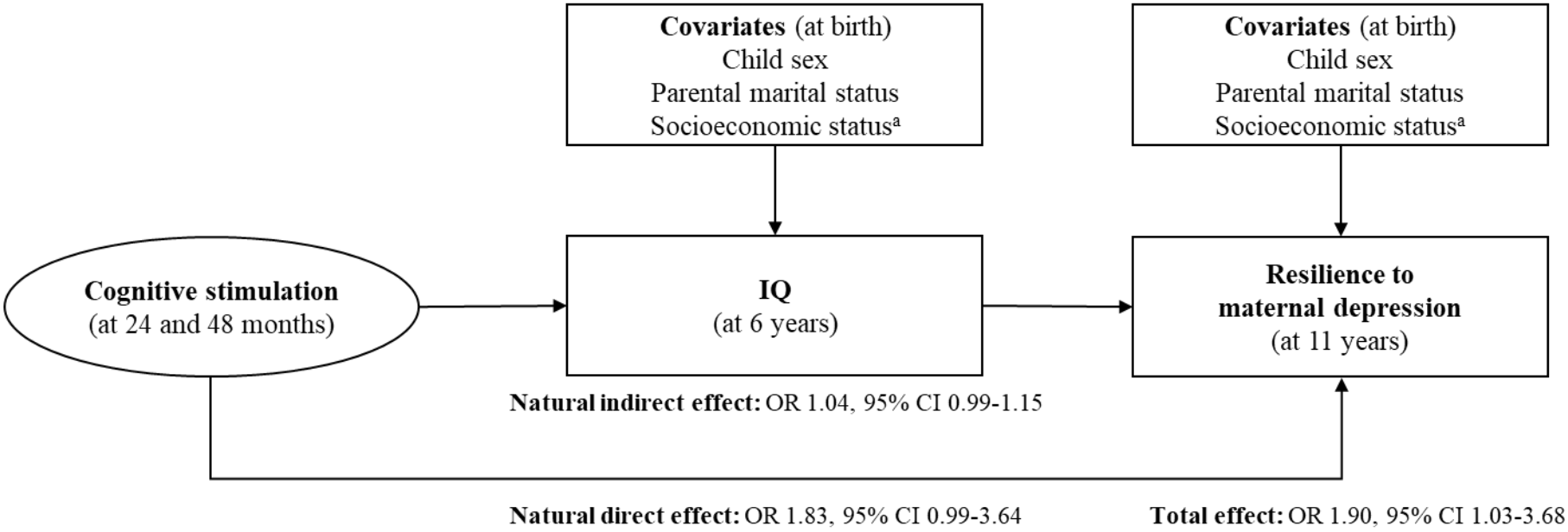
Note. Effect definitions for counterfactual mediation are provided in Appendix 2. Latent variables are presented as ellipses. Observed variables are presented as rectangles.

Figure 4 Single-mediator model representing the hypothesis that IQ is a mediator of the association between socioeconomic status and resilience to maternal depression, using counterfactual mediation and after adjusting for covariates



Note. Effect definitions for counterfactual mediation are provided in Appendix 2. Latent variables are presented as ellipses. Observed variables are presented as rectangles.

Figure 3 Single-mediator model representing the hypothesis that IQ is a mediator of the association between cognitive stimulation and resilience to maternal depression, using counterfactual mediation and after adjusting for covariates



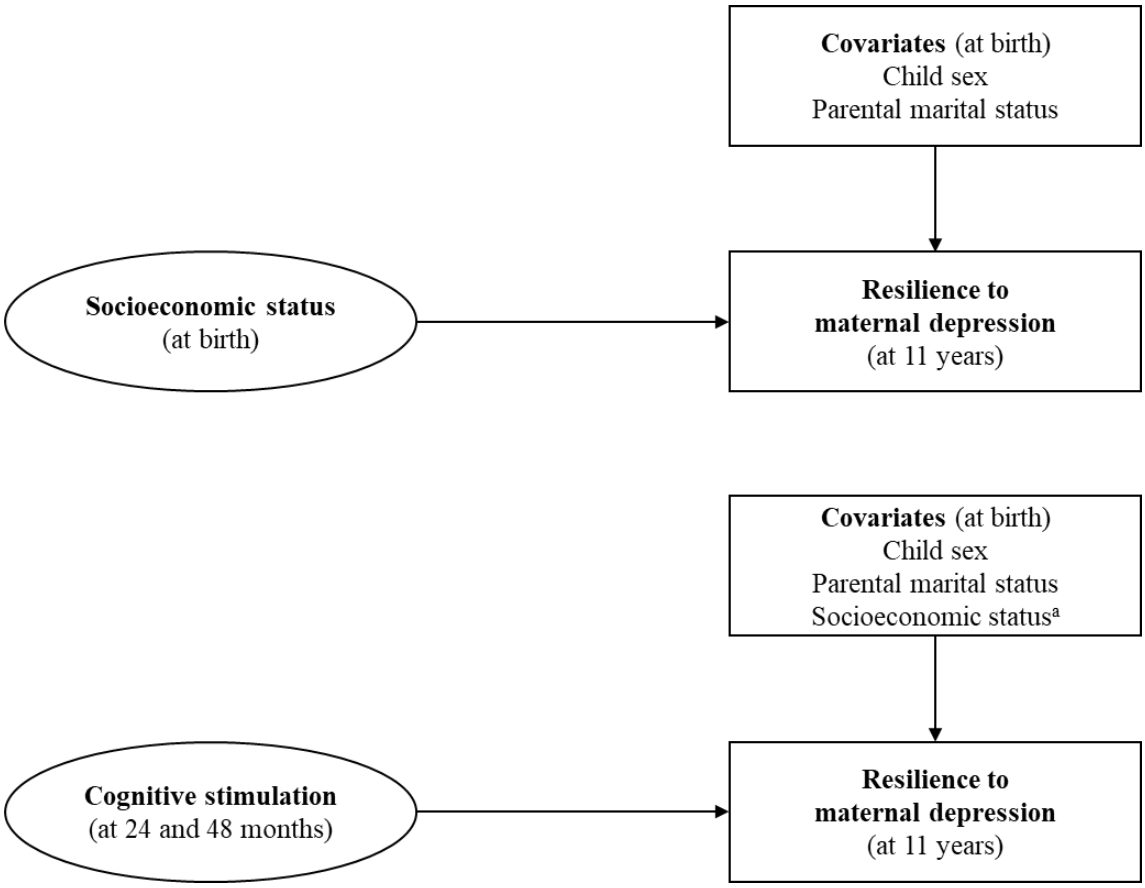
Note. Effect definitions for counterfactual mediation are provided in Appendix 2. Latent variables are presented as ellipses. Observed variables are presented as rectangles. [a] Latent variable.

Table 1 Descriptive statistics for those exposed versus unexposed to persistent maternal depression in the first 6 years of life

	Exposed (*n* = 1132) Mean (SD) or %	**Unexposed** (*n* = 2430) Mean (SD) or %	**Effect sizes of group differences**
Socioeconomic status			
Monthly family income (BRL)	567.9 (889.0)	911.9 (1164.4)	$r = .18$, $p < .001$
Maternal education (years)	7.0 (3.1)	8.7 (3.4)	$r = .28$, $p < .001$
Paternal education (years)	7.0 (3.4)	8.4 (3.7)	$r = .21$, $p < .001$
Cognitive stimulation (0-4)			
At 24 months	3.1 (1.1)	3.3 (1.1)	$r = .15$, $p < .001$
At 48 months	3.7 (1.1)	3.9 (1.0)	$r = .15$, $p < .001$
IQ (possible range: 40-160)	93.3 (14.4)	98.5 (15.6)	$r = .19$, $p < .001$
Sex (female)	50.0	47.8	OR 1.09, 95% CI 0.95-1.26

Note. Observed, rather than latent, variables are presented. BRL = Brazilian Real (2.89 BRL = 1 USD in January 2004 when recruitment of the families commenced). *r* = Correlation coefficient. OR = odds ratio. CI = Confidence interval.

Appendix 1 Univariable associations of SES and cognitive stimulation with resilience to maternal depression, after adjusting for covariates



Note. Latent variables are presented as ellipses. Manifest variables are presented as rectangles. [a] Latent variable.

Appendix 2 Effect definitions for counterfactual mediation

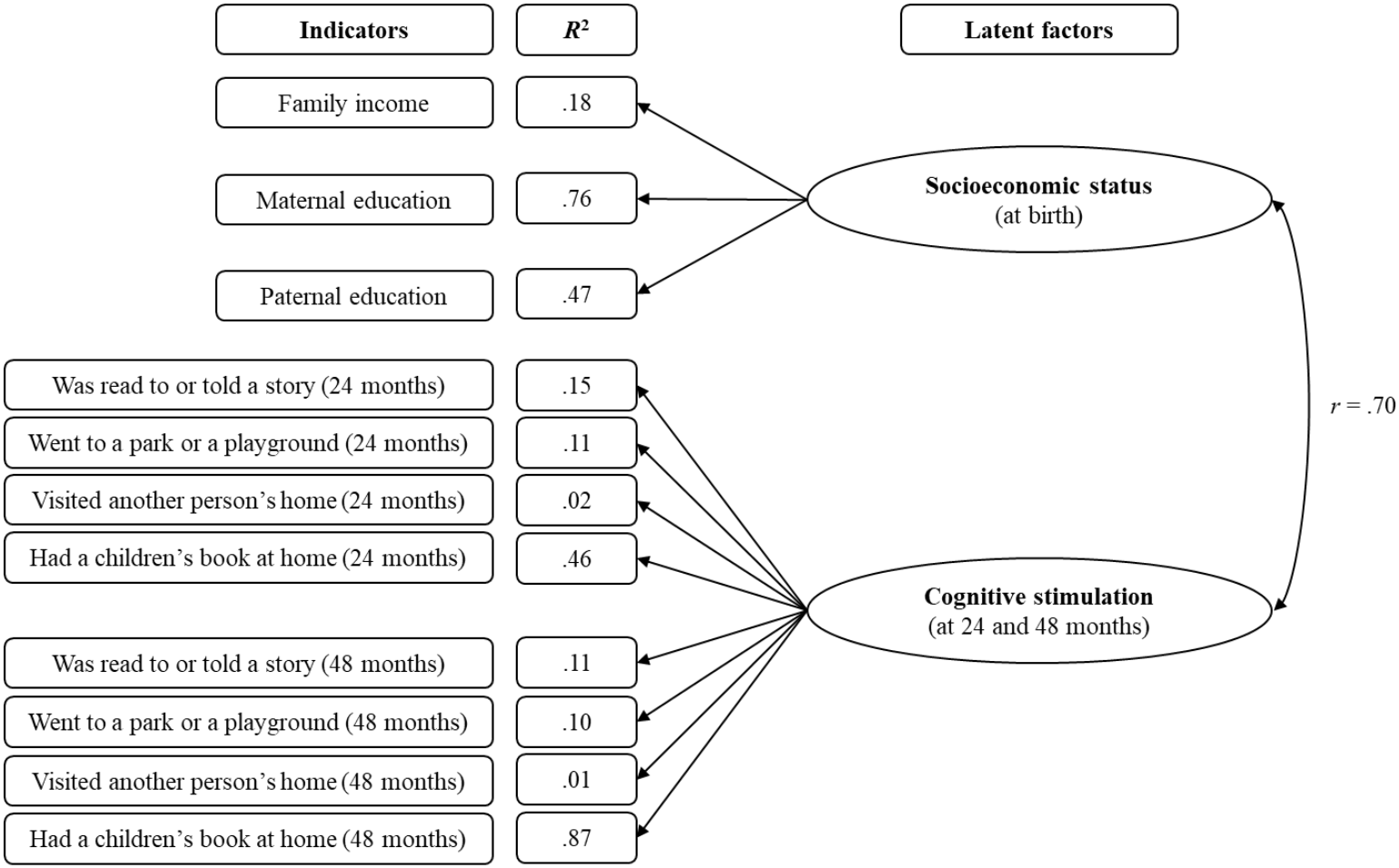
Natural direct effect $Y_i(a,M_i(0))$ to $Y_i(a^*,M_i(0))$	The effect of changing the exposure from the exposure level of interest a (e.g., the population mean) to the comparison level a^* (e.g., 1 standard deviation above the population mean), while fixing the mediator M to the level it would take under the exposure level of interest a.
Natural indirect effect $Y_i(a^*,M_i(0))$ to $Y_i(a^*,M_i(1))$	The effect of changing the mediator M to the level it would take in response to changing the exposure from the exposure level of interest a (e.g., the population mean) to the comparison level a^* (e.g., 1 standard deviation above the population mean), while fixing the exposure to the comparison level a^*.
Total effect	Natural direct effect + natural indirect effect

Appendix 3 Results from counterfactual mediation models when using the *inclusive* definition of resilience (i.e., a low Strengths and Difficulties Questionnaire total difficulties score)

	OR (95% CI)
SES → cognitive stimulation → resilience	
Natural direct effect	1.15 (0.73-1.84)
Natural indirect effect	1.19 (0.83-1.78)
Total effect	**1.36 (1.15-1.64)**
SES → IQ → resilience	
Natural direct effect	1.23 (0.99-1.56)
Natural indirect effect	1.11 (0.99-1.23)
Total effect	**1.37 (1.14-1.63)**
Cognitive stimulation → IQ → resilience	
Natural direct effect	1.21 (0.76-1.90)
Natural indirect effect	1.03 (1.00-1.10)
Total effect	1.25 (0.77-1.92)

Note. Bold values indicate statistically significant associations.

Appendix 4 Measurement model specified as a confirmatory factor analysis model with correlated factors



Note. R^2 = Proportion of explained variance. r = Correlation coefficient.

Chapter 7 – General Discussion

The current thesis examined the relationship between childhood adversities and child and adolescent psychopathology, especially focusing on population-based samples from low- and middle-income and non-Western countries and conduct problems. More specifically, it examined: (1) the potential contribution of childhood trauma exposure to psychiatric disorders in children; (2) longitudinal associations between harsh parenting and child conduct and emotional problems; (3) associations between timing and persistence of child abuse and developmental trajectories of conduct problems from childhood to adolescence; (4) the potential protective effect of neighbourhood collective efficacy on family violence and youth antisocial behaviour; and (5) individual, family, and social factors contributing to child resilience in the presence of maternal depression. Collectively, these studies provide a consistent picture in relation to the links between childhood adversity and mental health, across a range of developmental stages, types of exposure, and cultural and socioeconomic contexts.

Summary of empirical chapters and key findings

The first empirical chapter of this thesis used data from the 2004 Pelotas Birth Cohort, a population-based study from Brazil, a middle-income country. It examined whether the effects of trauma exposure on risk for psychiatric disorders at ages 6 and 11 years were general or specific. Furthermore, it contrasted the effects of interpersonal versus non-interpersonal trauma on risk for psychiatric disorders, while also accounting for their co-occurrence. More than one third of children in this middle-income country cohort had experienced a traumatic event before reaching adolescence. As such, this study provides unique evidence relating to the potential impacts of trauma exposure during development in a context in which childhood trauma is relatively common. By as early as age 6 years, childhood trauma was associated with a significant increase in the odds of 'any psychiatric disorder' in this sample, and this effect was similarly evident at age 11 years. Consistent with previous research, interpersonal trauma

exposure was associated with a particularly robust pattern of effects, being linked to increased odds of 'any psychiatric disorder' and all major diagnostic groups – anxiety, mood, ADHD/hyperactivity, and conduct/oppositional disorders. For non-interpersonal trauma, the pattern of effects was less consistent; nonetheless, non-interpersonal trauma was still associated with increased odds of 'any psychiatric disorder', and anxiety and mood disorders specifically, even after adjusting for co-occurring interpersonal trauma. Consistent with studies from high-income countries, such as the US, the findings provide stronger evidence for general, as opposed to specific, effects of trauma exposure on psychopathology, supporting transdiagnostic models of childhood trauma.

The second study in this thesis presented findings on cross-lagged associations between harsh parenting and child conduct and emotional problems in children aged 6 and 11 years, again using the 2004 Pelotas Birth Cohort. In this examination of child symptoms, it was possible to take full advantage of the prospective longitudinal design. The results demonstrated bidirectional associations between harsh parenting and child conduct problems (i.e., harsh parenting at age 6 years predicted child conduct problems at 11 years, and vice versa), and a unidirectional effect from harsh parenting to child emotional problems (i.e., harsh parenting at age 6 years predicted child emotional problems at 11 years, but *not* vice versa), with no robust evidence for sex differences in these associations. Overall, these findings present a crucial validation and extension of observations from high-income countries to a middle-income country cohort of different patterns of associations between harsh parenting and conduct versus emotional problems in children. The results suggest that links between harsh parenting and child psychopathology may be robust across different social and cultural norms, underscoring the potential universality of the detrimental effects of harsh and abusive parenting.

The third study in this thesis used data from the Avon Longitudinal Study of Parents and Children (ALSPAC), a population-based UK birth cohort, to investigate links between

abuse timing and developmental trajectories of conduct problems. The first stage of analysis involved identifying developmental trajectories of conduct problems in children aged 4-17 years, extending previous work by Barker and Maughan (2009) in the same sample. Whereas previous research using conduct problem trajectories has focused on abuse experienced during childhood, the current study incorporated exposure measures covering both childhood and adolescence, enabling investigation of the impact of timing and persistence of child abuse. The key findings were as follows: (i) abuse exposure was associated with substantially greater odds of being in the early-onset persistent and adolescence-onset conduct problem classes, particularly when abuse was present across both childhood and adolescence; (ii) abuse that only occurred in adolescence was not associated with conduct problems trajectory membership; (iii) there was no evidence of stronger associations between child abuse and membership of the early-onset persistent compared to the adolescence-onset class, which is in contrast to some previous findings; (iv) there was no evidence of an association between abuse exposure and increased odds of being in the childhood-limited class. Overall, the findings suggest that: conduct problems with an onset in adolescence show similar associations with abuse to conduct problems that emerge in childhood and persist, with any differences between these trajectories being quantitative rather than qualitative in nature; and timing of exposure to abuse is likely to be less relevant to our understanding of child conduct problems than the accumulation of exposure, as abuse in adolescence was still harmful, however, only as part of an ongoing pattern of abuse that spans childhood and adolescence. These observations reaffirm the critical importance of interventions to address harsh and abusive parenting behaviours, regardless of developmental stage.

The fourth empirical chapter examined potential effects of neighbourhood collective efficacy in mitigating against family violence and youth antisocial behaviour, using two nationally representative, longitudinal cohorts from South Korea. Across both the younger

(including children aged 10-12 years) and older (including children aged 15-17 years) cohorts, longitudinal analyses demonstrated that higher levels of collective efficacy predicted lower levels of family violence, and higher family violence predicted higher levels of youth antisocial behaviour. Thus, collective efficacy led to a decrease in youth antisocial behaviour through its effect on family violence. Contrary to expectations, there was no direct effect of collective efficacy on youth antisocial behaviour. The results were largely identical when examining the same relationships for domestic violence and child abuse separately. In line with previous empirical chapters using data from Brazil and the UK, the results reaffirm the importance of family-level adversities on the emergence of child behaviour problems in a South Korean context. However, this chapter also introduced potential protective factors, such as positive neighbourhood characteristics, that have been linked to antisocial behaviour and may operate more indirectly through proximal mechanisms, such as mitigating the effects of family violence.

Extending this focus on potential protective factors, the final empirical chapter of this thesis applied a different framework, examining the factors that may contribute to resilience to a well-established risk factor – persistent maternal depression. Again, this study used the data from the 2004 Pelotas Birth Cohort. Building on previous chapters, this study took a multi-level approach to examining the pathways through which social- (family SES), family- (child cognitive stimulation), and individual-level (child IQ) factors contribute to positive mental health outcomes in children exposed to maternal depression. There was evidence of an indirect effect from SES on resilience via cognitive stimulation (e.g., reading to the child) and child IQ. Furthermore, there was a evidence of a total effect (i.e., the joint influence of direct and indirect effects) from cognitive stimulation on resilience through IQ. In sum, cognitive stimulation seemed to be an early modifiable protective factor in predicting resilience in children exposed to persistent maternal depression.

Overall, the current thesis provides strong evidence of a relationship between childhood adversities and child and adolescent psychopathology. This link has been consistently demonstrated across five studies using population-based samples from three diverse international contexts: the 2004 Pelotas Birth Cohort (Brazil), a higher-risk, middle-income country sample; ALSPAC (UK), a low-risk, high-income country sample; and the Korean Youth Panel Survey (South Korea), again, a low-risk, high-income country sample, but from a collectivist culture.

More specifically, this thesis provides further evidence that childhood adversity is a transdiagnostic risk factor associated with multiple forms of psychopathology. This non-specific effect of adverse and potentially traumatic experiences has been shown when focusing on childhood psychiatric disorders using diagnostic assessment tools (i.e., DAWBA, see study 1), emotional and behavioural difficulties assessed using screening questionnaires (i.e., SDQ, see study 2), and distinct developmental trajectories of conduct problems spanning the period from early childhood to late adolescence (i.e., early-onset persistent and adolescence-onset conduct problems; see study 3).

Despite some evidence suggesting that childhood adversities may be particularly harmful at specific developmental stages (see study 3), collectively, the studies included in the current thesis provide evidence of the harmful effects of childhood adversities irrespective of timing. More precisely, childhood trauma exposure was associated with 'any psychiatric disorder' in children at ages 6 and 11 years (study 1); particularly abuse in childhood but also in adolescence when experienced as an ongoing pattern of abuse that spans childhood and adolescence ('persistent' abuse) was associated with the elevated conduct problem trajectories (see study 3); and family violence was associated with antisocial behaviour in both younger and older children (see study 4).

The current thesis confirms the well-documented effects of adverse family environments, including harsh and abusive parenting (see studies 2, 3, and 4) and maternal depression (see study 5), on child psychopathology. Beyond the family environment, it further shows the far-reaching consequences of childhood trauma across childhood psychiatric disorders (study 1). Importantly, study 1 further highlights the detrimental effects of interpersonal versus non-interpersonal trauma, a distinction that has been mostly limited to the PTSD literature, and especially non-interpersonal trauma which has been largely overlooked in relation to other forms of psychopathology.

The present thesis further highlights two important considerations when examining childhood adversities: (i) the possibility of bidirectional effects between exposure and outcome, as shown in study 2, in which a reciprocal relationship between harsh parenting and child conduct problems was found; and (ii) the multiple interacting levels of influence, as shown in study 4, in which family-level adversities (family violence) on individual-level psychopathology (youth antisocial behaviour) were mitigated by neighbourhood-level processes (collective efficacy), and study 5, in which individual, family, and social factors acted jointly in protecting against the deleterious effects of maternal depression and conferring resilience.

Strengths

Key strengths of the current thesis include the use of large, representative samples with prospective, longitudinal designs from three different countries, including the UK, Brazil, and South Korea. Furthermore, each empirical chapter used advanced statistical methods, including cross-lagged path analysis (study 2), latent class growth analysis (study 3), and mediation analysis (studies 4 and 5), with missing data being addressed using robust techniques, such as multiple imputation (studies 1 and 2), full information maximum likelihood (studies 3, 4, and 5), and inverse probability weighting (study 3). Furthermore, the use of latent variables

minimised measurement bias by allowing each item to contribute individually to the overall score of the underlying construct. Finally, in study 3, we extended existing conduct problem trajectories from ages 4-13 years up to 17 years. The resulting data can be requested from ALSPAC and will enable other researchers to examine associations between other risk and protective factors and conduct problem trajectories.

Limitations

The findings of the current thesis should be interpreted in the context of four major limitations. First, in relation to *how childhood adversities were measured*. Particularly in study 3, child abuse was assessed using retrospective self-report in adulthood, which may have been subject to recall bias. Although, false positives in adult retrospective reports have been shown to be rare (Hardt & Rutter, 2004), they show poor agreement with the kinds of prospective measures (Baldwin, Reuben, Newbury, & Danese, 2019) which have been used in studies 1, 2, and 4. While prospective measures may overcome some of the drawbacks associated with retrospective reports, there are still methodological caveats to consider. More precisely, studies 1 and 2 used caregiver reports of childhood trauma and harsh parenting. However, similar to prospective and retrospective measures, self- and caregiver reports show low agreement, with children reporting higher levels of child abuse than their caregivers (Cooley & Jackson, 2020). The issue of informant discrepancies further extends to officially documented cases of child abuse. More specifically, a recently published systematic review demonstrated that children reported higher levels of child abuse and lower levels of child neglect than documented in their case files (Cooley & Jackson, 2020). Similarly, in a birth cohort from Australia, most children who were registered as child abuse cases reported no history of child abuse when asked in adulthood, and vice versa (Najman et al., 2020). These issues may be particularly exacerbated when assessing child abuse, where caregivers may not disclose harsh and abusive parenting behaviours due to concern about the consequences (e.g., risk of losing their children or being

referred to child protective services), and less concerning when examining the effect of other types of adversity, such as maternal depression. In sum, childhood adversities, and particularly child abuse, are methodologically and ethically challenging to research, especially in large population-based cohorts where time constraints due to the high number of participants often do not allow a multi-informant approach.

Second, in relation to *how child and adolescent psychopathology was assessed*. The majority of the studies included in this thesis used caregiver reports to measure psychopathology. However, similar to the informant discrepancies in the assessment of child abuse, there is low concordance between self- and caregiver reports, with parents underestimating the levels of conduct and emotional problems in their children (De Los Reyes et al., 2015). Furthermore, studies 2 and 3 used the SDQ, a widely used measure with established psychometric properties (Stone, Otten, Engels, Vermulst, & Janssens, 2010). However, in both studies using data from population-based birth cohorts from the UK and Brazil, the conduct and emotional problems subscales showed low reliability, with Cronbach's alphas of mostly less than 0.60. Although low scores may be explained by the scales' efforts to cover a wide range of emotional and behavioural difficulties across a broad age range, these findings demonstrate the need for more reliable measures, which, additionally, may need to be adapted to different age groups. Interestingly, study 4 used a newly developed measure on antisocial behaviour that showed excellent internal reliabilities in both younger and older children, which, however, was criticised for the lack of formal psychometric assessment by peer reviewers. Undoubtedly, new measures need a thorough psychometric assessment before being applied widely. However, similarly, researchers need to continue testing established measures across a wider range of populations. Furthermore, even widely used measures should undergo some level of evaluation in each study sample, and if inadequate reliability and/or validity is detected, this should be clearly stated in the limitations section. Relatedly,

Hammerton et al. (2019) found partial measurement invariance (i.e., some parameters are allowed to differ across groups) between a Brazilian and British birth cohort when assessing the SDQ conduct problems subscale, and some researchers argue that full measurement invariance is required to perform unbiased comparisons (see e.g., Steinmetz, 2018). Thus, one could argue that the SDQ conduct problems subscale measures slightly different underlying constructs in Brazil than in the UK. Unfortunately, cross-cultural studies like the one published by Hammerton and colleagues (2019) are still rare, and demonstrate that testing the psychometric properties of measures should extend beyond assessments of internal reliability (e.g., Cronbach's alpha).

Third, in relation to *low frequencies of childhood adversities*. Despite using data from large, representative samples, prevalence rates of trauma exposure were low. Consequently, in studies 1 and 3, the primary analysis was restricted to an aggregate measure of 'any trauma' and 'any abuse', respectively. Although we were able to examine two broad exposure categories in study 1, namely interpersonal and non-interpersonal trauma, the data did not allow a more detailed examination of specific types of trauma. Similarly, in study 3, results based on physical and psychological abuse need to be interpreted with caution and the specific impact of sexual abuse could not be examined. Finally, the severity, duration, and frequency of childhood adversities were not fully captured in the current thesis, albeit their well-established influence on the association between childhood adversities and psychopathology. Although study 3 included an item on 'persistent' abuse, the measure may have captured two isolated instances of abuse, as opposed to an ongoing pattern of abuse spanning across childhood and adolescence.

Fourth, in relation to *underlying mechanisms*. While two of the studies included in the thesis examined pathways linking childhood adversities to psychopathology, including neighbourhood-level processes (study 4) and social-, family-, and individual-level factors

(study 5), little attention has been given to the potential biological, emotional, and cognitive underpinnings of these relationships. For example, McLaughlin et al. (2016; 2020) proposed accelerated biological aging, and changes in emotional and social information processing as transdiagnostic mechanisms in the association between childhood adversities and psychopathology.

Finally, in relation to *confounding*. First, due to limited data availability, we did not include child IQ as a covariate in most analyses, which, however, has been shown to be one of the strongest predictors of child conduct problems (Murray & Farrington, 2010) – the main outcome variable of the current thesis. Neuropsychological deficits, such as low IQ, may be especially important when examining early-onset persistent conduct problems, as outlined in Moffitt's (2018) developmental taxonomic theory of antisocial behaviour, as well as adolescence-onset conduct problems, as shown by Fairchild and colleagues (2013) in their revised taxonomic model of antisocial behaviour. However, results were almost identical when we examined associations between child abuse and conduct problem trajectories in a UK birth cohort (study 3), additionally adjusting for child IQ. Furthermore, in a systematic review of longitudinal studies specifically focusing on risk factors for antisocial behaviour in LMICs, Murray et al. (2018) found mixed evidence of low child IQ as a risk factor, with many studies reporting null or weak associations. Similarly, in another systematic review focusing on Brazil, Murray et al. (2013) reported that low child IQ was not an independent predictor of conduct problems, which should increase confidence in our findings based on a Brazilian birth cohort (studies 1 and 2). Thus, the importance of IQ as a determinant of conduct problems may depend on the social and cultural context, which requires future research. Nevertheless, future studies should include IQ as a covariate when examining outcomes related to child externalising problems. Second, we did not consider genetic confounding. Approximately 50% of the variance in antisocial behaviour may be explained by genetic factors (Burt, 2009a; Burt, 2009b;

Ferguson, 2010), which in itself is not problematic, because around 50% may be explained by shared and non-shared environmental factors. However, since our exposure variable – *parenting* – in studies 2, 3, and 4 may also be explained by genetic factors (Kendler & Baker, 2007), associations between harsh and abusive parenting and child conduct problems may be overestimated, particularly if more variance is explained by genetic than environmental factors (Barnes, Boutwell, Beaver, Gibson, & Wright, 2014). One way to account for genetic confounding is the use of family-based designs (e.g., twin studies). Alternatively, researchers could use Mendelian Randomisation, in which an instrumental variable is used to strengthen causal inference, without the need for family-based designs. In brief, researchers select a genetic variant that is associated with the exposure of interest (e.g., harsh parenting) but not potential confounders (e.g., maternal education). If there is no direct effect of the genetic variant on the outcome variable (e.g., conduct problems) after accounting for the exposure variable, it is argued that there is a causal relationship between the exposure and outcome variables (Pingault, Cecil, Murray, Munafò, & Viding, 2016). Using this method, it has been shown that previously significant associations between for example smoking and depression disappeared, using data from the UK-based ALSPAC birth cohort (Pingault et al., 2016).

Recommendations for future research

First, more research is needed from low- and middle-income countries. For example, we drew on a meta-analysis on cross-lagged associations between harsh parenting and child conduct problems to inform the research questions and the design of study 2 (Pinquart, 2017). However, the overwhelming majority of the studies included were conducted in high-income countries. Furthermore, a methods paper on conducting systematic reviews on risk factors for child conduct problems and youth crime and violence in low- and middle-income countries demonstrated that 15% of articles would have been missed if the search strategy had been limited to English language articles (Shenderovich et al., 2016). Consequently, there is not only

a need to conduct more studies in low- and middle-income countries, but also to identify the existing ones by using non-English language searches and regional databases. Related to this, study 3 examined developmental trajectories of conduct problems in a UK birth cohort. To date, there is only one published paper on conduct problem trajectories from a low- and middle-income country – namely Puerto Rico (Maldonado-Molina, Piquero, Jennings, Bird, & Canino, 2009). Thus, more research is needed to examine the developmental course of psychopathology in children and adolescents in other cultural and social contexts. The age 15 assessment of the 2004 Pelotas birth cohort has recently been completed and upcoming studies will show whether the same conduct problem trajectories can be identified in Brazil as in high-income countries. Second, more research is needed to take account of informant discrepancies when examining the impacts of childhood adversities on psychopathology, particularly for child abuse and neglect. If possible, researchers should use a multi-informant approach and collect data via self- and caregiver report. Third, we need more studies based in different cultural contexts to better understand risk and protective factors of child psychopathology. For example, unlike in studies from the US, study 4 showed no direct effect of neighbourhood collective efficacy on youth antisocial behaviour. While collective efficacy may be considered more the exception than the rule in Western countries, in a collectivist culture such as in South Korea, the perception of low efficacy may indicate aberrant views to common values or norms. Alternatively, it is possible that there is substantially less variation in collective efficacy in Eastern compared to Western cultures, which may explain the differences in results (although collective efficacy did influence family violence in the expected manner). Thus, more research is needed to establish the measurement invariance of constructs across cultural contexts.

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