The effects of exposure to domestic violence and direct child abuse on child and adolescent internalising symptoms

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Thesis submitted for the degree of Doctor of Philosophy

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2022

Acknowledgements

Summary

Childhood exposure to domestic violence (CEDV) and direct child abuse (DCA), defined as physical and emotional abuse, are serious health concerns. CEDV and DCA are associated with poor developmental outcomes in children and young people including internalising symptoms (IS; anxiety, depression, somatic complaints, social withdrawal), yet many are resilient. Studies have started investigating the causal pathway between both CEDV and DCA and IS, however much work is needed to understand how CEDV and DCA affect IS, and the best ways to provide support.

This thesis aimed to describe the effect of CEDV, and DCA, on child and adolescent IS, and identify factors that protect against IS. Potential protective factors were identified through literature reviews and data from the Avon Longitudinal Study of Parents and Children were used to explore the causal pathways between both CEDV and DCA, and IS. CEDV was measured when children were 0-3 years, DCA at 0-3 and 6-9 years, and IS at 6 and 13 years.

Controversially there was little evidence that CEDV affects IS at 6 or 13 years. DCA had a statistically significant effect on both child and adolescent IS. There was little evidence that any of the included factors protected against the effect of DCA at 0-3 years on IS at 6 years, but many factors appeared to protect against the effect of DCA on IS in early adolescence. Key protective factors were children's social skills, positive parent-child relationships, good maternal mental health, and friendships. These findings have implications for practitioners, providing potential assets to explore and include within formulations, and strengths to develop in preventative interventions.

Table of abbreviations

Abbreviation	Meaning
ACE	Adverse childhood experiences
ALSPAC	Avon Longitudinal Study of Parents and Children
BAME	Black and minority ethnic
CBCL	Child Behaviour Checklist
CEDV	Childhood exposure to domestic violence
CI	Confidence interval
COA	Children of alcoholics
DAG	Directed acyclic graph
DCA	Direct child abuse
DV	Domestic violence
IPV	Intimate partner violence
IPW	Inverse probability weighting
IS	Internalising symptoms
MI	Multiple imputation
NOS	Newcastle-Ottawa Scale
NBRM	Negative binomial regression model
SDQ	Strengths and difficulties questionnaire

Contents

Chapter One. Introduction

1 Overview

This thesis aims to describe the impact of child exposure to domestic violence (CEDV), and direct child abuse (DCA), on child and adolescent internalising symptoms which are recognised to contribute to long-term ill health. Additionally, it aims to describe and explore the relationships between these variables and identify factors to protect children and adolescents against developing internalising symptoms. In this introductory chapter, I provide an overview of the existing evidence first linking domestic violence (DV) to child and adolescent internalising symptoms, and then DCA to internalising symptoms. Finally, I present the gaps in knowledge that I will address in this thesis.

CEDV in the home and DCA are both serious health concerns for children and adolescents, with almost 1 in 5 in the UK exposed to at least one of the two during their childhood (L. Radford, 2011). CEDV and DCA have been shown to be associated with poor developmental outcomes including internalising symptoms (Artz et al., 2014; Leeb, Lewis, & Zolotor, 2011; Norman et al., 2012), yet it is recognised that many are resilient (Tracie O Afifi & MacMillan, 2011; Yule, Houston, & Grych, 2019). Studies have started to consider the causal pathways between both CEDV and DCA and internalising symptoms in terms of mediating and moderating factors (e.g., Miller et al. 2012; Lansford et al. 2006), however there is still much work to be done to understand the causal effects of CEDV and DCA on child and adolescent internalising symptoms and the best way to intervene to prevent such negative outcomes. It is particularly important to explore internalising symptoms in children and adolescents given that they are less recognised and under-researched in comparison to externalising behaviours. Furthermore, research has shown that over half of mental health problems in adults stem from childhood (Kessler et al., 2005) and there is evidence to suggest that if they are treated early they may fully recover and not experience mental health problems in adulthood (Patton et al., 2014). Therefore, it is key to identify causes of internalising disorders such as anxiety and depression, in order to develop preventative interventions.

Thus, in this thesis I aimed to describe the effect of CEDV, and DCA, on child and adolescent internalising symptoms. Additionally, I aimed to identify factors that

protect children and adolescents exposed to DV or DCA against developing internalising symptoms. I conducted a systematic review and a literature review to identify factors that may act as mediators or moderators of the effect of DV on internalising symptoms. I then created a Directed Acyclic Graph (DAG) to inform my analyses and used a population-based cohort study, namely the Avon Longitudinal Study of Parents and Children (ALPSAC), to explore the causal pathways between both CEDV and DCA and internalising symptoms. CEDV was measured when children were 0-3 years old, DCA when children were 0-3 and 6-9 years, and internalising symptoms were measured when children were six and 13 years old. Analyses were conducted using longitudinal multivariate regression modelling of multiply imputed and weighted data to account for significant amounts of attrition and missing data within the observational dataset.

The original plan for this thesis was to only explore the causal pathways between CEDV and internalising symptoms, however given that there was no evidence of a causal effect of CEDV on internalising symptoms at six or 13 years, it was concluded that it would be incorrect and futile to continue to explore the causal pathways between these two variables, given there was no total effect. I recognised that the factor which reduced the effect of DV the most was DCA, therefore I decided to change course and explore the risk and protective factors for internalising symptoms amongst those who had been exposed to DCA. This gave me the opportunity to learn how to conduct mediation analyses using data from a large longitudinal cohort study.

2 Background

2.1 Exposure to DV and child and adolescent behavioural symptoms: existing evidence and knowledge gaps

2.1.1 Introduction to DV

DV is a serious public health concern across the world. Definitions of DV vary around the world, yet it is widely accepted among researchers that DV includes not only physical assault such as hitting, kicking, and object throwing, but also emotional/psychological abuse including humiliation and controlling behaviours, as well as sexual abuse (Wathen & MacMillan, 2013). Within the United Kingdom DV is defined as "Any incident of threatening behaviour, violence or abuse (psychological, physical, sexual, financial or emotional) between adults who are or have been intimate partners or family members, regardless of gender or sexuality."

(Home Office, 2013). DV occurs "across society, regardless of age, gender, race, sexuality, wealth and geography" (Wood, Bellis, & Watts, 2010). In England and Wales 4.9 million women (28.3%) and 2.4 million men (nearly 14.7%) have experienced intimate partner violence (IPV), a form of DV, since 16 years of age (Stripe, 2020) and these figures are likely to be conservative; they include data from the Crime Survey for England and Wales, police recorded crime and numerous different organisations. Within this review, DV and IPV are used interchangeably and considered synonymous terms to describe abusive behaviour from one person towards another within an intimate relationship.

2.1.2 Prevalence of childhood exposure to domestic violence (CEDV)

CEDV between caregivers is a serious health concern for children and adolescents. Approximately 1 in 5 in the UK are exposed to DV during their childhood, and roughly 6% of children and young people are exposed to DV between caregivers in a given year (Lorraine Radford, Corral, Bradley, & Fisher, 2013), equating to over 750,000 children. Furthermore, DV appears on nearly two thirds of child protection Serious Case Views (Brandon et al., 2011). It has been estimated that support of children and adolescents exposed to IPV, until they are 28 years old could cost UK taxpayers up to £1.4 billion pounds in education, health and care, residential and crime costs (Economics, 2018). Research interest in CEDV has increased significantly over the past two decades, yet methodological issues regarding lack of standardised definitions of DV and CEDV may have hindered efforts to explore the impact of DV between caregivers on children and young people (Mohr, Noone Lutz, Fantuzzo, & Perry, 2000). Early research focussed on children's direct witnessing of violence between caregivers, and its impact on their functioning (Evans, Davies, & DiLillo, 2008). However, more recent research has recognised that children are affected by DV regardless of whether they witness it directly or not (Kitzmann, Gaylord, Holt, & Kenny, 2003; MacMillan & Wathen, 2014; Øverlien, 2009). Thus, "exposure to DV" is now preferred rather than "witnessing DV" (John W. Fantuzzo & Mohr, 1999) and there is general consensus that exposure includes direct involvement, seeing, hearing and experiencing the aftermath of DV incidents such as tension between partners (Edelson, Edelson, Kerr, & Grandin, 1999; Jaffe, Wolfe, & Wilson, 1990; Ernest N. Jouriles, Norwood, McDonald, & Peters, 2001; Kimball,

2016). Some researchers even prefer the term "experiencing" to "exposure" as it is direct rather than passive (Callaghan, Alexander, Sixsmith, & Fellin, 2018).

2.1.3 Outcomes associated with child exposure to domestic violence

DV is one of many adverse childhood experiences (ACEs), including child maltreatment and household disfunction factors, which have been shown to have long lasting effects into adulthood (Hughes et al., 2017). CEDV is associated with an increased risk of psychological and behavioural difficulties during childhood and adolescence (Evans et al., 2008; Holt, Buckley, & Whelan, 2008; Howell, Barnes, Miller, & Graham-Bermann, 2016), as well as physical health problems, substance misuse, interpersonal violence and self-harm (Hughes et al., 2017). Furthermore, adolescents who have been exposed to DV or community violence are more likely to be victims or perpetrators of dating and relationship violence (Foo & Margolin, 1995; O'Keefe, 1997; M. Schwartz, O'Leary, & Kendziora, 1997).

CEDV has been linked to numerous psychosocial difficulties, health issues, and mental health problems among children and young people (Holt et al., 2008; David A Wolfe, Crooks, Lee, McIntyre-Smith, & Jaffe, 2003). These consequences constitute serious psychosocial and developmental problems in a child's functioning that can have long term effects and create vulnerabilities over the life course (Alaggia & Donohue, 2018). Some argue that CEDV should be considered a form of child maltreatment as the negative impact is similar to that of DCA (Edelson, 2004).

Psychological consequences of CEDV can be considered to fall into 3 groups:

i.   **Internalising symptoms** which are those that are directed inwards such as anxiety, depression, and social withdrawal,

ii. **Externalising behaviours** are those directed outwardly including aggressive behaviour and conduct disorder in childhood,

iii. **Trauma symptoms** are experienced following extreme adversity and include nightmares, flashbacks, and physiological responses to trauma cues, usually alongside internalising and externalising symptoms.

The direct associations between DV exposure and children's internalising, externalising and trauma symptoms have been shown to be robust, with a meta-analysis by Evans et al. (2008) reporting mean weighted effect size d-values of 0.48, 0.47, and 1.54 respectively. However, the larger effect size value for trauma

symptoms was based on evidence from only six studies, and given significant heterogeneity amongst the effect sizes from the individual studies, it should be interpreted with some caution. Given these observed associations, researchers have started to explore potential risk and protective factors to better understand how DV impacts on child health and well-being and to inform the development of interventions that can mitigate the effects of exposure.

It is important to recognise that not all children exposed to DV experience negative outcomes and many function as well if not better than their non-exposed peers.

2.2 What is resilience?

Resilience has been defined as the maintenance of healthy/successful functioning or adaptation within the context of a significant adversity or threat (Garmezy, 1993; Suniya S Luthar, Cicchetti, & Becker, 2000a; Ann S Masten & Obradović, 2006). This healthy functioning includes behavioural competence such as behaviours toward others, emotional competence such as good mental health, social competence such as peer relationships, and academic achievement such as school performance (Walsh, 2011). One aspect of emotional competence is the lack of internalising symptoms (Thomas M Achenbach & Edelbrock, 1991).

Resilience has been studied in the face of many childhood adversities, including child neglect or abuse, separation and loss, family or neighbourhood violence, war, terrorism, natural disasters, poverty, hospitalisation, along with many others. Some studies have focused on a single traumatic experience, such as rape or loss of a parent, while others have measured cumulative risk of multiple negative events or ACEs (Felitti et al., 1998). It is important to recognise that adversities rarely occur in isolation, because the most severe forms of childhood adversity often reflect chronic, repeated, or combined exposures to traumatic events (A. Masten & Barnes, 2018). However, cumulative risk models provide limited insight into unique characteristics that provide protection in the context of a particular risk (e.g., DV). Although there is an argument for investigating risk factors in combination, in order to reflect real-life and the fact that ACEs often co-occur, focussing on an individual risk or adversity might better suit identification of protective factors or predictors of resilience among children and adolescents.

2.3 Impact of CEDV on child and adolescent internalising symptoms: existing evidence and knowledge gaps

Since establishing the association between CEDV and behavioural difficulties, including internalising and externalising behaviours, researchers have started to explore the causal pathways between the two, identifying factors that may contribute to the observed effect. These factors may increase or decrease the risk for those exposed and therefore may be considered to be risk or protective factors. To date, maternal factors such as positive mental health and supportive emotion coaching by mothers have been found to play a significant protective role in the association between CEDV and internalising symptoms. However, the evidence base is sparse, and the majority of studies have focused on risk (Carter, Paranjothy, Davies, & Kemp, 2020). Many findings have been limited by methodological limitations such as an over-reliance on DV shelter and/or clinical samples who may have unique challenges and suffer higher levels of distress than non-shelter and non-clinical samples (John W. Fantuzzo & Mohr, 1999). Furthermore, there has been a reliance on maternal reports of CEDV and internalising symptoms, studies have tended to adopt a cross-sectional design, preventing inferences about the direction of any causal effect to be estimated, and sample sizes have generally been small.

DV rarely occurs in isolation from other adverse childhood experiences (ACEs) such as child maltreatment, parental mental and/or physical health issues and substance misuse, making it a very complex issue. Studies have shown that DV commonly co-occurs with child maltreatment (Appel & Holden, 1998; Brown, Rienks, McCrae, & Watamura, 2019). If studies limit their focus only to DV and do not consider other ACEs, they may overestimate the impact of DV and overlook the possibility that ACEs overlap and have a cumulative effect, therefore affecting children to a greater magnitude. Furthermore, ACEs tend to cluster and therefore focussing on a single aspect of the broader range of issues is less helpful when trying to develop interventions to improve child health and wellbeing.

2.4 Association between CEDV and DCA, and the association between DCA and child internalising symptoms: what is known and gaps in the evidence

DV has been regarded as a form of child maltreatment (Gilbert et al., 2009) as children growing up in violent and abusive households have been found to experience similar rates of internalising and externalising problems to those who have been

directly abused (Moylan et al., 2010). However, it is not always easy to distinguish between the effects of DV and DCA given that that the two often co-occur (Appel & Holden, 1998; Ernest N Jouriles & LeCompte, 1991) and thus, children are often exposed to multiple types of family violence (Dodge, Pettit, & Bates, 1997; Margolin, 1998).

Despite often overlapping and sharing similar negative outcomes (T. I. Herrenkohl, Sousa, Tajima, Herrenkohl, & Moylan, 2008), DV and DCA have traditionally been addressed in isolation by researchers, policy makers and programmes. Interventions addressing one of these in isolation may be less effective if the other is experienced alongside, and targeting both simultaneously may be beneficial.

Understanding how CEDV, DCA and other factors relate to one another, and the relative strength of each as a risk factor, particularly using longitudinal studies, is an important first step in setting the context for intervention and policy level change efforts, to improve the lives of disadvantaged children and families. Moylan et al. (2010) studied the effects of both DV and child abuse and found that, when factors in the child's environment other than DV and child abuse are accounted for, neither DV or direct abuse alone increased the risk of children experiencing internalising symptoms, yet dual exposure did increase the risk compared to non-exposed adolescents. This, however, is likely the majority of exposed children given that many will experience both CEDV and DCA.

2.5 Knowledge gaps addressed in this thesis

CEDV has been associated with long-term negative outcomes including internalising symptoms. Researchers have started to explore factors that increase the risk or protect against internalising symptoms but this research needs consolidating and building upon, utilising longitudinal data and rigorous research methods. Similarly, although the impact of child maltreatment on children's mental health has been explored extensively, fewer studies have explored the effects of individual forms of abuse separately and identified risk and protective factors for internalising symptoms following DCA. I will attempt to address these gaps in the literature in this thesis.

3 Thesis aims and research questions

This thesis sets out to address the following research questions and to achieve that through systematically examining the evidence for the impact of DV on child and

adolescent internalising symptoms and the causal pathways between the two within the international literature; and then to examine the effect and risk and protective factors for internalising symptoms within an English cohort. The specific aims were as outlined below.

3.1 Aims

i. To identify mediators and moderators of the association between CEDV and internalising symptoms within the existing literature

ii. To identify factors that protect children and early adolescents from developing internalising symptoms following CEDV within a population-based birth cohort in England (ALSPAC)

iii. To identify factors that help protect children and adolescents exposed to DV or DCA from developing internalising symptoms

3.2 Research Questions:

i. What factors mediate and/or moderate the effect of exposure to DV on child and adolescent internalising symptoms? (Addressed in Chapter 2)

ii. What factors protect children and young people from the negative effects of trauma and increase resilience? (Addressed in Chapter 3)

iii. What is the effect of CEDV on child and adolescent internalising symptoms within a UK population-based cohort study? (Addressed in Chapter 5)

iv. Do protective factors identified from the literature mediate and/or moderate the effect of early childhood exposure to DV on adolescent internalising problems? (Addressed in Chapter 5)

v. What is the effect of DCA during early years and middle childhood on child and adolescent internalising symptoms? (Addressed in Chapter 6)

vi. What are the mediators and moderators of the effect of DCA on internalising symptoms during childhood and adolescence? (Addressed in Chapter 6)

3.3 Methods used to address research questions

In Chapter 2, I conducted a systematic literature review to consolidate the evidence for mediators and/or moderators of the effect of CEDV on child and adolescent internalising symptoms.

In Chapter 3, I conducted a less rigorous, but extensive review of the wider childhood trauma and resilience literature to identify potential protective factors, given that few

protective factors had emerged from the systematic literature review and I was particularly interested in taking a strengths-based approach. And focussing on protective factors rather than risk factors.

In Chapter 4, I consolidated the evidence from the first two chapters along with a non-structured review of covariates of the effect of CEDV on child and adolescent internalising symptoms, in the form of a directed acyclic graph. In order to identify covariates, I searched the literature for risk factors for CEDV and risk factors for internalising symptoms and then provided evidence for factors that were associated with both the exposure and the outcome. I also introduced the causal inference approach as it is important to understand the fundamentals of the approach, to understand the methods used in the chapters to follow.

In Chapter 5, I conducted the analyses in ALSPAC exploring the causal effect of CEDV on child and adolescent internalising symptoms, which is when I decided to change the course of the thesis, in the interest of my learning and development, as well as to address the gap in the literature. I also introduced methods of dealing with missing data and conducted multiple imputation and inverse probability weighting as a method to overcome large amounts of missing data in ALSPAC.

In Chapter 6, I conducted mediation and moderation analyses using the multiply imputed datasets created in Chapter 5, to identify significant causal pathways between DCA and child and adolescent internalising symptoms.

Chapter 2: Risk and protective factors for internalising symptoms among children and adolescents exposed to domestic violence: A systematic literature review

1 Key Messages
My systematic literature review found that:

- One individual level mediator was identified, namely emotional intelligence and two effect modifiers were identified: relational victimisation and participation in extracurricular activities.

- Familial mediators included maladaptive parenting and parenting stress, while effect modifiers included positive parenting (maternal warmth and availability) and family social support.
- No community level protective factors were identified
- Most research in the field has been conducted in the United States
- There is a lack of research exploring the causal pathway between CEDV and internalising symptoms during early childhood and adolescence and most studies only control for a couple of confounders
- Research has focussed on risk with few studies investigating the effects of protective factors which may be targeted by preventative interventions

My systematic literature review was published in the review journal Trauma, Violence, and Abuse and a copy of the published manuscript can be found here: https://doi.org/10.1177%2F1524838020965964

2 Introduction

2.1. Background

Although CEDV has been shown to be associated with internalising symptoms, as outlined within the first chapter of this thesis, it is important to recognise that not all children and adolescents exposed to DV will develop internalising or externalising problems and some continue to function the same or better than their non-exposed peers. The proportion of children and adolescents considered to be resilient in terms of psychological adjustment has been found to vary greatly depending on the sample used, the severity of DV to which they are exposed, and other underlying factors (Graham-Bermann, Gruber, Howell, & Girz, 2009; Martinez-Torteya, Anne Bogat, von Eye, & Levendosky, 2009). Within a sample of children residing in shelters for victims of IPV, Graham-Bermann et al. (2009) found that 20% did not display internalising or externalising problems. Alternatively, in a sample of children residing in the community, and thus including those exposed to less severe DV, as many as 90% were reported to be psychologically resilient.

Fong, Hawes, and Allen (2017) conducted a systematic review investigating risk and protective factors for externalising behaviours following IPV and reported that child age, gender, callous–unemotional traits, and cognitive appraisals were key

moderators, and maternal mental health and quality of parenting mediated the association between IPV exposure and externalising problems in children (Fong et al., 2017). Although there is likely to be an overlap, externalising behaviours are considered to result in part from poor self-regulation, whereas internalising symptoms are thought to occur from individuals overregulating their thoughts and emotions in a maladaptive way (Dante Cicchetti & Toth, 2014; Merrell, 2008). Internalising and externalising problems have been shown to have different sets of risk and protective factors (contextual and genetic factors) despite their comorbidity (Cotter, Wu, & Smokowski, 2016; Smokowski et al., 2017).

Another recent systematic review synthesised evidence for factors promoting emotional-behavioural resilience in children following exposure to IPV (Fogarty, Wood, Giallo, Kaufman, & Hansen, 2019). Emotional and behavioural outcomes were defined as internalising and externalising difficulties and therefore the authors identified protective factors against such problems. Sufficient evidence was found to suggest positive maternal mental health predicts emotional-behavioural resilience among children exposed to IPV. Other potential factors promoting resilience were emotion coaching, authoritative parenting, and easy child temperament.

My review aimed to extend this evidence base to identify risk and protective factors for internalising symptoms in children and adolescents exposed to DV. Thus, it explored the mechanisms underlying both negative and positive psychological adjustment among children and adolescents exposed to DV. The focus on internalising symptoms is important as such symptoms and disorders are often overlooked among children and young people (Merrell, 2008) meaning that many struggle through their school lives with these "secret illnesses" that are difficult to identify through external observation unlike externalising behaviours (Reynolds, 1992).

2.2 Aims and objectives

This systematic literature review aimed to answer the question "What factors mediate and/or moderate the effect of CEDV on internalising symptoms among children and adolescents?" This question was addressed using the following PICOS (participants, interventions, comparisons, outcomes, and study design):

P: Children and adolescents (age <= 18 years) who have been exposed to DV

I: Mediating or moderating factors

C: compared with those who do not have the factor(s)

O: internalising symptoms

S: cohort, case-control, or cross-sectional studies

Mediating factors are those that help explain the causal pathway through which a variable, in this case exposure to DV, is associated with an outcome (internalising symptoms). Exposure to DV is unlikely to directly cause internalising symptoms and therefore there must be other factors in between leading to negative outcomes. For example, the stress caused by witnessing DV may lead to changes in a child's ability to regulate their emotions and this emotion dysregulation may lead to internalising symptoms. In addition, moderator variables alter the strength of the association between the exposure and the outcome variable of interest so that the association is different under certain conditions of the moderator (Baron & Kenny, 1986). This is also known as an interaction between the moderator and the exposure variables. For example, if gender is a factor that moderates the association between exposure to DV and internalising symptoms, witnessing DV will be associated with internalising symptoms among males or females in a significantly different way.

Mediating factors for internalising symptoms among children and adolescents following exposure to DV are likely to include individual and family factors, and moderating factors are likely to include individual, family, and also community/societal level factors. Individual factors may include emotion regulation, interpretation of the violence, and child age and gender. Family level factors are likely to include maternal/paternal mental health and parenting practices. Community level factors may include peer relations and involvement in school/community activities. Mediators are unlikely to be community/societal level factors as it is unlikely that CEDV will directly impact factors such as peer relations or involvement in school activities, but rather it is likely to impact the victim and child's emotional regulation, which may in turn influence their ability to maintain friendships or take part in activities. These factors may be modifiable through therapeutic interventions such as maternal mental health and emotion regulation or unamenable to change such as gender and ethnicity and therefore non-modifiable. Modifiable factors are of most

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interest when looking to reduce the impact of DV exposure on children and adolescents as they may be focussed upon and altered within interventions.

Mediators and moderators may be protective factors that buffer children and adolescents against internalising symptoms or they may increase the severity of internalising symptoms following exposure to DV. It is important that all potential mediating and modifying factors are examined, regardless of whether they are protective or increase risk, so that we can not only help children and adolescents avoid the negative impact of exposure to DV through the reduction of risks but also support children by enhancing factors which buffer against internalising symptoms, empowering them to overcome and develop resilience against adversity.

3 Method

This systematic review was registered with the PROSPERO International Prospective Register of Systematic Reviews, registration number: CRD42019127012.

3.1 Information sources

The electronic databases EMBASE, PsychInfo and Medline were searched for articles from the "second generation" of research in this area including mediating and moderating variables published between 1st January 1990 and 6th November 2018, to exclude early, poor quality studies with methodological limitations (John W Fantuzzo & Lindquist, 1989). The search strategy included words synonymous with "intimate partner violence", "child or adolescent", and "psychopathology" along with "risk or protective", "mediat* or moderat*", "resilience", and related words. The full search strategy may be found in Appendix A. The past three years (2015-2018) of the key three journals (Child Abuse and Neglect, Journal of Family Violence, & Journal of Family Psychology) were also hand searched to ensure all important, recent research was included. A snowball technique was applied whereby backward reference tracking of all included studies and identified review articles was applied in order to identify further studies.

3.2 Study eligibility criteria

Studies were included if they were original, quantitative, longitudinal studies investigating factors that potentially mediate or moderate the association between child exposure to DV and internalising symptoms in children and adolescents aged from 0 to 18 years old, using validated measure(s) to assess internalising symptoms in

order to allow comparison across studies. If child maltreatment, family violence, stressful life events, or adverse childhood experiences (ACEs) have been studied, studies will be included if data specific to witnessing (being exposed to) DV can be extracted. Studies were excluded if they did not measure internalising symptoms specifically or used a sample of children and/or adolescents from a non-typical population (e.g., children/adolescents with a chronic illness, intellectual impairment, autism spectrum disorder, or physical disability). Papers were limited to peer reviewed articles published in English.

3.3 Study Records

3.3.1 Screening

The database search generated 18,988 results and all references were managed in Endnote v8. Following removal of duplicates and application of search limits, I screened all titles and abstracts of citations and determined the potential for inclusion in the review. A random 5% sample of titles were reviewed by three of my supervisors (SP, AK, AD; 180 each), and a 30% sample of abstracts were reviewed by the three other reviewers to reduce the likeliness of bias within the selection process (Felson, 1992). Inter-rater agreement was very good at both stages (K = .85 and K = .86 respectively). Full texts of relevant citations were obtained and read to determine study eligibility, and if the eligibility criteria were met, data detailing the study characteristics and outcomes were also extracted. A 40% sample were assessed by the three reviewers and again inter-rater agreement was high (K = .87). Any disagreements were discussed and resolved between all reviewers. A further 16 studies were identified through the snowballing techniques described above. Studies were then categorised into themes to detect particular risk or protective factors the mediated or moderated the effect of the exposure on the outcome studied at the individual, familial, and community levels.

3.3.2 Quality and Risk of Bias

Studies were assessed for risk of bias using a combination of the Newcastle-Ottawa Scale (NOS) for Cohort Studies (Wells et al., 2015), and a modified version of the NOS suitable for cross-sectional studies. The NOS assesses risk of bias in terms of selection into the study, comparability, and outcome. The item determining representativeness of the sample, assessed within the selection category, was removed as when investigating causal relationships between variables, the sample does not

need to be representative of the population from which it is drawn (see Rothman et al., 2013). The total number of stars used within the NOS that can be awarded to a study is eight; three for selection within cohort studies and four for cross-sectional studies, two for comparability in both study types, and three for outcome within cohort studies and two for cross-sectional studies.

3.3.3 Analysis
Due to heterogeneity between the studies in methods and outcomes, which was assessed qualitatively, a narrative synthesis of results was conducted and presented by the level of the factor: individual, familial or community.

4 Results

4.1 Study Characteristics and Overview

The search identified 7,772 articles after de-duplication and search limits applied. Following application of the eligibility criteria, the full texts of 121 articles were obtained, and 12 longitudinal cohort studies were included in the review (see Figure 1).
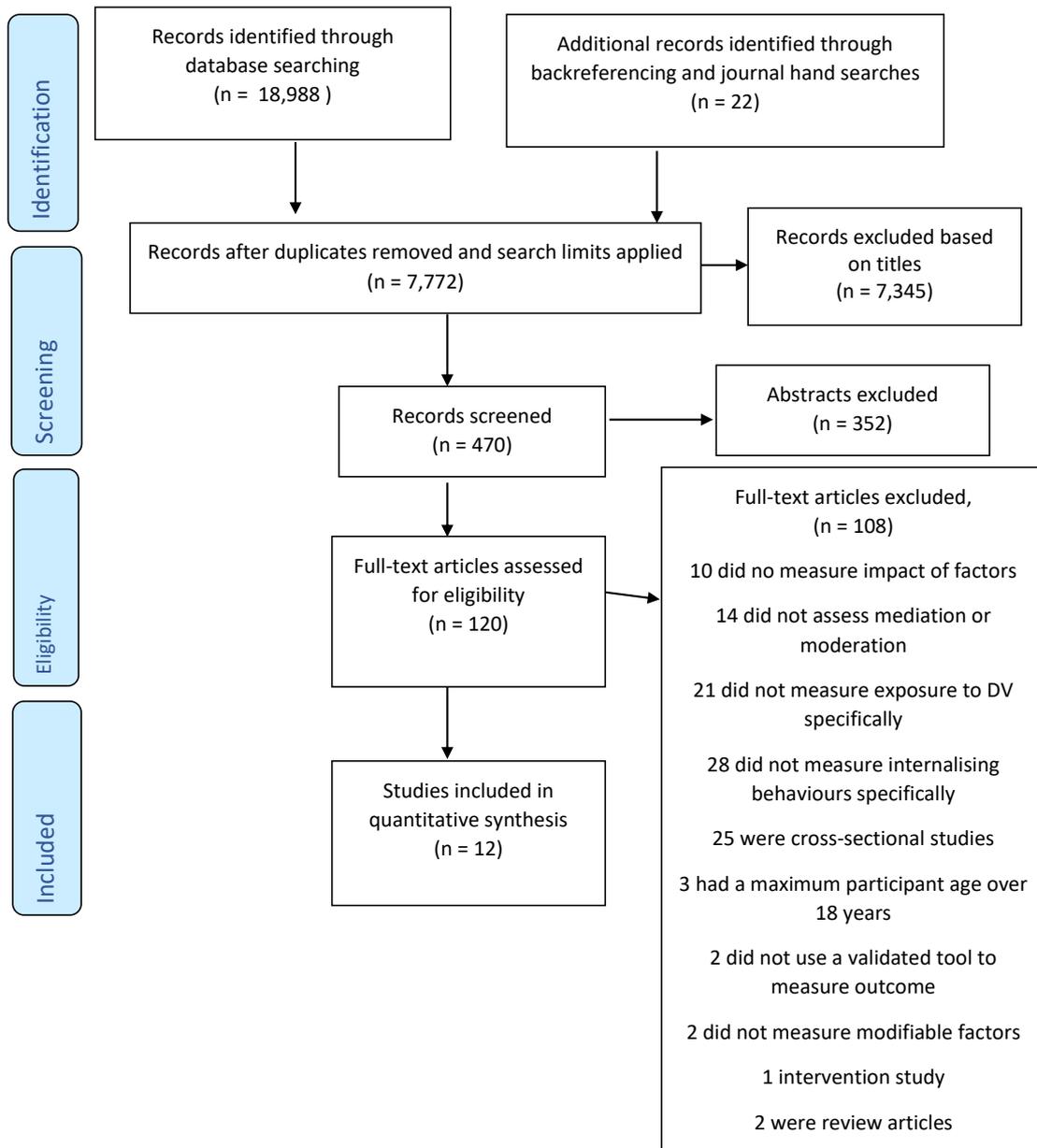
Figure 1. *PRISMA Flow Diagram of Included and Excluded Studies*

All studies were conducted in the United States. All studies focused on outcomes within the primary school–aged group (5–11 years) with four including adolescent outcomes (12–18 years). None of the studies investigated pre-schoolers' outcomes (under 5 years). Three studies investigated individual factors and nine explored familial factors. The average length of follow-up within the studies was 30 months, and the sample sizes ranged from 35 to 6,228 children. Study characteristics including type of DV studied and the measures of DV and internalising symptoms used are presented in Table 1.

Table 1: Characteristics of included longitudinal studies

Authors, Year	Study Description (age range (yrs), gender; follow-up time)	Sample type	N	Type of violence exposure and perpetrator	Measure of DV and informant	Factor(s) of interest	Measure of mediation of effect moderation	Measure of child internalising symptoms and informant
Bair-Merritt, et al 2015	6-9; 51% male; 2 years	Community	270 mother-child dyads	Verbal, physical, sexual abuse, and injury; maternal and partner	CTS-21	Maternal mental health (depression) and parenting stress	Mediation	CBCL[2]
Camacho, et al 2012	10-18; 44% male; 2 years	Community	129 mother-adolescent dyads	Physical abuse and injury; maternal or partner	CTS[3]	Peer relations	Moderation	CBCL[2] internalising subscale
Gardner, et al 2012	9-17; 50% male; 2 years	Community	6,228 children and adolescents	Physical abuse; maternal or partner	CTS[3] - physical aggression subscale	Community activity participation	Moderation	YSR[4]

Gewirtz et al., 2011	6-12; 50% male; 14 weeks	DV shelter and DV court	35 mother-child dyads	Physical abuse; male	Shelter and court reports of DV	Parenting skills and maternal distress	Mediation	CDI-S[5], Levonn[6] and adapted CFS[7]
Huang, et al 2010	1-5; 50% male; 5 years	Community	1234 mother-child dyads	Physical violence, emotional control, sexual abuse; partner	Physical, emotional and sexual abuse was reported by mothers	Maternal mental health, and parenting	Mediation	CBCL[2]
Katz, et al, 2007	4-11; 62% male; 4 years	Community	130 families	Physical violence; maternal and partner	CTS[3]	Emotional competence	Mediation	CBCL[2] - depression scale
Kennedy, et al 2009	8-14; 61% male; 2 years	Community	100 mother-child dyads	Physical and emotional abuse; partner	4-item Child Report of Witnessing IPV Scale[8]	Family social support	Moderation	RCMAS[9]
Kennedy, et al 2010	8-14; 61% male; 2 years	Community	100 mother-child dyads	Physical and emotional abuse; partner	4-item Child Report of Witnessing IPV Scale[8]	Family social support	Moderation	CDI[10]

Rea et al 2005	7-12; 45% male; 12 months	DV shelters and community	191 mother-child dyads	Physical and emotional abuse; maternal and partner	CTS[3] - physical and verbal aggression subscales	Parenting	Mediation	CBCL[2]
Renner et al., 2013	6-17; 49% male; 2 years		1,653 children	Physical and emotional abuse; partner	CTS[3] physical abuse questions and questions from WEB[11]	Parenting stress	Mediation	SSRS[12]
Yoo et al., 2012	5; 52% male; 5 years	Community	1,234 mother-child dyads	Physical, emotional, and sexual abuse; partner	Physical, emotional and sexual abuse reported by mothers	Maternal mental health, parenting, poverty, marital status	Mediation (maternal mental health and parenting), and moderation (poverty and marital status)	CBCL[2]
Yoo et al., 2013	5; 52% male; 5 years	Community	1,234 mother-child dyads	Physical, emotional, and sexual abuse; partner	Physical, emotional and sexual abuse reported by mothers	Maternal mental health, parenting	Mediation	CBCL[2]

[1]CTS-2: Revised Conflict Tactics Scale; Straus, Hamby, Boney-McCoy, & Sugarman, 1996; parent-reported
[2]CBCL: Child Behaviour Checklist; Achenbach & Edelbreook, 1981; parent-reported
[3]CTS: Conflict-Tactics Scale; Straus, 1979; parent-reported

[4] YSR: Youth Self-Report; Achenbach & Rescorla, 2001; child-reported

[5]RCMAS: Revised Measure of Children's Manifest Anxiety Scale; Reynolds and Richmond,

[5] CDI-S: Child Depression Inventory Short-Form; Kovacs, 1985; child-reported

[6]Levonn: Richters, Martinez, & Valla, 1990; child-reported

[7]CFS: Children's Fears Survey; Ramirez, Masten, & Miliotis, 1994; child-reported

[8]Child Report of Witnessing IPV Scale; Allen, Wolf, Bybee, & Sullivan, 2003; child-reported

[9] RCMAS: Revised Children's Manifest Anxiety Scale; Reynolds & Paget, 1983; child-reported

[10] CDI: Child Depression Inventory;

[11] WEB: Women's Experience of Battering Scale; Smith, Smith, & Earp, 1999; mother-reported

[12] SRSS: Social Skills Rating System; Gresham & Elliot, 1999; parent-reported

4.2 Individual Factors

4.2.1. Mediators

Emotional intelligence

Emotional intelligence is the ability to recognise, process, and regulate feelings (Mayer & Salovey, 1997). Emotion regulation, an aspect of emotional intelligence, was thought to lie on the causal pathway between exposure to DV and internalising symptoms. One study (L. F. Katz, Hessler, & Annest, 2007) showed that emotion dysregulation mediated the association between DV exposure at 5 years and internalising symptoms at 11 years such that DV exposure led to emotional dysregulation and subsequent internalising symptoms. Katz et al. (2007) also investigated emotional awareness and reported it to mediate the effect of DV exposure measured at age 5 on internalising problems at 11 years.

This study showed that awareness of negative emotions had a negative association with internalising problems, suggesting that emotional awareness acts as a buffer against internalising symptoms (Katz et al., 2007).

Peer relations

Camacho, Ehrensaft, and Cohen (2012) investigated the indirect effect of peer relation quality in the relationship between IPV and internalising symptoms in a longitudinal study of preadolescents and adolescents aged 10–18 years. The quality of peer relations was assessed by levels of peer support, prosocial behaviours, overt victimisation, and relational victimisation (exposure to behaviours aimed at damaging relationships or social reputation). They reported that peer support received and prosocial behaviour did not act as a mediator on the pathway between child exposure to DV and internalising symptoms.

4.2.2 Moderators

Extracurricular activities

One included study investigated the effect of participating in extracurricular activities and reported that intensive participation in either extracurricular activities or after-school programs, but not moderate participation, has been reported to moderate the impact of DV exposure on internalising symptoms among adolescents, suggesting that it may act as a protective factor within this developmental stage (Gardner, Browning, & Brooks-Gunn, 2012).

Camacho et al. (2012) reported that relational victimisation moderates the effect of DV on children's internalising symptoms such that those who experience high levels of relational victimisation and exposure to DV are at increased risk of more severe internalising symptoms compared to those who have been exposed to DV but have experienced low levels of relational victimisation.

4.3 Familial Factors

4.3.1 Mediators

Parenting skills

Parenting skills are likely to be diminished in violent families, and this in turn is likely to impact on children's internalising symptoms. The "spill-over" hypothesis proposes that "the emotions, affect, and mood generated in the marital realm transfers to the parent-child relationship" (Krishnakumar & Buehler, 2000, p. 26). Thus, hostility within the parental relationship is predicted to reduce parent's abilities to provide warm/responsive parenting and increased hostile and harsh parenting. Five cohort studies investigated the role of parenting skills on the causal pathway between child exposure to DV and internalising symptoms (Gewirtz, DeGarmo, & Medhanie, 2011; Huang, Wang, & Warrener, 2010; Rea & Rossman, 2005; Yoo & Huang, 2012, 2013).

In a longitudinal study of 905 young children, Huang et al. (2010) found insufficient evidence of a pathway between DV at one year of age, negative parenting (unresponsiveness, harshness, and poor communication skills) at year three, and internalising symptoms at year five. However, smacking in particular was found to mediate the pathway between DV and internalising symptoms among the children. Another two studies replicated these findings using the same longitudinal data set (Yoo & Huang, 2012; Yoo & Huang, 2013), although when exploring the moderating role of poverty, Yoo et al., 2012 found that smacking only mediated the effect of DV on internalising symptoms among "non-poor" families. Rea and Rossman (2005) conducted a cohort study with children aged 7–12 years and their mothers, the majority of whom had been exposed to IPV and reported that authoritarian parenting, and in particular verbal hostility, increased the severity of internalising symptoms over time, after controlling for the effects of initial psychological functioning, life

adversity, and exposure to IPV. This suggests that negative parenting acts as a mediator for primary school–aged children but not among preschool-aged children. No study included a sample of adolescents; therefore, the impact of parenting practices on internalising symptoms during adolescence following exposure to DV is unknown.

Maternal mental health

Three cohort studies that investigated the role of maternal mental ill-health on psychological adjustment were included (Gewirtz et al., 2011; Huang et al., 2010; Yoo & Huang, 2013). Overall, evidence suggested that poor maternal mental health does not lie on the pathway between DV exposure and internalising symptoms; however, these studies were conducted with young children under six years, and it was unclear whether maternal mental health may have a greater impact among older children and adolescents. Gewirtz et al. (2011) reported that maternal distress, defined as any current psychopathology or post-traumatic stress symptoms, was only positively associated with depression symptoms in children, among the range of internalising symptoms. In addition, maternal distress did not significantly impact changes in internalising symptoms over time nor did it mediate the association between parenting skills and internalising symptoms. A larger study with a sample of 905 young children also found that, although DV at year 1 was associated with maternal mental health problems at year 3, there was no pathway between DV at year 1, maternal depression at year 3, and children's internalising behaviour problems at year 5 (Huang et al., 2010). Similarly, Yoo and Huang (2013) reported that, among preschool children, maternal anxiety and depression did not mediate the association between exposure to DV at year 1 on internalising symptoms at year 5.

Parenting stress

Evidence for the role of parenting stress on the causal pathway between DV exposure and internalising symptoms among children and adolescents was mixed. Renner and Boel-Studt (2013) conducted a cohort study with approximately 3-years follow-up and found that parenting stress (stress caused by fulfilling the parenting role) fully mediated the association between exposure to psychological IPV and internalising behaviours among 6- to 12-year-olds, but no such pathway was found among adolescents (Renner & Boel-Studt, 2013). Moreover, in this study, no direct or

indirect pathways were found between physical IPV and internalising behaviours in either age group. Bair-Merritt et al. (2015) reported that parenting stress did not mediate the significant association between IPV and internalising symptoms among 6- to 9-year-olds. More research is needed to understand the conditions under which parenting stress accounts for significant variance in children's internalising symptoms following DV exposure. It may be that parenting stress is associated with internalising symptoms through additional mediational pathways such as via parenting skills.

<u>Family functioning</u>
Evidence to support family functioning as protective against developing internalising symptoms is mixed. Owen, Thompson, Shaffer, Jackson, and Kaslow (2009) found that family cohesion may be a protective factor in school-aged children. However, neither maternal reports of family cohesion or family adaptability nor child reports of family adaptability mediated the relationship of exposure to DV and children's internalising symptoms (Owen et al., 2009). Owen et al. (2009) also found weak evidence for a mediating role of family relatedness, defined as emotional quality and proximity seeking to primary attachment figures.

The effect of DV on internalising symptoms was found to be mediated through children's perceptions of the emotional quality of family relationships; however, no such mediating pathway was found for maternal reports of emotional quality or child reports of proximity (Owen et al., 2009).

It may be that family functioning is a more powerful mediator of the link between maternal IPV and children's internalising symptoms in European American children rather than African American families (Nievar & Luster, 2006), yet more research is needed to establish whether this is true.

4.3.2 Effect moderators
<u>Positive parenting</u>
Studies have explored the positive impact of effective parenting. Gewirtz et al.'s (2011) small (N=35), short-term longitudinal study following recent exposure to IPV found that effective parenting moderated the effect of IPV exposure on children's internalising symptoms such that the behaviours decreased over time among those whose mothers demonstrated positive parenting skills, whereas internalising symptoms remained stable over time among those with poorer parenting skills.

Family social support

Evidence suggesting family social support to be protective against developing
internalising symptoms following CEDV is mixed. In a longitudinal study with a
sample of 100 school-aged children, it was found that changes in family social
support moderated the effect of changes in witnessing DV on depression symptoms
over time. However, a three-way interaction showed that the impact of social support
depended on gender. Reductions in witnessing IPV over time were associated with
fewer depression symptoms over time except among boys who reported low levels of
family social support initially (Angie C. Kennedy, Bybee, Sullivan, & Greeson,
2010). This finding implies that social support is a protective factor against depression
following exposure to IPV, particularly among boys. When considering anxiety
symptoms as the outcome, Kennedy and colleagues (2009) found, using the same
sample, method, and procedure, that family social support did not moderate the
association between IPV exposure and anxiety among children.

4.4 Risk of bias in individual studies

Most studies were of reasonable quality given their observational nature and the
sensitive topic; however, all introduced some risk of bias for several reasons. Studies
were also limited by their lack of control group preventing comparison and suggesting
that the negative effect of CEDV on internalising symptoms is assumed to be true.
Furthermore, it cannot be concluded whether mediators and/or moderators have a
significant effect on child and adolescents given CEDV, or whether they have a
significant effect regardless. A full evaluation of the risk of bias using the NOS can be
found in Table 2. Most studies adjusted for several important confounders.

The lack of consistency among measures of DV made it difficult to compare results
and potentially introduced further bias. Few studies included emotional abuse, those
that did sometimes omitted physical abuse, and none mentioned measuring all forms
of DV described above, so the full extent of the abuse was not obtained. Most studies
only investigated DV events within the past year, so the impact of past exposure,
cumulative, and chronic exposure over time was not explored. Furthermore, a few
studies included child reports of DV exposure, yet the majority relied on parental
reports, which may not truly reflect childhood exposure and may have been affected
by social desirability. All the included studies introduced risk of bias using self-report
measures to assess internalising symptoms rather than data linkage to

medical/education records or blind assessment of behaviour by clinicians. Most of the studies also relied on maternal reports of their child or children's internalising symptoms rather than child self-reports introducing potential response bias, as mothers may not be aware of their child's internal suffering resulting in underestimates, or they may overestimate their children's psychological distress if they are stressed and distressed themselves. Studies that included both child and parental reports demonstrated that the two do not always correlate, and therefore, results may not be interpreted confidently. In this context, however, using record linkage to identify internalising symptoms could also introduce bias as only those with severe, clinical levels of internalising symptoms would be recorded, unlike self-report measures.

Table 2. Newcastle-Ottawa Scale Scores for included Cohort Studies

Study	Selection	Comparability	Outcome
Bair-Merritt, 2015	**	**	**
Camacho, 2012	*	**	**
Gardner, 2012	**		**
Gewirtz, 2011	***	**	**
Huang, 2010	*	**	*
Katz, 2007	**	**	**
Kennedy, 2009	***	**	**
Kennedy, 2010	***	**	**
Rea, 2005	**	**	*
Renner, 2013	**	**	**
Yoo, 2012	*	**	*
Yoo, 2013	*	*	*

5 Discussion

This review has consolidated evidence for mediators and moderators on the causal pathway between CEDV and internalising symptoms in children and adolescents that are modifiable. One individual-level mediator was identified, namely emotional intelligence, and two moderators were identified: relational victimisation and participation in extracurricular activities. Familial mediators included maladaptive parenting and parenting stress, while moderators included positive parenting

(maternal warmth and availability) and family social support. No factors were identified at a community level.

This review's findings partially support another recent systematic review, which synthesised evidence for factors promoting emotional–behavioural resilience in children following exposure to IPV (Fogarty et al., 2019). Emotional and behavioural outcomes were defined asinternalising and externalising difficulties, and the authors identified protective factors against such problems only, not risk factors. Our review identified low social support, emotion dysregulation, negative parenting practices (e.g., smacking), and parenting stress to increase the severity of internalising symptoms following exposure to DV. Potential factors promoting resilience identified by Fogarty et al. (2019) included emotion coaching, authoritative (warm and responsive) parenting, and easy child temperament. Emotion coaching may be a moderator that improves children's emotional intelligence, identifiedwithin this review to be a mediator on the causal pathway between DV exposure and internalising symptoms. Positive maternal mental health was also reported by Fogarty et al.(2019) to predict emotional–behavioural resilience among children exposed to IPV; however, this review has found that, when only longitudinal studies are included, poor mental health does not lie on the pathway between DV exposure and internalisingsymptoms; although, this has only been reported among young children (Gewirtz et al., 2011; Huang et al., 2010; Yoo & Huang, 2012; Yoo & Huang, 2013). The studies identified in this review did not include children over the age of 5 years, so it may be that maternal mental health has a greater impact when children are older and have a greater understanding of the situation.

The results from this review have highlighted that there is little evidence to date for the factors that impact the association between exposure to DV and internalising symptoms in adolescence. Increasing age has been found to be protective (Spilsbury et al., 2007), implying recent DV exposure has a smaller effect on internalising symptoms in adolescents. However, other studies have shown that internalising symptoms are still pronounced in adolescence following IPV exposure and argue that adolescence may be a sensitive period for the impact of IPV exposure on internalising symptoms, given the already increased vulnerability to psychopathology due to developmental, behavioural, hormonal, and neurological changes (Menon, Cohen, Shorey, & Temple, 2018; Vu, Jouriles, McDonald, & Rosenfield, 2016). A practical

limitation may be that cohort studies investigating life course effects are expensive, yet longitudinal data do exist and allow exploration of the effects of time between the exposure (DV) and the outcome as well as exposure duration.

5.1 Implications for interventions

This review has identified potential modifiable factors which may be important to target during interventions for children, young people, and their families who have suffered from DV. For example, among the child related factors identified, interventions targeting children's coping skills, emotion regulation, emotion awareness and self-esteem could help prevent children who have been exposed to DV developing internalising symptoms. In addition, challenging children's cognitive appraisals of violence may be a powerful way of breaking the cycle of abuse that is all too common with boys growing up to become perpetrators of DV and girls entering abusive relationships and becoming victims of DV themselves. Parental interventions may benefit from focussing on maternal mental health symptoms and positive parenting so that harsh, punitive discipline is avoided as this is likely to be a further risk factor for children developing internalising symptoms. At a familial level, interventions which aim to foster children's feelings of family cohesion and positive sibling relationships may help buffer children from experiencing negative psychological outcomes following DV exposure. Moreover, at a community/social level, as relational victimisation was found to moderate the link between DV and internalising symptoms (Camacho, 2012), helping children develop positive social skills and confidence, along with protecting them from victimisation by peers, may help prevent children and adolescents from domestically violent families from developing poorer psychological adjustment. Furthermore, encouraging intensive involvement in after-school/community activities may also protect children and adolescents exposed to DV from developing internalising symptoms.

5.2 Limitations

The main limitation of this review is the lack of studies investigating risk and protective factors influencing the link between exposure to DV in childhood and child and adolescent internalising symptoms. This is compounded by the fact that there are many different factors which may impact the relationship, meaning there are only a

couple of studies per factor on average. Therefore, the review provides breadth but not depth to our knowledge of underlying mechanisms in this field and the conclusions that can be drawn about each factor and its role in the relationship between DV and internalising symptoms among children and adolescents are limited.

Most research investigating the effects of DV on children's and adolescents' mental health problems has not teased apart the impact of direct abuse and indirect abuse i.e., exposure to DV. For example, studies investigating the impact of child maltreatment often include both child abuse and witnessing DV. Additionally, studies tend to focus on a limited number of mediating factors, with the majority only investigating one risk or protective factor at a time, disregarding the complex pathways of multiple factors that are likely to be involved in mediating or moderating the association between witnessing DV and negative psychological outcomes.

Another limitation is that, to date, no standardised definition of CEDV has emerged (Holmes, Yoon, & Berg, 2017). However, most researchers agree that exposure to DV occurs when children see, hear, are directly involved in (i.e., attempt to intervene), or experience the aftermath of physical or sexual assaults that occur between their caregivers (Edelson et al., 1999; Ernest N. Jouriles et al., 2001; Wolak & Finkelhor, 1998). This means that the exposure may vary across studies making it difficult to compare results and potentially introducing bias. Moreover, many studies included samples residing in shelters or involved with DV programmes/social services and thus may have oversampled disadvantaged populations with exposure to the most extreme DV and not included children and adolescents exposed to lower levels of DV.

The majority of studies only included male perpetrated violence towards the mother rather than female perpetrated abuse and none considered same-sex couple violence. This may confound the effects of child gender and it is unknown whether factors such as maternal mental health (or paternal mental health), along with other factors, would have the same impact if the DV was female perpetrated. In addition, most studies measured DV in the past year and therefore the impact of chronic versus less frequent violence has not been widely investigated.

Most studies included in this review focussed on risk factors. Risk and protective factors are conceptually distinct (Rutter, 2000). Studies included in this review tended to adopt the traditional risk-outcome approach, focussing on identifying and reducing

risks, and operationalising protection as absence of risk (e.g., poor maternal mental health a risk and absence of psychological disorders protective). In contrast, a strength-oriented approach considers protective factors that are distinct from risk factors (Rutter 2000).

All the included studies were conducted in the United States of America and different factors may be prominent risk factors or key to protecting children and adolescents exposed to DV against internalising symptoms in other countries and cultures. For example, in collectivist cultures, such as those in China and Japan, social support may play a greater protective role on the link between DV and internalising symptoms than in individualist Western cultures such as the USA and UK. Additionally, cultural views and responses towards DV may influence the complex association between DV and child and adolescent internalising symptoms. Thus, it is not clear whether the findings identified in this review are generalisable to other ethnic and cultural groups.

Many of the studies in this systematic review included wide age ranges or crossed developmental and educational stages (e.g., 1-16 and 6-17) which complicates interpretation of findings given that risk and protective factors are likely to vary across developmental stages. Within such studies spanning age ranges, it is unclear whether the examined factor is influential at all ages or whether effects among a certain age group are driving the positive or negative results.

Finally, this review is likely to be affected by publication bias with an emphasis on publication of significant results rather than null results. Thus, a number of studies may have investigated the factors identified as mediating or moderating the association between exposure to DV and development of internalising problems in children and adolescents and found that they did not have an effect but this is unknown if the study is not published in a peer reviewed journal. Furthermore, this review may be affected by the multiple (duplicate) publication bias as multiple studies with a significant result are more likely to published than more than a single study indicating a non-significant result yet both significant and non-significant results contribute to the overall effect of a factor within a systematic review.

5.3 Directions for future research
This systematic review has demonstrated that there is breadth but not depth within the area of study. Although many potential risk and protective factors have been

identified, more research is needed to establish the indirect pathways between exposure to DV and children and adolescents' psychological adjustment. In particular, taking a strength-oriented approach, exploring protective factors for children and adolescents exposed to DV and emotional resilience. It would also be valuable for studies to include a control group of those not exposed to DV during childhood to ensure that the total effect of CEDV on internalising symptoms is significant prior to preceding with mediation/moderation analyses, and to determine whether risk and protective factors are specific to children and adolescents exposed to DV or whether they are associated with internalising symptoms in the wider population.

It is unclear from the current literature whether certain risk and protective factors are important at different stages through childhood and adolescence, and further longitudinal research is needed to understand whether a potential factor has an impact at any stage or whether a null result in a particular age group may be significant in another. For instance, it may be that maternal factors are more influential during childhood however, when children enter adolescence and gain greater freedom, social factors such as peer support or bullying may be more prominent.

Other adverse childhood experiences (ACEs) should be considered when investigating internalising symptoms. This is because, although DV exposure has been shown to be a strong risk factor for poor psychological adjustment, other traumatic experiences such as direct child abuse are likely to co-occur with DV and also have a negative impact on children and adolescents' mental health. Inclusion of other ACEs would help understand the bigger picture of what children and adolescents are experiencing and which aspects are a priority for intervention. Furthermore, studies have shown the accumulation of ACEs to be associated with greater risk of psychopathology both in childhood (E. Flouri & Kallis, 2007) and across the lifespan (Chang, Jiang, Mkandarwire, & Shen, 2019). Investigating multiple risk and protective factors consecutively rather than in isolation would enable researchers and clinicians to identify the most important factors to target in order to prevent exposure to DV leading to mental health problems in childhood and adolescence or even later in adult life.

Another line of research that has been neglected is the role of paternal characteristics and whether risk/protective factors have differing roles depending on whether DV is

predominantly perpetrated by the male or female adult in the household. Understandably, the paucity of research in this area is likely due to issues of safety and a duty of care to the families participating in research. However, paternal parenting skills may be just as important as maternal parenting and it may be that factors such as a positive mother-child relationship are only protective if the mother is the victim of DV rather than the perpetrator.

Additionally, while much of the research surrounding CEDV has focused on violence perpetrated by males (Wolak & Finkelhor, 1998), it is important to recognise that children may also be exposed to DV in which their mother is the perpetrator or bidirectional acts of violence between caregivers occurs (Holmes et al., 2017). Outcomes and related risk or protective factors may be different when the mother is the perpetrator or violence is bidirectional.

5.4 Conclusion

This systematic review provided an overview of the current evidence on risk and protective factors for internalising symptoms in children and adolescents exposed to DV. This evidence is valuable for services working with children and young people who are experiencing internalising problems following exposure to DV, and their families. Key methodological issues have also been highlighted which may help improve future research in the field. Although another study has looked at protective factors effecting emotional-behavioural resilience more broadly (Fogarty et al., 2019), this is the first systematic review to specifically investigate the potential pathways between exposure to DV and internalising symptoms in childhood and adolescence in terms of risk and protective factors that may mediate or moderate the association between CEDV and internalising symptoms. Maternal mental health and direct parent-child abuse appear to play a critical role in the indirect pathway between DV and internalising symptoms, at all three developmental stages. This further demonstrates the need for mothers who have experienced DV to have access to a range of mental health services and support to maintain both their own and their children's mental wellbeing. Overall, this knowledge is vital for guiding clinical and community interventions for children and young people exposed to DV, and their families in order to prevent children experiencing psychopathology.

6 Contribution of this chapter to the thesis

This chapter has amalgamated evidence for risk and protective factors for internalising symptoms among children who have been exposed to DV. Factors identified may be further explored within a UK context to determine whether there is further evidence to support to claims made within the included papers. My systematic review has also identified the recent research that has been conducted in this area ensuring that I build upon rather than simply repeat work that has already been conducted.

Chapter 3 Identification of protective factors for internalising symptoms among children and adolescents following adverse childhood experiences – literature review

1 Overview

In the previous chapter I conducted a systematic literature review to identify risk and protective factors for internalising symptoms, at an individual, familial, and community level, among children and adolescents exposed to DV. These factors could potentially increase or decrease the risk of developing internalising symptoms through mediation and/or moderation. Furthermore, some of these factors are modifiable and therefore can be targeted in interventions to mitigate the impact of CEDV on internalising symptoms

This review has consolidated evidence for mediators and moderators on the causal pathway between DV exposure and internalising symptoms in children and adolescents that are modifiable. One individual-level mediator was identified, namely emotional intelligence, and two moderators were identified: relational victimisation and participation in extracurricular activities. Familial mediators included maladaptive parenting and parenting stress, while moderators included positive parenting (maternal warmth and availability) and family social support. No factors were identified at a community level.

In this chapter I will discuss the literature investigating the impact of factors which protect against internalising symptoms among children and adolescents who have experienced adversity more broadly, as the DV literature is limited, and the resilience literature has shown that certain factors protect against the impact of ACEs (Goldstein & Brooks, 2005; Ann S. Masten, 2019).

2 Background

2.1 Adverse Childhood Experiences (ACEs)

It has long been recognised that trauma in early years is linked to poorer developmental outcomes such as mental health and wellbeing (Eth & Pynoos, 1985).

For example, (Egeland, Sroufe, & Erickson, 1983) found in their longitudinal study that children who had been maltreated were more likely to develop internalising and externalising symptoms, as well as attachment issues, at preschool age. Other early studies have highlighted that these negative outcomes may be observed into early adulthood, such as Briere and Runtz's (1988) study which found that all forms of child maltreatment measured were associated with psychological symptoms amongst University students (Briere & Runtz, 1988). However, the ACEs agenda, introduced in the 1990s, brought multiple different traumatic experiences in childhood together under one umbrella term. A landmark ACEs study was conducted in the 1990s after the observation that almost half of patients at the Keiser Permanente obesity clinic in California dropped out of the programme despite losing weight. This led Dr. Vincent Felitti, chief of the obesity clinic, to interview patients who had dropped out, and he found that many had suffered childhood sexual abuse. He then proposed that weight gain was a coping mechanism to deal with the depression, anxiety, and trauma experienced by the victims of childhood sexual abuse. Felitti et al. (1998) then investigated the impact of seven child abuse and household dysfunction experiences on mortality and morbidity in adulthood. These comprised physical, psychological, and sexual abuse, substance abuse, parental mental illness, DV, and parental imprisonment, and were what we now know as ACEs. They found that of the 9,508 adults who responded to the mail survey, at least half had experienced at least one ACE and those who had experienced one ACE were likely to have experienced more than one, in other words ACEs were likely to co-occur. In addition, the presence of ACEs was associated with adult health conditions in an additive, dose-response fashion. Therefore, on average, the more ACEs an adult had experienced, the more health conditions they had later in life, particularly those associated with health-risk behaviours such as cancer and coronary heart disease.

Since the first ACEs study (Felitti et al., 1998), numerous studies have investigated relationships between ACEs and negative outcomes across the life-course including psychosocial outcomes (Ahmadi, Pynoos, Olango, & Molla, 2016; M. A. Bellis, Hughes, Leckenby, Perkins, & Lowey, 2014; Chang et al., 2019)and internalising symptoms in children and adolescents(Bright & Thompson, 2018; Brockie, Dana-Sacco, Wallen, Wilcox, & Campbell, 2015; Clarkson Freeman, 2014; Hunt, Slack, & Berger, 2017; Wang & Maguire-Jack, 2018). Research has led to the addition of

physical and emotional neglect, alcohol abuse and divorce/separation. More recently it has been argued that the definition of ACEs should also include poverty as poverty is strongly associated with ACEs and negative outcomes in adulthood (M. Hughes & Tucker, 2018). Researchers have also argued that other sources of early life adversity, including financial difficulties, residential instability, and living in a household with someone who is chronically ill or disabled (Manyema, Norris, & Richter, 2018; Nobari & Whaley, 2021; Zhang & Monnat, 2022), should be included in the "list" of ACEs. Additional possible ACEs include bullying, race-based discrimination, and exposure to neighbourhood violence or war (Bethel et al 2017; Lin et al 2021; World Health Organization 2018).Thus, there is no definitive list of ACEs, and the list is not exhaustive in terms of possible childhood trauma. Alternatively, ACE tools that have been developed are epidemiological tools rather than exhaustive lists of experiential risk factors for later health and social problems.

Researchers have also explored biological and hormonal pathways between ACEs and negative outcomes across childhood and adulthood. These studies have shown that that the stress caused by ACEs acts as a toxin within a child's brain causing changes in the neurodevelopment and social, emotional and cognitive impairments (Garner et al., 2012). These impairments increase the likelihood of adopting health harming behaviours in late childhood and early adolescence such as smoking, drinking alcohol, and taking drugs as coping mechanisms to alleviate the stress caused by adversity (Anda et al., 2006). This in turn can lead to the development of disease, disability, and social problems, and ultimately early mortality. The potential pathway between ACEs and early mortality is illustrated in Figure 3.
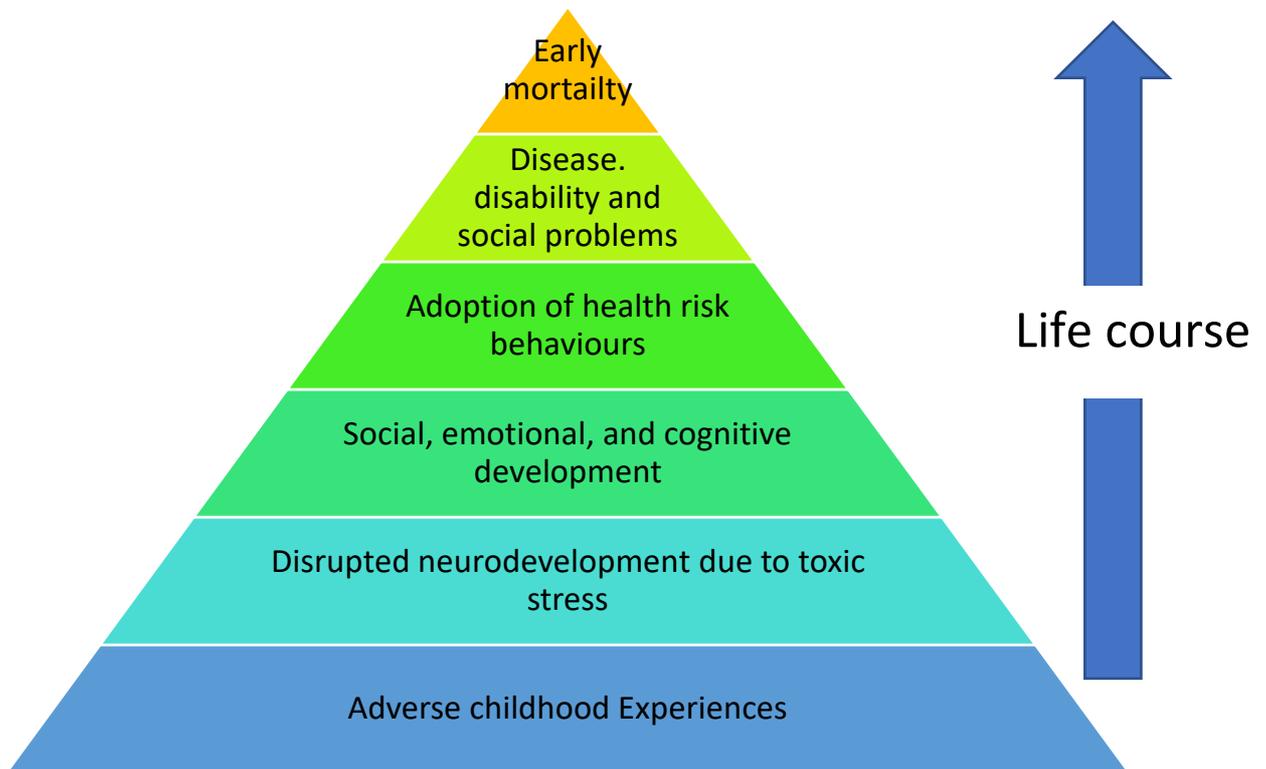
Figure 3. *The ACE pyramid displaying the impact of ACEs across the life course. Adapted from Felitti et al. (1998)*

However, this cascade of events from toxic stress caused by adversity leading to socioemotional impairments, health-harming behaviours and morbidity is not observed for everyone who has been exposed. There are a number of factors that may facilitate or inhibit these pathways. Understanding which factors are protective early in life, along with public policies and healthy development interventions may mitigate the harmful effects of toxic stress on the body and enable people to be resilient.

2.2 Protective factors for resilience

As described in the previous chapter, protective factors are those that buffer an individual against risk (Suniya S Luthar, Doernberger, & Zigler, 1993; Rutter, 1987). Such protective factors that support positive development and adjustment may be individual (neurobiology and behaviour), familial and relational, community, cultural, or societal. Masten (2018; 2015) gave the following shortlist of protective factors for healthy child development:

1. Caring family and sensitive, skilled parenting
2. Close relationships with other well-functioning adults
3. Close friends and romantic partners

4. Intelligence, problem-solving skills, planning, executive function skills
5. Self-regulation skills, emotion regulation
6. Motivation to succeed
7. Self-efficacy, positive view of the self or identity
8. Hope, faith, optimism, belief that life has meaning
9. Engagement in well-functioning schools
10. Connections with well-functioning communities

In general, a child or adolescent who has experienced more adverse experiences is less likely to have positive well-being. Additionally, a higher number of ACEs is significantly associated with having fewer overall protective factors. In other words, those who have experienced ACEs are less likely to possess the protective factors (Hughes et al., 2018). Regardless, the total number of protective factors enjoyed by a child is strongly and significantly related to better overall child well-being." (Moore and Ramirez, 2016).

A systematic review of protective factors associated with resilient outcomes in children following adversity found that the strongest evidence was available for cognitive skills, emotion regulation, relationships between children and caregivers and academic engagement (Gartland et al., 2019). These factors were associated with resilience, or positive outcomes, across a range of contexts suggesting that these are important starting points for public health interventions aiming to promote resilience and reduce negative outcomes, to focus on.

Martinez-Torteya et al. (2009) investigated risk and protective factors for child emotional and behavioural problems within the DV literature and identified that protective factors include positive caregiving/caregiver-child relationship, easy/engaging child temperament, and high cognitive ability.

More recently, Alaggia and Donohue (2018) conducted a literature review and reported that an extensive amount of research has been conducted identifying protective factors for child development among children exposed to DV. They reported protective factors at an intrapersonal, interpersonal, and community or cultural level. Factors at an intrapersonal level included easy child temperament, social competence, self-confidence, intelligence/academic success, good emotion regulation, optimism, motivation, problem-solving skills, spirituality, hobbies or

ability to distract oneself, and accurate cognitive appraisal of violence responsibility. At an interpersonal level, protective factors included at least one secure attachment and access to one safe adult, a protective mother, good maternal mental health, maternal warmth, and an in-home social network. At a community, contextual or cultural level, protective factors included peer and social support, job/further education prospects following secondary education, having a well-educated mother, spirituality, and having a bicultural influence. However, this review included all studies of resilience across the life-course and was not focussed on internalising symptoms during childhood and adolescence. Moreover, the vast majority of studies did not move beyond exploring associations between the protective factors and outcomes, to determining whether such factors were mediators or moderators of the effect of DV on child and adolescent internalising symptoms.

3 Aim and Objectives

3.1 Aim

To identify, from published literature, potential protective factors and covariates of the causal pathway between CEDV and internalising symptoms in childhood and adolescence through exploration of the wider ACEs and resilience literature.

3.2 Objectives

1. Conduct a systematic search of the childhood adversity literature to identify factors which promote childhood resilience in terms of protecting against internalising symptoms
2. To consider each potential covariate/confounder identified within the literature and its relationship with other variables in the pathway between exposure to CEDV and internalising symptoms

4 Method

4.1 Literature search

To identify relevant literature investigating the impact of factors which protect against internalising symptoms among children and adolescents who have experienced adverse childhood experiences, MEDLINE, EMBASE, and PsychInfo were searched in September 2019 using the following search strategy:

1. ("child abuse" or "child maltreat*" or ACE* or "child advers*" or "intimate partner violence" or IPV or "domestic violence" or "domestic abuse" or "alcohol

abuse" or divorce or "substance abuse" or "parental mental health" or "mental illness"
or neglect or incarceration or jail* or imprison*).mp.

2. (child* or adolescen* or "young person" or "young people" or teenage* or
youth*).mp.

3. ("mental health" or psychopathology or development or internali* or behav* or
depress* or anx* or somatic or withdrawal or well* or psych*).mp.

4. (mediat* or moderat* or resilien*).mp.

5. 1 and 2 and 3 and 4

6. limit 5 to english language

7. limit 6 to human

8. limit 7 to yr="2008 -Current"

9. limit 8 to (childhood or adolescence <13 to 17 years>)

10. limit 9 to (infant or child or preschool child <1 to 6 years> or school child <7 to
12 years> or adolescent <13 to 17 years>)

11. limit 10 to peer reviewed journal

12. limit 11 to (adverse effects or adverse effects - focussed or children or children -
focussed or "humans only (removes records about animals)")

13. limit 12 to (adverse effects or adverse effects - focussed or children or children -
focussed or "humans only (removes records about animals)")

14. remove duplicates from 13

This search produced 3,290 results, of which 135 were reviewed to identify
individual, family, and community level protective factors potentially on the causal
pathway between ACEs and internalising symptoms in children and adolescents.

4.2 Evidence Synthesis
In this narrative synthesis studies were qualitative or quantitative, employing cross-
sectional or longitudinal designs. Studies were grouped into those exploring
individual, parent and family, and community level protective factors.

5 Results

A summary of the results in terms of the protective factor, the exposure and outcome it has been explored with, and whether there was evidence of it acting as a protective factor may be found in Table 3.

Table 3. Evidence for each identified protective factor at an individual level including exposures measured, whether the factor has been considered as a mediator or moderator, and the strength of the evidence.

Protective factor name	Exposure(s) assessed	Mediation/ moderation	Strength of evidence
Cognitive function	DV and child sexual abuse	Moderator	Weak – cross-sectional and inconsistent findings
Internal locus of control	Child maltreatment and negative life events	Moderator	Moderate – cross-sectional studies, consistent findings
Temperament/ personality	Family conflicts or none	Mediation and moderation	Weak – most studies cross-sectional, one longitudinal
Communication/pro-social skills	Family/community violence and maltreatment	Mediation and moderation	Moderate – consistent findings, cross-sectional studies
Self-regulation – emotional and coping	DV, maltreatment, sexual abuse, family conflicts	Mediation and moderation	Moderate – consistent findings, some longitudinal evidence
Motivation to succeed	None		Weak – no studies assessed mediation/moderation
Self-esteem/efficacy	Child maltreatment	Mediation and moderation	Moderate – consistent findings, cross-sectional studies
Physical activity	Child maltreatment	Moderation	Moderate – consistent findings, cross-sectional studies
Perceived social support	None		Weak – research needed in high-risk populations
Hope, faith, optimism	Child maltreatment	Moderation	Weak – inconsistent findings, cross-sectional studies

5.1 Individual factors

Evidence was found for protective factors at the individual level within child and adolescent populations. Factors related to the individual may be particularly important

in the context of internalising symptoms among adolescents when children are becoming increasing autonomous (Deković, 1999).

5.1.1 Cognitive function – intelligence, problem solving skills, executive functioning, and mentalisation

High intelligence has been associated with resilience (Jaffee, Caspi, Moffitt, Polo-Tomas, & Taylor, 2007; Tiet et al., 1998) and is predictive of lower levels of psychiatric disorders, lower rates of conduct problems, and higher levels of overall functioning (Malcarne, Hamilton, Ingram, & Taylor, 2000). Studies with DV-exposed samples of children and young people are sparse but one cross-sectional study with 60 8-11 year olds suggested that IQ is negatively associated with behavioural problems including internalising symptoms (Kolbo, Blakely, & Engleman, 1996). Well-developed verbal cognitive abilities may enable verbal mediation of conflict leading to more appropriate behavioural choices and better coping strategies (Buckner, Mezzacappa, & Beardslee, 2003).

Another cross-sectional study, not included in the systematic review in the previous chapter, explored whether intelligence protected children exposed to DV from internalising symptoms and found that intelligence did not have a significant moderating effect (West, 2014). This study supported one that explored relationship between adverse life events and emotional and behavioural problems among 10-19 year olds (N=1,175) which also found insufficient evidence that general cognitive function moderated the association between adversity and internalising symptoms (Eirini Flouri & Mavroveli, 2013). However, intelligence moderated the effect of adverse life events on conduct problems such that adolescents with high cognitive ability did not experience conduct problems following adversity. It may be that those with higher cognitive abilities stay in school for longer and therefore avoid conduct disorders and delinquent behaviour, but school does not protect against internalising disorders. More research is needed utilising longitudinal methods to determine whether cognitive functioning moderates the effect of adversity on internalising symptoms.

Mentalisation, often measured as reflective functioning, is the ability to understand the thoughts and emotions behind the behaviour of oneself and others (Fonagy, Gergely, Jurist, & Target, 2002). Peter Fonagy's research team have conducted

several studies exploring the protective role of mentalisation against psychopathology in the context of child sexual abuse victims. One study with a sample of 74 7-12-year olds found that mentalisation was inversely associated with depression and partially mediated the link between child sexual abuse and depression (K. Ensink, Begin, Normandin, Godbout, & Fonagy, 2017). Again, this study was cross-sectional and thus more longitudinal research is needed to determine the causal pathway between DV, mentalisation, and internalising symptoms.

5.1.2 Internal locus of control

Locus of control is an aspect of personality psychology and describes how individuals attribute responsibility for events in their lives to themselves (internal control) or to other sources such as luck, chance, fate, or powerful others (external control) (Belter & Brinkmann, 1981; Rotter, 1966). If one has an internal locus of control, they perceive themselves to be in control of the outcome of events in their life, and therefore able to change them, whereas if one does not believe they are in control of events in their life, they may feel as if there is no point in trying to make changes to themselves or their lives.

Locus of control was examined as a moderator of links between negative life events and psychological symptoms in 238 young people eight to 16 years old by Kliewer and Sandler (1992). Findings indicated that having an internal locus of control buffered the effects of negative life events on psychological symptoms, and this moderating effect did not differ by age or gender. More recently Cheung et al. (2018) found that internal focus of control was associated with better mental health outcomes using a large (n = 10,123), cross-sectional, nationally representative sample of adolescents aged 13-17 years from the United States. regardless of their maltreatment history in line with resilience theory.

Bolger and Patterson (2001) reported that, within a sample of 785 7–13-year-olds, of which 59 had been maltreated, mediation analyses showed higher levels of perceived external control accounted substantially for relations between neglect, harsh parenting, and sexual abuse, and children's internalising symptoms. Analyses of interactions between the exposure and perceptions of control revealed that, among maltreated children, higher levels of perceived internal control predicted fewer internalising problems, suggesting that perceived internal control buffered the effect

of child maltreatment on internalising symptoms. However, maltreated children were more likely to have higher levels of perceived external locus of control rather than internal. This is thought to be because trauma and insecure attachments in children's lives can affect the development of self-efficacy and skills, possibly as a defence mechanism or a realistic adaptation to the child's environment (Roazzi, Attili, Di Pentima, & Toni, 2016).

5.1.3 Temperament and personality

Children at risk of psychopathology with easy temperaments (regularity, approachability, high adaptability, positive mood, low reactivity; Thomas & Chess, 1977) have been shown to develop fewer internalising symptoms than children with difficult temperaments (Kim-Cohen, Moffitt, Caspi, & Taylor, 2004; J. Smith & Prior, 1995; Tschann, Kaiser, Chesney, Alkon, & Boyce, 1996). Children with easy temperaments are less emotionally reactive to stress and more likely to use positive coping strategies to deal with stressors (Compas, Connor-Smith, & Jaser, 2004); they are also better able to regulate their negative emotions (Olson, Bates, Sandy, & Schilling, 2002). Research examining the moderating effect of child's temperament on DV-exposed children's adaptation is sparse. DeJonghe, Bogat, Levendosky, Von Eye, and Davidson (2005) conducted a cross-sectional study with a sample of 89 infants and found that temperament predicted infants' observed distress to verbal conflict among children not exposed to DV but children exposed to DV were distressed following verbal conflict regardless of their temperament. No studies to date have examined the link between temperament and internalising problems in the context of DV.

Temperament traits include negative affectivity, extraversion, and effortful control. Delgado, Carrasco, González-Peña, and Holgado-Tello (2018) found that effortful control and extraversion protected a large sample of children (N= 424) aged 3-6 years from internalising symptoms. Within the context of family functioning, Tschann et al. (1996) reported that approachability among 3-6-year olds was negatively associated with internalising symptoms, demonstrating its ability to act as a buffer against the negative impacts of family conflicts.

Among 2,230 preadolescents, Oldehinkel, Hartman, De Winter, Veenstra, and Ormel (2004) reported that effortful control and high-intensity pleasure were negatively

associated with internalising symptoms and may therefore be considered to be protective elements of temperament.

Within the general population, lower levels of effortful control, conscientiousness, flexibility, and task orientation have been identified in both cross-sectional (P. Davies & Windle, 2001; John, Caspi, Robins, Moffitt, & Stouthamer-Loeber, 1994) and longitudinal studies, (Caspi, Henry, McGee, Moffitt, & Silva, 1995) of childhood depression and anxiety, yet not to the same extent as they have been reported among children with externalising behaviours (Rettew & McKee, 2005). Low extraversion and high negative affectivity have also been linked with depression and anxiety disorders in later childhood and adolescence (Biederman et al., 1993; C. E. Schwartz, Snidman, & Kagan, 1999), suggesting that positive effect and extroversion may protect against anxiety and depression.

5.1.4 Communication/prosocial skills

Communication and/or prosocial skills haven't been explored as protective factors in the context of CEDV per se. However, prosocial skills can serve as a buffer between early negative experiences, such as exposure to violence in the home, and later negative developmental outcomes (Howell, 2011). Both physically abused and non-physically abused children with early social competence are less likely to develop internalising symptoms than those with low early social competence (Lansford et al., 2006). Prosocial skills help children form trusting and positive relationships with others, which is vital for children who are exposed to DV. Children with good social skills can gain support and protection from people outside of their family, such as teachers, often leading to better outcomes following exposure to violence (Alvord & Grados, 2005).

LeBlanc, Self-Brown, Shepard, and Kelley (2011) found evidence that after controlling for demographic variables and violence exposure in the home, adolescents' communication and problem-solving skills moderated the association between school and neighbourhood violence exposure and psychological distress. There was no moderation of the violence exposure-positive outcome association. The findings suggest that communication and problem-solving skills might serve a protective function for adolescents, thereby decreasing psychological distress in the face of school and neighbourhood violence exposure.

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Shonk and Cicchetti (2001) found that social competence fully mediated the association between child maltreatment and internalising symptoms among a sample of 229 socio-economically disadvantaged children aged 5-12 years, according to teachers' comprehensive evaluations, school records, and camp counsellors' ratings. However, this was a cross-sectional study and thus longitudinal studies are required to confirm the mediation effect.

5.1.5 Self-regulation – emotion regulation and coping skills
L. F. Katz et al. (2007) found that the emotion awareness was associated with fewer internalising symptoms among children exposed to IPV and L. F. Katz, Stettler, and Gurtovenko (2016) found that children's abilities to regulate their negative emotions of sadness and anger related to exposure to DV were less likely to experience internalising symptoms that those less able to regulate their emotions. This ability to regulate emotions is often taught by the caregiver and both parental emotion socialisation and emotion coaching has also been identified as a protective factor against internalising symptoms following exposure to domestic violence by Katz and colleagues (2006; 2007; 2016).

Within the context of marital and family conflict, Whitson and El-Esheikh (2003) have reported that emotion regulation moderated the association between conflict and internalising symptomology among 6–11-year-olds, such that among those who had experienced high levels of conflict, children with good emotion regulation had fewer internalising symptoms compared to those with poor emotion regulation. Longitudinal evidence supports this finding as greater vagal suppression to a simulated argument was protective against child and adolescent internalising problems associated with marital conflict (El-Sheikh & Whitson, 2006). This demonstrated that child vagal regulation can buffer against internalising symptomology in the context of exposure to marital conflict.

Among sexually abused children aged 7-12 years, avoidant coping strategies were found to be protective against internalising symptoms, which contrasts with findings from adolescents and adults. Contrary to expectations, active/social coping was not found to buffer against the effects of sexual abuse on child internalising symptoms (Chaffin, Wherry, & Dykman, 1997). Alternatively, among children of divorce, Sandler, Tein, and West (1994) reported that avoidant coping was positively

associated with internalising symptoms suggesting that avoidant coping is not beneficial following stress or trauma; however, longitudinal analysis suggested that the internalising symptoms may lead to increased avoidance rather than vice versa. Additionally, active coping and distraction were significantly associated with fewer later internalising symptoms. Interestingly, within the longitudinal analyses, support coping was associated with increased depression 5 months later suggesting social support is actually a risk factor rather than protective.

Cognitive appraisals of traumatic events may be a form of coping. The mediating and moderating role of cognitive appraisals have been studied in the context of ACEs. For instance, Mazur, Wolchik, Virdin, Sandler, and West (1999) reported that positive illusions protect children of stressful parental divorces (aged 9-12 years) against child-reported depression whereas attributions of self-blame following maltreatment account for variance in internalising symptoms, increasing risk of young people experiencing psychopathology (Feiring, Taska, & Lewis, 1998).

HPA-axis reactivity to stress may be a key moderator of the impact of trauma on children's internalising symptoms. Kuhlman, Repetti, Reynolds, and Robles (2018) found that both exaggerated and attenuated HPA-axis reactivity to stress may increase risk of psychopathology. In particular, it was reported that young people exposed to physical abuse had higher internalising symptoms if they had attenuated HPA-axis reactivity to acute stress whereas young people exposed to emotional abuse or non-intentional trauma had more severe internalising symptoms if they had exaggerated HPA-axis responses to acute stress.

5.1.6 Motivation to succeed

No studies were identified examining the protective impact of motivation to succeed on behavioural outcomes following exposure to ACEs. However, Deković (1999) reported that, among a sample of 508 adolescents from a Dutch cohort study, low motivation to succeed at school was associated with internalising symptoms. This implies that the opposite is also true, such that high motivation to succeed is associated with lower internalising symptoms.

5.1.7 Self-esteem and self-efficacy

Jungmeen Kim and Cicchetti (2003) investigated the protective role of self-efficacy and reported that young maltreated children (<8 years) with higher levels of social

self-efficacy showed significantly fewer internalising symptoms than young maltreated children with lower levels of social self-efficacy. For older children (>8 years), regardless of maltreatment status, higher levels of perceived social self-efficacy were related to lower levels of internalising symptomatology.

Roustit, Campoy, Chaix, and Chauvin (2010) examined the mediational role of adolescent self-esteem on the effect of parental psychopathology on adolescents' internalising symptoms and found that it formed a significant indirect pathway. Parental psychopathology was shown to reduce adolescents' self-esteem which in turn increased the severity of internalising symptoms. Therefore, improving the adolescents' self-esteem of those whose parents have poor mental health may reduce the risk of them developing internalising disorders.

5.1.8 Physical Activity

Physical exercise is known to be related to reduced depression symptoms (Knapen, Vancampfort, Morien, & Marchal, 2015; Rosenbaum, Tiedemann, Sherrington, Curtis, & Ward, 2014; Stanton & Happell, 2014), anxiety disorders, posttraumatic stress disorder, and obsessive-compulsive disorder (Asmundson et al., 2013) in the general population. Physical exercise, especially aerobic and strength training, has even been recommended as a form of, or supplement to, treatment for individuals with depression and other mental disorders (Asmundson et al., 2013; Knapen et al., 2015; Rosenbaum et al., 2014; Stanton & Happell, 2014).

A recent meta-analysis exploring the literature on the association between physical activity and depression among children and adolescents, including 50 independent studies, found that physical activity is associated with fewer concurrent depressive symptoms but the impact of physical activity on future depression symptoms is weak implying that physical activity needs to be continued to maintain its protective effects (Korczak, Madigan, & Colasanto, 2017).

Within a large, representative sample of young people who have experienced child maltreatment, including exposure to IPV, Cheung et al. (2018) reported that physical exercise was significantly associated with better mental health outcomes generally,

not just internalising symptoms. This finding is in line with previous research (Tracie O. Afifi et al., 2016; Morgan, 2010; Waechter & Wekerle, 2015). Cheung et al. (2018) concluded that "one area of research that requires further examination is physical exercise in relation to resilience after child maltreatment".

5.1.9 Perceived social support

Rueger, Malecki, Pyun, Aycock, and Coyle (2016) conducted a systematic review investigating whether social support is associated with depression in childhood and adolescents. They concluded from their meta-analysis that "although it is clear that social support offers positive benefits across different support sources and developmental level and gender of support recipient, more research is needed, especially with youth exposed to chronic stressors, to effectively intervene in the prevention and treatment of depression. With widespread interest in social support across diverse psychology subfields, as well as social work and other health care professions, there is a great need to coordinate efforts to study social support provided to youth."

5.1.10 Hope, faith, religion, and optimism

Having belief that life has meaning is an important motivational factor to persevere despite adversity. Brassai, Piko, and Steger (2011) reported that, among a large sample of adolescents in Romania, meaning of life was a protective factor against poor psychological health.

Jungmeen Kim, McCullough, and Cicchetti (2009) reported that child religiosity may largely contribute to stress coping processes among maltreated and non-maltreated children from low-income families. The results also indicate that the protective roles of religiosity varied by gender. Child reports of the importance of faith were related to lower levels of internalising symptoms among maltreated girls but not boys. However, a study published a year later by the same author reported that neither child nor parental religiosity predicted internalising problems among maltreated, low-income children.

Optimism has been found to be an important protective factor for fewer depressive symptoms among Hungarian adolescents (Bettina Franciska Piko, Kovacs, & Fitzpatrick, 2009). Lemola et al. (2010) also reported the dispositional optimism was significantly negatively associated with internalising symptoms among children and

adolescents suggesting it is a strong protective factor. Nevertheless, it is unclear whether optimism acts as a buffer against internalising symptoms in the context of DV exposure.

5.2 Family factors

Both longitudinal and cross-sectional studies of resilience in maltreated children have consistently found that family-level factors have the biggest impact on resilience during childhood. These include a stable caregiving environment and the presence of a supportive caregiver (Tracie O Afifi & MacMillan, 2011). However, Deković (1999) found that among adolescents, the effects of familial protective factors on internalising symptoms were relatively small. Several attributes of the adolescents' family (parental support, monitoring, and adolescents' attachment to parents) seem to be important but at this age, peer factors and individual factors may play a greater role (Deković, 1999). A summary of the evidence identified within this review can be found in Table 4.

Table 4. Evidence for each identified protective factor at a family level, including exposures measured, whether the factor has been considered as a mediator or moderator, and the strength of the evidence.

Protective factor name	Exposure(s) assessed	Mediation/ moderation	Strength of evidence
Sibling relationships	Stressful life events	Moderator	Moderate – longitudinal evidence but research needed
Positive maternal mental health and well-being	DV	Moderator	Weak – cross-sectional evidence and poor mental health measured rather than aspects of well-being
Positive parenting and parent-child relationships	ACEs, maternal mental health problems, maternal substance misuse, divorce, maltreatment	Mediator/ moderator	Strong – multiple studies including longitudinal evidence, consistent findings
Secure mother-child attachment	Paternal substance misuse, child sexual abuse	Mediator/ moderator	Weak – inconsistent evidence, insufficient evidence in longitudinal study
Perceived parental social support	DV and parental conflict	Mediator	Weak – cross-sectional evidence with risk focus

5.2.1 Sibling relationships

Sibling interactions are characterised by both strong positive features, such as warmth and intimacy, as well as negative qualities, such as intense conflict, which are associated with children's well-being (Feinberg, Solmeyer, & McHale, 2012). Despite sibling relationships playing an important role across the lifespan, they have received far less attention than other relationships in children such as peer relationships and parent–child relationships (Dirks, Persram, Recchia, & Howe, 2015).

Gass, Jenkins, and Dunn (2007) conducted a two-wave longitudinal study to examine the protective effect of positive sibling relationships on child adjustment for children experiencing stressful life events. Mothers reported on stressful life events and child adjustment. Older siblings assessed the quality of relationship between themselves and target children. Sibling affection moderated the relationship between stressful life events and internalising symptomatology. Notably, the protective effect of sibling affection was evident regardless of mother–child relationship quality.

The clinical utility of these findings will be strengthened by work mapping key moderators of these associations. For example, the buffering effects of a highly positive sibling relationship may be stronger for younger children who spend more time together than older children (Volling, 2003).

5.2.2 Positive parental mental health and wellbeing

The impact of poor maternal mental health has been extensively studied among women who have experienced DV and other adversities, yet positive mental health has been much less frequently explored, possibly because it is uncommon. Those who are resilient themselves and maintain positive mental health are more likely to have well-adjusted, resilient children (Graham-Bermann et al., 2009). Graham-Bermann et al. (2009) found that children of mothers who had poor mental health were less likely to be resilient and therefore concluded that the complement is likely to be true i.e., children of mothers without any mental health disorders are more likely to be resilient. Yet, they recognise that a better way of answering that question would be to measure positive maternal mental health constructs such as self-competence, self-esteem and optimism and their associations with child emotional problems over time following domestic violence exposure.

5.2.3 Positive Parenting and Parent-child relationship

The presence of a consistent, warm, nurturing, and supportive relationship with at least one parent has been found to be protective against behavioural problems (Bountress & Chassin, 2015; Bradley et al., 1994; David M Fergusson & Lynskey, 1996).

Some mothers of children exposed to IPV may be warm towards their child as a compensation for exposure to violence in their parenting interactions (Letourneau, Fedick, & Willms, 2007), creating a buffer by preventing the abuse from affecting their functioning as mothers, and protecting their children from violence (Peled & Gil, 2011; Pinto, Figueiredo, Pinheiro, & Canário, 2016). Graham-Bermann et al. (2009) found that, when investigating parenting as a protective factor against emotional and behavioural problems among children exposed to DV, maternal warmth and effective parenting practices (e.g., setting boundaries and appropriate discipline) were found to have a buffering effect against internalising symptoms. P. T. Davies and Lindsay (2004), Manning, Davies, and Cicchetti (2014), and G. Margolin and Gordis (2004) also reported that mothers sensitive parenting skills in the context of family violence protected children from negative emotional-behavioural outcomes.

Among children of parents with substance use disorders (SUDs), consistent parental support has been found to mediate the pathway between SUDs and internalising symptoms (Bountress & Chassin, 2015). This therefore suggests that interventions to improve or prevent children's internalising symptoms should focus on improving negative parenting as well as treating the parental SUD. Further evidence for this has been reported by (Burstein, Stanger, Kamon, & Dumenci, 2006) who found that negative parenting mediated the effect of parental internalising symptoms on child affective problems among children of parents with SUDs, but not parental externalising symptoms. However, the effects of parental externalising problems were moderated by high positive involvement such that children of parents with SUDs and externalising problems experienced fewer internalising symptoms when parents were more positively involved, yet only when the externalising problems were not too severe (Burstein et al., 2006).

Anthony, Paine, and Shelton (2019) reported that, among young children adopted from services, maternal warmth moderated the association between ACE scores and

internalising problems, measured using the Strengths and Difficulties Questionnaire (R. Goodman, 1997). However, the study relied on adopted parental reports which may have underestimated ACEs experienced and internalising symptoms. Furthermore, the sample size was small.

Within an intervention study for depressed mothers with children experiencing at least one internalising problem (n= 62), positive parenting skills (acceptance) were found to be the mechanism responsible for the improvement in child symptoms following improvement of maternal symptoms (Swartz, Cyranowski, Cheng, & Amole, 2018). Therefore, it was suggested that interventions focussing on positive parenting could positively contribute to reducing child internalising symptoms.

J. Kim and Cicchetti (2004) examined concurrent and longitudinal relations between mother–child relationship quality and adjustment of 206 maltreated and 139 non-maltreated disadvantaged children aged 7–12 years. A multi-method assessment was conducted to measure children's relatedness to their mother (i.e., psychological proximity seeking and emotional quality), their self-esteem, and internalising and externalising problems. Data were collected during two consecutive summer camps. Results indicated that relatedness to mothers in the first year predicted children's internalising and externalising problems in the second year. An intervention study by Tein, Sandler, MacKinnon, and Wolchik (2004) for children of divorced parents also found that the mother-child relationship mediated the pathway between divorce and children's internalising symptoms accounting for the reduction in post-test symptom scores. Mother-child relationship quality may exert its buffering role through moderating the effect of emotion regulation on internalising problems among children and young people who have been maltreated (Alink, Cicchetti, Kim, & Rogosch, 2009). Alink et al. (2009) reported that among those who were maltreated, children who had a secure relationship with their mother were better able to regulate their emotions and therefore less likely to experience internalising problems.

Gabalda, Thompson, and Kaslow (2010) also found that family adaptability and cohesion predicted reduced internalising symptoms among 152 8- to 12-year-old, low-income, African-American children at high risk of maltreatment however, causal inferences cannot be made as it was a cross-sectional study.

Burstein et al. (2006) reported that among children of parents receiving substance abuse treatment, high positive involvement moderated relations between parent externalising behaviours and their child's internalising problems. Associations between parent externalising behaviours and child anxiety and affective problems were significant only among families with high positive involvement, defined as praise and positive reinforcement of prosocial child behaviour and parental interest and interaction with the child.

More recently, Karin Ensink, Bégin, Normandin, and Fonagy (2017) investigated whether another aspect of positive parenting, namely parental mentalising – parents imagining the child's experience and understanding what their behaviour is communicating – is a protective factor against internalising problems among sexually abused children. It was concluded that maternal mentalising, or reflective functioning, moderated the relationship between child sexual abuse and child internalising difficulties among children aged 2-12 years. Children who had experienced sexual abuse but had mothers with good mentalising abilities experienced fewer internalising symptoms than those who had experienced child sexual abuse, and whose mothers also demonstrated less mentalising capacities. However, parental mentalising did not mediate the association between child sexual abuse and depression among older children (7-12 years), suggesting that parental mentalisation is more important for younger children (Ensink et al., 2017).

Father-child relationships and paternal parenting skills have also been explored. O'Hara, Sandler, Wolchik, Tein, and Rhodes (2019) conducted a prospective study of children (aged 9-18 years) of parents going through high-conflict divorces and found that father's time spent with children was associated with their children's internalising symptoms and this association was mediated by parenting quality. Analyses also implied that although more time spent with the child is associated with better father-child relationships, this is only up to a point after which the benefit does not increase.

5.2.4 Mother-child Attachment
The quality of attachment to parents is strongly related to adolescents' well-being and depression (Armsden & Greenberg, 1987). Edwards, Eiden, and Leonard (2006) investigated the buffering effect of secure attachment against internalising symptoms among toddlers of alcoholic fathers. Testing of an interaction effect of child age,

alcohol group, and attachment security with mothers on internalising problems suggested that at 24 and 36 months of age, mother-infant attachment security moderated the relationship between alcohol group status and internalising problems. Namely, within the alcohol group, those children with secure attachment with their mothers had significantly lower internalising compared to insecure children of paternal alcoholics.

Charest, Hebert, Bernier, Langevin, and Miljkovitch (2019) explored the mediational role of attachment on the direct association between child sexual abuse and behavioural problems. It was reported that secure attachment was negatively associated with child sexual abuse and secure attachment did not act as a mediator and therefore was not found to be a protective factor for sexually abused children aged 3-6 years (Charest et al., 2019).

5.2.5 Parental perceived social support

Owen et al. (2008) examined if mother or child's perceived social support decreased the emotional and behavioural consequences of intimate partner conflict for 148 African-American children ages 8–12 years. Results revealed that children's perceived social support mediated the relation between intimate partner conflict and children's internalising symptoms. Findings also indicated a mediational role of mother's perceived social support in the link between both physical and nonphysical partner abuse with children's internalising symptoms. Results from this study suggest that diminished levels of perceived social support are a risk factor for psychological problems in children from low-income, African American families. Thus, it may be inferred that high levels of perceived social support are protective against internalising symptoms in children, although more research is needed to confirm this.

5.3 Community factors

Most studies investigating protective factors tend to focus on individual and family level factors and research exploring community protective factors is limited. A summary of the evidence for each factor may be found in Table 5. It is important to note that I have considered the community factors in Table 5 to be individual level factors in my systematic literature review chapter and cohort analyses as they are measured at an individual level. However, here they are considered as community factors as they were described as such in the literature.

Table 5. Evidence for each identified protective factor at a community level, including exposures measured, whether the factor has been considered as a mediator or moderator, and the strength of the evidence.

Protective factor name	Exposure(s) assessed	Mediation/ moderation	Strength of evidence
At least one trusted adult	ACEs	Moderator	Moderate – cross-sectional and retrospective studies but consistent findings
Perception of school	None	Moderator	Weak – cross-sectional studies and inconsistent findings
Friendship and peer support	IPV and life events	Moderator	Weak – cross-sectional studies and inconsistent findings
Extra-curricular activities	ACEs and DV	Moderator	Weak – cross-sectional studies and inconsistent findings

5.3.1 At least one trusted adult

Trusted adults are any adult in the young person's life including parents, extended family, teachers, parents of friends, family friends, neighbours, youth workers, coaches, or tutors (Bryant & Zimmerman, 2003; Hendry, Roberts, Glendinning, & Coleman, 1992; Scales & Gibbons, 1996). Key to the definition is that they are chosen by the child or young person as someone whom they trust (Meltzer, Muir, & Craig, 2016).

A sparse but growing body of literature suggests that supportive/trusted adults, ranging from neighbours and teachers to extended adult family members, may be one of the most important factors contributing to resilience among children and young people living with adversity. For example, E. C. Herrenkohl, Herrenkohl, and Egolf (1994) found that, among high-functioning children who had experienced ACEs, those who had had at least one stable caregiver during their childhood were more resilient.

More recently Bellis et al. (2017) conducted a large retrospective study with adults and found that having an "always available adult" during childhood reduced risk of mental health problems among those who had experienced ACEs, therefore acting as a protective factor (Mark A. Bellis et al., 2017).

Among adolescents, reported "the more ACEs a child possessed positively predicted a higher likelihood of having a supportive adult outside of the child's family. This

could be due to the fact that children with ACEs seek or receive support from non-family adults to enhance their development. Despite this, non-family adult support was not found as an overall mediator, perhaps because the quality, regularity and duration of the relationship are not assessed."

In summary, these studies suggest that having a supportive adult in one's life may be an important protective factor for children and adolescent who have experienced adversity, including DV and DCA, however more longitudinal research is needed.

5.3.2 Perception of school/School environment

Schools can provide a safe environment for children living in chaotic homes. Piko and Fitzpatrick (2003) found that happiness with school was protective against depression symptoms among adolescents in Hungary. However, in a more recent study, Piko, Kovacs, and Fitzpatrick (2009) found that school-related factors played a limited protective role on depressive symptoms and that happiness with school was only a protective factor among boys.

5.3.3 Friendships and peer support

Within the general population, positive peer relationships have been identified as buffering against internalising symptoms in children (for a review see Licitra-Kleckler &Waas, 1993; Windle, 1992) and adolescents (Gaertner, Fite, & Colder, 2010; Waldrip, Malcolm, & Jensen-Campbell, 2008).

Among children who have experienced adversity, Camacho et al. (2012) reported that receipt of prosocial behaviours buffer against the effects of IPV exposure on internalising symptoms in adolescent girls. Alternatively, Margolin et al. (2006) found that among middle school African-American students, community support did not reduce internalising symptoms, and other research has reported mixed findings. It may be that peer relations do not have a greater influence than family relations until later adolescence.

Steinhausen and Metzke (2001), found that peer acceptance moderated the association between life events and internalising symptoms in a sample of 6-17-year-olds in Switzerland such that internalising symptoms decreased under high numbers of life events when the youths were accepted by their peers.

5.3.4 Extracurricular activities

Activity involvement, whether in or out of school, is associated with adaptive and personal development for both children and adolescents within the general population. Being active helps young people develop more fully and is protective against loneliness (Rokach, 2001). Activity involvement in school has been positively correlated with increased self-esteem in both black and white high school students in the U.S (Holland & Andre, 1987) and is negatively associated with loneliness (Pretty, Andrewes, & Collett, 1994). However, a systematic review (Farb & Matjasko, 2012) found that studies exploring the association between activity participation and psychological adjustment are inconclusive, depending on the type and duration of participation studied. Feelings and experiences whilst participating were more closely related with positive psychological outcomes than the participation itself (Farb & Matjasko, 2012).

Moore and Ramirez (2016) conducted a cross-sectional study using the large 2011-2012 US National Survey of Children's Health (n= 34,151) and found that activity participation did not mediate the association between ACEs and the wellbeing of adolescents aged 12-17 years, however this could also reflect the fact that the frequency of participation or the quality of the activities were not measured. Gabalda et al., (2010) also conducted a cross-sectional study with 152 mother-child dyads at high risk of maltreatment and found that participation in after school activities did not protect low-income, African-American children from internalising problems at 8-12 years of age. Longitudinal mediation analysis is required to confirm this finding.

When frequency of activity participation is considered, intensive involvement in activities has been found to have a buffering effect against internalising symptoms among children who have experienced domestic abuse compared to moderate participation, which did not have a significant effect (Gardner et al., 2012).

It is important to note that families dealing with adversity and poverty will have less access to extracurricular activities, which may reduce their ability to act as a buffer for those exposed to DV and other adversities.

6 Discussion and Conclusion

This literature review has identified some evidence, albeit fairly weak, for the role of protective factors among children and adolescent who have experienced adversity. At

an individual level factors with a moderate level of evidence included: internal locus of control; communication/pro-social skills; self-regulation (emotional and coping skills); and physical activity. There was weak evidence for motivation to succeed; cognitive function; temperament/personality; perceived social support; and hope, faith, and optimism. At a family level, there was strong evidence for positive parenting and parent-child relationships buffering against internalising symptoms and factors with a moderate level of evidence included: positive-parenting and parent-child relationships, and sibling relationships. Alternatively, there was weak evidence for the roles of parental social support, secure mother-child attachment, and positive maternal mental health and well-being as protective factors. At a community level, the only factor with moderate evidence was having a trusted adult, and there was weak evidence for perception of school, friendships and peer support, and extra-curricular activities.

Few studies have moved beyond identifying associations between protective factors and resilience within cross-sectional studies to investigate the causal pathways between ACEs and internalising symptoms reporting mediators and/or moderators of the direct associations utilising longitudinal methods. Thus, the factors identified within this study are only potential mediators and/or moderators and their effects need to be investigated.

Studies investigating protective factors for internalising symptoms following exposure to DV are scarce, however in the wider resilience literature, research has focussed on modifiable family level factors and identified several key factors buffering against the negative effects of various adversities. This suggests that the same factors may exert their buffering effects across adversities such as ACEs, however there may be some protective factors that are specific to certain contexts. For example, among children exposed to DV, it may be that a positive attitude towards school is particularly important as school may be their safe space away from the violent home. Alternatively, although a positive mother-child relationship may be highly beneficial in certain circumstances such as civil war or homelessness, if the child's mother is violent then this is unlikely to be a protective factor within the given context.

Furthermore, studies have not tended to investigate factors at different developmental stages during childhood and adolescence and therefore fail to determine whether certain protective factors are relevant for children of certain ages. Adolescence, for instance is a period in which numerous changes occur, both inside the individual (cognitive and physiological changes) and outside the individual (changes in the child's relationships with peers and parents). It might therefore be expected that both risk and protective factors that change as an individual enters adolescence (Kazdin, 1993; Tolan, Guerra, & Kendall, 1995). Given that resilience is a process and is changeable, it may be that some factors develop over time such as self-efficacy.

7 Contribution of this chapter to the thesis

This chapter has identified potential factors that may protect children exposed to DV from developing internalising symptoms, through exploring the wider literature ACEs and resilience literature. Further research is needed to explore whether these factors buffer against internalising symptoms among children and adolescents exposed to DV, particularly utilising longitudinal methods. Thus, I will design a research study assessing the mediating and/or moderating effects of the factors identified within this chapter and the previous chapter (systematic literature review). However, first I will amalgamate the information in both this chapter and the previous chapter to identify the numerous variables needed for such as study, within the following chapter.

Chapter 4 Mediators and moderators on the causal pathways between childhood exposure to domestic violence and adolescent internalising symptoms: A causal diagram approach

1 Overview

Chapter 2 presented evidence for mediators and moderators of the effect of CEDV on internalising symptoms in childhood and adolescence. Additionally, the previous chapter explored evidence from the broader ACEs and resilience literature to identify factors associated with internalising symptoms, following a form of childhood adversity. In this chapter I amalgamate that evidence and present a causal diagram (directed acyclic graph; DAG) to describe the complex web of factors involved in the between exposure to DV and internalising symptoms. I hypothesised that the factors identified within the previous two chapters would either increase the risk or protect against internalising symptoms among children and adolescents exposed to DV through mediation and/or moderation. The DAG will provide readers with an overview of the variables used to answer research questions 3-6 set out within the introduction, namely the exposure, outcomes, mediators/moderators and confounders.

First, I give some background on causal inference and causal diagrams. Next, I present the aims and objectives of the chapter followed by the DAG. Finally, I conclude the chapter with a discussion of the strengths and limitations of the DAG along with implications of this work for the remaining research in this thesis.

2 Background

DV has been shown to have an effect on children's and adolescents' internalising symptoms, but there are numerous other factors that contribute to experiencing internalising symptoms, with DV oftentimes starting a chain of effects on other factors, and or various combinations of factors, which alone, or in combination, lead to the experience of internalising symptoms. Consequently, the causal pathway is not a single chain but rather a complex web of multiple pathways, as described in chapters 2 and 3.

In the previous chapter I reviewed potential protective factors identified through the wider ACEs and resilience literature given the findings of my systematic review in the preceding chapter. I found that studies exploring the impact of mediators and moderators on the causal pathway between CEDV and internalising symptoms have tended to focus on risk factors with few researchers exploring factors that may protect children and adolescents following CEDV.

In this chapter I present a causal diagram, specifically an extended DAG, to describe my assumptions about the causal structure of the variables that have been reported to be involved in the pathways between CEDV and internalising symptoms in childhood and adolescence. The reason it is an *extended* DAG is that I have included an informal illustration of where the important moderators are likely to be, which will be explained later. DAGs are useful in supporting analyses that have a causal aim, since they allow us to organise the existing subject matter knowledge. They also display the assumptions we are prepared to make about the causal relationships between the important variables (some of which may be unmeasured) relevant to our question of interest. In addition, the graphical rules applied to the DAG can aid in deciding the best analysis of the data at hand so that as much as possible is done to reduce bias (e.g., due to confounding) in the results. In particular, when moderation by certain variables is of interest, but when these refer to measures that occur *after* the exposure, moderators may also simultaneously be mediators of the effect of the exposure on the outcomes, and DAGs can help to avoid further pitfalls such as unwittingly conditioning on consequences of exposure without allowing for this in the interpretation. Conclusions from this chapter will guide the analyses and interpretation of findings in my studies presented in chapters five and six.

3 Aim and objectives

3.1 Aim

To create a DAG of the causal pathways between CEDV and internalising symptoms. This will inform the analyses of longitudinal data to understand the impact of potential protective factors that can mitigate the impact of CEDV on internalising symptoms.

3.2 Objectives

1. To conduct a literature search identifying factors associated with both DV and internalising symptoms which may therefore be considered potential confounders or mediators

2. To consider the role of each factor identified within the literature in the DAG

3. To use the results from both this and previous chapters to create a DAG of the causal pathways between DV and internalising symptoms in childhood and adolescence

4. To interpret the DAG within the context of the analyses to be conducted in the following chapters

4 Causal inference

Causal inference is concerned with defining and estimating (usually from non-experimental data) the causal effect that an exposure has on an outcome, placing emphasis on the underpinning assumptions. In more detail, some causal inference techniques are concerned with *explaining* this effect, that is, the extent to which it is mediated via one or more posited intermediate variables (mediation analysis), and also the extent to which the magnitude of the casual effect varies according to levels of a third variable (moderator), noting that a mediator can simultaneously be an important moderator. This is key to taking an epidemiological approach to improving health and wellbeing in societies. Research examining causal effects is distinctly different to that which explores associations between variables in the way that the research question is phrased and the extent to which covariates and confounding is considered (Hernán, 2018). Loosely speaking, confounders are variables that are associated with both the exposure and the outcome but are not on the causal pathway and are not colliders (Tyler J. VanderWeele & Shpitser, 2013). The counterfactual framework for causal effects is the most popular basis for approaching causal inference in epidemiology and health sciences (Höfler, 2005). Counterfactuals are unobserved, or hypothetical, variables within observational studies. For example, if we want to know the effect of DV on internalising symptoms we can divide study individuals into two groups, those who have been exposed to DV and those who have not been exposed to DV. However, we will never know what would have happened to an individual if they had been in the other group (the "counterfactual" outcome). Crucially, the two groups will differ in other ways than just the exposure, meaning that the naïve comparison between groups will be subject to confounding. A causal

effect is defined as a contrast between (usually) the mean of the two counterfactual variables, such as the difference in the mean internalising symptoms when "assigned" to DV versus not. However, it is arguably quite irrelevant to imagine a hypothetical world in which every child is exposed to DV. In this case, it is of more interest to determine what the causal effect is among those who have experienced DV compared to what it would be if they hadn't experienced DV or, alternatively, to compare the real world to a hypothetical world in which DV is eliminated. Measures of association such as odds ratios, risk differences, and risk ratios can be given a causal interpretation only if strong assumptions are met, exchangeability being the central one. Exchangeability refers to the notion that the risk of an outcome in Group A would have been the same as the risk in Group B if the participants in Group B had received the exposure in Group A, and vice versa. In other words, exchangeability holds for the DV – internalising symptoms setting if both counterfactual outcomes are independent of DV. In randomised controlled trials (RCTs), exchangeability is met as group allocation is independent of participant characteristics. Thus, measures of association (that compare study arms) can be given a causal interpretation, as long as there is no additional bias due to other reasons such as differential loss to follow up across arms. However, in the case of DV where an RCT would clearly be unethical, we must rely on observational data and crucially on confounder adjustment, with the aim that exchangeability is still met albeit now conditionally on confounders. In other words, given a set of confounders C, we hope that both counterfactual outcomes are independent of DV.

A statistical association between variables X and Y will exist regardless of whether X causes Y or Y causes X, or there is a Z that causes both X and Y. Furthermore, a conditional association may be created by conditioning on a common effect of X and Y. Within the context of interest, spurious associations may occur by conditioning on, for example, children's externalising symptoms or health harming behaviours such as smoking, consuming alcohol and taking illicit drugs, as these may be caused by both CEDV and child and adolescent internalising symptoms. These variables may be conditioned upon if they are not considered when identifying the sample population, resulting in selection bias. These four basic causal structures that give rise to associations between variables are illustrated in Figure 4. In the third structure, there is a marginal association between X and Y (because of Z), but not a conditional

association given Z. Contrastingly, in the fourth structure, there is no marginal association between X and Y, but there is a conditional association given Z. A causal DAG is simply an extension of such observations to more complex structures with many variables, made up of many structures of the four types illustrated.

Association within the population:

i.   X ⟶ Y   X causes Y (direct cause)

ii. X ⟵ Y   Y causes X (reverse causality)

iii.   Z
          ↙   ↘
        X   Y   Z causes X and Y (confounder/common cause)

Association within the subpopulation (conditional association):

iv. X - - - - - Y   X and Y have a common effect that has been conditioned on (collider/selection bias)
          ↘   ↙
           Z
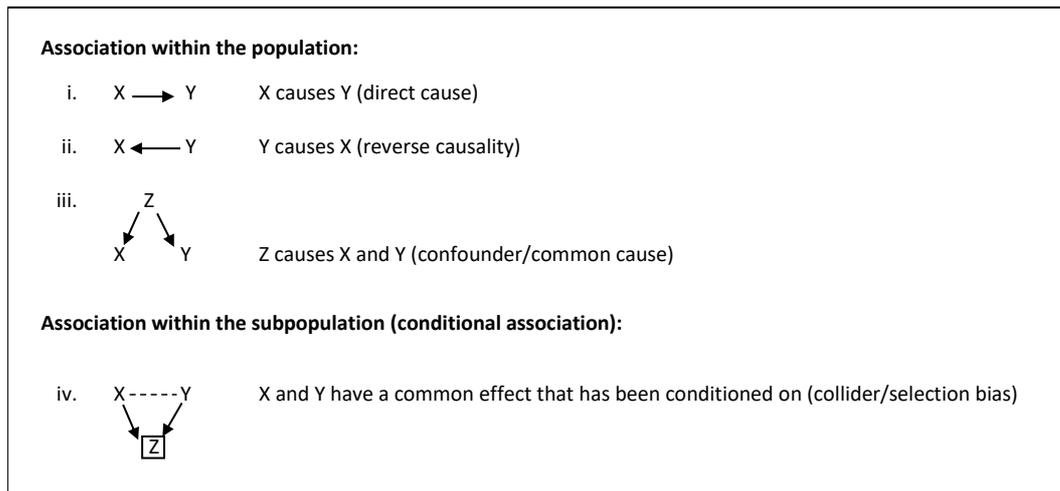
Figure 4. Four causal structures that can produce statistical association. Adapted from McGuire (2018) Epidemiology of Prenatal Alcohol Use and Fetal Alcohol Spectrum Disorder. Cardiff University: ORCA. Retrieved from https://orca.cf.ac.uk. X, exposure; Y, Outcome; Z, covariate

5 Covariates and potential confounders

In this section I will discuss the factors that increase the risk of both DV and internalising symptoms, and therefore must be considered in analyses as confounders of the causal effect of DV on internalising symptoms among children and young people.

Covariates are variables that are related to the outcome being studied such that they explain some of the variability in the outcome (Field-Fote, 2019). If the covariate is a cause of both the exposure and the outcome, then the covariate is a (common cause) confounder and should be statistically controlled for in order to avoid bias (Tyler J VanderWeele, 2019). This will also be the case if the covariate in question is a cause of either the exposure or the outcome and is associated (via an unmeasured common cause) with the other. It is important, however, when attempting to estimate the total effect of an exposure (in this case CEDV), not to control for mediators on the causal

pathway from this variable to the outcome (internalising symptoms in childhood and adolescence), as this would bias the estimate of the total effect (Robins, 1989; Schisterman, Cole, & Platt, 2009; Wilcox, Weinberg, & Basso, 2011). In this section I will provide evidence for covariates and potential confounders of the causal pathway between DV and internalising symptoms in children and adolescents that will need to be controlled for when estimating the strength of this pathway using multivariable modelling methods. It was not considered necessary for this evidence to be exhaustive, therefore studies were identified using Google Scholar. Possible covariates were identified by searching for studies that had identified risk factors for DV and child and adolescent internalising symptoms, as well as identifying factors that studies identified as part of the systematic literature reviewed had included as possible confounders. It is important to highlight here that this evidence synthesis is to be used as a starting point for justifying where the arrows are directed within the DAG; yet for confounder control, it makes sense to include any arrow that is plausible, based on a priori knowledge of the world, not just those for which there is evidence. Regardless, it is important to ensure that as many key potential confounders as possible have been identified and the best way to do that is by searching the relevant literature. Covariates identified were not grouped into individual, parent and family, and community factors as they are all related to the parent and family.

5.1 Maternal factors

5.1.1 Maternal age
Osborne, Lau, Britton, and Smith (2012) reported that risk of DV is increased among women aged 16-24 and men aged 16-19 years. There is also considerable research relating young mothers and teenage motherhood to internalising problems in children (D. M. Fergusson & Woodward, 1999; Harden et al., 2007; McGrath et al., 2014). Fergusson and Woodward (1999) investigated internalising symptoms at 18 years among 1025 New Zealand children using a birth cohort. They found that the young people of young mothers under 25 years were significantly more likely to have anxiety disorders compared to children of older mothers after controlling for significant covariates (SES, family type, pregnancy planning, and maternal relationship with mother). The children of young mothers were also significantly more likely to suffer from major depression than those of older mothers after again adjusting for significant covariates (maternal smoking during pregnancy and an

unhappy childhood). Harden et al. (2007) conducted a study with a cohort of 1,368 children of 482 female twins in Australia aged between 14 and 39 years at assessment. They found that children of adolescent mothers had more internalising symptoms than their younger siblings and their first cousins. Thus, these comparisons controlled for genetic and environmental variables shared by family members, but they did not control for paternal characteristics. Mother's age was collected as a covariate by Bair-Merritt et al. (2015) in their cohort study of the impact of DV on school age children's internalising and externalising behaviours, indicating that maternal age is associated with both DV and children's internalising symptoms. This evidence suggests that young maternal age increases the risk of DV, and young maternal age is also associated with more severe internalising symptoms, thus making DV a mediator on the causal pathway between young maternal age and internalising symptoms (see Figure 5). However more research is needed and that is not a question of interest within this thesis.

Figure 5. *Domestic violence as a mediator between young maternal age and child and adolescent internalising symptoms*

5.1.2 Maternal education

Maternal education is associated with multiple characteristics such as family income, background, and genetics (Harding, Morris, & Hughes, 2015). Education has been found to predict DV, however unemployment and low income have been shown to be stronger risk factors (Capaldi, Knoble, Shortt, & Kim, 2012). Capaldi et al. (2012) conducted a systematic review of risk factors for DV and reported that studies have shown an association, however this tends to dissipate when other factors are controlled. Sorenson (1996) conducted a study using the National Survey of Families and Households data in the U.S ($n = 6,779$) and reported that less educated participants were more likely to report physical violence in the past year compared to those considered highly educated. Cunradi, Caetano, and Schafer (2002) assessed the impact of SES factors on DV across races in the U.S while controlling for other

potential risk factors including alcohol abuse. It was reported that education predicted female perpetrated DV in White and Hispanic couples in the U.S but not male perpetrated DV or DV among Black couples as income was a greater predictor within those populations.

Poor maternal education has also been shown to be a risk factor for child and adolescent internalising symptoms. A study exploring behavioural problems (including internalising symptoms) among large samples of British and U.S children aged 7-17 years found that among the British children, poor maternal education levels were associated with higher behaviour problems, independent of family financial status; yet among American children the association between maternal education and behavioural problems did not exist after controlling for income (McCulloch, Wiggins, Joshi, & Sachdev, 2000). Maternal education has also been shown to be associated with both DV and adolescent depression by Gallo et al. (2017), and was therefore included as a confounder in their study exploring the effect of childhood maltreatment on depression at age 18 years, using a population-based cohort study in Brazil. An illustration of how education is associated with DV and internalising symptoms is presented in Figure 6. In conclusion, maternal education is an important confounder to be included in studies assessing the causal pathway between DV and internalising symptoms among children and adolescents.
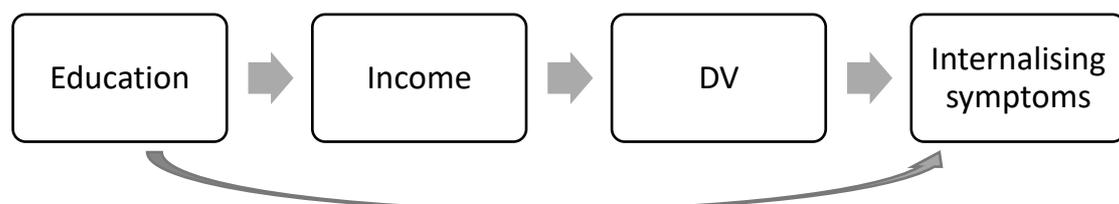
Figure 6. *Proposed causal pathway between mothers' education level and child and adolescent internalising symptoms*

5.2 Parental factors

5.2.1 Parental alcohol and substance abuse
The National Institute for Health and Care Excellence (NICE) states that the role played by alcohol and substance abuse in DV is poorly understood as people may be less inhibited and more likely to be abusive when under the influence of alcohol and/or drugs, but DV may also lead one to drink or take drugs to numb the pain or guilt. Research has suggested that among those who reported DV within the past year,

21% reported the perpetrator was under the influence of alcohol and 8% said they were under the influence of drugs (Osborne et al., 2012). Parental alcohol abuse has been shown to be a risk factor for internalising symptoms in children of alcoholics (COAs). DeLucia, Belz, and Chassin (2001) showed that children and adolescents of alcoholics and former alcoholics had higher levels of internalising symptoms than children whose father was never an alcoholic. Furthermore, these symptoms did not decrease when parental alcoholism decreased (DeLucia et al., 2001). These findings are in line with previous research suggesting that COAs are at increased risk of internalising symptoms in childhood as well as continued negative developmental trajectories (Loukas, Piejak, Bingham, Fitzgerald, & Zucker, 2001; Peterson Edwards, Leonard, & Eiden, 2001; Tubman, 1993). Hussong, Flora, Curran, Chassin, and Zucker (2008) conducted a longitudinal study using a large sample of data (N = 596) from a large cohort study, the Michigan Longitudinal Study MLS, and also showed the enduring effects of parental alcoholism on COAs internalising symptoms after controlling for both parent depression and antisocial personality disorder.

Studies have generally linked parental substance abuse to children's internalising symptoms. For example, de Cubas and Field (1993) found that children of substance-abusing mothers aged 6-13-years scored had more internalising symptoms, as measured using the Child Behaviour Checklist (CBCL; Achenbach and Edelbrock, 1991) , compared to matched controls, and Wilens, Biederman, Kiely, Bredin, and Spencer (1995) reported increased levels of internalising symptoms among school age children of opioid abusers compared to control children. Therefore, parental alcohol and substance abuse should be included as potential confounders within studies exploring the effect of DV on children and adolescents' internalising symptoms.

5.2.2 Divorce
Research has shown that divorce is associated with an elevated risk of domestic abuse including lethal violence and injury, particularly around the time of separation (Campbell et al., 2003; Logan & Walker, 2004; McFarlane et al., 1999). Research conducted by Amato and Anthony (2014) using child fixed effect models with large U.S child and adolescent samples have shown divorce is associated with increases in internalising symptoms among both children and adolescents. Research in the UK has also shown divorce has detrimental effects on internalising symptoms yet the impact depends on child age. Fitzsimons and Villadsen (2019) explored the impact of

parental divorce/separation resulting in father departure. They reported that children and adolescents display a 16% rise in internalising symptoms and an 8% rise in externalising behaviours if their parents split when they are seven to 14 years old. Alternatively, if parents split earlier, the risk of emotional or conduct disorders does not increase, either immediately or by the age of 14 years, compared to those with intact family units.

5.2.3 Parental mental health

Research suggests that women who experience DV are more likely to experience a mental health problem, while women with mental health problems are more likely to be domestically abused, with 30-60% of women with a mental health problem having experienced DV (Howard et al., 2010). Additionally, women with mental health problems are more vulnerable to unsafe situations and relationships (McHugo et al., 2005).

A large number of studies have explored the relationship between parental mental health problems and child mental health, a systematic review summarising this literature (74 studies) reported that children of parents with psychiatric diagnoses are more likely to have mental health problems themselves, and often children develop the same problems as their parent(s) (van Santvoort et al., 2015).

5.3 Family factors

5.3.1 Child maltreatment

A review of 31 studies estimating the overlap of DV and child maltreatment reported co-occurrence rates between 30% and 60% in the studies examined (Edelson, 1999). Appel and Holden (1998) reviewed studies examining the co-occurrence of DV and child physical abuse specifically, and identified a co-occurrence rate of approximately 40% despite using conservative criteria to define child physical abuse. Nevertheless, rates varied depending on the child samples and the definition of violence used, yet clearly there is support of a substantial overlap between DV and child maltreatment.

A wealth of research has also reported the deleterious impact of child maltreatment on child and adolescent internalising symptoms (D Cicchetti & Toth, 1995; Kim & Cicchetti, 2010) and emotional disorders (Gilbert et al., 2009; Moylan et al., 2010).

5.3.2 Low socioeconomic status (SES) and financial difficulties

Although DV occurs within families across all income statuses, studies consistently indicate that as the financial/social status of a family decreases, the risk of DV increases (Benson, Fox, DeMaris, & Van Wyk, 2003; Benson, Wooldredge, Thistlethwaite, & Fox, 2004). Furthermore, those with financial difficulties may experience increased stress likely to impact on relationships. Benson et al. (2004) analysed data from the National Survey of Households and Families, which uses a large nationally representative sample of U.S. households, and data from the 1990 U.S. census. They found that as the ratio of household income decreases, the likelihood of DV increases. These findings are in line with earlier analyses of National Crime Victimization Survey data, another large nationally representative U.S. sample, that showed DV rates were five times greater in households with the lowest annual incomes compared with households with the highest annual incomes (Greenfeld, 1998).

Low socioeconomic status also increases the risk of mental health problems among children and adolescents (F. Reiss, 2013). Reiss et al. (2013) conducted a systematic review consolidating research investigating socioeconomic inequalities and mental health problems in children and adolescents from 55 studies. They reported that children and adolescents with low SES are at higher risk of mental health problems than those with high SES. Furthermore, results from a cohort study of 2,111 participants aged 7-17 years at baseline in Germany showed that all indicators of SES (household income, parental education and parental unemployment) significantly predicted internalising symptoms at the 2-year follow-up (Reiss et al., 2019).

5.3.2 Parental/child chronic illness

Osborne et al. (2012) reported that the risk of DV almost doubles among people with a chronic illness or disability, making them vulnerable. The risk is also increased among those with a mental health problem (Trevillion & Seneviratne, 2016). The impact of having a child with chronic disabilities on the likelihood of a relationship being abusive has not been explore, however the risk of child maltreatment is greater for children with chronic illnesses or disabilities compared to the general population(Jaudes & Mackey-Bilaver, 2008; Svensson, Eriksson, & Janson, 2013), and as DCA and DV are correlated, it may be they influence each other. Maternal mental health problems have also been shown to increase internalising symptoms in

childhood within a large Australian cohort study (Bayer et al., 2011). Furthermore a classic study reporting findings of the Ontario Child Health Study, a survey of 3,294 children aged 4 to 16 years in the general community, found that children with chronic illnesses and disabilities are at greater risk of experiencing psychiatric disorders compared to those without chronic illnesses and/or disabilities (Cadman, Boyle, Szatmari, & Offord, 1987).

5.3.3 Stressful life events

Stressful life events impacting on families, particularly those that cause economic stress, have been suggested to lead to an increased risk of DV (N. Smith & Weatherburn, 2013). For instance, Fagan and Browne (1994) argued that life transitions leading to economic stress are particularly important risk factors for male-to-female DV. Additionally, Smith & Weatherburn (2013) conducted a large-scale longitudinal study in Australia (N= 9,393) exploring the impact of personal stress, financial stress and social support on DV and indicated that women were more likely to have experienced DV if they had had personal stress, economic stress, and/or low social support. Negative and stressful life events such as violence, poverty, abuse, bereavement/loss of loved ones, house/school moves, or parental separation, have been reported to increase the risk for childhood anxiety (Stein et al., 1996), depression (Maughan & McCarthy, 1997; Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999; Toth & Cicchetti, 1996), and somatic complaints (Friedrich & Schafer, 1995).

5.3.4 Race/Ethnicity/Religion

DV has been shown to vary by ethnicity and religious culture (Ellison, Trinitapoli, Anderson, & Johnson, 2007). The Office of National Statistics (ONS) reported that within the years 2015 to 2017 women of Mixed/Multiple ethnic groups were most likely to experience DV (10.1%) compared to all other ethnicities (Bradley and Cooper, 2018). Asian/Asian British women were the least likely to have experienced DV (2.8%) according to the Crime Survey for England and Wales. In terms of religion, Christian women were more likely to have experienced DV than Muslim and Hindu women in the UK, according to the census data. Alternatively, in Iraq, Al-Tawil (2012) reported that the prevalence of psychological, physical and sexual violence against women were all higher in Muslim culture compared to Christian culture, and Muslim culture was found to be a significant risk factor for DV. Capaldi et al. (2012) concluded in their systematic review that ethnicity, and being in a

minority ethnic group, is a risk factor for DV. In the U.S the greatest risk of DV has been found among African Americans (Caetano, Ramisetty-Mikler, & Field, 2005; Huang et al., 2010), and in a New Zealand study, Marie, Fergusson, and Boden (2008) found that, after controlling for confounders, male and female participants reporting Maori ethnicity were at higher risk of experiencing DV than were non Maori participants. These studies suggest an association between ethnicity and DV; however other studies have shown that, after controlling for SES, differences in DV rates across ethnic groups are reduced. For example, Field and Caetano (2004) conducted a review of both cross-sectional and longitudinal research studies in the U.S investigating ethnic differences in IPV rates along with the roles of SES and alcohol use. They concluded that both SES and alcohol use play important roles in the association between ethnicity and IPV as differences in crude IPV rates diminished after adjusting for SES and alcohol use. However, Black couples were still at greater risk of IPV compared to their White and Hispanic peers, after controlling for such factors, suggesting that ethnicity still plays a role. The issue of unmeasured confounding remains, however, as it is very unlikely all possible confounders were measured. Thus, these results should be interpreted with some caution.

One's ethnicity may also increase the risk of experiencing internalising symptoms, however research is mixed. For depression, some studies report no differences across ethnic groups (Brooks, Harris, Thrall, & Woods, 2002; Costello et al., 1996), yet others report a higher prevalence of depressive symptoms among minority groups compared to White groups (Wickrama, Noh, & Bryant, 2005; Wight, Aneshensel, Botticello, & Sepúlveda, 2005). Hispanic and Native American children may be at greater risk than white children, while African American and Asian children appear to experience the lowest prevalence rates of depression (Saluja et al., 2004). In the U.S, researchers have found in several studies that Chinese children and adolescents experience equal or higher levels of internalising symptoms compared to North American peers (Chen, Rubin, & Li, 1995; Chun, Enomoto, & Sue, 1996; Lee, Okazaki, & Yoo, 2006; Sue, Nakamura, Chung, & Yee-Bradbury, 1994).

There does not appear to be an ethnic pattern to anxiety disorders, however inter-race differences may exist for specific anxiety diagnoses (Austin & Chorpita, 2004). For example, post-traumatic stress disorder may be more prevalent in African American children than White children; but White children appear to experience more

obsessive-compulsive disorder symptoms and social anxiety disorder (Last, Perrin, Hersen, & Kazdin, 1992). Somatic complaints also vary across cultures. Crijnen, Achenbach, and Verhulst (1999) conducted a study of nine countries, and found Australia and Jamaica had the highest number of complaints, followed by Puerto Rico, the USA, the Netherlands, Sweden, Thailand and Israel.

 McLaughlin, Hilt, and Nolen-Hoeksema (2007) examined the prevalence of internalising symptoms among a large sample of Black, White, and Hispanic/Latino adolescents in the U.S and reported that there were differences across cultures such that Hispanic females reported experiencing higher levels of internalising symptoms than other groups and Black males reported the highest levels of physiologic anxiety compared to males from other racial/ethnic groups. However, these results were limited by a lack of SES data, and the Hispanic/Latino American group was significantly larger; therefore there was more power to identify significant differences in this group. The lack of SES data is of particular concern given that in more economically developed countries (MEDCs), ethnic minority groups are likely to be in lower SES groups, and the social inequalities may account for the differences in internalising symptoms. Other cross-cultural studies have found few differences in childhood internalising symptoms across cultures (Austin & Chorpita, 2004), with the exception of cross-cultural differences in specific anxiety disorders. In the U.S., a study exploring ethnic differences in internalising symptoms among adolescents found that the effect of ethnicity diminished after controlling for SES (Kennard, Stewart, Hughes, Patel, & Emslie, 2006). The lack of research in the UK means it is unclear whether ethnicity should be included within analyses as a confounder but the evidence suggests it is likely that when SES is considered, ethnicity no longer has a significant effect on either DV or internalising symptoms.

6 Directed Acyclic Graphs (DAGs)

In this section I will provide an overview of DAGs and explain their purpose. Then I will present the DAG that I created displaying the risk factors, protective factors, and covariates identified through the evidence synthesis within this chapter and the previous chapters (systematic review and literature review). Finally, I will describe how it will inform the statistical modelling strategy for factors that protect children and adolescents against internalising symptoms following DV exposure.

Causal DAGs were introduced by Judea Pearl(Pearl, 1995), although routed in much earlier work on path diagrams by the geneticist Sewall Wright (1918; 1934). They are used to enhance causal inferences in epidemiology (Tennant et al., 2021). Pearl advocates that observed data plus causal knowledge drawn from the literature allows progression from statistical association to causality (Pearl, 2000). DAGs display assumptions about the causal relationships between exposures, covariates, and outcomes, including variables that may not have been measured. The causal relationships are represented by arrows from the cause to the effect and the following two rules apply: the DAG must be acyclic and therefore not contain any feedback loops, and the common cause of any other two variables within the DAG must be included (along with the relevant arrows) for the DAG to be considered causal (Sauer & Vanderweele, 2013). The graphical rules upon which DAGs are developed provide a means to identify which variables should be controlled for and which should not, in order to reduce bias in effect estimates (Williams, Bach, Matthiesen, Henriksen, & Gagliardi, 2018). DAGs can become complex and cumbersome to interpret (at which point algorithms can be employed) when they contain a large number of variables, but they still provide a theory driven approach to statistical modelling strategies as the hypothesised causal relationships within the DAG guide variable selection. Furthermore, the complexity is not a limitation of the DAG: it is simply a reflection (hopefully) of reality, enhancing transparency. The complexity is inherent to the problem and should be evident in the way in which the question is answered. For instance, if a question is answered using a limited number of factors chosen for convenience, then there is a high risk that the resulting inferences cannot be given a causal interpretation due to the many other potential confounders that were not considered.

Since DAGs may be used as both visual representations of hypothesised causal processes and as aides to support statistical modelling methodologies, I developed a DAG to describe the causal pathways of interest and used it to inform the statistical modelling strategy for the analyses in the following chapters.

The DAG depicts the hypothesised causal pathway between CEDV and child and adolescent internalising symptoms, focussing on factors that protect against internalising symptoms and therefore reduce the likelihood of children and adolescents suffering from internalising disorders.

The potential mediators and moderators of the causal pathway between CEDV and internalising symptoms have been identified through the systematic review in Chapter 2 and literature review in Chapter 3.

I colour coded the different elements so that mediators, potential moderators, and confounders at an individual, familial, and community level could be easily identified. This also allowed for the addition of hypothesised moderators within the DAG, which was important to me, given that many of the risk and protective factors have been found to exert their effects through moderating the effect of DV on child and adolescent internalising symptoms.

For illustrative purposes alone, I have extended the DAG to include arrows from potentially important moderators to the arrow representing the effect they are thought to modify. This is not strictly in keeping with the meaning of a DAG. A DAG is nonparametric and thus is agnostic as to the *nature* of the effects of the input nodes on their output node. As such, whenever two variables affect a third variable, it is perfectly possible for the effect of one to be moderated by the other. Indeed, such a statement is anyway ambiguous when we do not commit to how the effect will be expressed. For example, when the outcome variable is binary, there is at most one effect estimate measure (e.g. risk difference, risk ratio, odds ratio) that can be constant across levels of the other variable, and so the concept of moderation is inherently parametric and scale dependent. However, to illustrate why I am interested in particular variables in the DAG (and in my subsequent analysis), I have extended the DAG to include these additional arrows from the moderators to the effects they are thought to modify, where these are of particular interest. I have also included arrows illustrating how the identified variables contributing to the causal pathways between CEDV and internalising symptoms are related to each other, adding further to the complexity of the DAG. The associations between mediators are not based on review of the literature, as this was deemed to be outside the scope of the thesis, rather they were based on a priori knowledge of the subject area (see Figure 7).
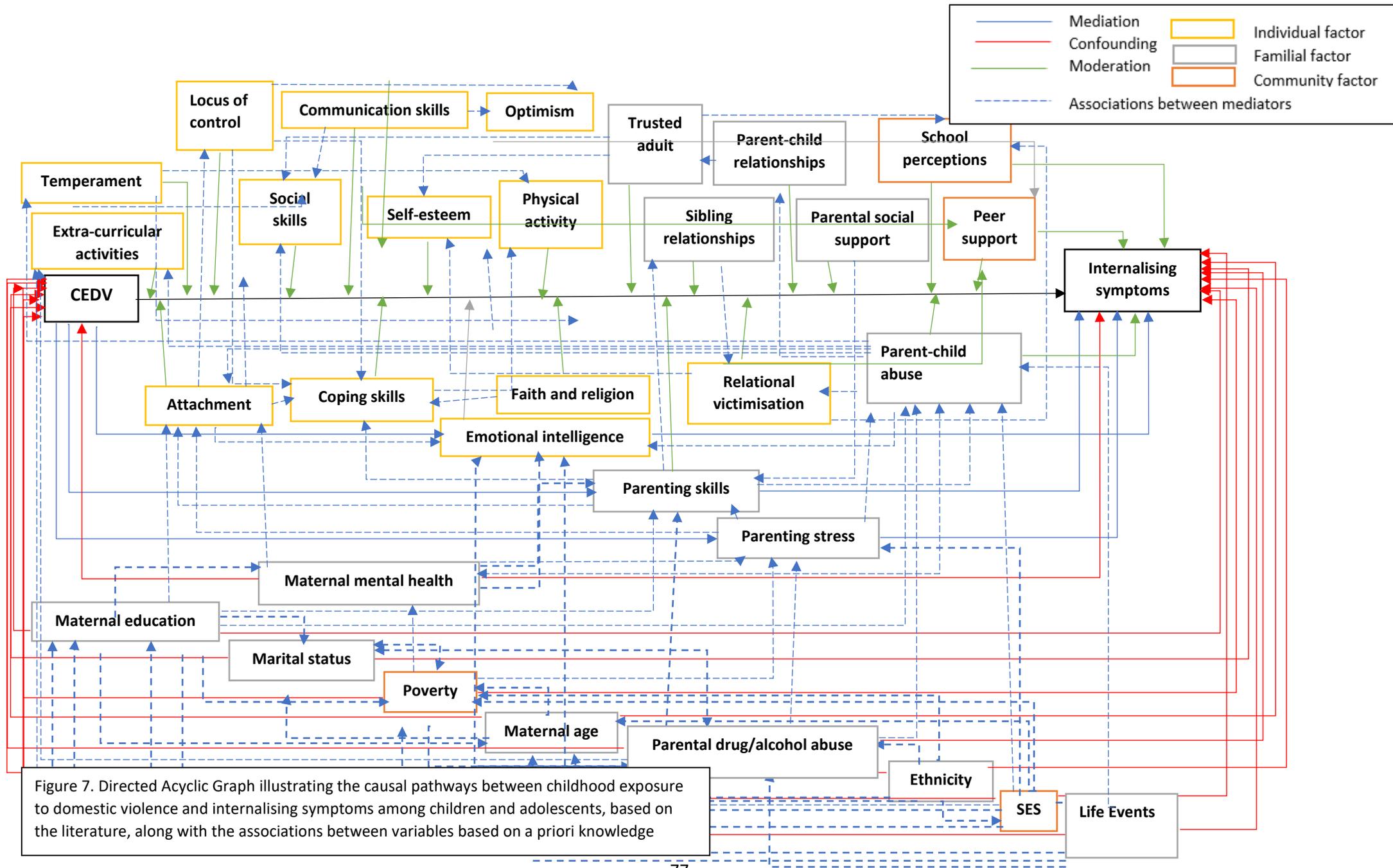
Figure 7. Directed Acyclic Graph illustrating the causal pathways between childhood exposure to domestic violence and internalising symptoms among children and adolescents, based on the literature, along with the associations between variables based on a priori knowledge

7 Discussion

Research has shown that the causal pathway between CEDV and internalising symptoms is multifaceted and complex. The DAG I have created highlights the pathways and the numerous factors which may increase or decrease the risk of experiencing internalising symptoms among children and adolescents exposed to DV through effect moderation. Following CEDV, the risk of experiencing internalising symptoms is determined by a range of individual, familial, and community factors that mutually contribute to child and adolescent outcomes. This causal diagram can be used to inform statistical modelling strategies for epidemiology studies exploring the causal pathways between CEDV and internalising symptoms.

7.1 Strengths and limitations of the DAG approach

This DAG builds upon evidence from existing studies of the causal pathway between CEDV and internalising symptoms. Formally bringing this evidence together in a DAG has not been attempted previously within this field, where discussions thus far have predominantly been associational. The approach synthesises the latest evidence from both the DV and ACEs literature fields, in terms of protective factors for children and adolescents, and the DAG provides a visual, unified summary.

The DAG is likely to be subject to change in light of new evidence, given that it is based on the current evidence as of 2019, in a field that is relatively new and expanding extensively. This is not a limitation of the DAG approach, but simply highlights the realities of making causal inferences within a developing evidence base.

Researchers may use this DAG as a starting point and build upon it, providing further pathways, moderators, and covariates. It may also be used as a tool to guide analyses, ensuring bias is reduced as much as possible.

The causal inferences derived from a DAG depend upon it being valid. As DAGs require interpretation of the available evidence, it is possible that other researchers may interpret the same evidence differently, leading to a different DAG displaying different assumptions. However, the fact that DAGs increase the transparency of the assumptions upon which variables and adjustment sets are based, means that they are open to scrutiny which is of benefit to science (Tennant et al., 2021).

The limitations inherent within the DV literature will also apply to this DAG. Therefore, the causal assumptions made may be based on research of limited quality often relying on cross-sectional data, studies without control groups and often with non-representative samples of mother-child dyads living within shelters or within clinical services. Furthermore, although it is possible to measure a variety of co-occurring variables in observational studies, the evidence thus far has focussed on a limited number of factors at a time. Thus, relationships between factors have not been explored sufficiently yet (see systematic review for full review of evidence quality).

Finally, as mentioned above, DAGs are nonparametric and one cannot be anything other than agnostic about moderators in a nonparametric model. This is a challenge when trying to explore the causal pathway between CEDV and internalising symptoms as many factors increase or decrease the likelihood that a child will experience internalising symptoms through moderation. However, it is possible to augment a DAG with indications of where one thinks moderation is likely to be important. I have followed Weinberg's suggestion in the DAG by illustrating effect moderation with an arrow that intersects the causal pathway between CEDV and internalising symptoms. This representation is purely for visual illustration.

Despite their growing popularity, some argue that DAGs and the counterfactual approach are becoming almost synonymous with causal inference and as such, limit the field when many other approaches are available (Krieger & Davey Smith, 2016). Kreiger and Davey Smith (2016) highlight that DAGs can lead to spurious causal inference, potentially causing harm, if assumptions are invalid. However, this is the case with any tool or statistical method. Regardless, it is important to highlight that DAGs require a great deal of thought and interpretation of the literature.

8 Contribution of this chapter to the thesis

The DAG presented in this chapter amalgamates and illustrates current knowledge of the causal pathways between CEDV and child and adolescent internalising symptoms. In the following chapters I will investigate factors that may protect children and adolescents from experiencing internalising symptoms following exposure to DV using secondary cohort data. I will use the DAG to identify the possible mediators and moderators of interest available within the dataset (those protective factors within the DAG that are potentially modifiable), and to determine the confounders that need to be statistically controlled for within the

analyses. Based on the DAG above, analyses attempting to estimate mediation and/or moderation should include at least the following covariates: maternal education, maternal age at birth, marital status, poverty, socioeconomic status, life events, ethnicity, and parental drug/alcohol abuse. In addition, the DAG makes clear that it will be important to take care when performing mediation analyses since some mediators affect other mediators. I return to this in Chapter 6.

In the next chapter, I will adapt the DAGs based on the data available within the cohort dataset, which will then aid the design and execution of the statistical analyses.

Chapter 5. Child and adolescent internalising symptoms following exposure to domestic violence: a population-based cohort study

1 Overview

In this chapter, I investigate protective factors for internalising symptoms following CEDV using data from a population-based birth cohort in England (ALSPAC). First, I present the aims and objectives of this chapter, followed by the methods, utilising the causal diagram from the previous chapter to inform the statistical modelling strategy. I then present the challenge faced by missing data in the dataset and how this was overcome, along with results from the negative binomial regression modelling. I conclude the chapter with a discussion of the strengths and limitations of this work.

1.1 Background

As previously discussed, children and adolescents exposed to DV may experience internalising symptoms; however, there is variation in outcomes and many children and adolescents do not develop internalising symptoms at all, demonstrating resilience in that aspect of health. Therefore, it is important to identify factors that protect against internalising symptoms as well as those that increase risk, in order to inform preventative strategies and interventions.

In the previous chapters I identified various factors that are involved in the pathway between CEDV and internalising symptoms. Some of these factors act as mediators and/or moderators and I summarised results from the literature in a DAG. Following exposure to DV, a child's risk of developing internalising symptoms may be influenced by individual traits and personal skills, sociodemographic characteristics, family characteristics and functioning, and social structures. However, to date, literature has primarily focussed on risk and neglected the numerous positive personal attributes and assets that children and young people may possess, which may protect them against negative developmental outcomes (e.g., internalising symptoms) following adversity (e.g., CEDV). Research that has explored protective factors has rarely taken a causal approach. Thus, studies have seldom used longitudinal data or controlled for multiple confounders factors. Furthermore, the majority of research within the field of DV and child and adolescent internalising symptoms has been conducted in the USA, with very few studies conducted in UK datasets. Moreover, studies that have explored

protective factors have tended to examine them in isolation, therefore it is unclear which factors have the greatest influence, and thus should be focussed upon within interventions aimed at reducing or preventing internalising symptoms among children and young people. Given these limitations within the current evidence base, the study presented in this chapter aimed to assess the impact of modifiable protective factors that may mediate and/or moderate the pathway between CEDV and internalising symptoms.

I considered ALSPAC to provide an excellent opportunity to address the aims of this study given that it is a population-based birth cohort study in the UK and the vast amount of data collected. Data collected in ALSPAC includes information on childhood adversities including DV, and developmental outcomes including internalising symptoms, along with numerous assets and behaviours of both parents and children, and demographic and social factors necessary to include as confounders within multivariable regression analyses. Furthermore, both DV and internalising symptoms have been measured at multiple time points across the children's lives, making it possible to investigate the extent to which early exposure to DV leads to internalising symptoms later in life (adolescence) whilst controlling for factors such as earlier internalising symptoms.

1.2 Aims and objectives

1.2.1 Aim

To quantify the effect of potentially modifiable protective factors on internalising symptoms among children and adolescents in the ALSPAC cohort who have been exposed to DV.

1.2.2 Objectives

1. To use statistical methods to overcome the challenges posed by missing data within this cohort study

2. To estimate the total effect of early CEDV on internalising symptoms at early childhood and adolescence (six years and thirteen years respectively) adjusting for covariates identified within the literature

3. To examine the extent to which potentially protective factors (from the DAG developed in Chapter Four) mediate and/or moderate the effect of CEDV on internalising symptoms

2 Method

2.1 Data Source

The dataset comes from the Avon Longitudinal Study of Parents and Children (ALSPAC), a prospective longitudinal birth cohort study. ALSPAC recruited 14,541 pregnant women from the Bristol area between 1990 and 1992. Follow-up of participants is ongoing and children of mothers from the original cohort are now roughly 31 years old (mean age ~ 28 years in May/June 2020). The ALSPAC dataset includes repeated measures of DV and other ACE exposures, child behavioural and psychological outcomes, child developmental factors, family characteristics and functioning, lifestyle, community, and sociodemographic factors. The data were collected through a range of questionnaires, in-clinic assessments and data linkage (including the National Pupil Database and the Pupil Level Annual School Census). ALSPAC sample characteristics, study phases, methodology and representativeness have been described in previous publications (Boyd et al., 2013; Fraser et al., 2013), and in detail on the study website (http://www.alspac.bris.ac.uk/welcome/index.shtml). Participants recruited into the ALSPAC cohort were of higher socioeconomic status and less likely to be of non-White ethnicity compared to both the general population of mothers with children under the age of one in Britain at the time of recruitment, and those in the Avon area who were not enrolled in the ALSPAC study (Boyd et al., 2013; Fraser et al., 2013). These differences present important limitations when attempting to generalise prevalence estimates from the ALSPAC cohort to the general population of the UK but representativeness is somewhat less of a concern for the validity of the protective factor regression analyses. As Rothman notes, careful consideration of causal mechanisms and effective control of confounding is arguably more important than the representativeness of the sample when making inferences about cause-effect relationships (Rothman et al., 2013).

2.2 Study Approval

Ethical approval for this study was obtained from the ALSPAC Ethics and Law Committee (IRB00003312) and the Local Research Ethics Committees. Project approval was granted by the ALSPAC Executive Committee on the 12th August 2019 (Project B3352).

2.3 Participants

This study included a sub-sample of all live births within the core ALSPAC sample (born between 1990 and 1992), alive at one year. Families who joined the study at a later date, those without data past the child's birth and missing important demographic characteristics, participants who did not speak English, and participants who were in the armed forces social

class category were excluded. Armed forces families were excluded due to their low number (n = 28), which caused computational problems in imputation models. This left a population sample of 10,182 children (see Appendix B for a flow diagram).

2.4 Variables

2.4.1 Exposure

DV exposure was measured using two questions to both mothers and partners asking whether their partner has been physically or emotionally cruel to them since the birth of the child or the previous wave of questionnaires. Both partners were asked about DV when the child was 8, 21, 33, 47 months and at nine years of age. A child was considered to have been exposed to DV at each time point if either partner responded positively ("Yes") to either physical or emotional cruel towards them (Flach et al., 2011). In this analysis, the measures at 8, 21, 33, and 47 months were combined to give a binary variable identifying any DV exposure at 0-3 years (described as early exposure). The binary variable was coded as "No" if neither mother or partner responded "Yes" to any of the DV items, and if there were missing observations but none were answered "Yes", they were also coded as "No". Previous studies investigating early childhood DV exposure (Bowen, 2015) have not included measures of DV at 47 months, therefore prevalence estimates are expected to be somewhat higher.

2.4.2 Outcome

The primary outcomes were internalising symptom scores at six and thirteen years. Mothers completed The Strengths and Difficulties Questionnaire (SDQ; about their children), a widely used instrument for assessing psychological morbidity in children Goodman (2001); (R. Goodman, 1997). The SDQ measures four domains of psychological health, on separate scales with five items each: emotional problems, peer problems, hyperactivity, and conduct problems (Goodman, 1997, 2001). The internalising subscale was used in this study which combines the emotional symptoms subscale and the peer problems subscale and includes items relating to anxiety, depression, somatic complaints, and social withdrawal symptoms. The SDQ asks 10 questions about internalising symptoms using a 0-2 Likert scale with zero meaning "Never", one meaning "Sometimes", and two meaning "Often". The total possible internalising symptoms score at one age point (the sum of the scores for these ten items) was thus twenty. This variable was treated as quantitative in the analysis, but its non-normal (conditional) distribution was accommodated using negative binomial regression. It is possible to interpret the scores within categories with scores of 0-5 reflecting roughly "normal" levels of internalising symptoms, a score of 6-8 suggesting "borderline" symptoms,

and nine or over implying "abnormal" internalising symptom levels (Becker, Rothenberger, & Sohn, 2015).

2.4.3 Potential moderators and/or mediators

These factors were identified from a review of the wider resilience and ACEs literature (see Chapter 3) due to a paucity of research exploring protective factors for internalising symptoms among children and adolescents exposed to DV. See Table 6 for a summary of all included variables and when they were measured in this study.

Table 6. Exposures, outcomes, explanatory factors and covariates included in analyses and when they were measured (child years).

Years	Pregnancy	0	1	2	3	4	5	6	7	8	9	10	11	12	13
Exposure		Domestic Violence														
Outcome								SDQ								SDQ
Explanatory factor																
Individual Level																
Organised activities													★		
Attitude to school										★					
LoC										★					
Emotion awareness										★					
Friendships										★					
Self-esteem										★					
Physical activity													★		
Activities with friends													★		
School problems											★				
Bullying										★					
Externalising behaviours												★			
Intelligence										★					
Temperament					★										
Family Level																
Domestic violence								◆——————							
Sibling interaction								★							
Mother social support											★				
Sibling bullying														★	
Parenting practices					★										
Maternal depression							★					★			
Maternal anxiety							★					★			
Maternal parenting					★										
Paternal parenting					★										
Parent-child relationships									★		★				
Covariates																
Direct child abuse		◆————					◆————							
DV in pregnancy		★													
Parental aggression		★													
Maternal age			★												
Maternal education		★													
Ethnicity		★													
Alcohol and drug abuse	◆———														
Occupational social class		★													
Financial difficulties		★	★			★									
Marital status				★	★				★						
Chronic illness (household)			★												
Mother's health					★										
Stressful life events							★			★					
Maternal historical MH prob	★														
Paternal historical MH prob	★														

2.4.3.1 Individual level factors

Protective factors were measured during the time between the exposure and the outcome, therefore in relation to the outcome of internalising symptoms at six years they were measured at 4-5 years, and in relation to internalising symptoms at thirteen years protective factors where measured when the children were aged eight to 11 years. Additionally, protective factors at 4-5 years were included when estimating the effect of early DV exposure on internalising symptoms at age 13 years. Ideally the protective factors would have been measured before the exposure, however this was not always possible given the exposure measure assessed DV from birth.

Child participation in organised activities

For this study children's participation in organised activities was measured when they were 11 years old using mother-reported child activity variables. Mothers were asked whether their child attended any special sport or group activities. Each of the ten items was scored on a Likert-scale from 1 "nearly every day" to 6 "not at all". These activity variables were then summed to give a total "organised activities" score variable from 10 to 60.

Children's attitude towards school

Children's attitude towards school was reported by children when they were eight years old. Three items asked whether children are happy at school, like school, and feel safe when playing in the playground. Responses to these questions were scored on a 4-point Likert scale from 1 "Strongly agree" to 4 "Strongly disagree". These were then summed to give a composite "attitude towards school" variable.

Locus of control

Children's locus of control (LOC) was measured in the ALSPAC clinic when children were eight years old using an adaptation of the Children's Nowicki Strickland Internal External scale (CNSIE, Norwicki and Strickland, 1973) consisting of 12 items (Nowicki, Gregory, Iles-Caven, Ellis, & Golding, 2018). The CNSIE has been used in hundreds of studies that have provided data supportive of their construct validity. Investigators read the LOC questions out loud to the child (to control for reading ability) and children responded with a yes/no answer. Children were considered to have either an internalised or externalised LOC overall. The summary score was used in these analyses.

Emotional awareness

Children's emotional awareness was measured during clinic visits when the children were eight years using the faces subset of the Diagnostic Analysis of Nonverbal Accuracy (DANVA; Nowicki & Carton, 1993). This computerised task measures a child's ability to identify emotions from facial cues. Participants were presented with images of children showing facial expressions of happiness, sadness, anger, or fear. Higher scores represent more errors, or misattributions, suggesting poorer emotional awareness (Kothari, Skuse, Wakefield, & Micali, 2013).

Friendships

Children's friendships were measured during the clinic assessment when children were eight years. Trained psychologists interviewed the children using the Cambridge Hormones and Moods project Friendship questionnaire (Goodyer, Wright, & Altham, 1989). Children were asked questions about their satisfaction with friendships, how often they see their friends outside school, and whether they can talk to their friends about problems and they are understanding. The seven items were then summed to give a composite summary score from 0-15.

Child self-esteem

Children's self-esteem was measured during the ALSPAC clinic assessment at eight years using a shortened form of Harter's Self Perception Profile for Children (Stewart, Roberts, & Kim, 2010). This version consisted of the global self-worth and scholastic competence subscales of the original measure, comprising 12 items. Responses to these items were summed to give a total self-esteem score, with lower scores indicating poorer self-esteem.

Child physical activity

Children's physical activity was measured when they were 11 years old during their ALSPAC clinic visit. Mean minutes of vigorous, moderate, and gentle exercise were measured over 2 weeks prior to their visit. Children were asked to wear an Actigraph AM7164 2.2 accelerometer (Actigraph LLC, Fort Walton Beach, FL, USA) around their waist, at the right hip, for seven days. A valid day was defined as providing data for at least 10 hours per day (excluding sequences of 10 or more minutes with consecutive zero counts).

Child activities with friends

Children's friendships were measured when they were 11 years during the clinic assessment. Children were asked by qualified psychologists whether or not (yes/no) they participate in a

range of 27 activities with friends including going to school, clubs, sports centres, town, and cafes. These items were then summed to give a total score between 0 and 54 with higher schools reflecting more activities participated in with friends.

Child problems at school

Children's problems at school were reported by mothers when children were nine years old. Mothers were asked whether their child has been identified as having a particular problem at school. This could include physical disabilities, learning difficulties, emotional and behavioural problems.

Bullying

Bullying was measured with a structured face-to-face interview with the child participants at the 8.5-year clinic visit, using the Bullying and Friendship Interview Schedule (Wolke, Woods, Bloomfield, & Karstadt, 2000). Children were asked five questions assessing whether they had been a victim or perpetrator of overt or relational bullying. Overt bullying includes having belongings stolen; being threatened or blackmailed; being hit or beaten up; having been called names; or nasty tricks played on you. Relational victimisation includes other children not wanting to play; trying to get someone to do something they do not want to do; withdrawing friendship; telling tales; spreading lies/rumours; deliberately spoiling games; and doing other things to upset people (Horwood, Waylen, Herrick, Williams, & Wolke, 2005). At no time during the interview was the word bullying mentioned, so as not to prompt the child, only behaviours were used. All interviewers were fully trained in the measure, and regular checks were made to ensure reliability and avoid any bias. Children were coded as peer victims or bullies if they reported victimization or perpetration of these items at least four times in the last six months (Wolke et al., 2013).

Intelligence

Children's verbal and non-verbal/performance ability was measured when they were eight years old, as part of the clinic visit, using the Wechsler Intelligence Scale for Children (WISC-III; Wechsler (1991). The questionnaire contents and details on how it was administered can be found here: https://www.closer.ac.uk/cross-study-data-guides/cognitive-measures-guide/alspac-cognition/alspac-age-8-5-wechsler-intelligence-scale-for/. For this study, the total score was used, which was the sum of the verbal and non-verbal scores.

<u>Temperament</u>

Temperament was measured using Buss and Plomin's Emotionality Activity Sociability (EAS) Temperament Survey for Children (Buss & Plomin, 2014), reported by mothers when their child was three years old. This survey includes 20 statements about behaviours and mothers rated the extent to which each statement describes their child on a scale ranging from one (not at all like) to five (exactly like). Scores on these items were combined to form four subscales (each comprising five items): emotionality (tendency to show distress, e.g., cries easily, reacts intensely when upset), activity level (e.g., is always on the go or is very energetic), shyness (e.g., tends to be shy, takes a long time to warm up to strangers), and sociability (e.g., likes to be with people, prefers playing with others to playing alone). The factor structure of the EAS has been demonstrated to correspond well to these four traits and remain stable over time (Bould, Joinson, Sterne, & Araya, 2013).

2.4.3.2 Family level factors

<u>Sibling interaction</u>

Mothers reported on their children's sibling interactions when they were six years old. They were asked eight questions determining how often the children play, read, sing, make things, go out, talk, eat, and argue with their siblings. These items were summed to give a composite sibling interaction score.

<u>Mother Social network/support</u>

Mothers' social network was reported by mothers when children were nine years old. The social network scale in this study comprises 16 items which ascertain the extent of the mother's social networks and support. Mothers reported on ten items assessing the number of people in their lives such as the number of people in their lives they can go to in order to discuss personal problems, get advice on important decisions or borrow money from, as well as the number of times in the last month they have got together with friends and/or relatives. They then responded to six questions with statements relating to emotional, financial and instrumental support such as: 'My partner provides the emotional support I need', 'I'm worried that my partner might leave me' and 'If I was in financial difficulty, I know my family would help if they could'. Mothers selected one of four responses: Exactly feel, often feel, sometimes feel and never feel. These items were then all summed to a score ranging from 12 to 47, with a higher score indicating more positive social networks.

Sibling bullying

Children reported on whether they were bullied by siblings when they were 12 years old using a standard sibling bullying questionnaire (Tucker, Finkelhor, Turner, & Shattuck, 2013) adapted from the widely used Olweus Bullying Questionnaire (Olweus, 2007a). Children were first asked whether they had a sibling. Then questions asked how often children were bullied by their siblings at home during the past six months. Responses were scored on a 4-point Likert scale from 1 "Several times a week" to 5 "Never. These items were summed to give a total "sibling bullying" score.

Maternal and paternal postnatal depression

Mother's postnatal depression symptoms were measured using the Edinburgh Postnatal Depression Scale (EPDS; Cox et al., 1987) when their child was eight months old. The 10-item EPDS (Cox, Holden, & Sagovsky, 1987) assesses depressive symptoms during the prior week and respondents rated the frequency of each symptom on a Likert-type scale with four response options (coded 0-3), the exact wording of which varies depending on the item. Positive questions were reverse coded and all items were summed to give a score ranging between 0 and 30, where higher scores indicate more depressive symptoms (Paul & Pearson, 2020). Mothers and fathers were considered to be depressed if they scored 13 or more (Plant, Jones, Pariante, & Pawlby, 2017), or 10 or more (Edmondson, Psychogiou, Vlachos, Netsi, & Ramchandani, 2010) respectively. Dichotomous variables were created with depressed versus non-depressed.

Maternal and paternal depression

Mothers were asked if they had experienced depression (yes/no) during the past year when their children were five years old. They were also asked if they had experienced depression in the past two years when their children were 10 years old. The variable at five years was used during analyses assessing internalising symptoms at six years and the measure at 10 years was used in analyses using internalising symptoms at 13 years.

Mothers were asked if their partner had experienced depression (yes/no) during the past year when their children were three years old. They were also asked if their partner had experienced depression in the past two years when their children were six years old. Again, the variable at three years was used during analyses assessing internalising symptoms at six years and the measure at six years was used in analyses using internalising symptoms at 13 years.

<u>Maternal and paternal anxiety</u>

Mothers were asked if they had experienced anxiety (yes/no) during the past year when their children were five years old. They were also asked if they had experienced anxiety in the past two years when their children were 10 years old. The variable at five years was used during analyses assessing internalising symptoms at six years and the measure at 10 years was used in analyses using internalising symptoms at 13 years.

Partner anxiety was measured by asking mothers if their partner had experienced anxiety (yes/no) during the past year when their children were six years old.

<u>Maternal and paternal parenting</u>

Mothers self-reported the frequency at which they, and their partners, completed activities with their children when they were three years old. Ten items, developed for ALSPAC, were used assessing activities including bathing, feeding, playing, and cuddling their child. These were summed to give total mother, and partner, parenting scores.

<u>Parent-child relationships</u>

Mothers were asked eight questions about their relationship with their child and the same eight questions for their partner and child's relationship when children were seven years old. These questions were developed for ALSPAC and assess aspects of the relationships including feelings towards the child and child's behaviour towards the parent. Responses were either yes, no, or sometimes. These responses were recoded 0-2 and negative items were reverse scored.

Children were asked about their relationships with their parents when they were nine years. Eight items were included in this study which asked about children's feeling towards their parents, time spent with them and closeness. Responses were recorded on a Likert scale from "not true" to "very true". These items were summed with the mother-reported items to give a total parent-child relationship composite variable.

2.4.4 Confounders

Possible confounders were also identified from the DV and child and adolescent internalising symptoms literature and evidence for these is discussed in Chapter 3. As mentioned previously, is important to note that these potential confounders (or later measurements thereof) may also be mediators of the effect of DV on internalising symptoms, the extent of which cannot be assessed from the data (Tyler J. VanderWeele & Shpitser, 2013).

Direct child abuse

Direct child abuse (DCA) was measured using four questions to both mothers and partners asking whether they or their partner has been physically or emotionally cruel to their children since the study child was born or since the previous wave of questionnaires. Direct child abuse questions were asked when the child was 8, 21, 33, 47 months and at nine years of age. A child was considered to have been a victim of child abuse at each time point if either the mother or her partner responded positively to being either physical or emotionally cruel towards their children. The measures at 8, 21, 33, and 47 months were combined to give a binary variable identifying any DCA from 0-3 years, or early exposure, and the questions at nine years were combined to give a binary, more recent DCA variable, covering the period of middle childhood from 6-9 years.

Domestic violence during pregnancy

DV during pregnancy was measured using two questions to both mothers and partners asking whether their partner had been physically or emotionally cruel to them since conception or the previous questionnaire. DV during pregnancy was measured at 18 weeks gestation and at roughly eight weeks after the child was born.

Parental aggression

The parental aggression score was derived from three questions: 'Does your partner get angry with you?', 'Do you have arguments with your partner?' and 'Do you get angry with your partner?' (http://www.bristol.ac.uk/alspac/researchers/resources-available/data-details/questionnaires/). This measure has been used in previous studies (Puckering et al., 2014; Washbrook, Gregg, & Propper, 2014). Mothers selected one of the following responses: almost always; often; sometimes; barely, and never (scored 1-4). These responses were then summed to give a composite parental aggression score variable. Scores were split at the median to create a dichotomous variable (high and low aggression).

Maternal age

Maternal age was recorded at around eight weeks after the child was born. Maternal age at birth was grouped into six categories: under 20, 18-22, 25-29, 30-34, 35-39, and 40 plus.

Maternal education

Maternal education levels were reported by mothers self-reporting their highest qualification during pregnancy. There were five categories: CSE, Vocational, O Level, A Level, and Degree.

Ethnicity

Mothers reported their own, their partner's, and their child's ethnicity during pregnancy. Mothers' ethnicity options for her and her partner were White, Black Caribbean, Black African, Black Other, Indian, Pakistani, Chinese, and Other. However, due to small number of non-White participants these variables were dichotomised into White and non-White groups. Child's ethnicity was reported as white or non-white by mothers.

Parental alcohol and substance abuse

Mothers and fathers self-reported whether they had ever had an alcohol or drug addiction during pregnancy and fathers reported if they had taken hard drugs since the child was born when their baby was roughly eight weeks old.

Maternal and paternal occupational social class

Maternal and paternal occupational social class was reported during pregnancy. Responses were grouped into six classes as categorised by the OPCS (1991): I (professional/managerial), II (managerial and technical), III (manual), III (non-manual), VI (semi-skilled manual), and V (unskilled manual labour). These classes were then grouped into three categories: I and II; III (manual and non-manual); VI and V. Note that those in the Armed Forces, a separate category making up 0.3% of the ALSPAC cohort (Khandaker, Zammit, Lewis, & Jones, 2014), were excluded due to small numbers.

Financial difficulties

The financial difficulties measure was constructed of a series of five questions. Mothers self-reported how difficult it currently is to afford food, clothes, heating, rent/mortgage and other things considered essential for the child, on a scale from zero to three, with higher scores indicating more difficulty. These measures were taken twice when the child was eight weeks and two years old. Fathers also self-reported financial difficulties using a binary (yes/no) measure when children were eight months old.

Mother's marital status

Mothers self-reported their marital status when their child was two, three, and seven years old. The five categories (never married, divorced, separated, widowed, and married) were combined into three groups: never married; divorced/separated/widowed; and married.

Household chronic illness

Mothers reported whether someone in the household had a chronic illness (yes/no) when the child was roughly eighteen months old. This could be the mother, study child, or another person living in the same household as the study child.

Stressful Life events

Mothers reported on children's stressful life events when their child was three and eight years old. They were asked 16 questions about potential life events such as parental separation, moves, new beginnings, accidents and abuse. These items were summed with higher scores indicating more stressful life events for the child.

Previous parental mental illness

Mothers and fathers were asked, before the study child was born, to self-report whether they had ever had depression or anxiety. These four variables were binary (yes/no). They were asked this again when children were seven years old.

2.5 Statistical Analyses

2.5.1 The threat to validity posed by missed data within cohort studies

Missing data are pervasive in clinical epidemiological research. In many settings, participants whose value for a particular variable is missing are likely to differ from those whose value for that variable is observed. In particular, they can differ in their distribution of the outcome and/or exposure of interest, as well as other important characteristics, including the partially-observed variable itself. There are many approaches to deal with missing data, ranging from simple methods, which tend to be inefficient and risk the greatest bias, to more complex and principled methods which are more efficient and – when applied sensibly – lead to less bias due to missing data in the final results.

2.5.2 Types of missing data and analytical methods for dealing with it

Missing data can be described and classified in many ways. One distinction that is often made is between missing completely at random (MCAR), missing at random (MAR) and missing not at random (MNAR) – see Rubin (1976); Donders et al., (2006); Pederson et al. (2017) for explanations. Broadly speaking, MCAR, MAR and MNAR represent situations of increasing severity in terms of the incompleteness of the data. For MCAR, the probability of an item being missing is independent of its value, and of the value of any other item in the dataset. At the other extreme, when an item is MNAR, the probability of it being missing is inextricably linked to the value itself, such as when the fact of being the victim of DV itself makes you

less likely to report this. In between, there is MAR, where although the probability of an item being missing is associated with its value, that association can be broken by conditioning on some information, e.g., gender, or the answer to another item, contained in the data.

Although MCAR missing data might realistically occur in some limited situations, the majority of missing data in clinical epidemiological research is likely to be MNAR. That said, analysis methods that are valid under the assumption of MAR dominate the literature on how best to analyse incomplete data. This is because a MAR analysis is usually the 'best that can be done' using the data available. In an MAR analysis, by including as much information as is feasible that plausibly affects both the value of the missing variable and the fact of its observation, the hope is that any remaining bias (due to MNAR missingness) is small, and smaller than it would have been had no attempt been made to 'deal with' the missing data in this way.

Another aspect of missing data to consider is the missing data pattern. Common terms used to describe missing data patterns, particularly within epidemiological studies, are univariate or multivariate, and monotone or non-monotone. A univariate missing data pattern is one where only one variable has missing values compared to multivariate in which many variables may have missing values. A monotone missing data pattern is one in which the variables can be ordered such that if the value for one variable is missing for an individual, all 'subsequent' variables (according to the order) are also missing for that individual. This may occur when, for example, participants drop out of a study and never return or stop completing a survey. Non-monotone missing data patterns are all those that cannot be ordered for all participants in this monotone fashion, and thus missing values occur haphazardly throughout the dataset. This is what occurs in the ALSPAC dataset.

2.5.3 Approaches to deal with missing data challenges

There are a number of different methods that may be adopted to deal with missing data in clinical epidemiological research. The most appropriate method depends not only on whether the missing data is MCAR, MAR or MNAR, but also on the pattern with which the missingness occurs across the variables, and also on the nature of the research question (e.g., causal vs predictive) and the role (e.g., outcome/exposure/covariate) played by the incomplete variables in the analysis. Here is a brief overview of some of the methods that may be used within cohort studies with missing data, such as ALSPAC.

2.5.4 Multiple imputation

Multiple imputation (MI), proposed by Rubin (2004), imputes missing data as a random draw from a conditional distribution estimated from a complete case imputation regression model (the *complete cases* are those with all relevant variables observed), but more than one (typically 5 or 10) random draw is taken for each missing value. The method of MI using *chained equations* (van Buuren, Boshuizen, & Knook, 1999), also termed full conditional specification by some authors, involves specifying a univariate imputation model for each of the incomplete variables. When each incomplete variable takes its turn to be imputed, the method 'pretends' that the other variables are fully-observed by using their currently imputed values where necessary. By taking multiple stochastic draws for each missing value, MI accounts for the additional uncertainty due to the fact that the missing data were imputed in its final estimates of uncertainty, and – when implemented using chained equations – can be implemented even when the pattern of missing data is non-monotone. This method is recommended as the only practical approach for non-monotone missing data, particularly when missingness occurs in the covariates in the analysis, and has a strong evidence base for its effectiveness (Peugh & Enders, 2004).

2.5.5 Inverse Probability Weighting

Weighting refers to a broad class of approaches for dealing with non-representative samples in which a weight is given to each observation in a dataset to increase or decrease its importance in the analysis, so that the resulting analysis is representative of some population. In particular, inverse probability weighting (IPW) is a commonly used method to correct for bias introduced by using complete-case analysis (i.e., the analysis based only on those who have all relevant variables observed) when data are not MCAR. In its simplest form, complete cases are weighted by the inverse of their (estimated) conditional probability of being a complete case, given fully-observed variables. In an extension to longitudinal data with drop-out, participants with data up to and including wave t are given a wave-specific weight for wave t equal to the (estimated) conditional probability that they remained in the study up to and including wave t, given the data available from waves 1 to t-1.

In IPW, rather than specifying an imputation model, as is the case with MI, a missingness model is specified (S. R. Seaman & White, 2013). This model should include variables thought to be associated with both the probability that an observation will be missing and the value of the incomplete variable itself; in other words, variables on which we should condition in order for the MAR assumption to be satisfied.

2.5.6 Missing data in longitudinal studies: combining MI and IPW

Longitudinal cohort studies such as ALSPAC contain multiple waves of data and even if we disregard individual item missingness within waves, the missing data pattern is not necessarily monotone; some individuals who miss one or more waves return to the study in later waves. In addition, non-monotone missingness arises from individual item missingness within waves. As noted above, IPW cannot be used to deal with such non-monotone missingness, but MI (with chained equations) can. For this reason, it can be beneficial to combine IPW and MI when datasets have entire waves missing for some individuals as well as individual items within those waves missing in a non-monotone pattern (Shaun R. Seaman & Vansteelandt, 2018). The combination uses IPW to avoid over-reliance on the distributional assumptions of the imputation model, and MI to deal with the non-monotone missingness within waves, thus utilising the strengths of both methods and overcoming their weaknesses. Seaman et al. (2012) described how the two methods could be combined (MI/IPW) such that only isolated missing values are imputed using MI i.e. missing survey items, and weights are used to account for remaining larger blocks of missing data i.e. whole surveys missing. These authors also showed that the variance estimator given by Rubin's rules is approximately unbiased for MI/IPW, both for linear regression with an imputed outcome and in more general settings (Shaun R Seaman et al., 2012). Therefore MI/IPW may be considered a superior method compared to either MI and IPW alone, within cohort studies with large amounts of data missing.

2.5.7 Data management

ALSPAC is well known to suffer from high attrition levels, and contains large amounts of missing data; for this reason is often cited in missing data methods papers (e.g. Hughes, Heron, Sterne, & Tilling, 2019; Madley-Dowd, Hughes, Tilling, & Heron, 2019; Spratt et al., 2010). Family attrition rates were highest during infancy and by adolescence, the attrition rate was almost 50 percent. Response rates suggest selective participation, as not all surveys were completed by participants 'active' within the study. However, over 3,000 participants had responded to all surveys up to late adolescence (Boyd et al., 2013). Psychosocial factors such as education levels, household numbers, and health are likely to influence the probability that people respond to the numerous questionnaires administered by the Bristol University team. Child participants lost to follow-up have been shown more likely to be male and eligible for free school meals (Boyd et al., 2013), and mothers more likely to be of lower SES (measured by maternal education; Howe, Tilling, Galobardes, & Lawlor, 2013), leading to an over-representation of higher SES families in later waves. These findings indicate that,

as would reasonably be expected, the data are not MCAR. Analyses that ignore this issue are thus likely to be introducing bias. Thus, to attempt to minimise the bias in my results and obtain valid inferences, actions must be taken to account for the missingness in a principled manner. I will now describe how I conducted the MI and IPW to overcome the challenges of missing data within the ALSPAC dataset.

Within the ALSPAC dataset, multiple questionnaires are sent within each of the waves, to be completed by the mother, father/partner, and the child in later waves. In total, there are five groups of data: the mother- father- and -child-completed questionnaires, child-based questionnaires (generally completed by the mother), and clinic visits. This complicates the situation, particularly for my proposal to combine MI and IPW, since it isn't clear if the entire wave (typically comprising several of the five groups) should be considered the level at which IPW is done, with missing questionnaires/clinic visits within waves imputed using MI as well as the missing items within those questionnaires/visits; or if the IPW should be done at the questionnaire/visit, as well as the wave level, with MI reserved only for missing items. Given that the pattern of missing questionnaires and visits within waves is itself non-monotone, it was deemed more practical to do the former. Therefore, the first step in the data management process was to create a missingness indicator for each wave for each family. Families were considered to be present in that wave if any one of the questionnaires within that time-point was (at least partially) completed. This was completed for the 11 waves from pregnancy to when the child was 13 years old. Details of the questionnaires within each wave can be found in Appendix C.

I then removed any variables that were not going to be used within the planned analyses such as the administrative variables and those that were requested as they had been considered potentially important before the research questions were refined. Individual questionnaire items were also summed to total scores for the factors such as sibling relationships or activities with friends, and then the individual questionnaire items were deleted. For a list of excluded variables see Appendix D. The DV exposure variable was not included in the final imputation model as although the imputation reduced the proportion of the population that had been exposed to DV from 54% of the non-missing cases to roughly 38%, this is still much higher than other population estimates (Radford, 2011). Therefore, as previously described, I decided that a better option would be to change the missing values to "No", so that the variable was complete prior to the imputation, and the proportion of children exposed to DV was 22%. This is because it appeared that participants did not complete any of the

stressful events questionnaire items if they considered them not applicable, rather than answer "No" to them all.

As I was concerned about imputing too much data, particularly if families had dropped out of the study, I created a rule that if more than two consecutive waves of data collection were missed, then the family were treated as if they had dropped out of the study, and they contributed to the subsequent waves only via the estimation of the drop-out weights. When a single wave was missed in a non-monotone pattern, all the data for this wave were multiply imputed so that observed data from later waves could be used in full. Thus, the binary "in-wave" variables, reporting whether children were present in each wave, were recoded to "no" if the previous two waves were missing and "yes" if they were present in one of the two previous waves. Following that, logistic regression models were conducted, including variables that may have influenced the likelihood of families completing surveys, to determine the probability of a child being present within each wave. The probability for a given wave was multiplied by the previous wave's probability, and they were inverted to give the inverse probability weights.

In the MI procedure, I imputed 10 datasets using 100 burn-in iterations and the results (after using IPW in each imputed dataset) were combined using Rubin's rules. There were no material differences when the original and imputed datasets were compared univariately, using the available records for each variable in the original data. All analyses were conducted using STATA version 16 and the code for the multiple imputation procedure is presented in Appendix E. It should be highlighted that whenever a variable referred to sibling/partner characteristics and there was no siblings/partner, a not applicable category was created and if values were missing these were not imputed. Mothers were asked to report whether they had a partner, and whether the child had siblings at multiple time points.

2.5.8 Descriptive Statistics

Frequencies and percentages were calculated for the key categorical child participant demographic characteristics such as sex and ethnicity, maternal characteristics including social class and education level, and the outcomes (internalising symptoms at six and thirteen years) among those who had and had not been exposed to DV during early childhood (0-3 years). These were calculated using only complete cases and the imputed dataset for comparison, and to assess the validity of the imputation. The imputed data figures are those from the first of the ten imputed datasets. The outcome variables have also been described graphically in histograms, separately by exposure group.

2.5.9 Statistical modelling

All analyses were conducted using the imputed data given the high levels of missing data within the original dataset.

2.5.9.1 Preliminary hypothesis tests

Initially Wilcoxon-rank sum tests were conducted, chosen because the outcome data were non-normally distributed, to test the hypothesis that the distribution of internalising symptoms at both six and thirteen years is the same across DV exposure groups, namely early exposure versus no early exposure (prior to controlling for confounders).

2.5.9.2 Multivariate regression modelling

Multivariable negative binomial regression models (NBRM) were fitted to each of the imputed datasets separately, with IPWs (and hence sandwich estimators of standard errors), since this distribution provided a good fit to the conditional outcome distribution. Each model, for the internalising symptoms outcomes at six years and 13 years, respectively, included early DV exposure (0-3 years) as a predictor (see Appendix F for STATA code).

A "change in Mean Squared Error (MSE)" approach was used to select which of the identified potential confounders to include in the final models (Greenland, Daniel, & Pearce, 2016). This approach attempts to find a balance between on the one hand reducing unnecessary imprecision in the estimates due to including too many confounders, and on the other reducing bias due to omitting important confounders. Table 7 shows which variables were included in the two analysis groups as a result. The impact of each confounder was explored by adding them to the NBRMs one by one and assessing the difference made to the estimate of the total effect. The code used for the sequential addition of confounders is found in Appendix G.

Table 7. Confounder measurement details and those selected to be included in the negative binomial regression models measuring internalising symptoms at six years and thirteen years.

Confounder	Age of child when measured	Reporter	Variable type	Was the confounder selected for the model estimating the effect of DV on IS at 6 years? (Y/N)	Was the confounder selected for the model estimating the effect of DV on IS at 13 years? (Y/N)

Early childhood direct child abuse	0-3 years	Mother and father/partner	Binary	Y	N
DV in pregnancy	18 weeks gestation and 8 weeks postpartum	Mother and father/partner	Binary	Y	N
Relationship conflict	Pregnancy	Mother	Continuous	N	N
Maternal age	8 weeks	Mother	Ordinal	Y	N
Maternal education	8 weeks	Mother	Categorical	Y	N
Mother's ethnicity	8 weeks	Mother	Categorical	Y	Y
Partner's ethnicity	8 weeks	Mother	Categorical	N	N
Parental alcohol and drug abuse	8 weeks	Mother and father/partner	Binary	N	N
Occupational social class (mother)	8 weeks	Mother	Categorical	Y	Y
Occupational social class (father)	8 weeks	Mother	Categorical	Y	N
Financial difficulties (mother)	8 weeks and 2 years	Mother	Continuous	Y	Y
Serious financial difficulties (partner)	8 months	Father/partner	Binary	Y	Y
Family income (per week)	2 years	Mother	Ordinal	Y	N
Marital status	2, and 3 years	Mother	Categorical	Y	N
Crowding	2 years	Mother	Ordinal	Y	Y
Chronic illness (household)	18 months	Mother	Binary	Y	N
Mother's health	2 years	Mother	Ordinal	Y	Y
Stressful life events	Pregnancy	Mother	Continuous	Y	Y
Paternal history of depression	Pregnancy	Father/partner	Binary	Y	N
Mother's anxiety	3 years	Mother	Binary	Y	Y

Mother's depression	3 years	Mother	Binary	Y	Y
Partner's anxiety	3 years	Mother	Binary	Y	Y
Partner's depression	3 years	Mother	Binary	Y	Y
Mother's neurotic symptoms (CCEI)	8 months	Mother	Continuous	Y	Y
Partner's neurotic symptoms (CCEI)	8 months	Father/partner	Continuous	Y	N
Mother's post-natal depression symptoms (EPDS)	8 months	Mother	Continuous	Y	Y
Father/partner's post-natal depression symptoms (EPDS)	8 months	Father/partner	Continuous	Y	N
Child's conduct problems	3 years	Mother	Continuous	Y	N

2.5.9.3 Post-processing of regression parameters for ease of interpretation

Estimated parameters from the NBRM were used to generate more easily-interpretable effect measures. There are three reasons for this. First, after making the necessary assumptions of 'no unmeasured confounding' etc, the direct causal interpretation afforded by parameters of regression models (in general) is of the form "what would happen if everyone versus no one were exposed". With an exposure such as DV, the hypothetical world in which all children witness DV between the adults in their household is far-fetched and thus makes the resulting estimated effects difficult to interpret. Furthermore, parameters from NBRMs are already difficult to interpret, since they operate on a log scale. The exponent of the estimated coefficient of the exposure in such a model, under the assumption of no unmeasured confounding, is interpreted as the factor by which the mean internalising symptoms score would be inflated if every child were exposed versus no child. Such a multiplicative interpretation is especially unwieldy when effect moderation is considered, as is important in this study. The interpretation for moderation by mother's education, for example, would be "the factor by which the mean internalising symptoms score would be inflated if every child were exposed versus no child, is estimated to be C times higher among children whose

mothers had higher education relative to those whose mothers' had no secondary education", where C is the exponent of the estimated coefficient of the product term (between DV and mother's education) in that NBRM. Finally, when it comes to assessing mediation, an additive decomposition of direct and indirect effects is appealing ("x% of the effect of X on Y is mediated through M"), but this is again not possible (without further manipulation) from the parameters of multiplicative models. Thus, as well as directly interpreting the output of the regression models mentioned above, post-processing steps were performed in order to express the effects of, for example, how much smaller the mean internalising score would have been in this group had none of the children been exposed to DV. Due to its monotone nature, bootstrapping was used to estimate the standard error of such an estimated effect.

3 Results

3.1 Missing Data

Missing data led to substantial attrition in the complete case sample and Appendix G provides a full summary of the proportion of missing data for key variables. The composite "Early DV" variable had 59% missing, the internalising symptoms (SDQ) score at six years was 38% missing and at thirteen it was 51% missing. Of the 10,182 included in this study, 27% were missing the exposure and outcome variables..

I used MI and IPW to account for missing values for exposure, moderator, mediator, confounder and outcome variables, resulting in a restored sample size of 10,182 for all explanatory factor analyses. There were 44 complete cases that did not need imputing and 10,138 incomplete cases with imputed data. Of these 6,065 (59.82%) had at least one entire wave of data missing. The analysis made use of 117 fields of data across 11 waves, and out of the total possible 1,191,294 (10182*117) items of data, 512,978 (43.06%) were missing. A further 15,680 items (on 681 children) were artificially deleted due to being observed after at least two entirely missed waves. Of the total 512,978 missing items, 284,172 (55.40%) were imputed, whereas the remaining 228,806 (44.60%) were left as missing, to be dealt with by IPW.

3.2 Sample Characteristics

It is important to compare the key variables within the complete case data (incomplete dataset) with the MI dataset to check that the MI appears valid. Comparisons of the incomplete dataset and the first iteration of the imputed data are presented in Table 8.

Table 8. Mean and standard deviation of internalising symptoms at six and thirteen years in the incomplete data compared to within the first iteration of the MI (complete) dataset

Outcome Variables		
Variable	Mean (SD) in incomplete data	Mean (SD) in complete (MI) data
Internalising symptoms at 6 years	2.57 (2.56)	2.57 (2.51)
Internalising symptoms at 13 years	2.65 (2.62)	2.68 (2.64)

Table 9 describes the sociodemographic characteristics of participants included in the mediation and moderation analyses, by DV status, and data status (non imputed versus the first imputed dataset). The table suggests that children in non-white families with lower income, higher levels of crowding, lower maternal age at child's birth and of mothers with lower educational status are more likely to be exposed to DV.

Overall, 2,250 (22.1%) children were exposed to early DV reported by either mother or father/partner. Internalising symptoms scores at six and 13 years by DV status are presented in Figures 8a and 8b. They illustrate that the distribution of internalising symptoms was negatively skewed and internalising symptoms tended to be higher for the DV exposed group at both time points.

Table 9: Participant sociodemographic characteristics within the original dataset (m = 0) and the first iteration of the multiply imputed data (m = 1) by DV exposure status.

	Original dataset (non-imputed)		Imputed dataset (m=1)	
Variable	DV exposure at 0-3 years (n (%))	No DV exposure at 0-3 years (n (%))	DV exposure at 0-3 years (n (%))	No DV exposure at 0-3 years (n (%))
N	2,250 (22.10)	7,932 (79.90)	2,250 (22.10)[1]	7,932 (79.90)
Sex Male Female	 4,123 (51.98) 3,809 (48.02)	 1,157 (51.42) 1,093 (48.58)	 4,123 (51.98) 3,809 (48.02)	 1,157 (51.42) 1,093 (48.58)
Maternal age at birth <20 20–24 25–29	 102 (4.53) 422 (18.76) 845 (37.56)	 360 (4.54) 1,489 (18.77) 3,090 (38.96)	 102 (4.53) 422 (18.76) 845 (37.56)	 360 (4.54) 1,489 (18.77) 3,090 (38.96)

30–34	624 (27.73)	2,217 (27.95)	624 (27.73)	2,217 (27.95)
>=35	257 (11.42)	776 (9.78)	257 (11.42)	776 (9.78)
Missing	0	0	0	0
Marital status				
Not married	280 (14.89)	532 (10.17)	356 (16.01)	792 (11.31)
Separated/divorced/widowed	227 (12.07)	294 (5.62)	279 (12.55)	460 (6.57)
Married	1,373 (73.03)	4,404 (84.21)	1,588 (71.43)	5,752 (82.12)
Missing	370	2,702	27	928
Family income per week (mother report)				
<£100	229 (12.91)	350 (7.29)	305 (13.72)	578 (8.25)
£100-£199	343 (19.33)	813 (16.92)	451 (20.29)	1,255 (17.92)
£200-£299	481 (27.11)	1,400 (29.14)	593 (26.68)	2,074 (29.61)
£300-£399	331 (18.66)	1,059 (22.04)	400 (17.99)	1,497 (21.37)
>=£400	390 (21.98)	1,182 (24.60)	474 (21.32)	1,600 (22.84)
Missing	491	3,175	27[2]	928
Maternal social class				
I & II	686 (38.87)	2,123 (37.07)	816 (36.27)	2,778 (35.02)
III	880 (49.86)	2,894 (50.53)	1,145 (50.89)	4,069 (51.30)
IV & V	199 (11.27)	710 (12.40)	289 (12.84)	1,085 (13.68)
Missing	485	2,205	0	0
Child ethnicity				
White	1,988 (88.36)	6,550 (82.58)	1,988 (88.36)	6,550 (82.58)
Non-white	128 (5.69)	315 (3.97)	128 (5.69)	315 (3.97)
Unknown	134 (5.96)	1,067 (13.45)	134 (5.96)	1,067 (13.45)
Maternal highest qualification				
Low	1,336 (62.17)	4,624 (65.32)	1,415 (62.89)	5,166 (65.13)
Medium	533 (24.80)	1,534 (21.67)	549 (24.40)	1,741 (21.95)
High	280 (13.03)	921 (13.01)	286 (12.71)	1,025 (12.92)
Missing	101	853	0	0
Household crowding index at 2 years				
<= 0.5	350 (18.81)	940 (18.34)	405 (18.22)	1,296 (18.50)
>0.5 - 0.75	670 (36.00)	1,962 (38.28)	794 (35.72)	2,576 (36.78)
>0.75 - 1	679 (36.49)	1,834 (35.78)	815 (36.66)	2,569 (36.68)
> 1	162 (8.70)	390 (7.61)	209 (9.40)	563 (8.04)
Missing	389	2806	27	928
Variable	**DV exposure at 0-3 years (Median (IQR))**	**No DV exposure at 0-3 years (Median (IQR))**	**DV exposure at 0-3 years (Median (IQR))**	**No DV exposure at 0-3 years (Median (IQR))**
SDQ Internalising symptoms at 6 years	2 (1-4)	2 (1-3)	2 (1-5)	2 (0-4)
Missing	661 (29.38)	3,453 (43.53)	189 (8.40)	2,123 (26.77)
SDQ Internalising symptoms at 13 years	2 (1-5)	2 (0-3)	2 (1-5)	2 (0-4)
Missing	1,080 (48.00)	4,499 (56.72)	719 (31.96)	3,503 (44.16)

[1]No missing data in the DV variable as missing values were considered to be "No"

[2]Missing data remains in the imputed dataset due to the rule applied whereby families who missed two consecutive waves of data were thereafter considered to have "dropped out" of the study to avoid imputing whole waves of data
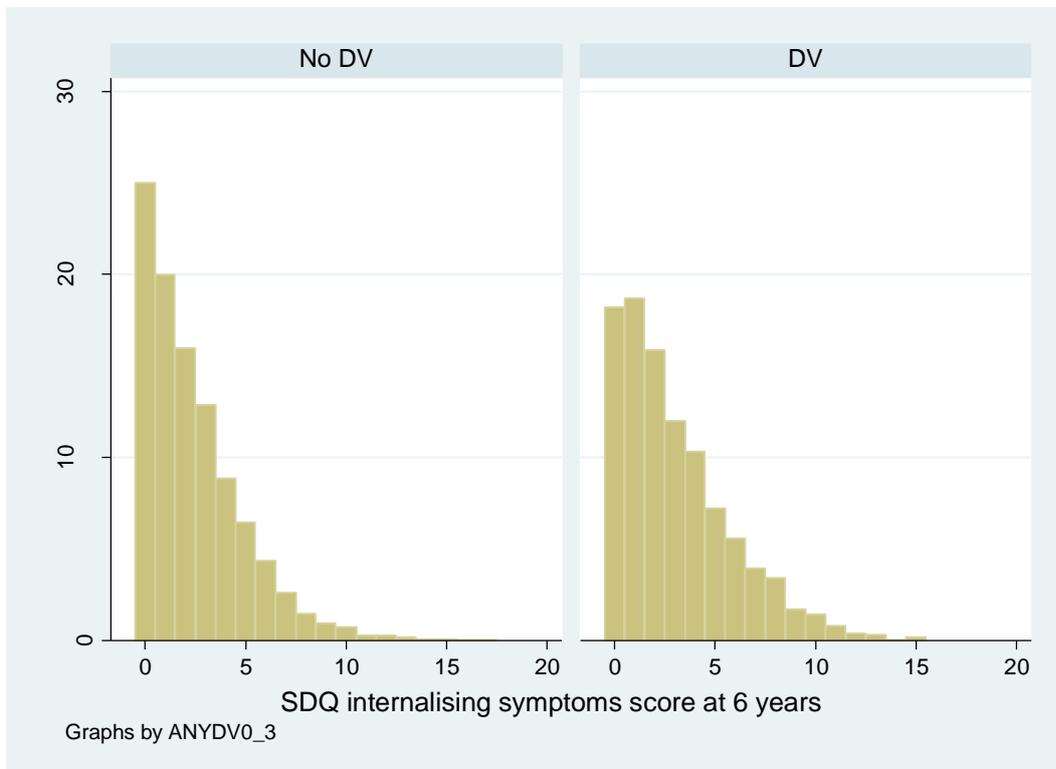
Figure 8a. Histogram illustrating the percentage of six-year-olds within the cohort with each internalising symptom score by DV exposure status using the first imputation of data
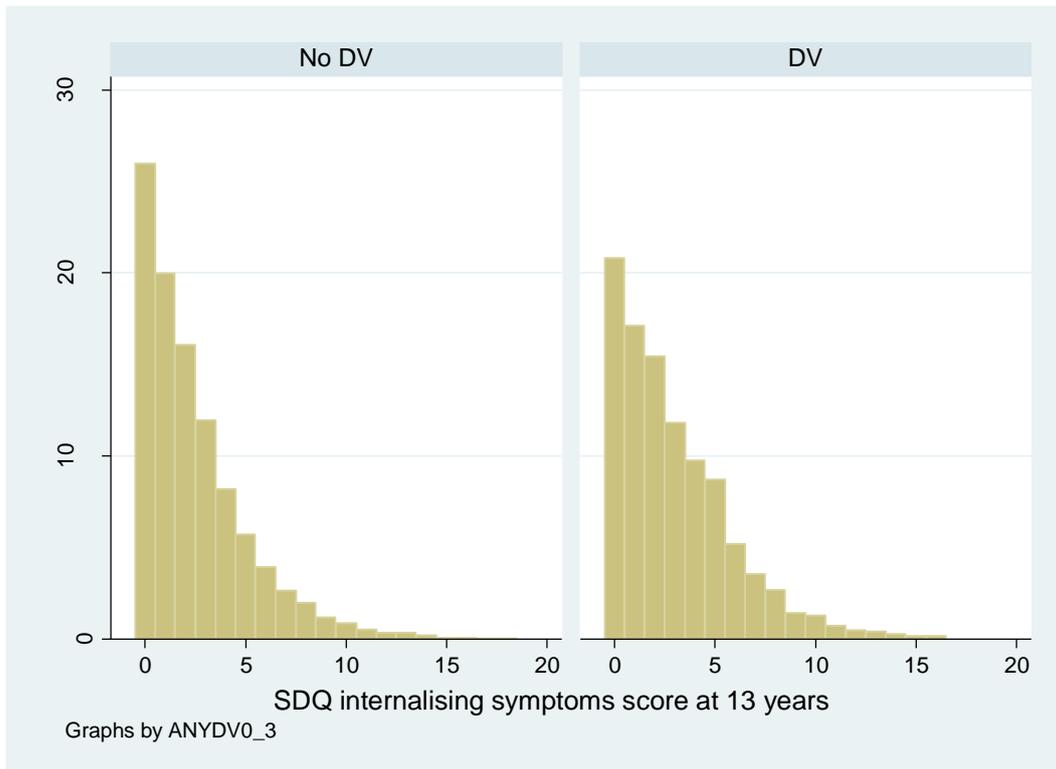
Figure 8b. *Histogram illustrating the percentage of six-year-olds within the cohort with each internalising symptom score by DV exposure status using the first imputation of data*

3.3 Preliminary Analyses

3.3.1 Wilcoxon-Rank Sum Tests

The results of Wilcoxon-rank sum tests are presented in Table 10. In summary, internalising symptom scores at both six and thirteen years for those exposed to DV in early childhood, were significantly greater than those who were not exposed to DV within the same exposure group.

Table 10. Wilcoxon-rank sum test results measuring the association between exposure to DV at 0-3 years and internalising symptoms at six and 13 years

Exposure group	SDQ age measured	Median internalising symptom score	z-score	P-value
0-3 years Yes No	6 years	 2 2	-8.801	< .001
0-3 years Yes No	13 years	 2 2	-6.571	<.001

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3.3.2 Crude negative binomial regression models (NBRM) without all necessary confounders
The NBRM with no confounders showed strong evidence for a difference in mean internalising symptoms score between the two DV groups. The logarithm of the mean of the internalising symptoms scores at six years was estimated to be 0.23 (SE = 0.02, p<0.001) higher than for those who were not exposed to DV. When the outcome was changed to internalising symptoms in early adolescence (13 years), the crude NBRM indicated that early DV also has a significant effect on internalising symptoms at 13 years. The logarithm of the mean internalising symptoms at thirteen years was estimated to be 0.17 (SE=0.03, p<0.001) higher than among those who were not exposed to DV.

As described in Section 2.5.9.3 above, the results in the previous paragraph were converted to a more easily-interpretable scale, and the results are presented in the top half of Table 11.

Table 11. Top half of the table displays the mean internalising symptoms score and standard errors (without any confounders) for those exposed to DV compared to the same group of children in an alternative scenario that assumes their exposure to DV had been avoided, along with the difference between those means and significance value from z-test. Bottom half of the table displays the same results with the confounders included that were selected by the change-in-estimated-MSE method.

No confounders				
DV exposure (physical and/or emotional)	**SDQ AGE measured**	**Mean internalising symptoms score (standard error)**	**Mean predicted internalising symptom score after setting DV (0-3 years) to absent for all**	**Difference (significance value from z-test)**
0-3	6	2.72 (0.04)	2.50 (0.05)	0.21 (0.04) (p<.001)
0-3	13	2.91 (0.06)	2.74 (0.07)	0.17 (0.05) (p<.001)
Including selected confounders				
DV exposure (physical and/or emotional)	**SDQ AGE measured**	**Mean internalising symptoms score (standard error)**	**Mean predicted internalising symptom score after setting DV (0-3 years) to absent for all mean internalising symptoms score**	**Difference (significance value from z-test)**
0-3	6	2.69 (0.04)	2.68 (0.05)	-0.03 (0.04) (p=0.42)
0-3	13	2.86 (0.05)	2.90 (0.06)	-0.03 (0.03) (p=0.33)

3.4 Total effect of DV on internalising symptoms at six and 13 years

3.4.1 Internalising symptoms at age six years

The NBRM including all selected confounders (as determined by the change-in-estimated MSE approach), did not provide evidence for a conditional association between exposure to DV from 0-3 years and internalising symptoms at age six, when conditioning on the confounders included in the model (estimated log mean difference = -0.03, SE = 0.04, p = 0.42). When confounders were added to the NBRM sequentially, it was evident that the inclusion of DCA reduced the estimated effect of DV exposure on internalising symptoms the most, followed by mother's neurotic symptoms (Figure 9).
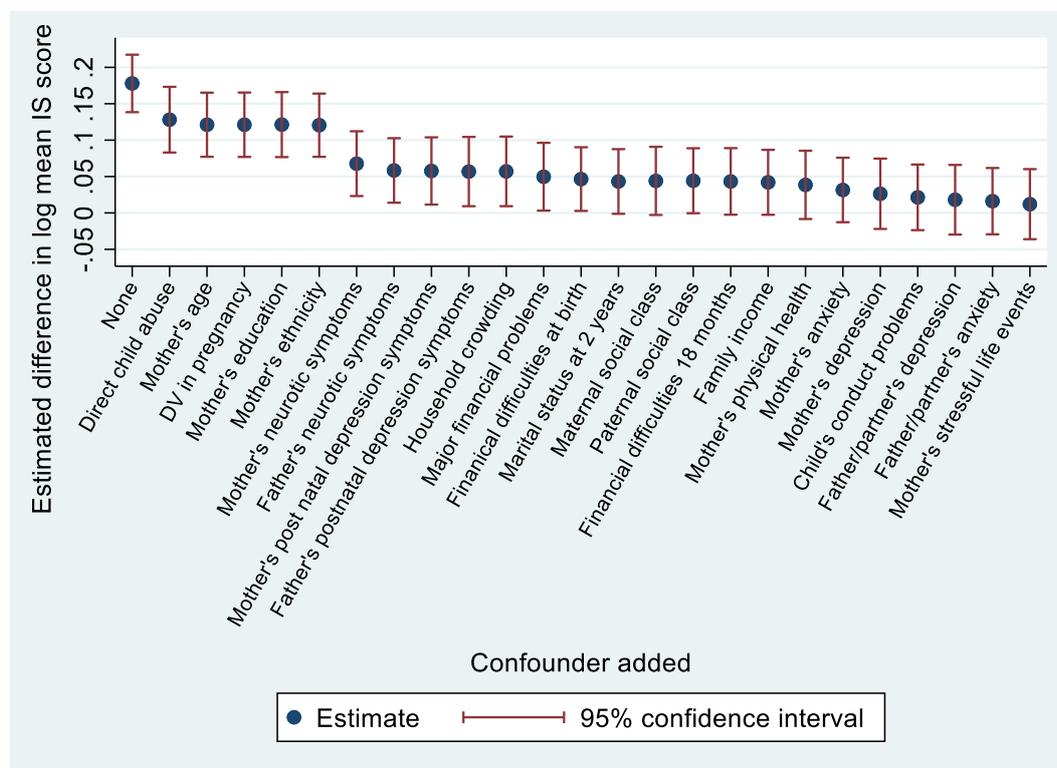
Figure 9. Estimates of the log mean difference in internalising symptoms scores at six years by DV exposure status with confounders added sequentially, and 95% confidence intervals.

Converted to an additive mean scale (see Table 12), the estimated difference between the internalising symptoms in the real compared to the hypothetical world without DV (at age 0-3) was 0.05 (SE = 0.04, p = 0.56).

3.4.2 Internalising symptoms at 13 years

The NBRM including all necessary confounders, after determining the confounder set with the lowest MSE, did not provide evidence for a conditional association between exposure to DV from 0-3 years and internalising symptoms at age 13, (estimated mean log difference = -

0.03 (SE = 0.03, p = 0.33). When confounders were added to the NBRM sequentially, it was evident that the mother's neurotic symptoms and mother's depression reduced the estimated effect of DV exposure on internalising symptoms the most (Figure 10).
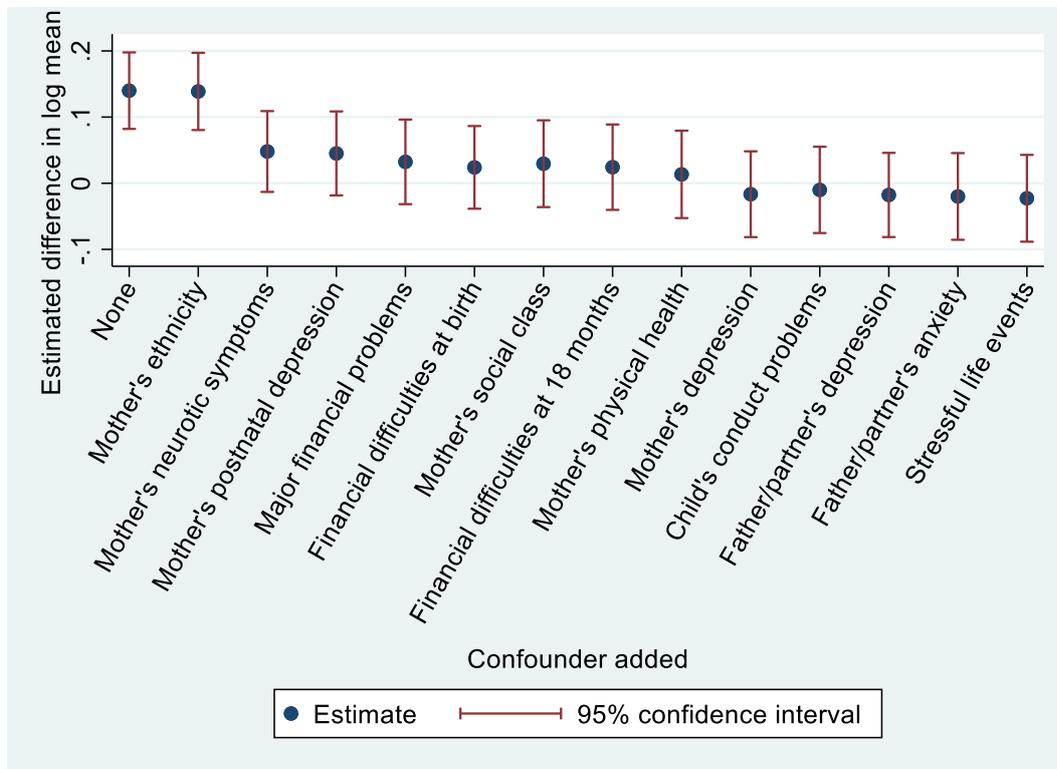
Figure 10. Estimates of the log mean difference in internalising symptoms scores at thirteen years by DV exposure status with confounders added sequentially, and 95% confidence intervals.

Results following post-processing are presented in Table 11. The estimated difference between the internalising symptoms as inferred in the ALSPAC cohort compared to the ALSPAC cohort had DV been removed (at age 0-3) was -0.04 (SE = 0.05, p = 0.28).

3.6 DV exposure during middle childhood as a mediator or moderator of early DV exposure on internalising symptoms at six and thirteen years

I ran NBRMs with the simulation scheme to determine whether DV exposure at 6-9 years mediates or moderates the effect of early exposure to DV on internalising symptoms at thirteen years and found no evidence of an effect. The estimate of the indirect effect was 0.01 (SE=0.02) and the estimate for effect moderation was -0.01 (SE = 0.05)

3.7. Correlation between DV and DCA

I cross-tabulated CEDV with DCA to observe how frequently they co-occur. Of those who had been exposed to DV within the first imputation (n=2,250), 1,345 (60%) also experienced

direct child abuse. Within the original dataset, 62% (658/1,062) of children who were exposed to DV were also victims of DCA.

4 Discussion

This study has found that 22% (2,250) of the ALSPAC sample of children had been exposed to DV during their first three years of life. This is approximately in line with other studies suggesting that, in the UK, approximately one in five, or 20% of children witness DV during childhood (Radford,2011).

The NBRM analyses provided insufficient evidence that CEDV at 0-3 years has an effect on children's internalising symptoms as measured by the mother-reported SDQ at six years or 13 years, with estimated effects close to zero. These findings are in contrast to a wealth of previous research, using standardised instruments of known reliability and validity such as the SDQ (Goodman, 1999), that has reported the negative impact of DV on children and adolescents' internalising symptoms (Evans et al., 2008). The majority of studies included in Evans and colleagues' (2008) meta-analysis used the Child Behaviour Checklist (CBCL), however the SDQ and the CBCL have been shown to be highly correlated, and equally able to detect psychiatric cases (Goodman & Scott, 1999). A further key difference between this study and other literature is that many previous studies have not controlled for important confounders such as DCA, parental mental health and socio-economic status (Carter et al., 2020). In this study, when these are taken into account the estimated effect of DV exposure on internalising symptoms is reduced. This is not to say that DV does not have a damaging effect on children's mental health, but that other factors which are highly correlated with exposure to DV, such as DCA and maternal mental health, have a greater impact on internalising behaviours. Nonetheless, given the limitations described below on the recall and response bias, in addition to the challenge posed by missing data, these results should be interpreted with some caution.

Limitations

Ascertaining exposure to DV in childhood is challenging. Firstly, there is no common, world-wide definition of DV. Different studies explore combinations of physical, emotional, sexual, and/or financial abuse, and coercive control making prevalence estimates difficult to compare. In this study I included only physical and emotional abuse, and therefore may have underestimated the true levels of DV exposure. A further challenge is that whilst there are validated tools to capture self-reported DV, it is subject to recall and response bias given the

sensitivity of the subject. In this study the completion of postal questionnaires may have reduced response bias. Nevertheless, the measure of DV used in ALSPAC is very broad and somewhat subjective and has not been validated against other measures of DV. It only asks parents two questions, specifically whether their partner has been physically or emotionally cruel to them since the previous wave of data collection. These questions are open to interpretation of what the word "cruel" means and people may have differing views on this. It is quite possible that a genuinely cruel person might not consider their behaviour to have been cruel, while those answering 'yes' might be reflecting on something they once said about which they feel guilty. Alternatively, measures such as the CTS (Straus, 1979) ask about specific actions and behaviours that are less open to interpretation such as threatening to hit and kicking.

Furthermore, the DV items in ALSPAC do not actually measure exposure to DV. I may have under estimated exposure in childhood as I have relied on the reports of parental DV experience and assumed this is evidence of DV in the childhood environment. Whilst this is not a direct capture of the child's exposure, research has shown that even if children do not technically "witness" the abuse, or are too young to remember it, they are still likely to experience deleterious effects on their health and development (Devaney, 2015; Överlien & Hydén, 2009; Rosser-Limiñana, Suriá-Martínez, & Mateo Pérez, 2020).

A further challenge was the high proportion of missingness in the exposure, requiring attempts to be made to mitigate response bias in the questionnaire design and delivery. Replacing all missing values with "No", essentially assuming that all those who did not complete the questionnaires had not experienced DV, is almost surely not entirely correct. It may well have led to an underestimation of DV, and potentially an underestimate of the difference in internalising symptoms between the exposed and unexposed groups. However, given the questionnaire included many different levels of "Yes" options, with different levels of severity, it may be argued that it is at least plausible that someone who suffered no DV felt that this question was not relevant to them, and omitted ticking "No". Furthermore, when I allowed the MI to impute the missing exposure levels under a missing at random assumption (results not shown) the imputed prevalence of DV was unrealistically high, and thus it was deemed that the missing equals no strategy, although not exactly correct, was closer to reality than the alternative.

It is important to note that the measure of early CEDV in this study differs from that used in other studies using ALSPAC data (Bowen, 2015) as we have included DV exposure at 47 months given the data was available providing another opportunity to identify the exposure and because CEDV at 3 years is likely to have a similar impact on internalising symptoms as CEDV at two years. However, this led to my estimated prevalence being 7.5% higher.

The outcome measure in this study utilised parent-reported internalising symptoms, however internalising symptoms reported by parents have been shown to vary greatly from children's self-reports (Van der Meer, Dixon, & Rose, 2008). It may well be that when internalising symptoms are assessed using child self-report measures, the results are markedly different; unfortunately, child reports of internalising symptoms were not included in ALSPAC.

It should be noted that the approach taken to overcome the challenge of dealing with a large amount of missing data is based on the important untestable assumption that the missing data are MAR, as well as requiring that the chosen imputation and weights models be correctly specified. The inevitable departures from these assumptions would lead to some degree of bias in all reported associations. Alternative approaches would typically be expected to be even more biased, however, as they would rely on the even stronger assumption of MCAR. Even from a pragmatic viewpoint, some strategy for dealing with missing data was rendered necessary given that only 44 children had complete data for all the variables involved in the analysis.

Another important limitation for the interpretation of the analyses is the assumption of no unmeasured confounding needed to attribute differences between DV groups to a causal effect of DV. Although ALSPAC provides rich confounder data on SES etc, there is always an important possibility that these only partially adjust for the confounding. There could plausibly be other aspects of the child's environment, not captured in the ALSPAC data, or this dataset in particular, that make DV more or less likely, as well as impacting on the internalising symptoms of the child.

Participants recruited into the ALSPAC cohort were of higher SES and less likely to be of non-White ethnicity compared to both the general population of mothers with children under the age of one in Britain at the time of recruitment, and those in the Avon area who were not enrolled in the ALSPAC study (Boyd et al., 2013; Fraser et al., 2013). These differences present important limitations when attempting to generalise prevalence estimates from the ALSPAC cohort to the general population of the UK but representativeness is somewhat less

of a concern for the validity of protective factor regression analyses. As Rothman notes, careful consideration of causal mechanisms and effective control of confounding is arguably more important than the representativeness of the sample when making inferences about cause-effect relationships (Rothman, Gallacher, & Hatch, 2013).

Further research would benefit from conducting a longitudinal study over a shorter time span, and measuring internalising symptoms roughly one year after CEDV. This method would likely reduce attrition and help overcome the challenges faced by missing data. Additionally, this study could be improved by using a validated measure of CEDV and children's reports of internalising symptoms given previous findings that child and parental reports of internalising symptoms often differ (Thomas M. Achenbach, McConaughy, & Howell, 1987; Becker, Hagenberg, Roessner, Woerner, & Rothenberger, 2004; Sourander, Helstelä, & Helenius, 1999). Furthermore, I recommend that future research checks the validity of the measures used in ALSPAC against social care records to quantify measurement error.

In conclusion, this study has found that, using a large population-based cohort study in England and adjusting for a large number of potential confounding factors, there is little evidence to suggest an effect of CEDV on child and adolescent internalising symptoms. However, this finding should be interpreted with caution and more research is needed to confirm or disprove the results. If these findings are confirmed in other studies, it will advance our understanding of how to support children exposed to DV.

5 Contribution of this chapter to the thesis

This chapter has highlighted that when important confounders are included in multivariate modelling, there was no evidence that CEDV significantly increases internalising symptoms in children and adolescents. Given that no total effect of CEDV on internalising symptoms was found, and therefore the investigation of mediation and effect moderation by protective factors would be inappropriate, the following chapter will explore the causal pathways between DCA and internalising symptoms instead.

Chapter 6. Mediators and moderators of the effect of direct child abuse on child and adolescent internalising symptoms: A cohort study

1 Overview

In this chapter, I explored the pathways between DCA and internalising symptoms, and in doing so I investigated which factors are protective, and whether they act as mediators and/or moderators, using data from a population-based birth cohort in England (ALSPAC). Previous research has been predominantly conducted in the USA and studies have rarely considered the complex web of factors that are likely to contribute to the association between DCA and child and adolescent internalising symptoms. This study aimed to address such gaps in the literature by exploring factors with a potential mediating or moderating role on the effect of DCA on internalising symptoms across childhood (at six and 13 years) using a UK based cohort study, and considering the complex nature of the pathway between DCA and internalising symptoms by including a large number of confounders.

Of course, the ultimate aim is to stop DCA yet unfortunately eradication of DCA is unlikely. The factors identified within this study as mediators and/or moderators are modifiable and should be targeted within interventions for children who have experienced DCA to protect them from developing internalising symptoms.

2.1 Background

2.2 Aims and objectives

2.2.1 Aim

To quantify the total effect of DCA on children's internalising symptoms at six and thirteen years and identify potentially modifiable protective factors for internalising symptoms among children and adolescents in the ALSPAC cohort who have been exposed to DCA.

2.2.2 Objectives

1. To estimate the total effect of DCA at two time points on internalising symptoms at early and middle childhood (six years and 13 years respectively) controlling for covariates identified within the literature

2. To examine the extent to which potentially protective factors (from the DAG developed in Chapter Four) mediate or moderate the effect of DCA during childhood on internalising symptoms

2.2 Literature Review

Child abuse has been shown to have significant effects on child and adolescent internalising symptoms among other negative outcomes (Norman et al., 2012; Pears, Kim, & Fisher, 2008; Toth, Manly, & Cicchetti, 1992; D. A. Wolfe, Scott, Wekerle, & Pittman, 2001). As was the case with DV, researchers have investigated factors that increase the risk or protect against internalising symptoms among those who have experienced child abuse (Bolger & Patterson, 2001; Kim & Cicchetti, 2010) yet few of these have taken a formal approach to quantifying direct and indirect effects, as well as the extent of effect moderation, in this context. Understanding the way in which child abuse leads to internalising symptoms and other negative outcomes is complicated by evidence that, like DV, child abuse does not consistently have a negative effect on child development and some children do not develop maladaptively (David M Fergusson & Horwood, 2003; Werner & Smith, 1992). In order to determine individual differences in negative outcomes among children who have been abused, it is important to explore the causal pathways to identify risk and protective factors. As described in Chapter 2, risk factors increase the probability of negative outcomes while protective factors buffer against adverse outcomes and "modify the effects of risks in a positive direction" (Suniya S Luthar, Cicchetti, & Becker, 2000b). In the broader resilience and ACEs literature, as described in Chapter 3, protective factors at the individual, family and community levels include self-esteem, family support, and community connectedness (Benard, 2004; Suniya S. Luthar, 1991; Ann S Masten, Best, & Garmezy, 1990; Werner & Smith, 1992). Identification of such factors within the context of DCA, defined here as physical and emotional abuse, could help clinicians develop treatment plans for patients and preventative interventions for children who have been abused. However, the majority of studies exploring protective factors have tended to include all forms of child maltreatment without considering that outcomes and protective factors may vary depending on the form of maltreatment.

A review of protective factors following any type of child maltreatment (Tracie O Afifi & MacMillan, 2011) has reported that collectively studies have shown that children and adolescents who develop in a caring family environment and feel supported are more likely to be resilient. Furthermore, personality traits were found to protect against internalising symptoms at an individual level. However, studies were heterogeneous limiting comparison across studies and studies were across all forms of maltreatment.

A recent meta-analysis of protective factors among children exposed to violence across multiple contexts (Yule et al., 2019) included a number of longitudinal studies investigating protective factors in the context of child maltreatment and internalising symptoms. These studies provided evidence for factors at an individual, family, and community level, namely self-perceptions, coping skills, family support, peer support, and community cohesion buffer maltreated children and adolescents against internalising symptoms. This implies that interventions that seek to bolster supportive relationships may be effective across social contexts including families, schools, and communities and schools are well-placed to deliver coping and self-regulation interventions. Additionally, Yule et al (2019) reported that the protective factors were consistent across type of violence exposed to (maltreatment. IPV or community violence). Therefore, this meta-analysis suggests that preventative interventions for children and young people may be effective following exposure to multiple forms of violence. However, this meta-analysis suffered from similar limitations to my systematic review, with limited evidence for each protective factor and very few studies controlled for relevant covariates within the analyses, limiting the interpretation of the results. Despite the lack of covariates within studies included in their meta-analysis, Yule et al (2019) examined whether adjusting for covariates was likely to reduce effect sizes within protective factors studies and found insufficient evidence to suggest that covariate adjustment would have changed the estimated effect sizes for protective factors. Regardless, evidence is limited and more studies are needed to confirm whether the protective factors identified within the meta-analysis are consistently found to be significant moderators, particularly within UK settings, as opposed to the US where the majority of research has been conducted. Thus, the analyses within this chapter aim to identify protective factors and explore the causal pathway between direct child abuse and child and adolescent internalising symptoms. This study aimed to address said gaps by exploring factors with a potential mediating or moderating role on the effect of DCA on internalising symptoms across childhood (at six and 13 years) using a UK

based cohort study, and considering the complex nature of the pathway between DCA and internalising symptoms by including a large number of confounders.

2 Method

All the methods for this analysis chapter were the same as those within Chapter 5 except of course, the exposure was changed from DV to DCA. DV was included within these analyses as a potential confounder, mediator and moderator as its relationship with DCA is unclear. It may be that that DV is an antecedent of both DCA and internalising symptoms, in which case it should be considered as a potential confounder. However, when DV is measured after DCA, it may be that DV lies on the pathway between DCA and internalising symptoms or it may interact with DCA to increase the severity of internalising symptoms compared to those who experienced DCA without DV. The DCA variables are described below.

2.1 Variables

All the variables included in these analyses, the age of the child when they were measured, and the person who reported them are presented in Table 13. Further descriptions of the variables and the tools used to measure them can be found in Chapter 5.

Table 13. Variables included in the analyses, who reported on them and the age of the child when they were reported. Variables considered potential mediators and/or moderators are in bold.

Age of child (years)	Variables measured	Responder
Pregnancy	Domestic violence	Mother and partner
	Parental aggression	Mother
	Maternal education	Mother
	Ethnicity	Mother
	Alcohol and drug abuse	Mother and partner
	Occupational social class	Mother
	Financial difficulties	Mother and partner
	Parental mental health problems	Mother and partner
	Stressful life events	Mother
0-3 years	Direct child abuse	Mother and father/partner
	Domestic violence	
1	Mother's age at birth	Mother
3	**Child's temperament – emotionality, activity, sociability**	Parent/caregiver
	Maternal parenting	Mother
	Paternal parenting	Mother

	Parental interaction	Mother
	Outside activity	Mother
	Discipline variation	Mother
4	Crowding	Mother
5	**Maternal anxiety**	Mother
	Maternal depression	Mother
6	Internalizing symptoms (SDQ[1])	Mother
	Sibling interaction	Mother
7	Parent-child relationships	Child
8	**Attitude towards school**	Child
	Locus of control	Child (clinic)
	Self-esteem	Child (clinic)
	Emotional awareness	Child (clinic)
	Friendships	Child (clinic)
	Bullying	Child (clinic)
	Intelligence	Child (clinic)
6-9	**DCA**	Mother and
	DV	father/partner
9	**School Problem**	Parent/caregiver
	Maternal social support	Mother
10	**Maternal anxiety**	Mother
	Maternal depression	Mother
11	**Physical activity**	Child (clinic)
	Activities with friends	Child (clinic)
12	**Sibling bullying**	Child
13	Internalizing symptoms (SDQ)	Mother

2.1.1 Exposure

DCA was measured using two questions to both mothers and partners asking whether they or their partner had been physically or emotionally cruel to their child(ren) since the birth of the child or the previous wave of questionnaires. Both partners were asked about DCA and were asked when the child was eight, 21, 33, 47 months and at nine years of age. A child was considered to have been directly abused at each time point if either partner responded positively ("Yes") to either physical or emotional cruel towards their child(ren) (Flach et al., 2011). In this analysis, the measures at eight, 21, 33, and 47 months were combined to give a binary variable identifying any DCA at 0-3 years (described as early exposure), and the questions at nine years, asking about abuse since the previous questionnaire three years ago, were combined to give a more recent DCA exposure at 6-9 years (described as middle childhood). Thus, these two binary exposure variables cannot be directly compared as they are created using different variables. The binary variables were coded as "Yes" if mother or

partner responded "Yes" to any of the questions and "No" if neither mother nor partner responded "Yes" to any of the DCA items or the responses were missing.

2.2 Change in MSE to determine confounders

As described in the previous chapter, a "change in Mean Squared Error (MSE)" approach to confounder selection (Greenland, Daniel, & Pearce, 2016) was utilised to identify the 'optimal' set of confounders in terms of the trade-ff between bias and imprecision. The 'optimal' set of confounders was identified for DCA at 0-3 years and DCA at 6-9 years. The variables within the 'optimum' set for each group were then included across the analyses with the addition of variables measured after six years being included in the analyses using middle childhood DCA as the exposure. Table 14 shows which variables were considered as confounders and which were included in the two analysis groups. DV was not included in the analyses as a confounder, given this method, however it was explored as a mediator and a moderator of the effect of DCA on internalising symptoms.

Table 14. Variables included in the analyses investigating the effects of early and middle childhood DCA on internalising symptoms, determined by the confounder set with the least amounts of bias and imprecision possible.

Confounder	Was the variable included in the modelling for DCA at 0-3 years?	Was the variable included in the modelling for DCA at 6-9 years?
Child sex		
Domestic violence in early childhood		
Domestic violence[1] in middle childhood		
Domestic violence in pregnancy		
Gestation at birth	✓	✓
Parental aggression		
Maternal age	✓	✓
Maternal education		
Mother's ethnicity	✓	✓
Partner's ethnicity	✓	✓
Child ethnicity	✓	✓
Parental alcohol and drug abuse		✓
Occupational social class (mother)		
Occupational social class (mother)		

Financial difficulties (mother)	✓	✓
Financial difficulties (partner)	✓	✓
Family income (per week)	✓	✓
Marital status		
Crowding		
Chronic illness (household)		`
Mother's health	✓	✓
Mother's stressful life events	✓	✓
Child's stressful life events		✓
Child's conduct problems		✓
Paternal history of depression	✓	✓
Mother's anxiety	✓	✓
Mother's depression	✓	✓
Partner's anxiety		✓
Partner's depression	✓	✓
Mother's neurotic symptoms (CCEI[2])	✓	✓
Partner's neurotic symptoms (CCEI)	✓	✓
Mother's post-natal depression symptoms (EPDS[3])	✓	✓
Partner's post-natal depression symptoms (EPDS)		
Mother's peer problems		✓
Mother's conduct problems		✓

[1]Domestic violence is defined as physical and/or emotional abuse between parents/caregivers
[2]Crown-Crisp Experiential Index
[3]Edinburgh Postnatal Depression Scale

2.3 Multivariate regression models to assess mediation and moderation

NBRM were used to accommodate the skewed nature of the distribution of internalising symptom scores. However, interpreting NBRMs is especially unwieldy when effect modification is considered. The interpretation for effect modification by mother's education, for example, would be "the factor by which the mean internalising symptoms score would be inflated if every child were exposed versus no child, is estimated to be C times higher among children whose mother's level of education is A relative to those whose mother's level of education if B", where C is the exponent of the estimated product term (between DCA and mother's education) in that NBRM. Finally, when it comes to assessing mediation, an additive decomposition of direct and indirect effects is appealing ("x% of the effect of X on Y is mediated through M"), but this is again not possible (without further manipulation) from the parameters of multiplicative models. Thus, to test mediation, I again used simple non-parametric averaging of carefully chosen predictions from the fitted NBRM but it was more

complicated than previously described as it involved comparing four "alternative worlds" where the mediator of interest and/or DCA did not exist.

Additionally, a sequential multiple mediator analysis (T. J. VanderWeele & Vansteelandt, 2014) was conducted to explore the extent to which many mediators within the child's immediate family life mediated the effect of DCA on internalising symptoms. This method was chosen rather than including one mediator at a time as when mediators were handled one by one, the sum of the proportion mediated on the additive scale for the mediators totalled more than one hundred per cent. This was despite the direction of mediation being the same, and due to the mediators in fact affecting one another or mediator-mediator interactions being present (see Figure 11 for a DAG illustrating the relationships between mediators). I used an estimation-by-simulation approach involving many regression models introducing one mediator at a time. Mediators were added sequentially based on the age of the child when they were measured, so the mediators measured first were added first and so on (see Table 13). An analytic expression for the standard errors of these mediated effects is intractable, therefore I used the non-parametric bootstrap with 100 bootstrap samples. Under strong assumptions including no unmeasured common causes of exposure and outcomes, exposure and either set of mediators, nor either set of mediators and the outcomes, this method allows us to partition the estimated overall effect of DCA on internalising symptoms. This is achieved by partitioning the overall effect into both an indirect effect through some or all of the mediators and a direct effect not through any of the mediators considered. The indirect effects refer to the sequential effects is, i.e., that there is an indirect effect through the first mediator, M1 (which may also include effects that pass through M1 to the later mediators and on to the outcome), and then an effect through either M1 or M2 or both (and then also possibly the later ones), and so on.

Moderation effects were estimated directly from the NBRMs described above, with an additional product term between each potential moderator and the exposure. Standard errors and hence 95%CIs were also calculated. Product terms were created by converting continuous variables to binary through median splits, and then multiplying the binary moderator variable by the exposure (DCA).
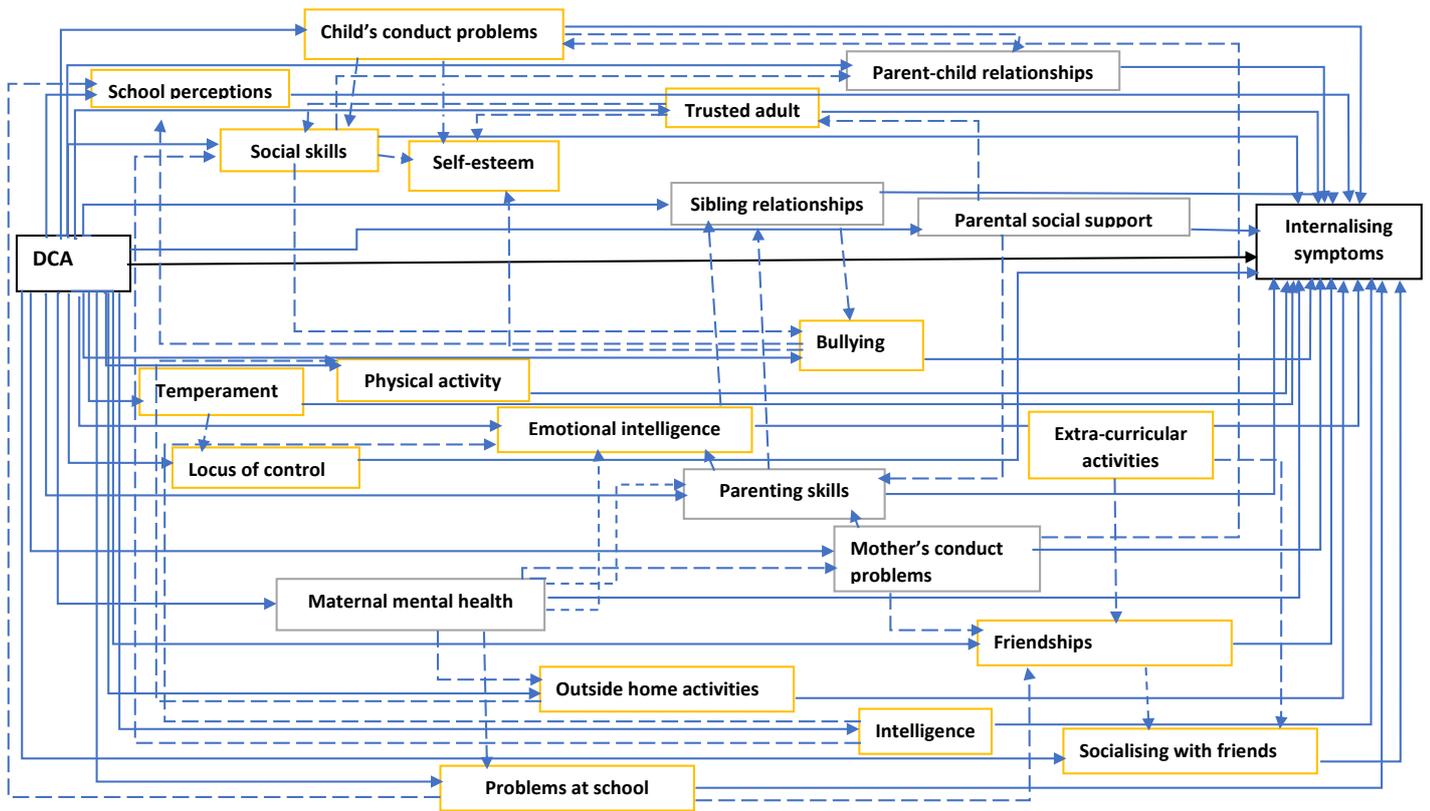
Figure 11. DAG illustrating the proposed pathways between DCA and internalising symptoms along with hypothesised relationships between the mediators. The solid lines indicate indirect pathways between DCA and internalising symptoms and the dash lines indicate hypothesised relationships between mediators.

3 Results

3.1 Sample Characteristics

Overall, 872 (8.56%) children were exposed to DCA at 0-3 years reported by either mother or father/partner; 614 (6.0%) were exposed to DCA at 6-9 years old, and 369 (3.6%) children were reported to have experienced DCA at both time points. DCA at 0-3 years was controlled for in analyses using DCA at 6-9 years as the exposure variable.

Figures 12a and 12b illustrate the sociodemographic characteristics of participants included in the mediation and moderation analyses, by DCA status at 0-3 and 6-9 years respectively, using the first imputed dataset. These tables suggests that children of older, never married mothers, with lower income, lower social class, living in households with higher levels of crowding are more likely to experience DCA and those who have experienced DCA have higher internalising symptom scores. Frequency tables can be found in Appendix H.
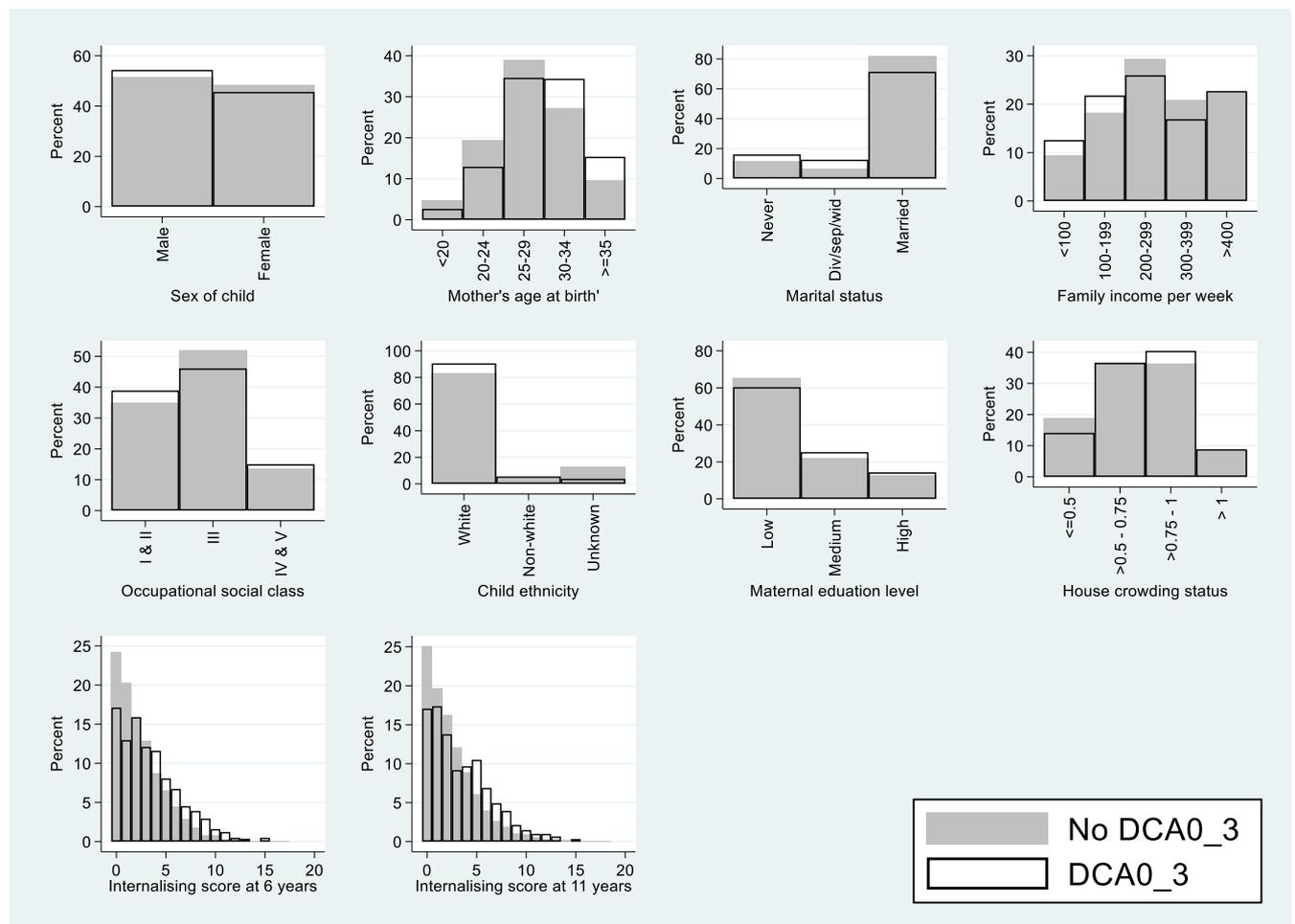
Figure 12a: *Sociodemographic characteristics of participants who had experienced DCA between 0-3 years and those who had in the first iteration of the multiply imputed data (m = 1).*
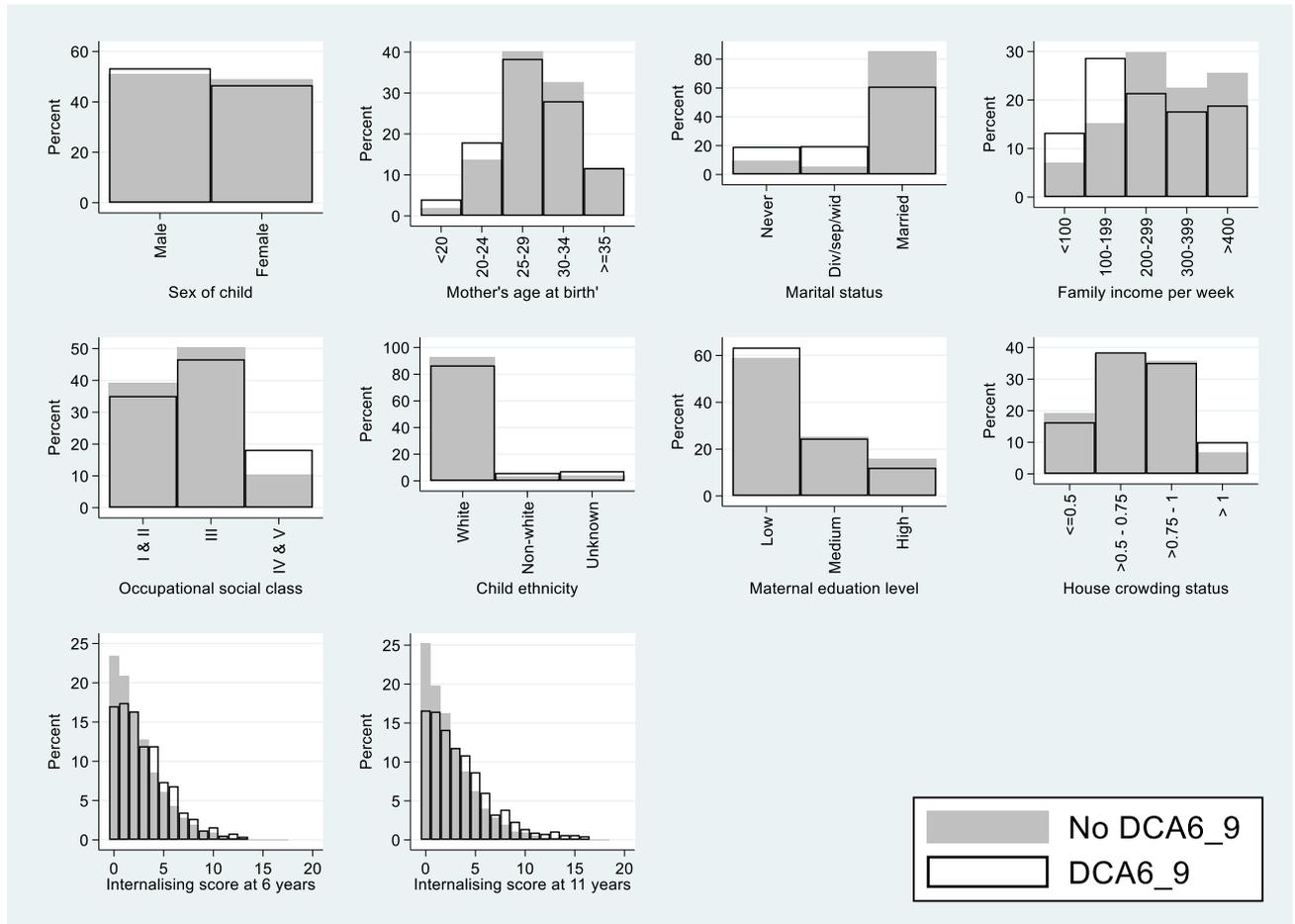
Figure 12b: *Sociodemographic characteristics of participants who had been exposed to DCA between 6-9 years and those who had not been exposed to DCA in the first iteration of the multiply imputed data (m = 1).*

3.2 Preliminary Analyses

3.2.1 Wilcoxon-Rank Sum Tests

The results of Wilcoxon-rank sum tests are presented in Table 15. In summary, internalising symptom scores at both six and thirteen years for those exposed to DCA in early childhood, or middle childhood were significantly greater than those who were not exposed to DCA within the same exposure group.

Table 15. Wilcoxon-rank sum test results estimating the association between DCA at 0-3 years and 6-9 years and internalising symptoms at six and 13 years.

Exposure group	SDQ age measured	Median internalising symptom score	Mann-Whitney U Test Statistic	z-score	P-value
0-3 years Yes No	6 years	 3 2	2.68	-30.53	<0.001
0-3 years Yes No	13 years	 3 2	1.54	-24.18	<0.001
6-9 years Yes No	13 years	 3 2	1.35	-30.54	<0.001

3.2.2 Understanding crude associations between explanatory and outcome variables

The unadjusted NBRM revealed that early exposure to DCA is significantly associated with internalising symptoms score at six years, at a significant level of 0.05. The logarithm of the mean internalising symptoms score at six years was estimated to be 0.32 (SE = 0.04) higher for those exposed to DCA compared with those who were not (p <0.001). When the outcome was changed to internalising symptoms in early adolescence (thirteen years), the unadjusted NBRM indicated that DCA exposure at 0-3 years was also significantly associated with internalising symptoms at thirteen years. The logarithm of the mean internalising symptoms score at 13 years was estimated to be 0.31 (SE=0.04) higher for those exposed to DCA at 0-3 years compared with those who were not (p < 0.001). The unadjusted NBRM for DCA at 6-9 years and internalising symptoms at thirteen also showed DCA was significantly associated with internalising symptoms. The logarithm of the mean internalising symptom score at 13 years was estimated to be 0.39 (SE=0.07) higher for those who had experienced DCA compared to those who had not (p < 0.001).

As described in the previous chapter, the results above were re-expressed on a more meaningful scale, as comparisons between the mean internalising symptom scores in the real world and in a hypothetical world where the children had not experienced DCA. The prediction in the hypothetical world where the children had not experienced DCA is of course only valid under the assumption that all relevant confounders (of DCA and internalising symptoms) have been well measured and appropriately included in the model. The calculation can still be performed, however, even without including any confounders in the

analysis; the comparison between the adjusted and unadjusted results then gives an indication of the extent of (measured) confounding. The results are presented in the top half of Table 16 below.

Table 16. Negative binomial regression model results measuring the total effect of DCA on internalizing symptoms first with no confounders (top half) and then with selected confounders, using multiply imputed data. This table displays the mean internalizing symptoms score and standard errors for those who have experienced DCA. Column 4 shows the mean internalizing symptoms score and standard errors for those who have experienced DCA with the confounders included that were selected by the change-in-estimated-MSE method, Column 5 shows the estimated mean internalizing symptoms score and standard errors for the same group of children in an alternative scenario that assumes their DCA experience had been avoided (with the same confounders), and Column 6 shows the difference between those means and significance value from z-test.

No confounders					
DCA exposure	SDQ AGE measured	Number of observations included in model	Mean internalising symptoms score (standard error)	Mean of the predicted internalising symptoms score had all DCA been avoided	Difference (significance value from z-test)
0-3	6	7,924	2.70 (0.04)	2.60 (0.04)	<0.001
0-3	13	7,924	2.83 (0.04)	2.73 (0.05)	<0.001
6-9	13	6,449	2.83 (0.04)	2.67 (0.06)	<0.001
Adjusting for the chosen confounder set					
DCA exposure	SDQ AGE measured	Number of observations included in model	Mean internalising symptoms score (standard error)	Mean of the predicted internalising symptoms score had all DCA been avoided	Difference (significance value from z-test)

0-3	6	7,924	2.69 (0.04)	2.64 (0.04)	<.001
0-3	13	7,924	2.81 (0.04)	2.77 (0.05)	0.05
6-9	13	6,449	2.82 (0.04)	2.76 (0.05)	0.05

3.3 Total effect of direct child abuse on internalising symptoms at six and 13 years

3.3.1 Internalising symptoms at age six years

The NBRM suggested that when children are victims of DCA from 0-3 years (and everything else in the model is held constant), the logarithm of the mean internalising symptoms scores at six years is estimated to be 0.13 (SE = 0.04) higher compared to those who had not experienced DCA, and this difference is considered to be significant (p <.001).

Again, a post-processing procedure was used in order to convert the results in the previous paragraph to a more meaningful scale and the results are presented in Table 16. In summary, there was evidence that DCA at 0-3 years had an effect on internalising symptoms at six years, and after a 'chosen' set of confounders were included in the NBRM there remained marginal evidence for an effect of DCA on internalising symptoms at a significance level of 0.05. However, the mean predicted outcomes were closer to those in the real world when confounders were controlled for. In other words, some, but not all, of the crude "effect" found was indeed due to the impact of the measured confounders.

3.3.2 Internalising symptoms at 13 years

The NBRM suggested that when children experience DCA from 0-3 years (and everything else in the model is kept constant), the logarithm of the mean of the internalising symptoms scores at 13 years was estimated to be 0.09 (SE = 0.03) higher for those who had experienced DCA compared to those who had not experienced DCA, with some evidence against the null hypothesis of no effect (p=0.03). The converted results showed that there was evidence that DCA at 0-3 years had an effect on internalising symptoms at 13 years, and after a 'chosen' set of confounders were included in the NBRM there remained marginal evidence for an effect of DCA on internalising symptoms at a significance level of 0.05. Thus, quite a large proportion of the crude association found was indeed due to the impact of the measured confounders, but not all. The same was found when children experienced DCA at 6-9 years and internalising symptoms were measured at 13 years. The logarithm of the mean internalising symptoms score was estimated to be 0.23 (SE = 0.06) higher amongst those who had experienced DCA compared to those who had not (p = 0.04), with some evidence of an

effect at a 0.05 significance level. The converted results showed evidence that DCA exposure at 6-9 years had an effect on internalising symptoms at 13 years after the inclusion of confounders (See Table 16).

3.4 Effect moderation by confounders

Note that, as is commonplace in the causal inference literature, we do not exclusively mean "common-cause confounder" when we refer to a "confounder". That is, a variable is also considered to be a potential confounder (i.e. prior to applying the change-in-MSE criterion) if it is lies on any directed path from such a common-cause confounder to either the exposure or the outcome. Also note that all confounders (likewise mediators) are also potentially effect moderators. For example, DV is a potential confounder (albeit not a common cause confounder) of the effect of DCA on internalizing symptoms, and it is also plausibly an important moderator, since the harmful effect of DCA on internalizing symptoms may be even more pronounced when the child is also exposed to DV.

3.4.1 Internalising symptoms at six years

There was insufficient evidence (at a 5% significance level) that any of the confounders listed in Table 14 above moderated the effect of DCA at 0-3 years on internalising symptoms at six years, when each of them were included as interaction terms with DCA individually in multivariate NBRMs.

3.4.2 Internalising symptoms at 13 years

There was insufficient evidence (at a 5% significance level) that any of the confounders moderated the effect of DCA at 0-3 or 6-9 years on internalising symptoms at 13 years.

3.5 Potential moderators

3.5.1 Direct child abuse at 0-3 years and internalising symptoms at six years

There was insufficient evidence of moderation at the 5% significance level for any of the factors at either an individual or at a familial level. Results can be found in Table 17.

Table 17. Table showing the results of the negative binomial regression models (NBRM) including moderation of the effect of DCA at 0-3 years on internalising symptoms at six years. Moderation estimates are logarithms of the mean difference in interaction effects with significance values from z tests.

Factor	Moderation Estimate (SE)	95% confidence interval	Moderation p-value
Mother parenting	-0.03 (0.04)	-0.10 – 0.04	0.39

Father parenting	0.00 (0.02)	-0.04 – 0.05	0.87
Emotionality	-0.02 (0.03)	-0.09 – 0.04	0.04
Activity levels	0.01 (0.03)	-0.05 – 0.08	0.36
Sociability	-0.00 (0.05)	-0.08 – 0.05	0.93
Outside activity	-0.00 (0.04)	-0.07 – 0.08	0.93
Discipline variation	0.02 (0.03)	-0.04 – 0.08	0.46
Father/partner interaction	0.00 (0.03)	-0.05 – 0.06	0.90
Mother interaction	-0.01 (0.02)	-0.05 – 0.04	0.81
Domestic violence	0.02 (0.02)	-0.03 – 0.06	0.40

3.5.2 Direct child abuse at 0-3 years and internalising symptoms at 13 years

Individual factors for which there was some evidence of moderation of the effect of DCA on internalising symptoms at thirteen included emotionality ($p<0.001$) and sociability ($p=0.01$). The effect of DCA on internalising symptoms was different for the different levels of emotionality such that high emotionality increased the negative effect of DCA on internalising symptoms. Alternatively, high sociability was associated with a less pronounced effect of DCA on internalising symptoms compared to the effect in those with low sociability. Maternal moderating factors included mothers internalising symptoms ($p<0.001$) and anxiety ($p=0.06$). High maternal internalising symptoms, and anxiety increased the effect of DCA on higher internalising symptoms at thirteen years such that young adolescents who have experienced DCA and have mothers with mental health problems have higher levels of internalising symptoms compared to those of mothers without mental health conditions. There was evidence of effect moderation for one family level variable namely parent-child relationships ($p<0.001$). The effect of parent-child relationships was negative suggesting that, as one might expect, more positive parent-child relationships reduce the negative effect of DCA on internalising symptoms. See Table 18 for full list of factors and results and Figure 13 for an illustration of the results.

Table 18. Table showing the results of the negative binomial regression models (NMBMs) measuring moderation of the effect of DCA at 0-3 years on internalising symptoms at 13 years. Moderation estimates are logarithms of the mean interaction effects with significance values from z tests.

Factor	Moderation Estimate (SE)	95% confidence interval	Moderation p-value
Bullying victim	0.01 (0.01)	-0.04 – 0.06	0.71
Bullying perpetrator	0.01 (0.03)	-0.00 – 0.03	0.14
Problem at School	-0.00 (0.01)	-0.02 – 0.02	0.66

Externalising behaviours	-0.01 (0.05)	-0.05 – 0.05	0.35
Sibling bullying	0.00 (0.01)	-0.03 – 0.03	0.87
Domestic violence	0.01 (0.01)	-0.02 – 0.03	0.98
Mother internalizing symptoms	0.05 (0.01)	0.05 – 0.09	<0.001
Maternal depression	0.01 (0.01)	0.02 – 0.04	0.43
Maternal anxiety	0.01 (0.01)	0.01 – 0.04	0.06
Parent-child relationships	-0.03 (0.01)	-0.08 – -0.02	0.001
Partner parenting	-0.01 (0.01)	-0.03 – 0.00	0.18
Mother parenting	-0.00 (0.01)	-0.02 – 0.00	0.73
Child emotionality	0.03 (0.01)	0.03 – 0.06	<0.001
Chid activity	-0.00 (0.01)	-0.04 – 0.00	0.58
Child sociability	-0.02 (0.01)	-0.07 – -0.01	0.04
Outside activity	-0.03 (0.01)	-0.05 – 0.00	0.17
Discipline variation	0.01 (0.01)	-0.01 – 0.03	0.35
Partner interaction	-0.01 (0.01)	-0.02 – 0.00	0.45
Mother interaction	-0.00 (0.01)	-0.01 – 0.01	0.68
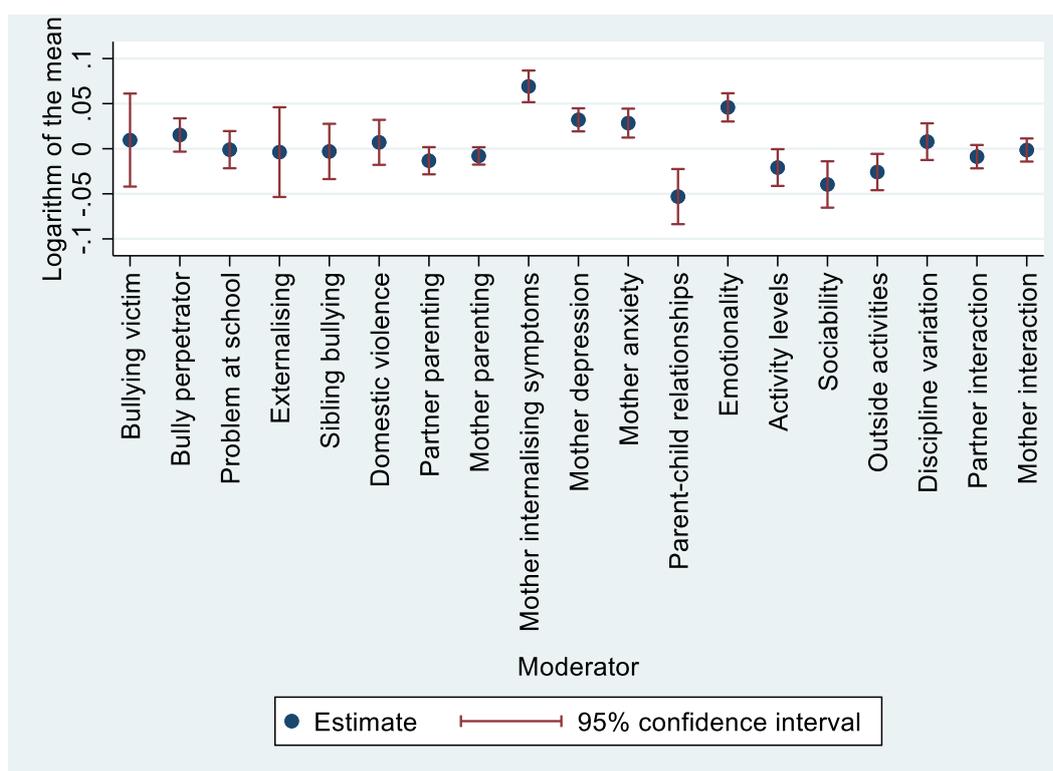
Figure 13. *Effect moderation estimates for DCA at 0-3 years and internalising symptoms at 13 years with 95% confidence intervals. Estimates are the logarithms of the means obtained from the negative binomial regression models.*

3.5.3 Direct child abuse at 6-9 years and internalising symptoms at 13 years

There was some evidence that two factors moderated the effect of DCA at 6-9 years on internalising symptoms at 13 years: children's activities such as taking part in clubs and playing a musical instrument (p=0.05), and parent-child relationships (p=0.001). The association between the activities completed by the child and internalising symptoms is positive but that is because the variable is negatively scored so in fact participation in activities, such as special groups and sports clubs, moderates the effect of DCA at 6-9 years on internalising symptoms at 13 years such that those who experience DCA and participate in high levels of activities have fewer internalising symptoms compared to those who experience DCA but partake in low levels of activities. Similarly, and as was the case when children experienced DCA in their early years, positive parent-child relationships moderated the effect of DCA at 6-9 years on internalising symptoms at 13 years such that the effect of DCA on internalising symptoms at 13 years was weaker amongst those with positive parent-child relationships compared to those with poor parent-child relationships, suggesting that parent-child relationships are a protective factor. It should be highlighted here that the parent-child relationships variable includes the study child's relationships with their mother and their father/mother's partner, one (or both) of whom would have been the DCA perpetrator but it is not possible to disentangle. See Table 19 and Figure 14 for a summary of the results.

Table 19. Table showing the results of the negative binomial regression models estimating moderation of the effect of DCA at 6-9 years on internalising symptoms at 13 years. Moderation estimates are "differences-in-differences" of log conditional mean internalising symptom scores effects with p-values from z tests comparing each difference-of-differences to the null value of nought.

Factor	Moderation Estimate (SE)	95% confidence interval	p-value
Bullying victim	0.00 (.04)	(-0.09, 0.09)	0.97
Bully perpetrator	0.00 (.02)	(-0.03, 0.03)	0.82
Problem at School	-0.02 (.02)	(-0.06, 0.02)	0.36
Externalising problems	0.01 (.04)	(-0.07, 0.09)	0.83
Sibling bullying	0.01 (.02)	(-0.03, 0.06)	0.82
Domestic violence	-0.03 (.03)	(-0.09, 0.03)	0.32

|			
Maternal depression	0.01 (.01)	(-0.01, 0.03)	0.45
Maternal anxiety	0.01 (.01)	(-0.01, 0.03)	0.22
Parent-child relationships	-0.02 (.01)	(-0.04, -0.01)	0.01
Child activities	0.02 (.01)	(-0.00, 0.03)	0.05
Locus of control	0.01 (.02)	(-0.02, -0.04)	0.43
Emotional awareness	0.00 (.02)	(-0.04, 0.04)	0.87
Intelligence	-0.00 (.02)	(-0.04, 0.03)	0.99
Friendships	0.01 (.03)	(-0.05, 0.07)	0.89
Child's self-esteem	0.00 (.02)	(-0.03, 0.04)	0.80
Activities with friends	-0.00 (.02)	(-0.06, 0.04)	0.89
Vigorous physical activity	-0.01 (.02)	(-0.05, 0.03)	0.62
Sibling relationships	-0.01 (.03)	(-0.05, 0.05)	0.58
Maternal social support	0.00 (.02)	(-0.05, 0.05)	0.99
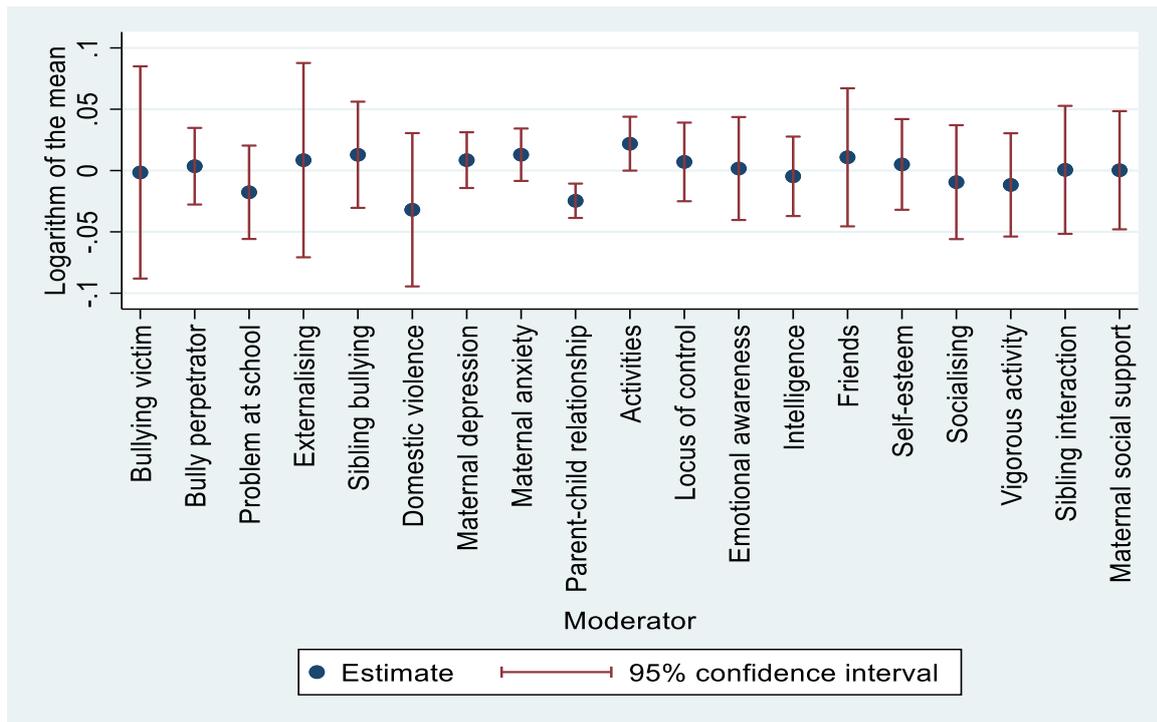
Figure 14. *Effect moderation estimates for DCA at 6-9 years and internalising symptoms at 13 years with 95% confidence intervals*

3.6 Mediation analyses

3.6.1 DCA at 0-3 years and internalising symptoms at six years

Overall, the 11 included mediators were estimated to account for 57% of the effect of DCA on internalising symptoms at six years. Mother's emotional symptoms and conduct problems, and father/partner's parenting appeared to account for the largest proportions of the effect that were mediated. This is illustrated in Figure 15 as having the greatest increase in estimated log of the mean internalising symptoms score compared to the previous set of factors given that the mediators were added sequentially meaning that the previous model includes all those from the first to the n-1 mediator.
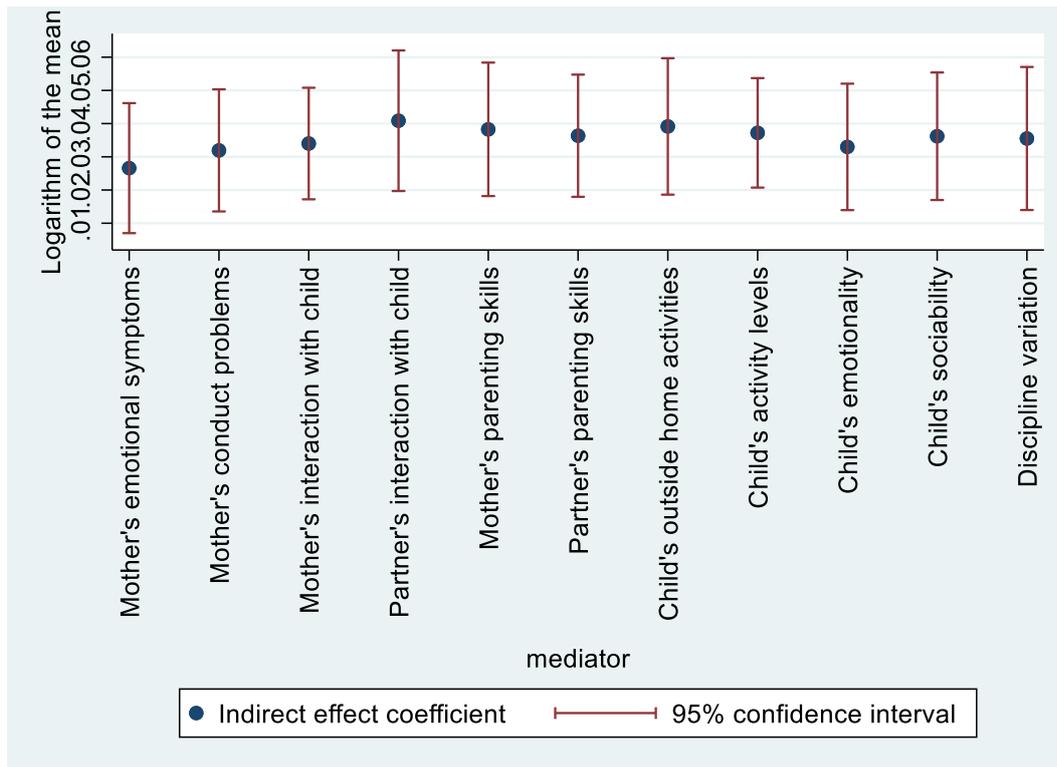
Figure 15. *Estimates of the indirect effects of DCA at 0-3 years on internalising symptoms at six years from a multivariate sequential mediation analysis, with 95% confidence intervals. Estimates are the log of the mean internalising symptom score at six years.*

3.6.2 DCA at 0-3 and internalising symptoms at 13 years

The 26 included mediators together were estimated to account for 85% of the effect of DCA at 0-3 years on internalising symptoms at thirteen years (see Figure 16). It is not surprising this model accounted for a greater proportion of the effect given the higher number of mediators included due to the large time gap between the exposure and the outcome. Mother's internalising symptoms and conduct problems, and child's conduct problems appeared to account for the largest proportions of the effect that were mediated, as the differences between the estimate for those factor sets had the biggest increase compared to the previous factor set.
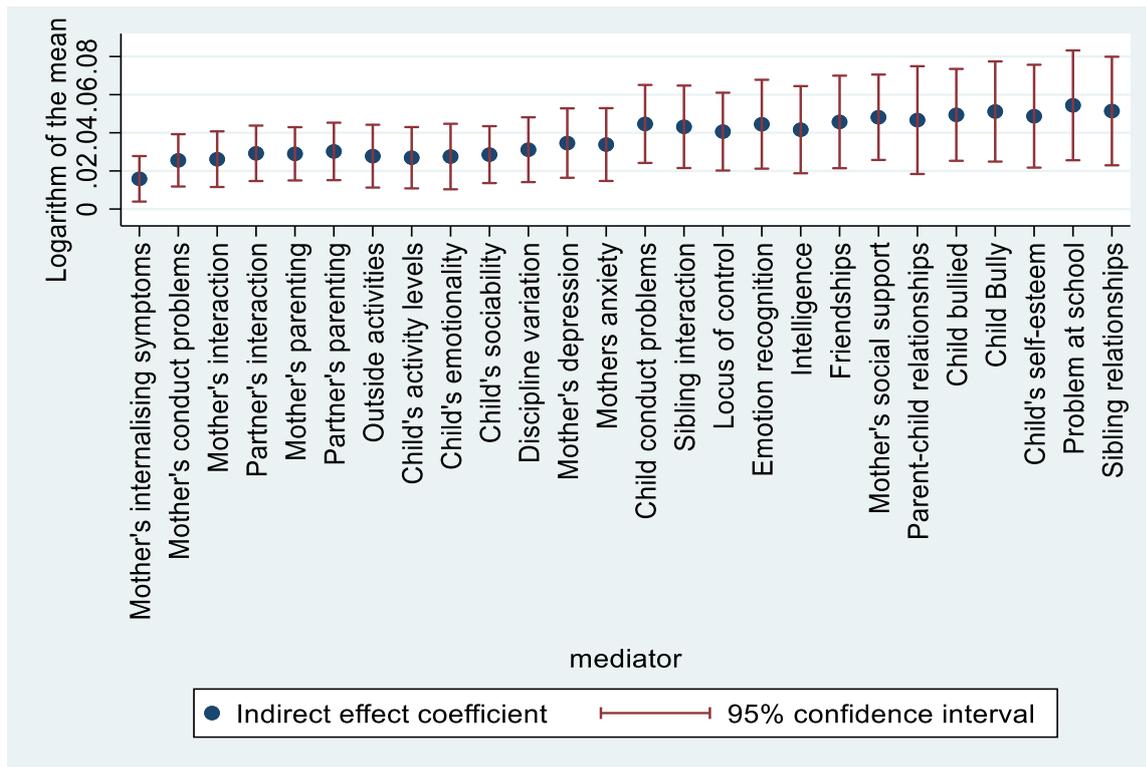
Figure 16*. Estimates of the indirect effect of DCA at 0-3 years on internalising symptoms at 13 years from a multivariate sequential mediation analysis, with 95% confidence intervals. Estimates are the log of the mean internalising symptom score at 13 years.*

3.6.3 DCA at 6-9 years and internalising symptoms at 13 years

Together, the 13 included mediators accounted for 42% of the effect of DCA at 6-9 years on internalising symptoms at thirteen years (see Figure 17). Parent-child relationships and child's conduct problems appeared to account for the largest proportions of the effect that were mediated, but there is a large amount of uncertainty around the estimates.
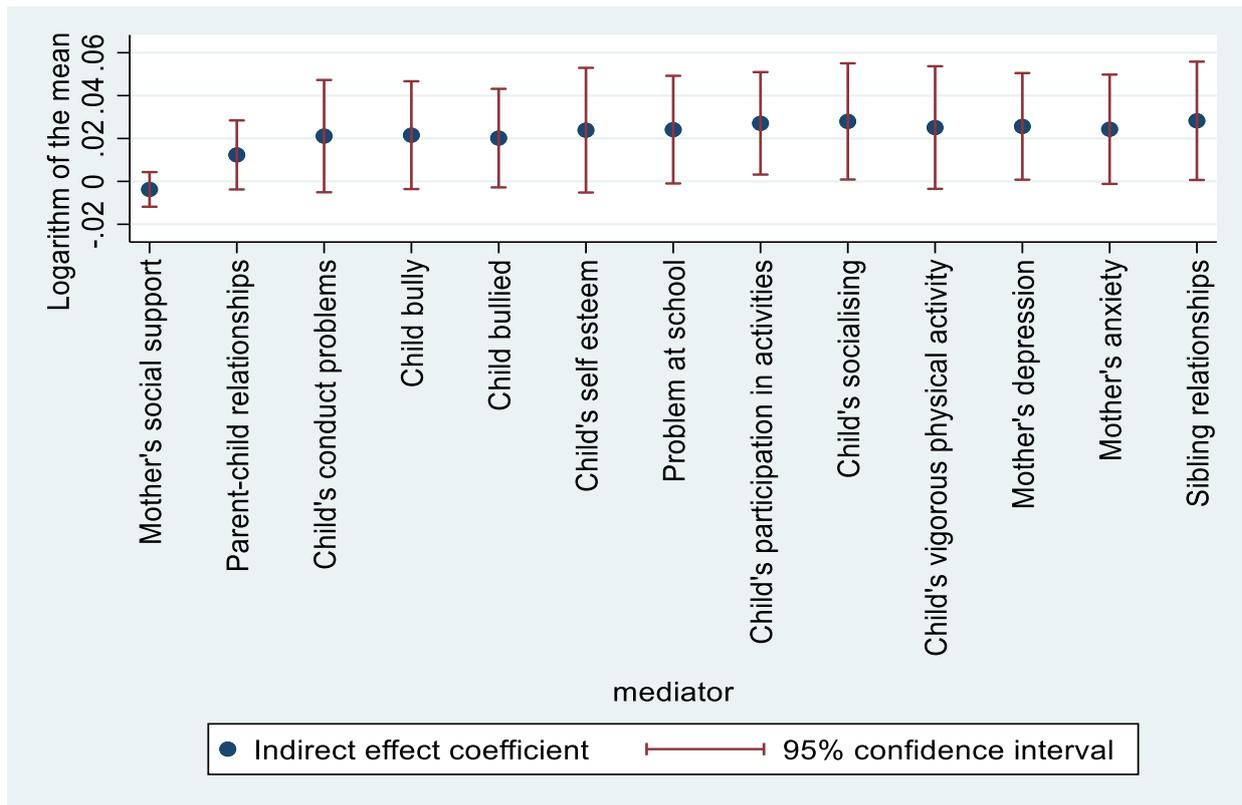
Figure 17. *Estimates of the indirect effect of DCA at 6-9 years on internalising symptoms at 13 years from a multivariate sequential mediation analysis with 95% confidence intervals. Estimates are the log of the mean internalising symptom score at thirteen years.*

4 Discussion

This study aimed to estimate the effects of DCA on internalising symptoms both in childhood and adolescence, and finally it explored the causal pathways between DCA and internalising symptoms, with a particular focus upon protective factors that mediate or moderate the effect of DCA on internalising symptoms.

4.1 Prevalence of DCA within ALSPAC

This study has found that within the ALSPAC dataset, 872 (9.6%) children had experienced DCA within the family when they were 0-3 years old. Within the imputed dataset 749 (7.4%) children experienced DCA within the family when they were 6-9 years old. These results are roughly comparable to ONS Crime Survey for England and Wales in 2019 (Elkin, 2019) which found that 7.6% of adults reported being physically abused and one in eleven reported being emotionally abused by the age of sixteen. It is important to highlight that the measures of DCA at 0-3 years and 6-9 years are not directly comparable as the early DCA measure included items from five waves of data whereas the measure at 6-9 years was taken from one

wave of data. Thus, there were many more opportunities for parents to report DCA during the child's early years and the measure during middle childhood is likely to result in a conservative estimate given many families will have dropped out of the study, although efforts have been made to account for this using MI and IPW.

4.2 Effect of DCA at 0-3 years and 6-9 years on internalising symptoms at six and 13 years
Using cohort data, this study has shown that DCA within the family has a significant negative effect on internalising symptoms at both six and 13 years. There was evidence that early DCA led to more severe internalising symptoms at both six and 13 years and DCA during middle childhood led to more severe internalising symptoms at 13 years. These effects remained significant after sets of confounders were included in the models, suggesting that the effect is causal. However, it is important to note that although major known confounders were in included in the analyses, there is a lot of uncertainty around the estimates and these could suffer from residual confounding.

4.3 Summary of findings for early DCA and internalising symptoms at six years
As total effects were observed, mediation and moderation were explored utilising multivariate modelling and a post-processing of the results in order to produce more easily interpretable statistics. When children experienced early DCA, there was no evidence for moderators impacting the effect of DCA on internalising symptoms at six years at a 5% significance level. This may be because the tests of moderation using interaction terms suffered from low power, particularly because we dichotomised the potential moderators, and therefore subtle moderation might not have been detected.

4.4 Summary of findings for early DCA and internalising symptoms at 13 years
When the causal pathway between DCA at 0-3 years and internalising symptoms at 13 years was explored, evidence of effect moderation of the total effect was also found for a number of factors. Individual factors that moderated the effect of DCA on internalising symptoms at 13 years included emotionality and sociability. Sociability, measured when the child was three, buffered children against internalising symptoms in early adolescence. Alternatively, emotionality at three years was associated with more severe internalising symptoms at 13 years among those who had experienced DCA at 0-3 years.

Maternal internalising symptoms moderated the direct effect of DCA on internalising symptoms in early adolescence, and increased the risk of more severe internalising symptoms at 13 years.

At a familial level, positive parent-child relationships buffered against internalising symptoms but this may have been because more positive relationships were associated with less severe levels of DCA within the family. It was unclear in this analysis whether the DCA was mother or partner perpetrated, yet that would give a clearer picture of the situation. It may have been that one parent figure compensated for the abuse of the other by being extra caring however this theory needs to be explored further.

The 26 included mediators together were estimated to account for 85% of the effect of DCA at 0-3 years on internalising symptoms at thirteen years. Mother's internalising symptoms and conduct problems, and child's conduct problems appeared to account for the largest proportions of the effect that were mediated, although it is difficult to tell whether other factors, not included in the factor set, and therefore not measured, were contributing to the findings.

4.5 Summary of findings for DCA during middle childhood and internalising symptoms at thirteen years

Among children who experienced DCA at 6-9 years, there was evidence that parent-child relationships and children's activities moderated the effect of DCA during middle childhood on internalising symptoms at 13 years. Both these factors acted as buffers, interacting with DCA to led to reduced internalising symptoms compared to those with poor parent-child relationships and those who did not participate in many activities.

There was also evidence found for parent-child relationships and children's conduct problems acting as mediators of the effect of DCA on internalising symptoms. However, it is unclear how much of the effect of DCA on internalising symptoms is accounted for by these possible mediators given the way in which mediation was measured.

In this study maternal mental health has been shown to have a great impact on child and adolescent mental health. This is in line with a large amount of previous research (Argent, Kalebic, Rice, & Taylor, 2020; Essex, Klein, Cho, & Kraemer, 2003) and studies have shown that maternal mental health problems have a negative effect on their children regardless of whether they have experienced DCA (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007; Noonan, Burns, & Violato, 2018). This again highlights the importance of working with mothers to improve their mental health as this may have a big impact on their child(ren)'s health and wellbeing. However, the results from this study also suggest that improving social skills, along with activities for children outside of the house, can have a

positive impact, particularly for older children and adolescents. This is in line with a previous study exploring internalizing symptoms following abuse which found that high social competence and proactive parenting were protective in a community sample of children, regardless of abuse type, among other factors (Lansford et al., 2006).

Schultz, Tharp-Taylor, Haviland, and Jaycox (2009) also found that social competence had a significant effect on internalizing symptoms such that children and adolescents with high social competence were more likely to be within the normal range of internalizing symptoms two years after first assessment. Daily living skills and peer relationships scores also had a significant effect when the protective factors were included in the logistic models separately however when all included, they no longer had a significant effect. This may have been because these factors were highly correlated with social competence. It is important to note that Schultz et al. (2009) used a sample of children with a child protection investigation and therefore the sample also included those who had been sexually abused and/or neglected, as well as those who had suffered physical and/or emotional abuse.

4.6 Implications of findings

These findings have implications for practitioners working with children who have been maltreated as they provide potential strengths to explore and include within psychological assessments, such as sociability and peer relationships, and protective factors to include and develop within interventions, including positive parent-child relationships and positive maternal mental health. They also provide evidence-based factors for preventative interventions to address, to help children who have been abused to be resilient and reduce the risk of them developing internalizing symptoms. Interventions will benefit from focussing not just at an individual level, but with the family and improving peer relationships as well.

4.7 Limitations

It is important to note that the variables used to measure DCA in the study do not state whether mother's and/or partners directly abused the particular child, only that a child within the family were abused. Therefore, the children within this study could have been observers rather than direct victims of DCA. However, it is likely that if one child in a family is abused, others in the family are likely to be too.

The study is limited by many of the same issues with the data and methods used in the previous chapter. For instance, ALSPAC relies on parental reports of both DCA and internalising symptoms and it is likely that parents under-report both of these phenomena

given DCA is socially inappropriate behaviour and few parents would like to think that their child is suffering with internalising symptoms, so they may be in denial or unaware of their child's symptoms. However, it is also possible that parents with their own history of ACEs or poor mental health view their children more negatively and consider their internalising symptoms to be more severe than they are in reality. The measure of DCA in ALSPAC, like the DV measure, is also broad and vague. Moreover, the measure does not ask if the study child themselves had been physically or emotionally abused, only that the mother and/or her partner had been cruel to their children. This measure has not been validated against other measures of child maltreatment either, and therefore it is recommended that future research validates this measure against others and against social care records.

Additionally, the large amount of data missing, particularly within the later waves of data, mean that I had to rely on methods to overcome potential bias due to attrition and missing data, namely IPW and MI. Although these methods have been demonstrated to address bias in longitudinal cohort studies such as ALSPAC, they are not perfect and results may be different if issues around missing data were not present. There are also risks to assuming that no response on ALSPAC questionnaires means that DCA did not occur. It might be that people choose not to answer a particular question because it triggers uncomfortable thoughts associated with personal experience for example. So it might be that exposure is higher among those who chose not to answer than in the general population. Whereas I considered no responses to be because respondents deemed the questions not applicable, given that they often did not complete any of the life events questions if they did not complete the DCA questions. This means that I have underestimated the number of children exposed to DCA and thus underestimated, or missed, causal pathways between DCA and internalising symptoms.

It is not clear whether the risk and protective factors identified within this study are specific to those who have experienced DCA or whether they are in fact compensatory factors that have the same effect among all children and young people, regardless of whether they have experienced DCA. For instance, it seems likely the positive relations with peers will have a positive impact on the well-being of all young people.

It is also important to highlight that dichotomising the potential moderators before testing for effect moderation throws away a lot of information and means that the tests for moderation

were probably quite underpowered, therefore it is likely that there are more moderators than just those identified in this study.

Finally, the causal interpretation of my estimates, both of the overall effects of DCA on internalising symptoms, and of the extent to which these are mediated and moderated by numerous potential risk and protective factors, all rely on strong untestable assumptions, predominantly that there are no unmeasured common causes of any two or more of the groups of variables considered. For example, there could be other elements of SES, not captured within this study, that confound the relationship between DCA and internalising, and/or between the mediators and the outcomes or exposure, such as housing status e.g., rented or owned property and family size. If these unmeasured components of low SES increase the probability of having internalising symptoms, decrease coping scores and increase maternal low mood scores, for instance, then both the overall effect of DCA and the extent to which it is mediated by maternal low mood would likely be exaggerated.

5 Conclusion

In summary, DCA has a significant effect on children and young adolescents' mental health regardless of when the DCA and internalising symptoms are measured. This finding supports previous research that has reported the long-term negative effects of DCA (D. T. Cicchetti, 1995; Flisher et al., 1997). Thus, efforts to reduce/prevent DCA should continue to be prioritised, yet given that some children will unfortunately continue to experience DCA, it is also important to ensure children are supported in the best way possible and have the necessary skills and resilience so that the negative impacts of such maltreatment can be mitigated. This study has shown that a number of factors may be targeted within treatment and preventative interventions for older children and young people. At an individual level, encouraging participation in activities and improving social skills may help prevent young people who have been physically and/or emotionally abused from developing internalising disorders. At a familial level reducing maternal mental health problems, and improving parent-child relationships may reduce internalising symptoms among young people.

Chapter 7. Summary of findings and conclusions

1 Overview

The main aims of this thesis were:

i. To identify evidence for risk and protective factors for internalising symptoms among children and adolescents exposed to DV

ii. To quantify the roles of protective factors on the effects of child exposure to DV and DCA on internalising symptoms using a population-based cohort dataset in England (ALSPAC)

This thesis presented novel methodological and empirical contributions to further the understanding of the epidemiology of CEDV and DCA on child and adolescent internalising symptoms, specifically exploring risk and protective factors and the causal pathways between CEDV/DCA and internalising symptoms. In this final chapter, I summarise the key results for each of the research questions specified in the introduction, consider the main strengths and limitations of my analyses, propose avenues for future research, and present the overarching implications and conclusions of this work. A detailed discussion, including comparison of my results with the existing literature, has been provided in each of the results chapters within this thesis.

2 Main results

2.1 Research Question 1: What factors mediate or moderate the effect of exposure to DV on child and adolescent internalising outcomes?

I addressed this research question through the systematic literature review of risk and protective factors for child and adolescent internalising symptoms among those who had been exposed to DV. The review included evidence over years 1990-2020 and included 48 studies (presented in Chapter 2). The key findings from the review were:

- One individual level mediator was identified, namely emotional intelligence and two moderators were identified: relational victimisation and participation in extracurricular activities.

- Familial mediators included maladaptive parenting and parenting stress, while moderators included positive parenting (maternal warmth and availability) and family social support.

- No community level protective factors were identified

- Most research in the field has been conducted in the United States

- There is a lack of research exploring the causal pathway between CEDV and internalising symptoms during early childhood and adolescence and most studies only control for a couple of confounders

- Research has focussed upon risk with few studies investigating the effects of protective factors which may be targeted by preventative interventions

My systematic literature review was published in the review journal Trauma, Violence, and Abuse and a copy of the published manuscript can be found here: https://doi.org/10.1177%2F1524838020965964

2.2 Research Question 2: What factors protect children and young people from the negative effects of trauma and increase resilience?

I conducted a systematic search of the ACEs and resilience literature to identify factors that protect children against internalising symptoms and other negative outcomes following traumatic experiences during childhood. I found there are number of factors proposed at an individual, family, and community level:

- Individual – cognitive function/intelligence, temperament, emotion regulation, social/communication skills, religion, internal locus of control, physical activity, self-esteem, optimism
- Family – At least one trusted adult, positive parent-child relationships, sibling relationships, and parental mental health, good parenting skills, parental perceived social support, secure attachment
- Community – friendships/peer support, extracurricular activities and perception of school/school environment

I used this information along with the results of the systematic review to develop a DAG to be used to inform the analysis chapters within this thesis in terms of which variables to include as potential protective factors and covariates/confounders.

2.3 Research Question 3: What is the effect of CEDV on child and adolescent internalising symptoms within a UK population-based cohort study?

I utilised ALSPAC cohort data to conduct a longitudinal analysis determining the effect of exposure to DV at 0-3 years on internalising symptoms at six and 13 years, controlling for

confounders measured in the study and informed by potential confounders identified within Chapter 4.

Overall, I found that:

- Crude estimates of the effect of DV on child and adolescent internalising symptoms implied a significant effect of DV on internalising symptoms at both time points
- When confounder sets were included within analyses, there was no longer evidence that DV exposure has an effect on internalising symptoms at six or 13 years, yet there was evidence that DCA has an effect.

2.4 Research Question 4: Do protective factors identified from the literature mediate or moderate the effect of early childhood/recent exposure to DV on adolescent internalising problems?

Although I set out to answer this question, given insufficient evidence of a total effect of CEDV on child or adolescent internalising symptoms was observed, I concluded it would be inappropriate to explore the causal pathways between the exposures and outcomes. Alternatively, as DCA was observed to have a significant effect when controlling for it as a confounder, I changed course and explored the causal pathways between DCA and child and adolescent internalising symptoms instead.

2.5 Research Question 5: What is the effect of DCA during early years and middle childhood on child and adolescent internalising symptoms?

I conducted multivariate modelling on multiply imputed ALSPAC, population-based, cohort data and found:

- Evidence for the effect of early DCA on internalising symptoms at six and 13 years and DCA during middle childhood on internalising symptoms at 13 years.
- That effects of DCA remained significant at both time points after inclusion of the chosen confounder sets

2.6 Research Question 6: What are the mediators and moderators of the effect of DCA on internalising symptoms during childhood and adolescence?

I conducted multivariate modelling on multiply imputed ALSPAC, population-based, cohort data and found:

- Insufficient evidence that the factors measured during early years mediated or moderated the effect of DCA at 0-3 years on internalising symptoms at six years.

- Evidence (at a 5% significance level) that a number of factors moderated the effect of DCA at 0-3 years on internalising symptoms at 13 years and had a buffering effect. At an individual level, the effect of DCA on internalising symptoms was smaller when children also had high sociability, activity levels and participated in high levels of outdoor activities compared to those who had low sociability, activity levels and took part in few outdoor activities. Alternatively, children who had experienced DCA and had high levels of emotionality had more internalising symptoms compared to those with low levels. At a family level, the effect of DCA on internalising symptoms was different with different levels of parent-child relationships such that those who experienced DCA and also had positive parent-child relationships had reduced internalising symptoms compared to those with poor parent-child relationships. Mothers' mental health symptoms (anxiety, depression, and internalising symptoms) also moderated the effect of early DCA on internalising symptoms as the effect of early DCA led to more severe internalising symptoms at 13 years when children had mothers with more mental health symptoms compared to those whose mothers had few mental health symptoms.

- Maternal anxiety and depression, children's activities such as attending groups, and parent-child relationships moderated the effect of DCA at 6-9 years on internalising symptoms at 13 years. The effect of DCA in middle childhood on internalising symptoms at 13 years was moderated by maternal anxiety and depression such that those with mothers who had high levels of maternal anxiety and depression experienced higher internalising symptoms compared to those with mothers who had low levels of anxiety and depression. Alternatively, high levels of activities and good parent-child relationships were associated with a less pronounced effect of DCA on internalising symptoms compared to the effect in those with low levels of activities and poor parent-child relationships.

- The mediators investigated explained a sizeable proportion of the effect of DCA on internalising symptoms at six and 13 years. Key mediators appeared to be mother's emotional/internalising symptoms, and both mother and child conduct problems. There was also some evidence for father/partner parenting skills mediating the effect of DCA at 0-3 years on internalising symptoms at six years, and parent-child relationships mediated the effect of DCA at 6-9 years on internalising symptoms at 13 years.

3 Strengths and limitations of the key results

3.1 Data Considerations

A strength of this research was the use of large population-based birth cohort (ALSPAC) data. This rich dataset enabled me to derive exposure variables based on reports from multiple informants, and multiple time points, increasing the likelihood of true DV or DCA reports. Furthermore, to the best of my knowledge, the sample sizes in the studies exploring causal pathways are the largest in the DV literature within the United Kingdom to date.

However, as described in detail in previous chapters, one important caveat of the data is the likelihood of measurement error due to the use of self-report, subjective methods to measure exposure to DV and DCA, and parental reports of child symptoms. Most notably, potential misclassification, and underreporting, of both the exposures and outcomes will have implications for the validity of the effect estimates. Yet, there is no gold standard method for measuring exposure to DV, and this method is likely to capture more positive cases than police or social services reports for example, and such reports only capture the most severe cases, or in other words the tip of the iceberg. Furthermore, the postal nature of ALSPAC surveys and reassurance that data will be anonymous may have increased honesty among parents and caregivers.

Compared to other UK-based cohorts (such as the Millennium Cohort Study (Connelly & Platt, 2014) which has also collected information about DV, the ALSPAC dataset benefits from including father/partner reports of abuse and measures emotional abuse and as well as physical abuse which can have an equally damaging impact (Vu et al., 2016). Furthermore, the higher number of data collection waves ALSPAC reduces the risk of recall bias and counters some of the potential reasons for underreporting, such as social desirability bias and the fear of repercussions from disclosing DV or DCA. With regards to DCA measures, ALSPAC benefits from asking comparable questions for child abuse as intimate-partner abuse and at the same, multiple time points (during early and middle childhood), whereas the MCS only asks about harsh parenting and physical child abuse when the child was three years. Nevertheless, the use of psychometrically tested measures of DV and DCA would have improved the external validity of this research.

I have had to make the assumptions that parental reports of DV mean that the child is "exposed" and that parental reports of DCA towards their children in general meant that the

cohort child had been abused. This may lead to an over-estimate of exposure to DV and DCA and the studies would have benefitted from having child reports to validate parental reports. Furthermore, there are risks to assuming that no response on ALSPAC questionnaires means that DV or DCA did not occur. It might be that people choose not to answer a particular question because it triggers uncomfortable thoughts associated with personal experience for example. So it might be that exposure is higher among those who chose not to answer than in the general population. Whereas I considered no responses to be because respondents deemed the questions not applicable, given that they often did not complete any of the life events questions if they did not complete the DCA questions. This means that I may well have underestimated the number of children exposed to DCA and thus underestimated, or missed, causal pathways between DCA and internalising symptoms.

Internalising symptoms are measured within ALSPAC utilising the SDQ. This is a validated measure of internalising symptoms, and is comparable to the widely used CBCL(R. Goodman & Scott, 1999). However, as discussed in previous chapters, the reliance on mother's reports of their child's internalising symptoms reduces the validity and introduces reporting bias given that mothers are likely to underreport their child's internalising symptoms for a number of reasons including social desirability bias, denial, or lack of awareness.

Finally, participants in the ALSPAC sample tended to be of higher socioeconomic status, and children tended to have better educational outcomes, than those in the general population of the UK. These differences should be considered when attempting to generalise prevalence estimates to the rest of the UK (Boyd et al., 2013).

3.2 Statistical methods

As in many cohort studies, missing data were common in the ALSPAC dataset. The epidemiological studies in this thesis were strengthened by the use of MI methods to reduce selection bias and maximise effective sample size, whilst ensuring that MI was not relied upon too heavily by also using IPW to account for bias due to attrition. However, the actual values of the missing data remain unknown and therefore it is not possible to determine whether these methods provided an adequate solution to the problem, nor whether or not the assumptions made, such as missingness at random, were appropriate. That said, my investigation comparing the distribution of the observed and imputed values univariately offered some reassurance that the imputation procedure was sensible.

The use of causal inference methods (DAGs) to guide the statistical modelling strategy and the interpretation of protective factors is a further strength of this work. This approach enabled me to identify plausible causal protective factors for internalising symptoms following DCA, and to use clear and reproducible methods for confounder selection to minimise bias in effect estimates. The DAG that I created was informed by subject-matter knowledge and based on a synthesis of the current available evidence within the resilience and childhood trauma literature. This approach is consistent with the view that DAGs should represent a researcher's understanding and beliefs about how the world works for a particular causal question (Glymour & Greenland, 2008).

Given the potential mediators in my analyses exploring the causal pathways between DCA on internalising symptoms were likely to affect one other, I performed a sequential multiple mediator analysis (T. J. VanderWeele & Vansteelandt, 2014) to investigate the extent to which threat perception and emotions mediated the effect of DCA on internalising symptoms. Although I used a causal inference approach to strengthen my analyses, it is important to again note the limitations of observational data. The protective factor effect estimates (mean differences and standard errors) that I have presented are still based on measures of association, and will only resemble the true causal effect to the extent to which there is no unmeasured confounding. The assumption of no unmeasured confounding is unlikely to hold for most epidemiological investigations. Therefore, the results I have presented represent my attempt to obtain the most rigorous estimates, given the limitations of the data.

4 Implications for future research

4.1 Research gaps and extensions

There has been a paucity of research investigating the effects of DV and DCA on children within the UK, particularly those utilising a causal approach and longitudinal methods. The results I have presented in this thesis controversially found no effect of DV on child and adolescent internalising symptoms, however due to limitations more research is needed to support or disprove this finding. Alternatively, I have presented evidence to indicate the burden of DCA on children's internalising symptoms and thus the wider society. They also provide evidence for protective factors that may be focussed upon by practitioners or researchers developing preventative interventions for maltreated children and adolescents. Future research is needed to develop such evidence-based preventative interventions

increasing factors including children's emotionality and social skills and maternal mental health, and relationships between parents and children, presumably as this would also reduce DCA.

4.2 Methodological and statistical recommendations

Results from this research indicate the differences that can occur when rigorous analyses of DV on internalising symptoms are conducted including a myriad of confounders, and assessing the effects of DV and DCA alongside each other. I recommend that future research also utilises a causal approach with longitudinal data and numerous important confounders in order to determine whether the same results are found. Additionally, studies would benefit from utilising validated child-report measures of both exposure to DV and internalising symptoms rather than relying upon parental reports. This point also applies to analyses exploring the causal pathway between DCA and internalising symptoms, however future research should also ensure that the questions asking about DCA refer to the study child not children with the family in general, so that it is clear whether the study child has in fact experienced DCA as opposed to have only witnessed it, yet the effects of witnessing and experiencing abuse may be similar (Moss, 2003; Teicher & Vitaliano, 2011).

5 Main conclusions

This thesis has identified evidence for risk and protective factors for children and adolescents exposed to DV within the literature and explored the causal pathways between both DV and internalising symptoms and DCA and internalising symptoms. It has found that when unvalidated parental reports of DV and internalising symptoms are utilised, DV does not appear to effect internalising symptoms among children and adolescents, yet evidence suggests DCA does have a significant effect on both child and adolescent internalising symptoms. Furthermore, in line with previous research I have found maternal mental health factors and parent-child relationships to play a major role in this causal pathway suggesting these are key areas for interventions for children and young people to focus on. Other factors include both maternal and child conduct problems and partner parenting skills. Given the paucity of research investigating these causal pathways, particularly within the UK, more research is needed to conclude whether DV really does not have a causal effect on internalising symptoms, when better measures of the exposure and child reports of internalising symptoms are used, and whether the protective factors identified are protective amongst other samples who have experienced DCA, such as Black, Asian and minority ethnic (BAME) groups who were under-represented within the ALSPAC sample. There also

remains important questions about whether the items used to measure child exposures such as DV and DCA in ALSPAC are actually measuring what they aim to measure. For example, the measure for DV in ALSPAC does not actually measure child exposure to DV, rather whether mothers and/or their partners have been victims or perpetrators of DV. The questions are very broad, including emotional cruelty and it is quite possible that a genuinely cruel person might not consider their behaviour to have been cruel, while those answering yes might be reflecting on something they once said about which they feel guilty. Thus further research should conduct sensitivity analyses with other measures of DV, such as child reports of validated measures (e.g. CTS) or social care records, to determine the validity.

Appendices

Appendix A. Systematic Review search strategy

1.   partner violence/ or partner violence.mp. or domestic violence/ or domestic violence.mp. or battered woman/ or battered wom*.mp. or family violence/ or family abuse.mp. or family violence.mp. or conjugal violence.mp. or marital conflict.mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fx, dq, nm, kf, px, rx, ui, sy, tc, id, tm]

2.   (intimate partner violence or intimate partner abuse).mp. or partner violence/ or interparent* violence.mp. or interparent* abuse.mp.

3.   1 or 2

4.   adolescen*.mp. or adolescence/ or teen*.mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fx, dq, nm, kf, px, rx, ui, sy, tc, id, tm]

5.   child/ or child*.mp. or infant.mp. or offspring.mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fx, dq, nm, kf, px, rx, ui, sy, tc, id, tm]

6.   4 or 5

7.   3 and 6

8.   (mechanism* or pathway* or mediat* or moderat* or associat* or cause or risk or protective or resilience or correlat* or relat* or link* or indirect).ti,ab.

9.   7 and 8

10.   (internali* or psychopathology or social or emotional or mental or psycho-social or developmental or adjustment or behav* or depression or anxiety or somatic or withdrawal or health).mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fx, dq, nm, kf, px, rx, ui, sy, tc, id, tm]

11.   9 and 10

12.   limit 11 to english language

13.   limit 12 to yr="1990 -Current"

Appendix B. Questionnaires within each timepoint in ALSPAC and age of child when they were completed.

W = weeks; M = months; Y = years; (G) = gestation

All questionnaires and age of child by type – mother reported (mo), father reported (fa) child based (chcb), child completed (chc), and clinic												
mo	D	B	C	E	F	G	H	J	L	N	P	S
	12W (G)	18W (G)	32W (G)	8W	8M	1.9Y	2.9Y	3.11Y	6.1Y	8.1Y	9.2Y	12.1Y
fa	PA	PB	PC	PD	PE	PF	PG	PL	PM	PN	PP	
	12W (G)	18W (G)	8W	8M	1.9Y	2.9Y	3.11Y	8.1Y	9.2Y	10.2Y	11.2Y	
chbs	KA	KG	KJ	KQ	KT	KU	KV	KW	TA	TB		
	4W	3.1Y	3.5Y	6.75Y	8.6Y	9Y	10Y	11Y	16Y			
chc	CCC	CCF	CCL	CCQ	CCR	ACES						
	8.1Y	9.7Y	12Y	13Y	14Y	18Y						
Clinic	f8	fe										
	8Y	11Y										

Questionnaires in each wave of ALSPAC data and age												
Questionnaire(s)	D, PA	B, PB	C	E, PC, KA, KZ	F, PD	G, PE	H, PF	PG, J, KG	KJ	L	KQ	N, PL, CCC	KT, f8
Age	12W (G)	18W (G)	32W (G)	8W	8M	1.9Y	2.9Y	3.11Y	3.5Y	6.1Y	6.75Y	8.1Y	8.6Y
Timepoint	1	2	3	4	5	6	7	8	9	10	11	12	13

Questionnaires in each wave of ALSPAC data and age continued								
Questionnaire(s)	KU	P, PM	CCF	KV	PP, KW, fe	S, CCL	CCQ, TA, TB	CCR	ACES

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Age	9Y	9.2Y	9.7Y	10Y	11Y	12.1Y	13Y	14Y	18Y				
Timepoint	**14**	**15**	**16**	**17**	**18**	**19**	**20**	**21**	**23**				

Questionnaires in my study waves and age of child

Questionnaire(s)	D, PA, B, PB, C	E, PC, KA, KZ, PD, F	G, PE	H, PF	P G, J, K G, K J	L, KQ	N, PL, CCC, KT, f8,	KU, P, PM, CCF	KV, PP, KW, fe	S, CCL	CCQ, TA, TB	CCR	ACES
Age	Preg	0-1Y	1Y	2 Y	3 Y	6 Y	8 Y	9 - 10 Y	10-11 Y	12 Y	13 Y	14 Y	18 Y
Timepoint	1	2	3	4	5	6	7	8	9	10	11	12	13

Appendix C. List of variables excluded from dataset for the multiple imputation

kz011b - Participant was alive at 1 year of age

chcb_preferredbw_2 – Preferred birth weight

chcb_EASemotionscorecomp_5 – EAS child emotionality score

chcb_EASemotionalitymissing_5 – EAS child emotionality score missing

chcb_EASactivityscorecomp_5 – EAS child activity score

chcb_EASactivitymissing_5 – EAS child activity score missing

chcb_EASsocialscorecomplete_5 – EAS child sociability score

chcb_EASsociabilitymissing_5 – EAS child sociability score missing

admin_mocompleted_5 – Mother completed the questionnaire

admin_ptnrcompleted_5 – Partner completed the questionnaire

admin_othercompleted_5 – Other completed the questionnaire

chcb_genderplayscoreM_5 – Male gender play score

chcb_genderplayscoreF_5 – Female gender play score

chcb_otherplayscore_5 – Other play score

chcb_emodiffscore_5 – Emotional difficulties score

chcb_conductdiffscore_5 – Conduct problems score

chcb_prosocialscomissing_6 – Prosocial score missing

chcb_hyperscoremissing_6 - Hyperactivity score missing

chcb_emosymptomsmissing_6 – Emotional symptoms score missing

chcb_conductprobsmissing_6 – Conduct problems missing

chcb_peerprobsscoremissing_6 – Peer problems missing

chcb_totaldiffscoremissing_6 – Total difficulties score

chcb_eventsscoresinc5_6 – Stressful events since 5 years score

chcb_eventssinc5missing_6 – Stressful events since 5 years score missing

chcb_anyeventssinc5years_6 – Any stressful event since 5 years

chcb_eventssinc5missing2_6 – Number of events since 5 years missing

chcb_cognitivemissing_6 – Cognitive skills missing

chcb_socialskillsmissing_6 – Social skilling missing

chcb_communicationmissing_6 – Communication skills missing

chcb_chactivitymissing_6 – Children's activity missing

chcb_maleparentingmissing_6 – Male parenting score missing

chcb_questionnairemo_7 – Mother completed the questionnaire

chcb_questionnairefa_7 – Father/partner completed the questionnaire

chcb_questionnaireother_7 – Other person completed the questionnaire

chcb_disciplineprobschool_8 – Discipline problem at school

chcb_learningprobschool_8 – Learning problem at school

chcb_relationshipprobschool_8 – Relationship problem at school

chcb_emoprobschool_8 – Emotional problem at school

chcb_otherprobschool_8 – Other problem at school

chcb_punished_8 – Child punished

chcb_runaway_8 – Child has runaway from home

chcb_afraidofsomeone_8 – Child is afraid of someone

chcb_afraidoffaorstepfa_8 – Child is afraid of stepfather

chcb_afraidofmoorstepmo_8 – Child is afraid of stepmother

chcb_afraidofbro_8 – Child is afraid of brother

chcb_afraidofsis_8 – Child is afraid of sister

chcb_afraidofotherrel_8 – Child is afraid of other relation

chcb_afraidneighbour_8 – Child is afraid of a neighbour

chcb_afraidteacher_8 – Child is afraid of teacher

chcb_afraidotherch_8 – Child is afraid of another child

chcb_afraidanother8 – Child is afraid of another person

chcb_faorstepfasticksup_8 – Father sticks up for child

chcb_mostepmosticksupforch_8 – Mother sticks up for child

chcb_brosticksupforch_8 – Brother sticks up for child

chcb_sissticksupforch_8 – Sister sticks up for child

chcb_otherrelsticksupforch_8 – Other relation sticks up for child

chcb_neighboursticksupforch_8 -Neighbour sticks up for child

chcb_teachersticksupforch_8 – Teacher sticks up for child

chcb_otherchsticksupforch_8 – Other person sticks up for child

chcb_anothersticksupforch_8 – Another person stick up for child

chcb_SMFQdepscore_8 - Depression score

chcb_SMFQdepmissing_8 – Depression score missing

chcb_SDQprosocialscore_8 - prosocial score

chcb_SDQprosocialmissing_8 – prosocial score missing

chcb_SDQemocompletecases_8 – emotional symptoms score, complete cases only

chcb_SDQemomissing_8 – emotional symptoms score, missing

chcb_SDQconductcomplete_8 – conduct problems score, complete cases only

chcb_SDQconductmissing_8 – Conduct problems score, missing

chcb_SDQpeerprobcomplete_8 – Peer problems, complete cases only

chcb_SDQpeerprobsmissing_8 – Peer problems, missing

chcb_birthweightobstadata_2 – Baby's birth weight

chcb_birthweightclinicdata_2 – Baby's birthweight from clinic

chcb_questionnairebiomo_11 – Questionnaire completed by biological mother

chcb_questionnairemofig_11 – Questionnaire completed by mother figure

chcb_questionnairebiofa_11 – Questionnaire completed by biological father

chcb_questionnaireteenager_11 – Questionnaire completed by teenager themselves

chcb_questionsomeoneelse_11 – Questionnaire completed by someone else

chcb_ADHD13yrs_11 – ADHD at 13 years

chcb_hyperkenesis13yrs_11 – Hyperkenesis at 13 years

chcb_condictdis13yrs_11 – Conduct disorder at 13 years

chcb_depression13yrs_11 – Depression at 13 years

chcb_eatingdos13yrs_11 – Eating disorder at 13 years

chcb_genanxiety13yrs_11 – General anxiety at 13 years

chcb_OCD13yrs_11 – Obsessive-compulsive disorder at 13 years

chcb_ODD13yrs_11 Opposite defiance disorder at 13 years

chcb_separationanx13yrs_11 – Separation anxiety at 13 years

chcb_separationanx13_11 – Separation anxiety at 13 years

chcb_socialphobia13yrs_11 – Social phobia at 13 years

chcb_socialphobia13_11 – Social phobia at 13 years

chcb_anydisorder13_11 – Any disorder at 13 years

chcb_anyemodisorder13_11 – Any emotional disorder at 13 years

chcb_anyanxdisorder13_11 – Any anxiety disorder at 13 years

chcb_anybehdisorder13_11 – Any behavioural disorder at 13 years

chcb_questbiomo_11 – Questionnaire completed by biological mother

chcb_questmofig_11 – Questionnaire completed by mother figure

chcb_questfafig_11 – Questionnaire completed by father figure

chcb_questbiofa_11 – Questionnaire completed by father

chcb_questteenager_11 – Questionnaire completed by teenager themselves

chcb_questsomeoneelse_11 – Questionnaire completed by someone else

chbs_nocontributers_5 – Number of people who contributed to the questionnaire

chbs_ageofchildatcomp_5 – Age of child at questionnaire completion

chbs_mocompleted_5 – Questionnaire completed by mother

chcb_ptnrcompleted_5 – Questionnaire completed by father

chcb_othercompleted_5 – Questionnaire completed by other

chcb_ageatcompletion_5 – Age of child at completion of questionnaire

chcb_mocompleted_6 – Questionnaire completed by mother

chcb_ptnrcompleted_6 – Questionnaire completed by mother's partner

chcb_othercompleted_6 – Questionnaire completed by other

chcb_ageofchildcompleted_6 – Age of child when questionnaire completed

chcb_mocompleted_8 – Questionnaire completed by mother

chcb_mofigcompleted_8 – Questionnaire completed by mother figure

chcb_facompleted_8 – Questionnaire completed by father

chcb_fafigcompleted_8 – Questionnaire completed by father figure

chcb_othercompleted_8 – Questionnaire completed by other

chcb_mocompleted_9 – Questionnaire completed by mother

chcb_mofigcompleted_9 – Questionnaire completed by mother figure

chcb_facompleted_9 – Questionnaire completed by father

chcb_mothercompleeted_9 – Questionnaire completed by mother

chcb_motherfigcompleted_9 Questionnaire completed by mother figure

chcb_fathercompleted_9 Questionnaire completed by father

chbs_agechildatcomp_11 – Age of child at completion of the questionnaire

chc_liketogotoschool_12 – Child likes to go to school

chc_likeschool_12 – Child likes school

chc_happyatschool_12 – Child is happy at school

chc_hadhelp_12 – Child had help with questionnaire

cl_locscore_7 – Locus of control score

cl_locsumscoreI_7 – Locus of control summary score

cl_locmissing_7 – Locus of control score, missing

cl_locmeanscore_7 – Locus of control mean score

cl_locmeansumscoreI_7 – Locus of control mean summary score

cl_DANVAall_missing_7 – DANVA emotion recognition score, missing

cl_DANVAlowintens_noerrors_7 – DANVA number of low intensity face errors

cl_DANVAlowint_5pluserrors_7 – DANVA low intensity faces, 5 or more errors

cl_DANVAlowintens_missing_7 – DANVA low intensity faces score, missing

cl_DANVAhighintens_errors_7 – DANVA high intensity emotion face errors

cl_DANVAhighint_3pluserror_7 – DANVA high intensity faces, 3 or more errors

cl_DANVAallhighint_missing_7 – DANVA high intensity faces score, missing

cl_DANVAmishappy_noerrors_7 – DANVA number misattributed as happy

cl_DANVAmishap_4pluserror_7 – DANVA misattributed as happy, four or more errors

cl_DANVAmisattsad_noerrors_7 – DANVA number misattributed as sad

cl_DANVAmissad_3pluserrors_7 – DANVA faces misattributed as sad, more than 3 errors

cl_DANVAmisangry_noerrors_7 – DANVA number of faces misattributed as angry

cl_DANVAmisangry_2pluserror_7 – DANVA faces misattributed as angry, two or more errors

cl_DANVAmisattfear_noerrors_7 – number of faces misattributed as fearful

cl_DANVAmisfear_2pluserror_7 – DANVA faces misattributed as fear, two or more errors

cl_overtvictim_missing_7 – Overt bullying victim, missing

cl_overtbully_missing_7 – Overt bully, missing

cl_overtbull_missing_7 – Overt bully missing

cl_relvictim_missing_7 – Relational bullying victim, missing

cl_relbully_missing_7 – Relational bully, missing

cl_relcomp_missing_7 – Relational bully victim or perpetrator, missing

cl_nofriends_9 – Number of friends child has

cl_friendsotherplaces_9 – Child goes with friends to other places

cl_friendsotherthing_9 – Child does other things with friends

cl_MVPAnovalidweekdays_9 – Physical activity, number of valid weekdays

cl_MVPAlightminsalldays_9 – Number of light minutes of physical activity, all days included

cl_MVPAtotalsedminssweekday_9 – Total sedentary minutes on weekdays

cl_MVPAtotalcountweekdays_9 – Total number of weekdays

cl_MVPAnominswornvaliddays_9 – Number of minutes activity watch worn during study period

chc_agechildatquestmonths_7 – Age of child when questionnaire completed

chc_agechildatcompmonths_8 – Age of child when questionnaire completed in months

chc_agechildatcompmonths_10 – Age of child at completion in months

chc_agechildatcompmonths_11 – Age of child when questionnaire completed in months

chc_agechildatcompmonths_12 – Age of child when questionnaire completed in months

cl_childageatcl_8 – Age of child when they attended the clinic

 cl_childnewcaseF8 – Was the child a new study case at the clinic?

cl_childsfirstcontactF8 – Was the clinic the child's first contact with the study?

cl_ageinmonthsF11_9 – Age of child in months when they attended the clinic

cl_newcase_9 - Was the child a new case in the study?

IncoreALSPACsample – Was the child in the core ALSPAC sample?

Pregnancysizesummary

Survivorsat28days – was the study child alive at 28 days?

Survivorsat1year – was the child alive at one year?

Groupedageofmotheratdelivery – age group of mother when baby born

mo_cigsperday_1 – number of cigerettes mother smoked per day in pregnancy

mo_highestedqual_1 – Mother's highest education qualification

mo_partneredqual_1 – Partner's highest education qualification

mo_aggscoremissing_1 – Relationship aggression/ parental conflict score

mo_aggscorenomissing_1 – Relationship aggression/parental conflict score, missing

mo_CCEIanxietymissing_2 – Mother's anxiety score, missing

mo_CCEIsomaticmissing_2 – Mother's somatic symptoms score, missing

mo_CCEIdepmissing_2 – Mother's depression symptoms score, missing

mo_CCEImissing_2 – Mother's total CCEI score, missing

mo_EPDSmissing_2 – Mother's postnatal depression score, missing

mo_tookotherdrug2overoneyr_5 – Mother took 2nd other drug over the past year

mo_tookotherdrug3overoneyr_5 – Mother took a 3rd drug over the past year

mo_statescoreSTAI_7 – Mother's state anxiety score

mo_traitscoreSTAImissing_7 – Mother's trait anxiety score, missing

mo_alcintakenormalwk_7 – Mother's alcohol intake on a normal week

mo_sulks_8 – Mother sulks

mo_peopletalktroubles_8 – Mother has people she can talk to about her troubles

mo_moretiredthanothers_8 – Mother is more tired than others

mo_sadnotangrywhenscoulded_8 – Mother is sad rather than angry when she is scoulded

mo_agechildatcompmths_2 – Age of child when questionnaire completed in months

mo_personcompleted_2 – Person who completed the questionnaire

mo_mocompleted_3 – Mother completed the questionnaire

mo_ptnrcompleted_3 – Partner completed the questionnaire

mo_othercompleted_3 – Other person completed the questionnaire

mo_ageofchildatcomp_3 – Age of child when questionnaire was completed

mo_mocompleted_4 – Mother completed the questionnaire

mo_ptnrcompleted_4 – Partner completed the questionnaire

mo_othercompleted_4 – Other person completed the questionnaire

mo_nocontributers_4 – Number of people who contributed to the questionnaire

mo_ageofchildatcomp_4 – Age of child when the questionnaire was completed

mo_mocompleted_5 – Mother completed the questionnaire

mo_ptnercompleted_5 – Partner completed the questionnaire

mo_othercompleted_5 – Other person completed the questionnaire

mo_nocontributers_5 – Number of people who contributed to the questionnaire

mo_ageofchildatcomp_5 – Age of child when questionnaire was completed

mo_ptnrcompleted_6 – Partner completed the questionnaire

mo_othercompleted_6 – Other person completed the questionnaire

mo_nocontributers_6 – Number of people who contributed to the questionnaire

mo_agechildatcomp_6 – Age of child when questionnaire was completed

mo_mofigcompleted_7 – Mother figure completed the questionnaire

mo_othercompleted_7 – Other person completed the questionnaire

mo_nocontributers_7 – Number of people who contributed to the questionnaire

mo_agechildatcomp_7 – Age of child when questionnaire completed

mo_mofigcompleted_8 – Mother figure completed the questionnaire

mo_othercompleted_8 – Other person completed the questionnaire

mo_nocontributers_8 – Number of people who contributed to the questionnaire

mo_agechildatcomp_8 – Age of child when questionnaire completed

mo_mofigcomp_10 – Mother figure completed the questionnaire

mo_othercomp_10 – Other person completed the questionnaire

mo_nocontributors_10 – Number of people who contributed to the questionnaire

mo_agechildatcomp_10 – Age of child when questionnaire was completed

fa_CCEIanxsubscaleI_2 – Father's anxiety subscale score

fa_CCEIanxmissing_2 – Father's anxiety score, missing

fa_CCEIsomaticsubscaleI_2 – Father's somatic symptoms subscale score, missing

fa_CCEIsomaticmissing_2 – Father's somatic symptoms score, missing

fa_CCEIdepsubscaleI_2 – Father's depression symptoms subscale score

fa_CCEIdepmissing_2 – Father's depression symptoms score, missing

fa_CCEItotalI_2 – Father's total internalising symptoms score 2

fa_CCEItotalmissing_2 - Father's total internalising symptoms score, missing

fa_EPDSscoreI_2 – Father's postnatal depression score 2

fa_EPDSmissing_2 - Father's postnatal depression score, missing

fa_majorfinancprobs_2 – Major financial problems

PAdaydata – Unknown ALSPAC administrative variable

PAmthdata – Unknown ALSPAC administrative variable

PAyrdata – Unknown ALSPAC administrative variable

PBdaydata – Unknown ALSPAC administrative variable

PBmthdata – Unknown ALSPAC administrative variable

PByrdata – Unknown ALSPAC administrative variable

PCdaydata – Unknown ALSPAC administrative variable

PCmthdata – Unknown ALSPAC administrative variable

PCyrdata – Unknown ALSPAC administrative variable

PDdaydata – Unknown ALSPAC administrative variable

PDmthdata – Unknown ALSPAC administrative variable

PDyrdata – Unknown ALSPAC administrative variable

PEmthdata – Unknown ALSPAC administrative variable

PEdaydata – Unknown ALSPAC administrative variable

PFmthdata – Unknown ALSPAC administrative variable

PFyrdata – Unknown ALSPAC administrative variable

PGdaydata – Unknown ALSPAC administrative variable

PGmthdata – Unknown ALSPAC administrative variable

PGyrdata – Unknown ALSPAC administrative variable

PMdaydata – Unknown ALSPAC administrative variable

PMmthdata – Unknown ALSPAC administrative variable

PMyrdata – Unknown ALSPAC administrative variable

PPdaydata – Unknown ALSPAC administrative variable

PPmthdata – Unknown ALSPAC administrative variable

PPyrdata – Unknown ALSPAC administrative variable

PAdobdat – Unknown ALSPAC administrative variable a

PBdobdata – Unknown ALSPAC administrative variable

PCdobdata – Unknown ALSPAC administrative variable

PDdobdata – Unknown ALSPAC administrative variable

PEdobdata – Unknown ALSPAC administrative variable

PFdobdata – Unknown ALSPAC administrative variable

PGdobdata – Unknown ALSPAC administrative variable

PMdobdata – Unknown ALSPAC administrative variable

PPdobdata – Unknown ALSPAC administrative variable

PEyrdata – Unknown ALSPAC administrative variable

PFdaydata – Unknown ALSPAC administrative variable

matchDOB_B3352 – Unknown ALSPAC matching variable

matchDOB2_B3352 – Unknown ALSPAC matching variable

matchday_B3352 – Unknown ALSPAC matching variable

matchday2_B3352 – Unknown ALSPAC matching variable

matchmth_B3352 – Unknown ALSPAC matching variable

matchmth2_B3352 – Unknown ALSPAC matching variable

matchyr_B3352 – Unknown ALSPAC matching variable

matchyr2_B3352 – Unknown ALSPAC matching variable

fa_gestoncomp_1 – Baby's gestation when questionnaire completed

fa_babyageifcomppostnatal_1 – Baby's age if questionnaire completed after birth

fa_babyageatcompwks_2 – Baby's age in weeks when questionnaire completed

fa_agechildatcomp_2 – Child's age when questionnaire completed

fa_personcompleted_2 Person who completed the questionnaire

fa_mocompleted_3 – Mother completed the questionnaire

fa_ptnercompleted_3 – Partner completed the questionnaire

fa_othercompleted_3 – Other person completed the questionnaire

fa_agechildatcomp_3 – Age of child when questionnaire completed

fa_mocompleted_4 – Mother completed questionnaire

fa_facompleted_4 – Father completed questionnaire

fa_othercompleted_4 – other person completed questionnaire

fa_nocontributers_4 - Number of people who contributed to the questionnaire

fa_fathercompleted_5 – Father completed questionnaire

fa_mocompleted_5 – Mother completed questionnaire

fa_othercompleted_5 – Other person completed questionnaire

fa_nocontributers_5 – Number of people who contributed to the questionnaire

fa_fathercomp_7 – Father completed the questionnaire

fa_fafigcomp_7 – Father figure completed the questionnaire

fa_othercomp_7 – Other person completed the questionnaire

fa_nocontributors_7 – Number of people who contributed to the questionnaire

fa_facomp_8 – Father completed the questionnaire

fa_fafigcomp_8 – Father figure completed the questionnaire

fa_othercomp_8 – Other person completed the questionnaire

fa_nocontributors_8 – Number of people who contributed to the questionnaire

fa_facomp_9 – Father completed the questionnaire

fa_fafigcomp_9 – Father figure completed the questionnaire

fa_othercomp_9 – Other person completed the questionnaire

fa_nocontributors_9 – Number of people who contributed to the questionnaire

fa_datecompquestmonth_9 – Month questionnaire completed

fa_datecompquestyear_9 – Year questionnaire completed

Appendix D. Flow diagram of the ALSPAC cohort

```
┌─────────────────────────────┐
│  Cases in the original      │
│  dataset                    │
│  (n = 11,613)               │
└─────────────────────────────┘
           │                          ┌─────────────────────────────┐
           │─────────────────────────▶│  Records excluded that      │
           │                          │  were not in original       │
           ▼                          │  cohort                     │
┌─────────────────────────────┐       │  (n = 712)                  │
│  Cases in the original      │       └─────────────────────────────┘
│  cohort                     │
│  (n=10,901)                 │
└─────────────────────────────┘
           │                          ┌─────────────────────────────┐
           │─────────────────────────▶│  Children excluded who      │
           │                          │  died before their 1st      │
           ▼                          │  birthday                   │
┌─────────────────────────────┐       │  (n = 496)                  │
│  Cases alive at one year    │       └─────────────────────────────┘
│  (n = 10,405)               │
│                             │
└─────────────────────────────┘
           │                          ┌─────────────────────────────┐
           │─────────────────────────▶│  Children excluded as       │
           │                          │  family was not in wave 1   │
           ▼                          │  or wave 2 (n = 202)        │
┌─────────────────────────────┐       └─────────────────────────────┘
│  Cases present in wave 1    │
│  or wave 2                  │
│  (n = 10,203)               │
│                             │
└─────────────────────────────┘
           │                          ┌─────────────────────────────┐
           │─────────────────────────▶│  Families in the armed      │
           │                          │  forces (n = 21)            │
           ▼                          └─────────────────────────────┘
┌─────────────────────────────┐
│  Cases in final study       │
│  cohort                     │
│  (n = 10,182)               │
└─────────────────────────────┘
```

Appendix E. Multiple imputation code

mi impute chained (regress) o_* (regress, omit(i.mo_* i.mofa_* i.fa_* i.cl_* c_mo_* c_fa_* c_cl_* i.e_*)) c_chcb_* (regress, omit(i.mo_* i.mofa_* i.fa_* i.cl_* c_mo_* c_fa_* c_cl_* i.e_*)) c_chc_* (regress, omit(i.mo_* i.mofa_* i.fa_* i.chcb_* c_mo_* c_fa_* i.e_*)) c_cl_* (regress, omit(i.chcb_* i.fa_* i.cl_* c_chcb_* c_fa_* c_cl_* i.e_*)) c_mo_* (regress, omit(i.mo_* i.mofa_* i.chcb_* i.cl_* c_mo_* c_chcb_* c_cl_* i.e_*)) c_fa_* (mlogit, omit(i.fa_* i.chcb_* i.cl_* c_chcb_* c_cl_* c_fa_*)) mo_* (mlogit, omit(i.fa_* i.chcb_* i.cl_* c_chcb_* c_cl_* c_fa_*)) mofa_* (mlogit, omit(i.chcb_* i.cl_* c_chcb_* c_cl_* c_mo_* i.mo_*)) fa_* (mlogit, omit(i.mo_* i.mofa_* i.fa_* i.cl_* c_mo_* c_fa_* c_cl_*)) chcb_* (mlogit, omit(i.mo_* i.mofa_* i.fa_* i.chcb_* c_mo_* c_fa_* c_chcb_*)) cl_* (mlogit, omit(i.chcb_* i.cl_* c_chcb_* c_cl_* c_fa_* i.mofa_* c_mo_*)) e_* , augment noisily add(10) burnin(10) rseed(190121)

Appendix F. Total effect of DV with post-processing

Total effect of early DV on internalising symptoms at 6 years

use "C:\Users\betha\OneDrive\Documents\PhD\ALSPAC\Data\MI and IPW\ALSPAC_imputed_data_weights_250121.dta", clear

capture program drop PA_comparison_SQ_vs_noDV

program define PA_comparison_SQ_vs_noDV, rclass

capture drop mybs_*

nbreg o_internalising_6 i.ANYDV0_3 i.e_mofa_directchabuse0_3 i.age_mo_matageatdel_1 i.anydvpreg i.mo_highestedqual1_1 i.eth_mo_ethnicegrp_1 c_mo_CCEI_2 c_fa_CCEItotalII_2 c_mo_EPDS_2 c_fa_EPDSscoreII_2 i.mo_crowding_4 i.fa_majorfinancprobsyn_2 c_mo_finandiff2_2 i.mo_maritalstatus_4 i.mo_matsocialclass_1 i.mo_patsocialclass_1 c_mo_finandiffsinc18mths_4 i.mo_famincomeperwk_5 i.fa_assptnrshealth_4 i.mo_anxoveroneyear_5 i.mo_depoveroneyear_5 c_mo_conductprobsrecoded_5 i.mo_ptnrdepoveroneyear_5 i.mo_ptnranxoveroneyear_5 c_mo_weightedlifeevents_1 [pw = prw6]

predict mybs_lp, xb

gen mybs_alph=e(alpha)

gen mybs_lp0=mybs_lp-_b[1.ANYDV0_3]*ANYDV0_3

gen mybs_exp_lp=exp(mybs_lp)

regress mybs_exp_lp [pw=prw5]

local mY =_b[_cons]

gen mybs_exp_lp0=exp(mybs_lp0)

regress mybs_exp_lp0 [pw=prw5]

local mY0 =_b[_cons]

return scalar mY=`mY'

return scalar mY0=`mY0'

return scalar m_diff=`mY'-`mY0'

end

```
save full_MI_dataset2, replace

forvalues j=1(1)10 {

        drop if _mi_m!=`j'

                                *note that the following line changes to reflect the
new parameters:

        bootstrap r(mY) r(mY0) r(m_diff), reps(100): PA_comparison_SQ_vs_noDV

        mat b`j'=e(b)

        mat V`j'=e(V)

        use full_MI_dataset2, clear

}

mat sum_b=b1

mat sum_V=V1

forvalues j=2(1)10 {

                mat sum_b=sum_b+b`j'

                mat sum_V=sum_V+V`j'

}

mat mean_b=sum_b/10

mat mean_V=sum_V/10

*We have 7 parameters now rather than the 3 we had before (because we have the 3 in the
males, the same 3 in the females, and the difference between the two differences)

*So all the final lines need new versions for parameters 4-7:

mat define between_V=(0,0,0)

forvalues j=1(1)10 {

                mat between_V[1,1]=between_V[1,1]+(b`j'[1,1])^2

                mat between_V[1,2]=between_V[1,2]+(b`j'[1,2])^2

                mat between_V[1,3]=between_V[1,3]+(b`j'[1,3])^2

}

mat between_V=between_V/10

mat between_V[1,1]=between_V[1,1]-(mean_b[1,1])^2

mat between_V[1,2]=between_V[1,2]-(mean_b[1,2])^2

mat between_V[1,3]=between_V[1,3]-(mean_b[1,3])^2
```

```
mat define Rubin_V=(0,0,0)

mat Rubin_V[1,1]=mean_V[1,1]+(6/4)*between_V[1,1]

mat Rubin_V[1,2]=mean_V[2,2]+(6/4)*between_V[1,2]

mat Rubin_V[1,3]=mean_V[3,3]+(6/4)*between_V[1,3]


***The estimate and SE for the mean in the real world:

di mean_b[1,1]

di sqrt(Rubin_V[1,1])

***The estimate and SE for the mean in the hypothetical world is:

di mean_b[1,2]

di sqrt(Rubin_V[1,2])

***The estimate and SE for the difference between these means is:

di mean_b[1,3]

di sqrt(Rubin_V[1,3])

***The p-value for the effect of DV is:

di 2*(1-normal(abs(mean_b[1,3])/sqrt(Rubin_V[1,3])))
```

Appendix G. Proportion of data missing in key variables

Variable description	N (%) missing
Domestic violence at 0-3 years	6,040 (59.32)
Direct child abuse at 0-3 years	7,084 (69.57)
Direct child abuse 6-9 years	7,574 (74.39)
SDQ emotional symptoms at 6 years	3,902 (38.32)
SDQ peer problems at 6 years	3,900 (38.30)
SDQ emotional symptoms at 13 years	5,179 (50.86)
SDQ peer problems at 13 years	5,183 (50.90)

Appendix H. Direct child abuse (DCA) at 0-3 years and 6-9 years by key covariates in the original dataset and the first of the 10 imputed datasets

Variable	Original dataset (before imputation)		First (of ten) imputed datasets	
	DCA at 0-3 years (n (%))	No DCA at 0-3 years (n (%))	DCA at 6-9 years (n (%))	No DCA exposure at 6-9 years (n (%))
N	872	9,310	749	9,433
Sex				
Male	475 (54.47)	4,805 (51.61)	400 (53.40)	4,880 (51.73)
Female	397 (45.53)	4,505 (48.39)	349 (46.60)	4,553 (48.27)
Maternal age at birth				
<20	23 (2.64)	439 (4.72)	30 (4.01)	432 (4.58)
20–24	113 (12.96)	1,798 (19.31)	134 (17.89)	1,777 (18.84)
25–29	302 (34.63)	3,633 (39.02)	288 (38.45)	3,647 (38.66)
30–34	300 (34.40)	2,541 (27.29)	210 (28.04)	2,631 (27.89)
>=35	134 (15.37)	899 (9.66)	87 (11.62)	946 (10.03)
Marital status				
Not married	111 (12.73)	1,209 (12.99)	144 (19.23)	1,004 (10.64)
Separated/divorced/widowed	123 (14.11)	657 (7.06)	147 (19.63)	592 (6.28)
Married	638 (73.17)	7,444 (79.96)	458 (61.15)	6,882 (72.96)
Missing	0	0	0	955 (10.12)
Family income per week (mother report)				
<£100	110 (12.61)	887 (9.53)	99 (13.22)	784 (8.31)
£100-£199	196 (22.48)	1,688 (18.13)	214 (28.57)	1,492 (15.82)
£200-£299	237 (27.18)	2,773 (29.79)	160 (21.36)	2,507 (26.58)
£300-£399	148 (16.97)	1,933 (20.76)	133 (17.76)	1,764 (18.70)
>=£400	181 (20.76)	2,029 (21.79)	143 (19.09)	1,931 (20.47)
				955 (10.12)
Maternal social class				
I & II	111 (12.73)	1,209 (12.99)	262 (35.12)	2,295 (39.28)
III	123 (14.11)	657 (7.06)	348 (46.65)	2,941 (50.34)
IV & V	638 (73.17)	7,444 (79.96)	136 (18.23)	606 (10.37)
Child ethnicity				
White	791 (90.71)	7,747 (83.21)	646 (86.60)	5,430 (92.95)
Non-white	48 (5.50)	395 (4.24)	45 (6.03)	193 (3.30)
Unknown	33 (3.78)	1,168 (12.55)	55 (7.37)	219 (3.75)
Maternal highest qualification				
Low	525 (60.21)	6,101 (65.53)	473 (63.40)	3,446 (58.99)
Medium	222 (25.46)	2,026 (21.76)	183 (24.53)	1,478 (25.30)
High	125 (14.33)	1,183 (12.71)	90 (12.06)	918 (15.71)
Household crowding index at 2 years				
<= 0.5	122 (13.99)	1,712 (18.39)	122 (16.35)	1,126 (19.27)
>0.5 - 0.75	316 (36.24)	3,449 (37.05)	287 (38.47)	2,225 (38.09)

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>0.75 - 1	354 (40.60)	3,356 (36.05)	262 (35.12)	2,092 (35.81)
> 1	80 (9.17)	793 (8.52)	75 (10.05)	399 (6.83)
SDQ Internalising symptoms at 6 years				
0	138 (15.83)	2,365 (25.40)	127 (17.02)	1,370 (23.45)
1	126 (14.45)	1,695 (18.21)	130 (17.43)	1,220 (20.88)
2	128 (14.68)	1,421 (15.26)	122 (16.35)	954 (16.33)
3	107 (12.27)	1,176 (12.63)	89 (11.93)	746 (12.77)
4	105 (12.04)	915 (9.83)	89 (11.93)	498 (8.52)
5	69 (7.91)	627 (6.73)	55 (7.37)	358 (6.13)
6	65 (7.45)	435 (4.67)	51 (6.84)	251 (4.30)
7	39 (4.47)	264 (2.84)	26 (3.49)	161 (2.76)
8	31 (3.56)	174 (1.87)	20 (2.68)	110 (1.88)
9	23 (2.64)	93 (1.00)	9 (1.21)	62 (1.06)
10	15 (1.72)	73 (0.78)	12 (1.61)	51 (0.87)
11	13 (1.49)	25 (0.27)	<5	25 (0.43)
12	6 (0.69)	20 (0.21)	6 (0.80)	16 (0.27)
13	<5	15 (0.16)	<5	10 (0.17)
14	<5	6 (0.06)	<5	<5
15	<5	<5	<5	5 (0.09)
16	0	<5	<5	<5
17	0	<5	0	0
18	0	0	0	0
19	0	0	0	0
20	0	0	0	0
SDQ Internalizing symptoms at 13 years				
0	291 (10.15)	1,145 (15.75)	107 (16.61)	1,329 (25.21)
1	258 (9.00)	890 (12.24)	106 (16.46)	1,042 (19.76)
2	203 (7.08)	742 (10.20)	91 (14.13)	854 (16.20)
3	151 (5.27)	546 (7.51)	76 (11.80)	621 (11.78)
4	153 (5.34)	378 (5.20)	70 (10.87)	461 (8.74)
5	121 (4.22)	265 (3.64)	56 (8.70)	330 (6.26)
6	87	164 (2.26)	39 (6.06)	212 (4.02)
7	(3.03)	116 (1.60)	21 (3.26)	149 (2.83)
8	54 (1.88)	83 (1.14)	25 (3.88)	100 (1.90)
9	42 (1.46)	43 (0.59)	15 (2.33)	54 (1.02)
10	26 (0.91)	42 (0.58)	9 (1.40)	47 (0.89)
11	14 (0.49)	21 (0.29)	6 (0.93)	27 (0.51)
12	12 (0.42)	14 (0.19)	5 (0.78)	18 (0.34)
13	9 (0.31)	13 (0.18)	7 (1.09)	13 (0.25)
14	7 (0.24)	10 (0.14)	<5	10 (0.19)
15	<5	<5	<5	<5
16	<5	<5	<5	<5
17	<5	<5	0	<5
18	0	<5	0	<5
19	0	0	0	0
20	0	0	0	0

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