Childhood and Adolescent Anxiety, Depression and Trauma Symptoms: The Role of the Parent-Child Relationship and Family Context Factors

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Thesis declaration form

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

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Date: 22nd June 2018

Overview

The overall focus of the thesis is the role of parent-child relationship factors and family context factors and their relationship to childhood and adolescent mental health, specifically depression, anxiety and trauma symptoms.

Part one is a meta-analysis exploring the association between witnessing intimate partner violence and trauma symptoms in children and adolescents. The meta-analysis reviewed 49 studies and considered whether the magnitude of the association varied across a number of moderator variables.

Part two of the thesis reports findings from a longitudinal study designed to explore how parent-child relationship factors, namely parenting sensitivity and attachment, affected the development of child and adolescent depression and anxiety symptoms using archived data from the NICHD Study of Early Child Care and Youth Development. Latent growth curve modelling was used to examine the association between these parent-child relationship factors and the development of child and adolescent depression and anxiety symptoms. In addition, the study examined whether family context factors moderated this relationship, namely, parental depression and anxiety, negative life events and the interparental relationship.

Part three is a critical appraisal of the research process which: situated the researcher's context within the area; explored the benefits and challenges of working with a large archived dataset; considered the arbitrary classification of the p-value and its contribution when interpreting results; and reflected on the implications of the study findings for future research and clinical practice.

Impact Statement

Worldwide 15.5 million children lived in households with intimate partner violence (IPV) in the past year. Furthermore, research suggests that witnessing IPV is associated with childhood and adolescent trauma symptoms. The current study was the first to conduct a meta-analysis examining the association between witnessing IPV and childhood and adolescent trauma symptoms as an individual outcome and its relation to moderators. An association between IPV and childhood and adolescent trauma symptoms was found which remained consistent across a range of moderators.

Given that depression and anxiety symptoms are relatively common during childhood and adolescence, with estimated incidence rates of up to five percent, this study secondly examined the role that parent-child relationship factors, namely attachment and parenting sensitivity, and family context factors, parental mental health, negative life events and the interparental relationship, played in the development of childhood and adolescent anxiety and depression symptoms. Findings showed that both parent-child relationship factors and family context factors were associated with childhood and adolescent anxiety and depression symptoms.

This research has implications for many stakeholders including children and their families experiencing anxiety, depression or trauma symptoms; clinicians working with these families; commissioners guiding service development; researchers and those responsible for policy guidelines. This research aims to deliver this impact by disseminating this thesis' findings, ultimately aiming to improve outcomes.

Specifically, the findings have implications regarding supporting interventions aimed at improving parenting sensitivity and strengthening attachment security. Moreover, the findings suggest providing attachment and parenting sensitivity interventions beyond infancy.

Findings also suggest that for mothers experiencing anxiety symptoms, delivering interventions that aim to increase parenting sensitivity could be beneficial. Additionally, findings suggest that children experiencing depression symptoms who also experience greater parenting sensitivity could benefit more from interparental relationship interventions when needed whereas children experiencing less sensitive parenting may benefit more from concentration on parenting sensitivity first.

Clinical implications regarding the findings for both the interparental relationship and IPV may include integrating a relationship component into existing parenting provision and transforming the culture to viewing the interparental relationship as a key integrative piece of treatment. This could involve skilling up the current workforce, more integrated multi-agency working and the provision of key professionals within existing clinical teams.

The findings also suggest supporting parental mental health interventions where needed. It may be that integrating parental and child mental health interventions could improve outcomes, reduce service duplication and increase service accessibility.

The findings also emphasise the role of negative life events in children's mental health. Implications could include incorporating negative life events routinely into formulations and signposting to community resources to help reduce their impact on interventions.

Overall these findings suggest utilising a family-wide approach and including multiple domains of the family environment alongside the parent-child relationship when supporting children and adolescents with depression, anxiety and trauma symptoms. Furthermore, embedding interparental relationship support, parental mental health provision and formulation of negative life events within the heart of services aimed at preventing and treating child and adolescent depression, anxiety and trauma symptoms should take precedence.

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Part 1: Literature Review

Children's Exposure to Intimate Partner Violence: A Meta-Analysis of

Childhood and Adolescent Trauma Symptoms.

Abstract

Aims: Previous research has investigated the association between children's exposure to intimate partner violence (IPV) and child and adolescent outcomes. This meta-analysis was the first to examine associations between children and adolescent's exposure to IPV and trauma symptoms and to consider whether the magnitude of the association varies across a number of moderator variables.

Method: This study reviewed 49 studies between 1990 and 2017.

Results: Results indicated a medium effect size where $r = .26$, 95% CI [.220, .299]. Moderator analyses revealed no significant effects for gender, age, sample type, conceptualisation of IPV (broader vs narrower conceptualisation), whose IPV was measured (mother only or both partners), IPV measure, validity of trauma symptom measure, who reported the child's trauma symptoms, whether the same reporter responded to both IPV and trauma symptom measures, and whether the IPV was measured across the lifetime or within the past year.

Conclusions: These results have both research and clinical implications when working with children and their families who experience intimate partner violence.

Introduction

It is estimated that around 15.5 million children worldwide reside in two-parent households where intimate partner violence (IPV) has happened in the last year (McDonald, Jouriles, Ramisetty- Mikler, Caetano, & Green, 2006), and about 16% of children have witnessed intimate partner violence (IPV) at least once in their lifetime (Finkelhor, Turner, Shattuck, & Hamby, 2015). In the UK, around one in five children have been exposed to domestic abuse (Radford et al., 2011). Despite this high prevalence of children exposed to intimate partner violence, the first empirical studies were not published until the early 1980's (Porter & O'Leary, 1980; Straus, Gelles & Steinmetz, 1980). Fantuzzo and Lindquist's (1989) qualitative review of this literature highlighted inconsistencies and methodological limitations. This included: poor operationalisation and definition of the types of violence children were exposed to; the use of unstandardised measures of IPV; and moderating variables such as age and gender not being included in the analysis (Fantuzzo & Lindquest, 1989). A second generation of research, primarily published since 1990, followed. These studies aimed to address the methodological concerns of their predecessors and utilised different research designs and assessed for both mediating and moderating variables.

Meta-analyses suggest that children's exposure to IPV is associated with a variety of negative adjustment outcomes, with effect sizes ranging between small to medium (Evans, Davies, & DiLillo, 2008; Kitzmann, Gaylord, Holt, & Kenny, 2003; Wolfe, Crooks, Lee, McIntyre-Smith, & Jaffe, 2003). These reviews have concentrated primarily on internalising and externalising outcomes. However, exposure to intimate partner violence may also lead to trauma symptoms including intrusive re-

experiencing of the events in flashbacks or dreams, hyperarousal and emotional withdrawal (Graham-Bermann & Levendosky, 1998; Kilpatrick & Williams, 1998; Lehmann, 1997; Rossman, 1998; Vickerman & Margolin, 2007). Further evidence has suggested that children who have been exposed to IPV have higher scores on posttraumatic stress disorder (PTSD) scales (Rossman, 1998) and frequently meet diagnostic criteria for PTSD (Kilpatrick & Williams, 1998). Remarkably, despite these indications, currently, only one meta-analysis has examined this relationship as part of a wider analysis of child outcomes (Evans et al., 2008).

Important questions about links between children's exposure to IPV and trauma symptomatology remain unanswered. For example, it is not clear what role moderators may play in this relationship. Since the publication of this prior meta-analysis, there has been an accumulation of research interested in the association with trauma symptoms. This may help address these questions and generate a further understanding of how children's trauma symptoms may be related to exposure to IPV. The current study, therefore, aimed to assess the strength of the association between IPV and child trauma symptoms in light of more recent evidence. Furthermore, it aimed to consider a number of sample and measurement characteristics as moderators of the outcomes of exposure to intimate partner violence with respect to children's traumatic stress reactions.

Sample Characteristics as Moderators of the Outcomes of Exposure to IPV

Previous reviews suggest that IPV exposure may impact boys and girls differently. In general, research suggests that boys show more externalising behaviours following IPV exposure than girls. However, no difference has been noted for internalising behaviours (Evans et al., 2008; Wolfe et al., 2003). At present, no meta-

analysis has considered the role of gender as a moderator of childhood trauma symptomatology from exposure to intimate partner violence.

Researchers have proposed that the effects of exposure to IPV may be expressed differently in children of different developmental stages, however, no clear pattern of symptoms is evident (Margolin, 1998). Previous studies have suggested that younger, rather than older, children are at particularly high risk for exposure to IPV (Fantuzzo, Boruch, Beriama, Atkins, & Marcus, 1997). Other suggestions have been put forward that as younger children: have less access to social influences outside of the family, such as friends; have less developed problem-solving and coping skills; and are more likely to be home when IPV may happen; they, therefore, may be more likely to come into contact with IPV. The organisational model of development proposes that early experiences shape subsequent adaptations that may moderate or exacerbate the impact of later events (Lamb, Gaensbauer, Malkin, & Schultz, 1985; Sroufe, 1979; Sroufe, Carlson, Levy, & Egeland, 1999). Following this, IPV exposure early in life, when emotional regulation strategies are developing and children's identification with their parents is strongest, may mean stronger and longer term negative effects on adjustment outcomes than for those exposed to IPV later in childhood (Sternberg, Lamb, Guterman & Abbott, 2006). In line with this, longitudinal research has found some evidence that younger children were more vulnerable to the effects of IPV than teenagers but that for at least some of the children who experience IPV earlier in childhood this might not continue through to adolescence (Sternberg et al., 2006). Changes in family and parenting practices, in addition to children's ability to appraise and cope with IPV, may help in mitigating the negative consequences of IPV.

Another factor to consider is that these associations may reflect changes over time in the types of IPV the children experienced. Still, with few longitudinal studies, it is difficult to determine how the effects of IPV may vary as a child develops. Previous meta-analyses have found no difference for internalising outcomes for children across the age range (Evans et al., 2008; Kitzmann et al., 2003; Vu, Jouriles, McDonald & Rosenfield, 2016; Wolfe et al., 2003). At present, no other meta-analysis has considered the role of age as a moderator of childhood trauma symptomatology from exposure to intimate partner violence.

Previous research has shown that IPV is more frequent and severe in help-seeking populations than in general community populations (Johnson, 1995). Furthermore, for these families with more frequent and severe IPV, they more often experience multiple other risk factors for children's outcomes, such as child abuse (Jouriles, McDonald, Slep, Heyman, & Garrido, 2008) which may then interact and potentiate adverse outcomes (Evans, Li, & Whipple, 2013). In light of these findings, the current review examines the nature of the sample studied - for example whether it was an intimate partner violence shelter sample, community sample or clinical sample, as these may influence the extent to which post-traumatic symptoms are observed at elevated rates amongst affected children.

Measurement Characteristics as Moderators of the Outcomes of Exposure to IPV

Following Vu et al.'s (2016) study, this meta-analysis aimed to explore how the conceptualisation and operationalisation of IPV may influence IPV's association with child trauma symptomatology. In most of the literature, IPV is conceptualised as acts of physical aggression (Evans et al., 2008; Kitzmann et al., 2003). However,

in more recent times researchers have started broadening this conceptualisation of IPV to also include psychological and/or sexual IPV (Huang, Wang, & Warrener, 2010; Jouriles, McDonald, Vu, & Sargent, 2016; Schnurr & Lohman, 2013; Zarling et al., 2013). A broader conceptualisation of IPV may more validly capture the whole picture regarding IPV exposure in children. Given that there is a possibility that children's exposure to psychological and/or sexual IPV in itself may lead to negative adjustment outcomes, these may be missed when a narrower conceptualisation of IPV focused on physical violence alone is used and may mean that the real impact of IPV exposure on children is underestimated. Research suggests that including both physical and psychological aggression when measuring IPV strengthens the prediction of adverse child outcomes, compared to when only physical IPV is assessed (Jouriles, Norwood, McDonald, Vincent & Mahoney, 1996). Other research has shown that children exposed to more types of IPV had more internalising and externalising symptoms rated by teacher reports (Lamers-Winkelman, Willemen & Visser, 2012). Furthermore, it has been suggested that broader conceptualisations of IPV may capture how these other forms of abuse may also lead to an increased risk for parental psychological problems which in turn may affect children's outcomes (Jouriles et al., 2016). This review evaluated whether this is also true for trauma symptomatology.

A second measurement characteristic that will be explored in this meta-analysis is whose violence is assessed: only that of the mother's intimate partner, or both the mother's and the intimate partner's violence. The majority of studies in the literature examine the violence perpetrated by the mother's intimate partner alone. It is likely that this bias in the research stems from much of the first-generation research into intimate partner violence which largely focused on male perpetrated violence

towards women and societal attitudes towards intimate partner violence at the time, where female perpetrators of IPV were less considered and subsequently so too were the effects these have on child outcomes. Despite this, studies have suggested that assessing both the mother's and her intimate partner's IPV improves the prediction of child outcomes compared to when only the mother's intimate partner's IPV is assessed (McDonald, Jouriles, Tart, & Minze, 2009). By narrowing research to concentrating only on violence against the mother, this may mean other witnessed violence within the household, which may also be crucial to child outcomes, is lost. This may lead to an underestimation of the level of IPV exposure that children witness and paint a less valid picture of its true impact.

Related to these measurement considerations, a third characteristic that this meta-analysis will consider is how children's exposure to IPV has been assessed. The Conflict Tactics Scales (CTS) and versions thereof (Straus, 1979; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) may be considered the most well-validated and most frequently used measure. Other studies have used measures of IPV (e.g., unstructured interviews or a couple of questions assessing violence) that have not been well-validated and this may have affected the subsequent findings regarding the association between IPV exposure and child adverse outcomes. Previous meta-analyses have found that studies of IPV employing the CTS produce larger effect sizes than studies employing other measures of child outcomes (Kitzmann et al., 2003; Vu et al., 2016). One possible explanation for this finding may be that the CTS considers a broader conceptualisation of IPV than physical violence alone and therefore assesses the broader picture of IPV and is less vulnerable to IPV underestimation. This study examined whether this finding is replicated for childhood trauma symptoms.

In a similar vein, the validity of the measure of trauma symptomatology was also included as an additional moderator in the analysis. This reflected the fact that a number of measures have been used to assess for trauma symptoms with the most common and most well-validated including the PTSD scale of the Child Behaviour Checklist (CBCL: Achenbach & Edelbrock, 1983), Trauma Symptom Checklist for Children (TSCC: Briere,1996) and UCLA PTSD Reaction Index (Steinberg, Brymer, Decker & Pynoos, 2004). However, similar to some of the IPV measures used, some studies have used measures that have no published psychometric properties and some have been created purely for the purpose of the research study they were used in. It is possible, therefore, that these less well-validated instruments may have over or underestimated the trauma symptoms of the children assessed. For this reason, further moderator analyses were planned to see if this had an effect on the relationship between IPV and child trauma symptoms.

A further factor that must be considered when evaluating the association between IPV and child trauma symptomatology measure is who the reporter is: self (child) or other report (parent or clinician). Previous research in medical settings has found that when a child experiences a trauma, the parent's own posttraumatic stress may affect their report and interpretation of the child's symptoms (Shemesh et al., 2005). Specifically, children's self-report was shown to be more highly correlated with clinicians' evaluation than parent report. Furthermore, parental reports of a child's trauma symptoms can provide insights into the parent's own trauma symptoms (McFarlane, 1987; Shemesh et al., 2005). One explanation proposed is that children are very sensitive to their parents' reactions and often disclose that they do not want to talk about a traumatic event or their reaction to it with their parents, as they do not want to distress their parents more. This can lead the parent to underestimate the

degree of distress experienced by their children (Jaberghaderi, Greenwald, Rubin, Zand & Dolatabadi, 2004). In an IPV population, it is likely that this may exacerbate these biases as the child may wish to protect the parent from further stress and the parent may also be influenced by their own reaction to the domestic abuse they are experiencing. This study, therefore, included whether trauma symptoms were reported by the child or another respondent as a moderator for the association between childhood trauma symptoms and IPV.

A related moderator is whether the same or a different reporter provides information for the IPV measure and the child trauma symptom measure. This is important as it could be that if the same reporter answers both outcomes, it is likely that variables such as the reporter's mood, motivation and alertness at the time of assessment are more similar, which can affect their responses, compared with when different reporters provide this information. In addition, when the same reporter provides information it is possible that their previous responses, for example, to the trauma symptoms measure may influence their later responses to the IPV measure and vice versa. This is an example of a common method bias caused by common rater effects where the degree of association between the variables is inflated, rather than reflecting these variables' true effects. These types of method bias are common in research (Doty & Glick, 1998).

A final moderator that was explored was whether IPV was measured over the past year or over the child's lifetime. This would allow for testing whether being exposed to IPV at any point in a child's life can lead to trauma symptoms or, instead, whether the exposure to IPV has to be recent for trauma symptoms to be present at assessment. This hypothesis had not been tested on trauma symptomatology in

children before and so was an exploratory moderator. Related to this is the duration of exposure to IPV and the course of IPV exposure over time, however, these were not included as moderators as most children in the research literature are exposed to long-lasting IPV and this information is often not reported (Straus, 1992).

Comparison of Present Study to Previous Meta-Analytic Reviews

Despite empirical studies and strong theoretical hypotheses recognising trauma symptomatology as a prevalent outcome of IPV exposure (Vickerman & Margolin, 2007) to date only one prior meta-analysis had investigated this as part of a wider set of child outcomes (Evans et al., 2008). Evans et al. (2008) included six studies that compared exposed children to non-exposed children and measured trauma symptomatology. However, that meta-analysis is less comprehensive than the current study in that it did not identify an additional 10 studies during that time period. Moreover, although this study identified the six studies that were included in Evans et al. (2008) meta-analysis only five were included in this analysis due to different inclusion criteria used. Furthermore, a large number of studies have been published since 2006 (n=34) when the previous meta-analysis authors had finished their search. The small sample size in the Evans et al. (2008) analysis also prevented an examination of moderator analyses. The present study was, therefore, the first to examine this association as an individual outcome through meta-analysis and consider whether the magnitude of the association changes across a number of moderator variables.

Method

Literature Search

Searches of PsycINFO, OVID MEDLINE®, Ovid MEDLINE® Daily, Ovid MEDLINE® Epub Ahead of Print, In-Process & Other Non-Indexed Citations and Embase databases were conducted through October 2017 to identify studies to include in the meta-analysis. A variety of combinations of the following search terms were used: children or adolescen* or teenage* or child or young person or young people or childhood, emotional trauma or exposure to violence or experience or event or trauma or witnessing or traumatic or PTSD, intimate partner violence or witnesses or domestic violence or partner abuse or marital relations or marital violence or battered women or battered females or battered husbands or interparental relationship or interpersonal violence or IPV. In addition, reference sections from all identified previous reviews of the research on IPV exposure were inspected.

Furthermore, the reference sections of studies identified following initial exclusion of the studies were manually searched. The search also included unpublished studies, such as dissertations, found within the aforementioned databases, which is necessary to offset the problem of publication bias (Begg, 1994; Rosenthal, 1998). This search yielded 5475 publications and dissertations and 3194 after duplicates had been removed (see figure 1.1 for Prisma flow diagram).

The primary eligibility criterion for inclusion in the meta-analysis was that the study examined the relationship between exposure to IPV, which included physical violence between intimate partners, and child trauma symptoms. To be included in the meta-analysis, a study had to meet the following criteria: 1) It reported the findings of a quantitative empirical study. Review papers, qualitative studies, and

case studies were excluded. 2) The study included as part of IPV measurement a measure of parental physical IPV. Thus, the definition of children's exposure to IPV was children residing in families in which occurrences of physical IPV were reported sometimes in addition to other forms of IPV. This definition is consistent with the definitions used in other reviews (Evans et al., 2008; Kitzmann et al., 2003; Wolfe et al., 2003). Studies that explored interparental conflict, but not parental physical IPV, were excluded. Studies examining childhood exposure to community violence, exposure to only verbal aggression or parent-sibling aggression were also excluded. Also, only witnessed IPV was included and not studies where the children were also victims of the IPV. 3) The study examined child trauma symptoms or PTSD. Studies that either only included other child adjustment variables (e.g., internalising, externalising or physical health outcomes) or where the statistics reported meant that child trauma symptoms were included amongst other adjustment variables that could not be separately analysed were excluded. 4) The study must have reported sufficient data to allow the calculation of an effect size estimate using the formulas described by Lipsey and Wilson (2001). 5) IPV was assessed when children were 18 years old or younger. 6) The study was written in the English language as it was beyond the scope of this thesis to include studies published in other languages. 7) The study must have been conducted between January 1990 and 10th October 2017. By restricting this meta-analysis to studies that were published or conducted after 1990, the average design quality of the studies is likely to have increased (Fantuzzo & Lindquist, 1989) and this follows a similar protocol as used by Evans et al. (2008).

Authors of dissertations and publications that met all inclusion criteria except for the reporting of appropriate statistics (criterion 4) were approached to request this

information; multiple efforts were made to contact non-responsive authors to acquire
this information.
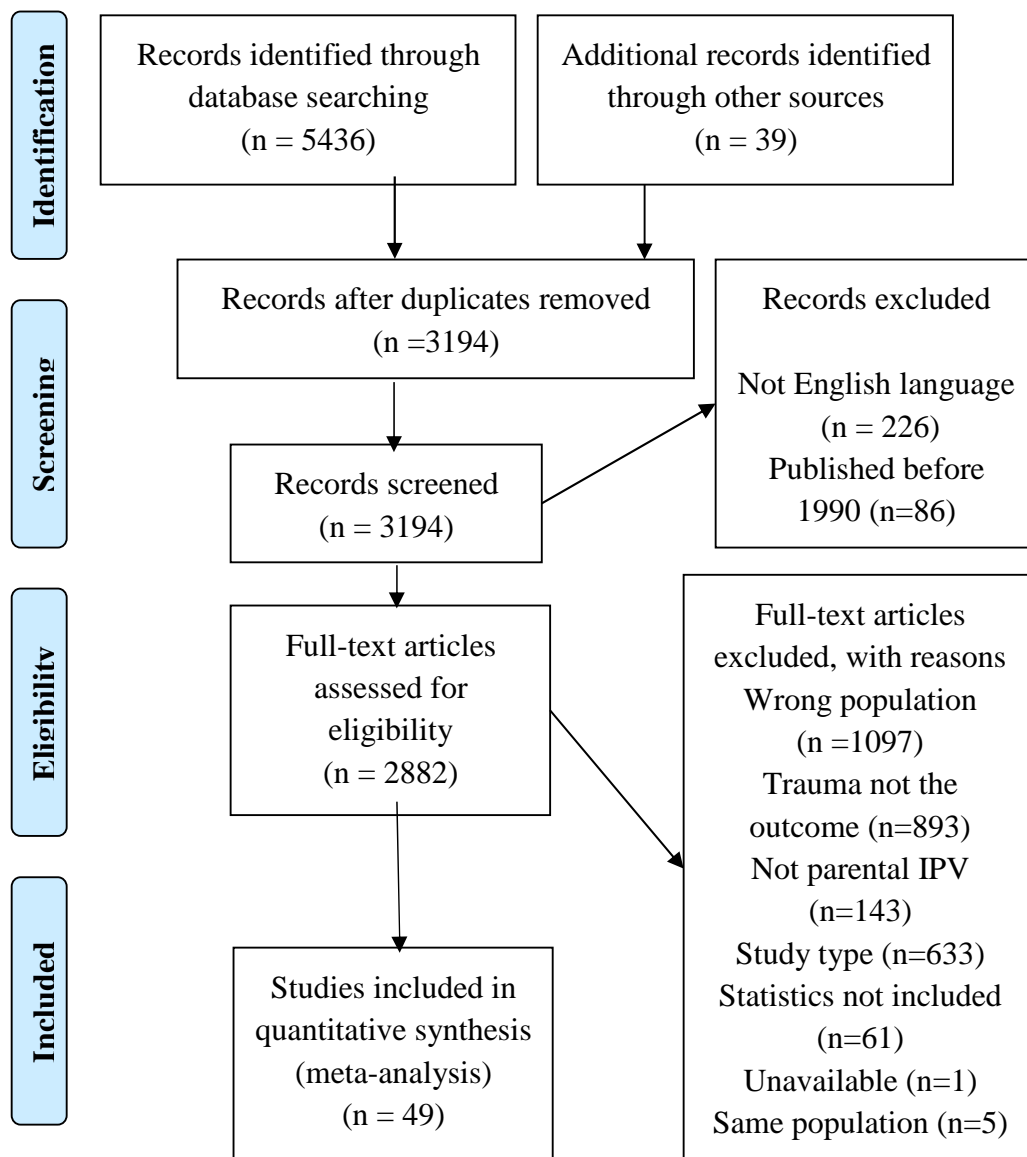
Figure 1.1. This figure illustrates the procedure employed to select studies for
the meta-analysis.

Of the 3194 publications and dissertations identified in the initial search, 2882 were assessed for full-text eligibility and 2833 were excluded because the studies: did not meet inclusion criteria, contained a correlation that had already been included, (e.g., when the same sample of participants was used for different studies), or authors did not supply the necessary statistics when contacted. A total of 49 publications and dissertations met the inclusion criteria and were thus included in the meta-analysis.

Coding Procedures

The following information was coded from each study: the number of male and female children in the sample, the mean age of the children in the study when IPV was assessed, whether children came from a shelter, clinic or community population, IPV conceptualisation (i.e., narrow = physical IPV only; broader = physical plus psychological IPV and/or sexual IPV), whose IPV was reported (partner only versus both mother and partner), whether IPV was assessed using the CTS or some version thereof versus IPV that was measured with a different assessment tool, whether the child trauma symptom measure used was well-validated or not, whether data on child trauma symptoms was reported by the child (self-report) or by a parent or clinician (other report), whether the same reporter completed both the IPV measure and the trauma symptom measure, time between measurement of IPV and measurement of child trauma symptoms (past year or lifetime), whether the study came from the same sample as another one of the 49 studies, sample size, and the correlation between IPV and child trauma symptoms.

Some studies supplied multiple statistics that could be included, resulting in more correlations than studies. Where multiple types of statistics were available, correlation coefficients were prioritised and total trauma symptoms were prioritised

over separate subgroups of trauma symptoms. These correlations were then averaged using Fisher's *r*-to-z transformation to create one effect size per sample. Where multiple studies reported on the same population, the studies with the largest sample sizes were selected and if this total sample size was the same, the study with the most validated measure of trauma symptoms was selected. For studies that included data from more than one source of report, effect sizes were averaged using Fisher's *r*-to-z transformation to create one effect size per sample. Additionally, for longitudinal data, an average effect size was taken across all time points using Fisher's *r*-to-z transformation.

Data Analyses

Meta-analysis is a statistical tool used for combining the effect on a common outcome domain (e.g. child and adolescent trauma symptoms) across individual studies. By combining study findings, the precision associated with a treatment effect becomes narrower (Sriganesh, Shanthanna & Busse, 2016). This study used the Comprehensive Meta-Analysis (CMA) program (Borenstein, Hedges, Higgins & Rothstein, 2014, Version 3) for analysis. This study aimed to produce an unbiased estimate of the population effect size and to examine the homogeneity of effect sizes within each of these analyses. The population effect size for these analyses is estimated by the average effect size, Pearson's *r*, with each *r* weighted by its precision. The resulting effect sizes were interpreted using Cohen's (1988) recommendations where an *r* of at least .10 is termed a small effect, an *r* of at least .24 is termed a medium effect, and an *r* of at least .37 is termed a large effect. For two of the studies only odd ratios were provided and so these were converted into *r* values using the CMA software. Due to the variability in the methods, settings, and recruitment procedures of the studies, it was assumed that both study-level sampling

error and subject-level sampling error were related to the effect sizes. Thus, as suggested by Lipsey and Wilson (2001), all meta-analyses were conducted using random effects models.

Results

The first research question evaluated whether exposure to IPV was systematically related to children's trauma symptoms (see table 1.1 for study characteristics). In subsequent sections, a series of moderator variables were tested to examine whether key study characteristics were associated with variation in the size of the IPV-to-trauma association.

Overall Average Study-Level Effect Size

Aggregation of the 49 studies yielded a weighted average correlation of $r = .26$, $z = 12.289$, $p < .001$, which as shown by its 95% confidence interval (.220, .299) and associated significance test differed significantly from zero (see figure 1.2 for forest plot). This weighted average correlation converts to a medium effect size (Cohen, 1988).

To determine whether the 49 effect sizes included in the weighted average effect size all estimate the same population effect size, homogeneity analyses were conducted. The homogeneity estimate (Q) follows a chi-square distribution and examines the likelihood that the variation in effect sizes within each analysis is different from what would be expected to result simply from sampling error. This Cochran's Q test for the effect sizes indicated between-study heterogeneity for trauma symptoms where $Q_B(48) = 194.690$, $p < .001$. The I^2 statistic, which describes the percentage of variation across studies that is due to heterogeneity rather than chance, also indicated

high heterogeneity (I^2=75%) (Higgins and Thompson, 2002; Higgins, Thompson, Deeks & Altman, 2003). This high heterogeneity also indicated that the planned moderator analyses would be deemed appropriate to try to explain this variance.

Publication Bias

To evaluate the extent to which null findings from unpublished studies might affect results, funnel plot asymmetry was examined using Egger's regression test (Egger, Smith, Schneider, & Minder, 1998). In a funnel plot, effect sizes are plotted against a measure of precision (e.g., standard error). Funnel plot asymmetry suggests that the data are biased and that small studies with small effect sizes are systematically underrepresented. The funnel plot for the data (see figure 1.3) was not asymmetric, t(47) = .989, p = .328, which suggests that null results from unpublished studies did not significantly influence the findings. Rosenthal's fail-safe N was also calculated, which delivers an estimate of the amount of unpublished studies with non-significant findings needed to render an average effect size non-significant (Rosenthal, 1979). In the current meta-analysis, a fail-safe N of 7017 studies was obtained. In addition, Duval and Tweedie's trim and fill procedure was conducted to see how many hypothetical, non-published studies with a negative effect size were missing from the meta-analysis. The analysis did not trim nor impute any studies (Duval & Tweedie, 2000a).

In summary, these analyses converge to suggest that the current meta-analytic findings are robust and are unlikely to be affected significantly by unpublished null findings as a consequence of a "file-drawer" problem of unpublished studies with null-findings.

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Table 1.1

Study characteristics of final sample

Study	N	Mean age (years)	Trauma measure	IPV measure
Black (2013)	143	4.25	PTSD Semi-Structured Interview & Observational Record & Child Dissociative Checklist	CTS
Boeckel (2017)	63	8.86	Child PTSD Symptom Scale	CTS 2
Briere (2001)	104	7.10	Trauma Symptom Checklist for Young Children	No information included
Castro (2017)	152	11.41	Child PTSD Symptom Scale	IPV to Woman Inventory
Chalovich (2004)	80	10.15	Trauma Symptom Checklist for Children	No information included
Costello (2016)	728	11.76	Trauma Symptom Checklist for Children	Violence Exposure Scale for Children-Revised
Edlynn (2008)	150	10.50	Trauma Symptom Checklist for Children	CTS
Ehrensaft (2017)	159	12.78	Child Behavior Checklist	CTS
El Sheikh (2008)	242	8.23	Trauma Symptom Checklist for Children	CTS2 & SVAWS
Erolin (2014)	87	10.20	UCLA PTSD Index	Family Violence Checklist
Georgsson (2011)	21	11.00	Children's Revised Impact of Event Scale	n/a all had IPV
Gewirtz (2011)	29	8.916	Levonn	n/a all had IPV
Ghasemi, Masoumeh	117	11.43	Trauma Symptom Checklist for Children	CTS2
Graham-Bermann (2006)	218	8.49	Trauma Symptom Checklist for Children	CTS
Hunt (2011)	257	11.70	Trauma Symptom Checklist for Children & UCLA PTSD Index	Caregiver report

Table 1.1 (continued)

Characteristics of studies identified

Study	N	Mean age (years)	Trauma measure	IPV measure
Jarvis (2005)	30	9.40	Child Post-traumatic Stress Reaction Index	CTS-R
Jouriles (2012)	88	15.90	The Los Angeles Symptom Checklist	CTS2
Kaslow (2008)	152	10.00	Trauma Symptom Checklist for Children	Index of Spouse Abuse
Kilpatrick (1997)	35	8.10	Child Post-traumatic Stress Reaction Index	CTS
Kimura (1998)	149	8.26	PTSD Reaction Index	CTS
Koolick (2016)	144	4.96	PTSD Semi-Structured Interview and Observational Record	CTS2
Lannert (2014)	182	0.98	Infant Social and Emotional Assessment Trauma-Related Symptoms Scale	SVAWS
Lehmann (1997)	84	11.00	Children's Impact of Traumatic Events Scale-Family Violence Form	The History of Violence Witnessed By Child Questionnaire
Lehmann (2001)	69	7.00	Parental Response To Child Trauma	Checklist of Controlling Behaviors
Levendosky (2013)	206	3.66	Infant Traumatic Stress Questionnaire & Child Traumatic Stress Questionnaire & Child Domestic Violence PTSD Scale	SVAWS
Luthra (2009)	157	12.20	Kiddie Schedule for Affective Disorders and Schizophrenia	Kiddie Schedule for Affective Disorders and Schizophrenia

Table 1.1 (continued)

Characteristics of studies identified

Study	N	Mean age (years)	Trauma measure	IPV measure
Mallah (1998)	160	8.27	PTSD Reaction Index & PTSD Reaction Index-Child Report	CTS & Perceptions of Adult Conflict Tactics Scale
McCloskey (1995)	24	Missing (range 5-12)	PTSD Reaction Index	CTS
McCloskey (2000)	337	9.30	Measure created for study	CTS & additional items
Mcguire-Schwartz (2017)	437	11.50	UCLA PTSD Index	UCLA PTSD Index
Mertin (2002)	56	10.00	Trauma Symptom Checklist for Children	Adapted Conflict Tactics Scale
Mohammad (2015)	89	12.32	Child Behavior Checklist PTSD Scale	CTS
Olaya (2010)	520	13.20	Diagnostic Interview for Children and Adolescents IV	Children's Perception of Interparental Conflict Scale
Piotrowski (2017)	184	9.38	CBCL PTSD Scale	CTS Physical Aggression Scale
Rigterink (2014)	35	9.50	Traumatic Stress Symptoms in Children & Child PTSD Symptom Scale & Child Dissociative Checklist & Child Behavior Checklist	CTS
Saltzman (2005)	48	8.42	Trauma Symptom Checklist for Children	CTS
Samuelson (2010)	62	11.70	Clinician-Administered PTSD Scale for Children	CTS2

Study	N	Mean age (years)	Trauma measure	IPV measure
Schechter (2011)	77	2.54	The Preschool Child Interview for PTSD and Observational Record	Demographic and Treatment History Questionnaire

Table 1.1 (continued)

Characteristics of studies identified

Study	N	Mean age (years)	Trauma measure	IPV measure
Schechter (2017)	59	2.29	Child Behavior Checklist PTSD Scale	CTS2 & Geneva Child Exposure to Violence Questionnaire
Silva (2000)	59	9.95	The Structured Clinical Interview for DSM-IV Childhood Diagnoses	The Structured Clinical Interview for DSM-IV Childhood Diagnoses
Skeen (2016)	972	8.90	Trauma Symptom Checklist for Children	CTS & UNICEF measure
Struthoff (2002)	35	13.05	The Los Angeles Symptom Checklist	CTS
Telman (2016)	120	9.85	Trauma Symptom Checklist for Young Children	CTS2
Turner (2012)	2016	Missing (range 2-9)	Trauma Symptom Checklist for Young Children	Juvenile Victimisation Questionnaire
Valjee (2016)	477	15.70	Davidson Trauma Scale	Developmental Trauma Inventory
Wherry (2015)	60	9.83	Trauma Symptom Checklist for Children-Alternate & Trauma Symptom Checklist for Young Children	The Health Resiliency Intervention for Violence Exposure
Williamson (2017)	151	77.7% aged 16–18	PTSD Checklist – Civilian Version	CTS2
Yoon (2016)	2064	11.12	Trauma Symptom Checklist for Children	Violence Exposure Scale for Children -Revised

| Zinzow (2009) | 3075 | 14.60 | PTSD module of National Survey of Adolescents | Measure created for study |

Note. Study names only list the first author of each paper. CTS= Conflict Tactics Scale; CTS2 = Conflict Tactics Scale 2; CTS-R = Conflict Tactics Scale Revised; SVAWS = Severity of Violence Against Women Scale.

Correlation and 95% CI

Study	Correlation	Std Err	Lower limit	Upper limit	Z-Value	p-Value	Total
Black 2013	0.251	0.14	0.091	0.399	3.035	0.002	143
Boeckel 2017	0.554	0.17	0.355	0.705	4.831	0.000	63
Briere 2001	0.260	0.15	0.071	0.431	2.674	0.007	104
Castro 2017	0.201	0.14	0.043	0.349	2.487	0.013	152
Chalovich 2004	0.197	0.16	-0.023	0.400	1.754	0.079	80
Costello 2016	0.370	0.12	0.306	0.431	10.459	0.000	728
Edlynn 2008	0.130	0.14	-0.031	0.284	1.585	0.113	150
Ehrensaft 2017	0.220	0.14	0.067	0.363	2.793	0.005	159
El Sheikh 2008	0.085	0.13	-0.042	0.209	1.317	0.188	242
Erolin 2014	0.204	0.16	-0.007	0.397	1.892	0.058	87
Georgsson 2011	0.545	0.26	0.148	0.791	2.593	0.010	21
Gewirtz 2011	0.210	0.23	-0.170	0.535	1.087	0.277	29
Ghasemi 2009	0.494	0.15	0.343	0.620	5.774	0.000	117
Graham Bermannn 2006	0.280	0.13	0.153	0.398	4.218	0.000	218
Hunt 2011	0.110	0.13	-0.012	0.230	1.767	0.077	257
Jarvis 2005	0.500	0.22	0.170	0.729	2.854	0.004	30
Jouriles 2012	0.040	0.16	-0.171	0.247	0.369	0.712	88
Kaslow 2008	0.130	0.14	-0.030	0.283	1.596	0.111	152
Kilpatrick 1997	0.183	0.21	-0.160	0.487	1.047	0.295	35
Kimura 1998	0.609	0.14	0.497	0.701	8.547	0.000	149
Koolick 2016	0.360	0.14	0.209	0.494	4.475	0.000	144
Lannert 2014	0.148	0.14	0.003	0.287	1.995	0.046	182
Lehmann 1997	0.389	0.16	0.190	0.557	3.696	0.000	84
Lehmann 2001	0.172	0.17	-0.067	0.393	1.414	0.157	69
Levendosky 2013	0.533	0.13	0.427	0.624	8.468	0.000	206
Luthra 2009	0.565	0.24	0.230	0.781	3.087	0.002	-
Mallah 1998	0.503	0.14	0.377	0.611	6.933	0.000	160

Figure 1.2 Forest plot of random effects meta-analysis   (continued)

-1.00   -0.50   0.00   0.50   1.00
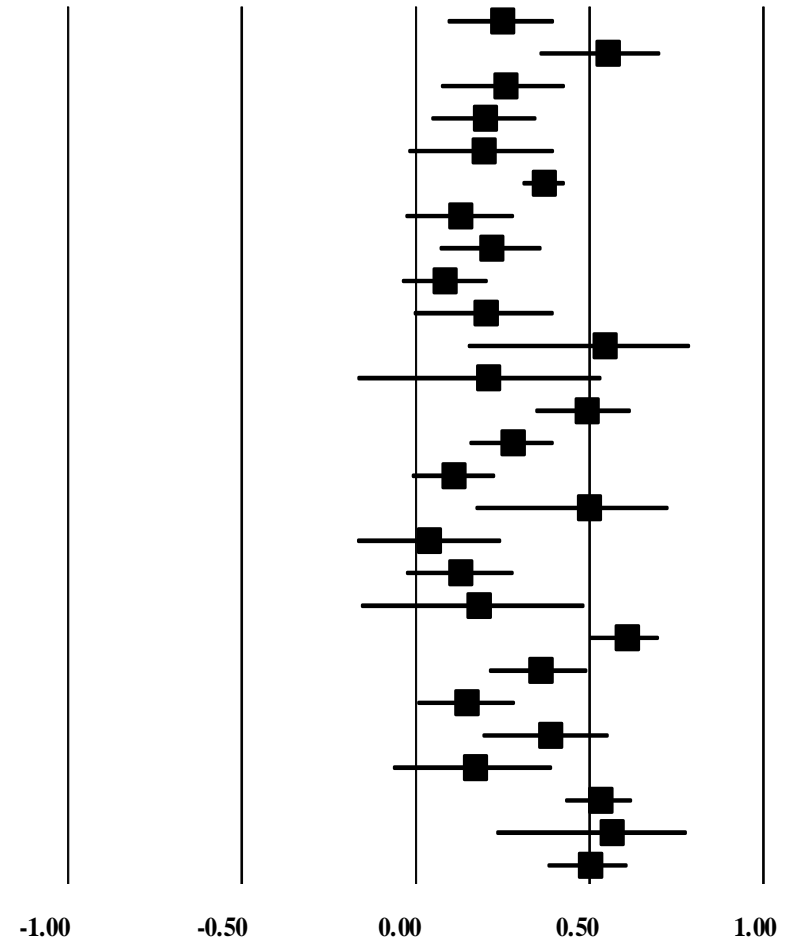
(continued)

Study	Statistics for each study							Correlation and 95% CI
	Correlation	Std Err	Lower limit	Upper limit	Z-Value	p-Value	Total	
McCloskey 1995	0.270	0.25	-0.150	0.607	1.268	0.205	24	
McCloskey 2000	0.274	0.13	0.172	0.370	5.129	0.000	337	
Mcguire-Schwartz 2017	0.080	0.12	-0.014	0.173	1.670	0.095	437	
Mertin 2002	0.020	0.18	-0.244	0.281	0.146	0.884	56	
Mohammad 2015	0.007	0.16	-0.202	0.215	0.065	0.948	89	
Olaya 2010	0.141	0.12	0.055	0.224	3.218	0.001	520	
Piotrowski 2017	0.340	0.14	0.205	0.462	4.764	0.000	184	
Rigterink 2014	0.160	0.21	-0.183	0.468	0.913	0.361	35	
Saltzman 2005	0.476	0.19	0.222	0.670	3.475	0.001	48	
Samuelson 2010	0.096	0.17	-0.158	0.337	0.736	0.462	62	
Schechter 2011	0.390	0.16	0.182	0.565	3.542	0.000	77	
Schechter 2017	0.305	0.18	0.053	0.520	2.357	0.018	59	
Silva 2000	0.076	0.18	-0.184	0.325	0.566	0.571	59	
Skeen 2016	0.193	0.12	0.132	0.253	6.084	0.000	972	
Struthoff 2002	0.150	0.21	-0.193	0.460	0.855	0.393	35	
Telman 2016	0.101	0.15	-0.080	0.275	1.096	0.273	120	
Turner 2012	0.283	0.12	0.242	0.323	13.053	0.000	2016	
Valjee 2016	0.290	0.12	0.206	0.370	6.500	0.000	477	
Wherry 2015	0.292	0.17	0.041	0.508	2.271	0.023	60	
Williamson 2017	0.260	0.14	0.105	0.403	3.237	0.001	151	
Yoon 2016	0.180	0.12	0.138	0.221	8.262	0.000	2064	
Zinzow	0.190	0.13	0.057	0.317	2.787	0.005	-	
Overall	0.260	-	0.220	0.299	12.289	0.000	-	
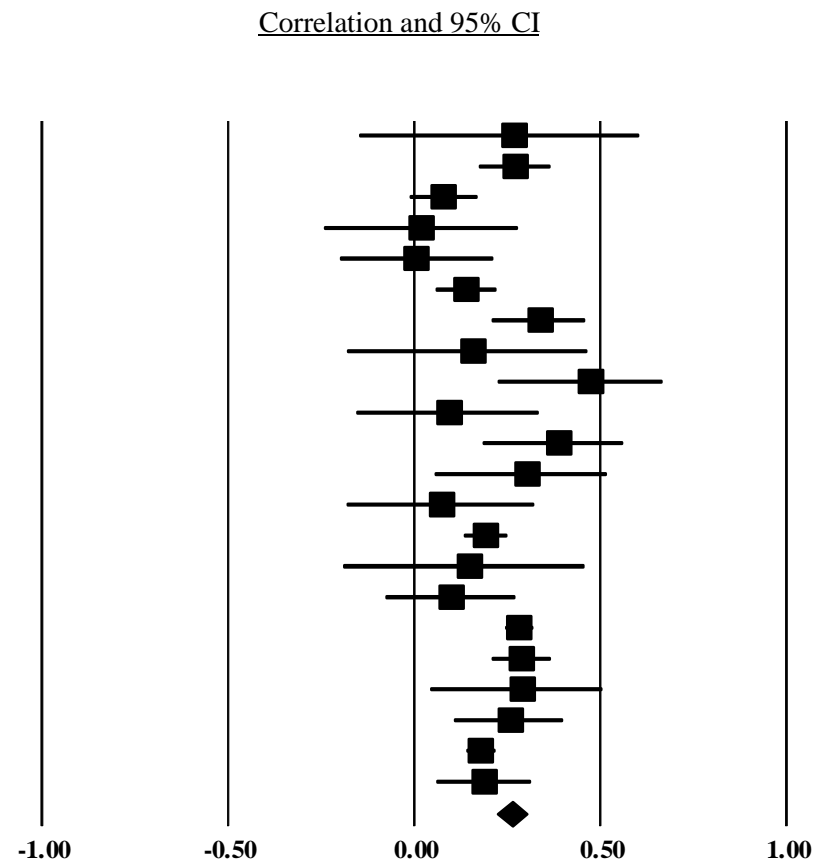
Figure 1.2 Forest plot of random effects meta-analysis   (continued)

Note. Study names only list the first author of each paper.
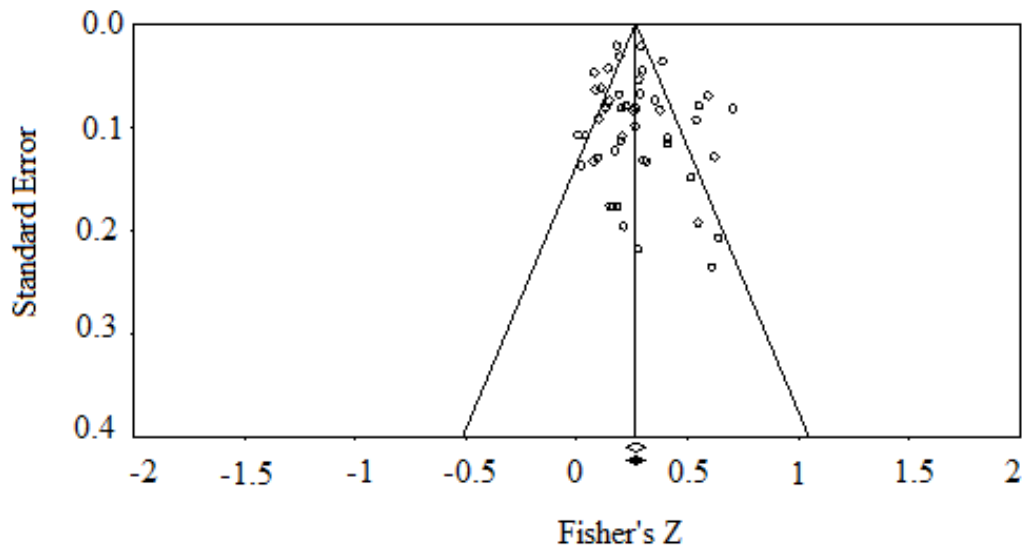
Figure 1.3 Funnel plot of standard error by Fisher's Z.

Moderator Analyses

To try to explain the heterogeneity in correlations, a mixed-effects model was employed to identify possible predictors of the correlation between IPV and child trauma symptoms. In a mixed-effects model, the effect sizes, in this case, correlations, are tested in a random-effects model, and predictors are estimated in a fixed-effects model. Separate analyses tested the effect of the continuous moderators on the association of IPV with child trauma symptoms (i.e., effect size) using meta-regression. These were the mean age of the children in the study when IPV was assessed and the percentage of males in the sample (see Table 1.2 for results). As suggested by Hedges (1994) weighted least squares regression procedures were employed, with effect sizes weighted by the inverse of the variance. For these analyses, the z-test is a two-sided test of the null hypothesis that the regression coefficient is equal to zero and the correlation is interpreted as normal.

Separate analyses were also conducted to examine the effect of each of the following categorical moderators on the association of IPV with child trauma symptoms (i.e., effect size): the sample type, IPV conceptualisation as broad versus narrow, whose

IPV was reported, the IPV measure used (CTS or some version thereof versus a different measure), how well-validated the trauma symptom measure was, whether the reporter of trauma symptoms was the child (self-report) or another reporter (parent or clinician), whether the same reporter completed both the IPV and trauma symptom measure and whether IPV was assessed over the past year or across the child's lifetime (see Table 1.2 for results). These were performed using procedures described by Cooper and Hedges (1994). Effect sizes are grouped according to the levels of the moderator variable and these groups are then compared to assess whether they differ significantly from each other. A significant between groups Q (Qb) indicates that these subgroups of effect sizes are significantly different from each other.

Continuous Moderators.

Child Gender.

The effect of child gender on the association between IPV and child trauma symptoms was assessed by using the proportion of males in the study as a predictor of the association. The overall proportion of males for the total sample was 51.82%. The proportion of males was not associated with the correlation between IPV and trauma symptoms (see table 1.2). The meta-regression showed that gender as a covariate explains zero percent of the variability in the model ($R^2 = .00$).

Child Age When IPV is Assessed.

The mean age of the children at the time of the IPV assessment in each study ranged from 11.77 months to 18 years. Because many studies recruited children of varying ages (e.g., 6–10 years old), the mean child age at the time of the assessment of IPV was used in the analyses. The mean age of the children at the time of the IPV assessment for the total sample was 9.56 years (SD= 3.24). The estimated correlation

between IPV and trauma symptoms was .299 when IPV was assessed at an average child age of five years, .253 at age 10 years, and .207 at age 15 years. The meta-regression showed that older child age at the time of the assessment of IPV was associated with lower correlations between IPV and trauma symptoms, however, this was not significant (see table 1.2). The meta-regression showed that age as a covariate explained four percent of the variability in the model ($R^2 =.04$).

Table 1.2.

Results of continuous moderator variables of IPV exposure and child trauma symptom association

Predictor	K	b	SE	Z	*p*	CI	$Q_B(df)$
Age	46	-.009	.008	-1.14	.256	-.025–.007	$Q_B(1)=.129, p=.256$
Gender	46	.159	.304	.52	.602	-.438-.755	$Q_B(1)=.027, p=.602$

K= number of studies

Categorical Moderators.

Sample Type.

Different sample types were examined to see whether similar estimates of effect sizes were attained from studies that recruited participants from intimate partner violence shelters, those that recruited participants from community or school populations, and those that recruited participants from clinical settings or agencies focused on child maltreatment. If the study recruited the exposure sample from more than one of these three settings, the study was excluded from these analyses. Shelter samples showed a higher correlation followed by community samples and finally clinic/other agencies samples (see table 1.3). However, homogeneity tests revealed that none of the groups were found to significantly differ from each other (see table 1.3).

Table 1.3.

Summary of meta-analysis results for categorical moderators

Moderator variable	k	*r*	z	*p*	95% CI
Sample type (k= 45)			$Q_B(2)$=.392, *p*=.822		
Shelter	10	.273	4.952	<.001	.168-.372
Community	21	.241	9.061	<.001	.191-.291
Clinic/other agency	14	.233	5.906	<.001	.157-.306
IPV Conceptualisation (k=45)			$Q_B(1)$ = .917, *p*=.338		
Narrow	19	.243	7.488	<.001	.181-.303
Broad	26	.286	8.556	<.001	.223-.346
Whose IPV was measured (k=46)			$Q_B(1)$ =.256, *p*=.613		
Partner and mother	28	.272	10.034	<.001	.220-.321
Partner only	18	.248	6.295	<.001	.173-.321
IPV measure (k=49)			$Q_B(1)$ =.906, *p*=.341		
CTS	24	.282	7.427	<.001	.210-.351
Non-CTS	25	.240	9.280	<.001	.191-.389
Trauma symptoms measure (k=49)			$Q_B(1)$ =.100, *p*=.752		
Well-validated	37	.257	11.326	<.001	.214-.299
Less well-validated	12	.275	4.814	<.001	.166-.378
Reporter (k=42)			$Q_B(1)$ = .072, *p*=.788		
Self-report (child)	29	.254	8.097	<.001	.195-.312
Other report (parent, clinician)	13	.242	6.693	<.001	.173-.309
Same or different reporter (k=35)			$Q_B(1)$ = .446, *p*=.504		
Same reporter	17	.273	20.281	<.001	.248-.298
Different reporter	18	.210	15.159	<.001	.184-.236
When IPV was assessed (k=36)			$Q_B(1)$ = .004, *p*=.953		
Last year	21	.274	8.022	<.001	.209-.336
Lifetime	15	.277	6.156	<.001	.191-.358

k=number of studies

Conceptualisation of IPV.

The way that IPV was conceptualised was coded into two categories. Narrow, meaning physical IPV only, and broader, meaning physical IPV plus psychological IPV and/or sexual IPV. Correlations were slightly higher when IPV was conceptualized more broadly (see table 1.3), however, the correlation between IPV and child trauma symptoms did not significantly differ across conceptualisations of IPV (physical IPV plus psychological and/or sexual IPV vs. physical IPV only) (see table 1.3).

Whose IPV.

Correlations were slightly higher when both the partner's and mother's IPV was assessed (see table 1.3). However, the correlation between IPV and child trauma symptoms did not significantly differ according to whether only the partner's IPV or both the mother's and the partner's IPV was assessed for child trauma symptoms (see table 1.3).

IPV Measure.

The estimated correlation between IPV and trauma symptoms was slightly higher when IPV was assessed using the CTS or some version thereof than when IPV was assessed using other measures (see table 1.3). However, the correlation between IPV and child trauma symptoms did not significantly differ when IPV was assessed using the CTS or some version thereof as compared to other measures see (see table 1.3).

Trauma Symptom Measure.

The estimated correlation between IPV and trauma symptoms was slightly higher when trauma symptoms were assessed using a less well-validated measure than when trauma symptoms were assessed using more validated measures (see table 1.3). However, the correlation between IPV and child trauma symptoms did not

significantly differ when childhood trauma symptoms were assessed using a more well-validated measure as compared to other measures (see table 1.3).

Self Versus Other Report.

The estimated correlation between IPV and trauma symptoms was slightly higher when trauma symptoms were self-reported by the child than when trauma symptoms were reported by another person (see table 1.3). In the category other reporter, parents formed 91.7% of reporters when just one other reporter was used to collect the trauma symptom measure. However, the correlation between IPV and child trauma symptoms did not significantly differ when information on child trauma symptoms was obtained via self-report from the child versus from another reporter, namely the parent or clinician (see table 1.3).

Same or Different Reporter

The estimated correlation between IPV and trauma symptoms was slightly higher when the same reporter completed both measures (see table 1.3). However, the correlation between IPV and child trauma symptoms did not significantly differ when both measures were completed by the same reporter or by different reporters (see table 1.3).

Whether IPV was Assessed Over the Past Year or Across the Child's Lifetime.

The estimated correlation between IPV and trauma symptoms was slightly higher when trauma symptoms were assessed over a lifetime than when trauma symptoms were assessed over the past year (see table 1.3). However, the correlation between IPV and child trauma symptoms did not significantly differ when IPV was assessed within the past year versus over a lifetime (see table 1.3).

Discussion

This section begins by summarising the key findings of the meta-analysis and linking these with previous research. Limitations of the research and implications for research and practice are subsequently discussed.

The results of this meta-analysis supported the hypothesis of an association between childhood IPV exposure and trauma symptoms in children. Average weighted correlation effect size estimates revealed an overall r value of .260 for the relationship between IPV exposure and trauma symptoms in children. Effect size estimates for child trauma symptoms signified a moderate degree of association between childhood exposure to IPV and psychosocial problems in children, thus supporting the conclusions of other reviews and meta-analyses (e.g., Evans et al., 2008; Howell, 2011; Howell, Barnes, Miller & Graham-Bermann, 2016; Margolin & Vickerman, 2007).

Despite methodological differences between the current study and prior meta-analyses in this field, the findings across these studies are largely consistent with respect to the possible effects of witnessing IPV. Notably, when inspecting the mean effect sizes for specific psychosocial outcomes, Evans et al. (2008) reported $d = 1.54$ for trauma symptoms, which translates to $r = .610$. The pooled effect size from Evans et al (2008) is considerably larger than the pooled effect size across the studies included in the current research, although the reason for this is unclear. The authors from Evans et al (2008) reported that follow-up analyses of homogeneity and the relatively small number of articles included in the analyses ($k = 6$) suggested that the effect size for trauma should be interpreted with caution. Additionally, for internalising problems, Evans et al., (2008) found a mean effect size of $d=.48$ (SE $=.04$) which translates to $r = .233$ and Vu et al. (2016) found $r = .10$ (SE $= .01$).

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These effect sizes are both more in line with the effect size of the present study and were based on a similar number of studies to the current research. Despite some differences, the results across these studies strengthen confidence in the finding that exposure to IPV between caregivers within the home is associated with an increased risk of emotional problems during childhood and adolescence.

This meta-analytic study followed previous meta-analytic reviews looking at other child and adolescent psychosocial outcomes in this area by exploring the effects of a number of moderator variables. The current findings converged with previous meta-analyses of other child outcomes (Evans et al., 2008; Kitzmann et al., 2003; Vu et al., 2016; Wolfe et al., 2003) in revealing that the associations between witnessing IPV and childhood psychosocial outcomes (specifically internalising symptoms) were not moderated by gender nor age. This study, therefore, adds to this picture in examining the moderating effects of gender and age on exposure to intimate partner violence, with trauma symptoms. It suggests that boys and girls are at similar risk for developing adjustment problems, including trauma symptoms, following IPV exposure.

The finding that age of the child did not affect the relationship between IPV and trauma symptoms is important. Previous studies have suggested that younger, rather than older, children are at particularly high risk from IPV exposure (Fantuzzo et al., 1997). Other suggestions have been presented that as younger children have less developed problem-solving and coping skills; less access to social influences outside of the family, such as friends; and are more likely to be home when IPV happens, then they may be more likely to come into contact with IPV. However, caution is warranted in that the majority of studies in this meta-analysis focused on whether the

child had been exposed to IPV during the previous 12 months before the study and therefore information regarding when the children were first exposed to IPV, or the course or nature of the IPV exposure over time were missing.

In order to explore this further, a moderator analysis was included representing whether IPV was assessed over the past year or across the child's lifetime. The majority of studies had included this information as the CTS measures across the past year. This analysis suggested that whether IPV was assessed over the lifetime or over the past year was not a significant moderator. However, caution is required regarding this finding given that these studies did not include information pertaining to whether the children assessed over the past year had witnessed IPV before this point and whether this was chronic. Furthermore, it did not ask those whose lifetime prevalence was assessed how recent the last exposure to IPV was; therefore, though suggestive that the chronicity of IPV measurement does not moderate the relationship between trauma symptomatology and IPV exposure, other important extraneous factors cannot be ruled out. Further research employing research designs and measures better positioned to capture this information would be vital in disentangling this relationship further.

When the hypothesis that children recruited from intimate partner violence shelters would exhibit more trauma symptoms than children from community samples (Margolin & Gordis, 2000) was tested, the results were consistent with previous reviews that investigated internalising symptoms in childhood (Evans et al., 2008; Vu et al., 2016). Results were similar in that the strength of the relationships between exposure to intimate partner violence and trauma symptoms did not differ between samples recruited from clinical settings, intimate partner violence shelters, and

community or other agency settings. This follows conclusions reached in some narrative reviews and further suggests that the recruitment setting of samples does not consistently affect the association between IPV and child and adolescent trauma symptoms.

In contrast to Vu et al.'s (2016) finding regarding the conceptualisation of IPV, the current study found no difference in association when IPV was conceptualised more broadly compared to when IPV was conceptualised more narrowly. Within the research literature, it has been suggested that excluding psychological and/or sexual violence when conceptualising IPV may have led to inaccurate conclusions about the effects and prevalence of IPV (Hamby, 2014). The present study focused more specifically on trauma symptoms and it may be that certain types of child adjustment are affected differently by different types of IPV. Further research including this as a moderator variable for other types of child adjustment as well as research considering the optimal operationalisation of a broader conceptualisation (i.e., the relative contributions of physical, psychological, and sexual IPV) may, therefore, prove useful.

The findings also support previous research on other IPV-related outcomes, in that the association between IPV exposure and trauma symptomatology did not vary as a function of whose IPV was measured (e.g. intimate partner only versus both partners) (Vu et al., 2016). One explanation of this may be that it is the violence witnessed affecting family members, whether mother or partner, which is the determining factor, as either may be a loved attachment figure to the child and therefore witnessing any IPV may be important to the development of child adjustment problems. However, further research should consider whether this same

pattern is observed when other domains of child adjustment or types of IPV are considered. Furthermore, capturing and operationalising different types of IPV in terms of its nature may be challenging because, for example, there is likely to be a spectrum of experiences between perpetration and retaliation, and ongoing severe interparental conflict, which may be hard to tease apart and measure accurately.

The results also add to the somewhat complex picture regarding the degree to which the association between IPV exposure and child adjustment is moderated by whether a well-validated instrument (such as the CTS) is used to measure IPV compared to other measures of IPV that may not have been well-validated. The current study's finding that this was not a significant moderator supports findings that have suggested the same for child internalising and externalising problems (Vu et al., 2016). However, in previous reviews (Kitzmann et al., 2003; Vu et al., 2016) when the CTS was used, child total adjustment problems had a stronger association with IPV exposure than other measures of IPV did. Similarly, the validity of the measure of trauma symptomatology was also not found to be a moderator in our analyses. This was an interesting finding given that the majority of less well-validated measures tended to be used in studies that assessed children under five and suggests that the conclusions drawn may be generalisable across the age range.

An additional moderator analysis that considered whether the reporter of the outcome variables was self vs other report did not show a moderator relationship. This differs from previous research investigating other child outcomes which have found that child self-report of trauma symptoms more valid than parental reports (McFarlane, 1987; Shemesh et al., 2005). It is possible that this finding reflects the fact that most of the other reports referred to younger children who were unable to

self-report and so relied on parental report. Furthermore, a number of studies

included both parental and self-report and so were excluded from this moderator

analysis which may have affected results.

The results did not show a moderator relationship for whether information was

obtained from the same reporter for both IPV and childhood trauma symptoms or

whether this information was collected from different reporters. This suggests that

using the same reporter does not lead to an inflated estimate of the association

between the variables. This also suggests that the association between IPV and

childhood trauma symptoms cannot be explained by common rater effects.

Nevertheless, future research may include measures from multiple reporters in order

to continue to test this and control for potential common method variance effects.

The fact that significant heterogeneity was found between the studies, yet the tested

moderator analyses did not have significant findings suggests that there may be other

moderators present which may provide a buffering effect. Future research could

include moderators such as whether siblings or grandparents were present in the

family, as there is currently the beginnings of a growing literature considering these

relationships and their potential role as protective factors for IPV and child

adjustment (Caldwell, 2014; Stafford, Stead & Grimes, 2007).

Limitations

Several limitations of this meta-analysis reflect the fact that this research in many

ways is still in its infancy as this is the first meta-analysis to consider child trauma

symptomatology and IPV. In that regard, it is notable that there is still little

agreement about the best way to define IPV exposure. For example, some studies

classify children who are aware of the violence but who have not witnessed the

violence themselves as being exposed to IPV. In contrast, others define exposure as witnessing two of the violent acts on the CTS. The current study did not include studies where the child had not witnessed the IPV themselves. In addition, few studies examined severity and frequency of IPV and many treated exposure to IPV as a dichotomous variable. Future research should seek to address these problems with operationalisation and reflect the more complex and multifaceted experience of IPV as opposed to its current simple dichotomisation.

The current research literature is also largely comprised of cross-sectional studies, meaning that causation cannot be inferred and extraneous variables which may not have been measured may bias the results. This is particularly true for IPV as it often co-occurs with other risk factors for child trauma symptomatology, such as child maltreatment (Jouriles et al., 2008). Related to this, it is likely that children who have been exposed to IPV once during their lives are often exposed to IPV at other times too (Margolin et al., 2009). This ongoing nature of IPV exposure needs to be explored in order for definitive conclusions to be reached regarding the impact of children's age at the time of IPV exposure, IPV duration and chronicity, and later child trauma symptomatology. Future research considering intervention studies and genetically informative studies may also help shed light on causal processes.

Another key limitation is that within the research literature many of the variables used to code for IPV, such as male partner only, do not accurately reflect the types of families we live in today. For example, same-sex couples also experience IPV yet the wording used in previous studies suggests that these relationships were not included in the sample populations and so the outcomes for these children were not available to be included as they had not been studied in the identified literature.

Another limitation is that a formal quality appraisal of the studies was not conducted. Although various methodological moderators were included which considered the quality of the study, for example, the validity of the measures, a formal quality appraisal would be important to consider for future research as the validity of the summary estimates depends on the quality of the included studies. Related to this, the study was coded by the author alone and in the future training additional coders would be beneficial to offset potential selection bias.

Closing Remarks

In summary, this meta-analysis provides additional evidence of the significant relationship between childhood exposure to IPV and trauma symptomology in children. The research literature remains at the early stages of development in contrast to other areas of IPV research. However, as this develops over time, the findings from these studies can be utilised to inform clinical practice and support effective prevention and intervention strategies when working with children and their families who experience intimate partner violence.

References

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Part 2: Empirical Paper

The Relationship Between Parent-Child Relationship Predictors and Childhood

and Adolescent Depression and Anxiety: The Role of Family Context

Moderators

Abstract

Aims: Using a longitudinal design, focusing on the transition from childhood to adolescence, this study assessed whether parent-child relationship factors, specifically attachment and parenting sensitivity, predicted childhood and adolescent depression and anxiety symptoms. Secondly, whether this relationship was moderated by the family context, specifically; parental depression and anxiety, negative life events and the interparental relationship.

Method: Participants were part of the NICHD Study of Early Child Care and Youth Development, a longitudinal study following, initially, 1364 children and their families from birth until age 15.

Results: Latent growth curve analyses revealed that parenting sensitivity predicted initial depression and anxiety symptoms and also anxiety symptoms over time. Attachment measured at 15 months was not a significant predictor. However, attachment measured at 36 months significantly predicted depression symptoms over time but not initial symptoms whilst the reverse pattern was found for anxiety symptoms. All family context variables were associated with childhood depression and anxiety symptoms. However, only maternal anxiety moderated the relationship between parenting sensitivity and childhood anxiety symptoms and only the interparental relationship moderated the relationship between parenting sensitivity and childhood depression symptoms. These interactions are noted with caution. Interestingly, there was a lack of main effects of attachment or parenting sensitivity when family-context variables were included as interactions.

Conclusions: This study provides evidence of the small yet significant link between parenting, attachment and childhood and adolescent depression and anxiety symptoms. The findings stress the importance of considering the whole family

context as well as parent-child relationship factors in theoretical models of childhood

depression and anxiety as well as suggesting the use of multiple potential pathways

for prevention and intervention strategies.

Introduction

Depression is a relatively common mental health problem worldwide among adolescents (Lopez, Mathers, Ezzati, Jamison & Murray, 2006). The level of depression in early childhood is low. Most studies report prevalence rates of one percent or less (Kessler, Avenevoli & Ries Merikangas, 2001). However, this rises rapidly through adolescence (Green, McGinnity, Meltzer, Ford & Goodman, 2005; Thapar, Collishaw, Pine & Thapar, 2012), with an estimated prevalence between four and five percent in mid-late adolescence (Costello, Egger & Angold, 2005; Costello, Erkanli & Angold, 2006).

Anxiety has also been shown to be among the most common mental health problems for children and adolescents (Breton et al., 1999; Canino et al., 2004; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Ford, Goodman & Meltzer**,** 2003; Lewinsohn, Hops, Roberts, Seeley & Andrews, 1993; Lewinsohn, Zinbarg, Seeley, Lewinsohn & Sack, 1997). Estimates indicate that between two and a half and five percent of children and adolescents meet criteria for an anxiety disorder at any given time (Breton et al., 1999; Costello et al., 2003; Ford et al., 2003; Lewinsohn et al., 1993, 1997). Research suggests that the rate of anxiety disorders remains relatively stable through childhood and adolescence (Benjamin, Costello, &Warren, 1990; Boyd, Gullone, Kostanski, Ollendick, & Shek, 2000; Last, Perrin, Hersen, & Kazdin, 1996). However, studies have reported slight increases in anxiety disorders in adolescence relative to preadolescence (Canino et al., 2004; Ford et al., 2003). Furthermore, across existing studies evidence strongly supports the view that childhood anxiety is a risk factor for adolescent depression (Schleider, Krause & Gillham, 2014).

In order to understand the development of both anxiety and depression in childhood and adolescence, a number of environmental risk factors have been considered in the literature, with the parent-child relationship perhaps being the most widely researched (McLeod, Weisz & Wood, 2007). In addition, research has suggested that the family context may also be significant when considering the development of child and adolescent anxiety and depression (Kerns, Siener & Brumariu, 2011; Sander & McCarty, 2005). This may play an important role in explaining the extent to which the parent-child relationship is associated with later childhood or adolescent depression or anxiety. The current research, therefore, considered the role of the parent-child relationship in the development of childhood and adolescent depression and anxiety as well as the role of specific family context moderators. These were parental depression, parental anxiety, negative life events and the inter-parental relationship.

Parenting

Theoretical models of child and adolescent depression and anxiety emphasise the importance of the parent-child relationship and in particular- parenting and attachment (Bowlby, 1980; Chorpita & Barlow, 1998; Cummings & Cicchetti, 1990; Downey & Coyne, 1990). However, despite these theoretical accounts, meta-analytic findings indicate that parenting accounts for less than four percent of the variance in childhood anxiety (McLeod, Wood & Weisz, 2007) and almost eight percent of the variance in childhood depression (McLeod et al., 2007).

Much empirical research has emphasised two key broad dimensions of parenting–rejection and control (Maccoby, 1992; McLeod et al., 2007; Rapee, 1997; Schwarz, Barton-Henry, & Pruzinsky, 1985). Furthermore, several reviews have summarised

evidence supporting an association between both these dimensions of parenting and child and adolescent depression and anxiety symptoms and disorders (Bogels & Brechman-Toussaint, 2006; McLeod et al., 2007; Rapee, 1997; Wood, McLeod, Sigman, Hwang, & Chu, 2003).

Given that different aspects of parenting are associated with child and adolescent depression and anxiety, this may suggest that there may be a more general style of parenting responsible. The current study combined these factors into an overall parenting composite -parenting sensitivity. This encapsulates both rejection and control, in that a sensitive parent is one who is responsive and accepting as well as cooperative and noninterfering (Belsky & Fearon, 2008). Therefore, children and adolescents may be less likely to experience depression or anxiety if they experience more sensitive parenting.

Within the literature parental rejection has been described as embodying excessive disapproval and criticism well as lack of warmth, responsiveness and contact with the child (Clark & Ladd, 2000; Maccoby, 1992; McLeod et al., 2007; Rapee, 1997). Theoretical models have posited that parental rejection contributes specifically to the development of childhood depression (Downey & Coyne, 1990; Marton & Maharaj, 1993) by encouraging a sense of helplessness, undermining self-esteem, and inducing the development of negative self-schemas, which have been argued to be the building blocks of depression (Garber & Flynn, 2001b; Hammen, 1992; Kaslow, Deering, & Racusin, 1994).

Parental rejection is also proposed to impact children's emotions by increasing sensitivity to anxiety (Gottman, Katz, & Hooven, 1997). Furthermore, the frequency and intensity of negative feedback may contribute to viewing the environment as

hostile and threatening and children viewing themselves as less than competent (Krohne, 1990; Krohne & Hock, 1991). Taken together, these theories suggest that parental rejection also places children at an increased risk for developing anxiety problems. McLeod et al. (2007) conducted a meta-analysis that found that parental rejection accounts for approximately eight percent of the variance in childhood depression and a further meta-analysis conducted by the authors found that parental rejection accounts for approximately four percent of the variance in childhood anxiety (McLeod et al., 2007).

Parental control has been described as parenting behaviours that include excessive regulation of children's activities and routines, encouragement of children's dependence on parents, and instructions to children on how to feel and think (Barber, 1996; McLeod et al., 2007; Steinberg, Elmer, & Mounts, 1989). One way these behaviours are proposed to increase the risk for childhood depression is by reducing perceived mastery (Chorpita & Barlow, 1998). Some theoretical models (Chorpita & Barlow, 1998; Krohne, 1990; Manassis & Bradley, 1994; Rapee, 2001; Rubin & Mills, 1991) propose that when parents are highly controlling in contexts when it is developmentally appropriate for children to act independently, children may experience decreased self-efficacy, and therefore, increased anxiety (Wood, 2006). Furthermore, parental encouragement of children's autonomy and independence (e.g., in novel contexts) may amplify children's perceptions of mastery over the environment, which has been hypothesised to lead to a reduction in the risk of childhood anxiety (Chorpita & Barlow, 1998; Wood et al., 2003).

Other factors that parental control is proposed to influence include: inducing perceived helplessness (Garber & Flynn, 2001b; Kaslow et al., 1994) and reducing

perceived personal control (Weisz, Southam-Gerow, & McCarty, 2001); well-documented risk factors for depression (Abramson, Metalsky & Alloy, 1989; Liu, Kleiman, Nestor & Cheek, 2015). McLeod et al. (2007) conducted a meta-analysis that found that parental control accounts for almost five percent of the variance in childhood depression and a further meta-analysis by the authors found that parental control accounts for almost six percent of the variance in childhood anxiety (McLeod et al., 2007).

Considering these factors together under the umbrella of parenting sensitivity, there is clear evidence that parenting sensitivity contributes to understanding the development of both childhood anxiety and depression. However, given the evidence that this may be a small albeit significant association, it is important to examine other factors that may also play a key contributory role. Furthermore, it is worthy to note how parenting sensitivity and another significant parent-child relationship factor, namely – attachment, are related. Parenting sensitivity, for example, has been found to be a reliable predictor of children's attachment classification (NICHD Early Child Care Research Network, 1997; 2001).

Attachment

Theorists have proposed that insecure attachment also has an important role to play in a variety of mental health problems including depression and anxiety (e.g., Bowlby, 1980; Cummings & Cicchetti, 1990). In line with this, many studies with adolescents (e.g., Armsden & Greenberg, 1987; Armsden, McCauley, Greenberg, Burke, & Mitchell, 1990; West, Spreng, Rose & Adam, 1999) have demonstrated that an insecure attachment style, usually measured by self-report questionnaires, is associated with a greater probability of experiencing depression. Similarly, research

conducted with children has found that greater levels of self-reported insecure attachment are significantly associated with greater levels of current depression symptoms (Graham & Easterbrooks, 2000; Muris, Mayer, & Meesters, 2000).

Likewise, in developmental studies where attachment is usually measured by direct observation of behaviour (e.g., using the Strange Situation Procedure) insecure attachment has been found to relate to a greater likelihood of developing anxiety disorders in pre-schoolers (Shamir-Essakow, Ungerer & Rapee, 2005) and adolescents (Warren, Huston, Egeland & Sroufe, 1997) and depression in adolescence (Murray, Arteche, Fearon, Halligan, Goodyer & Cooper, 2011). Insecure attachment has also been related to higher levels of anxiety symptoms (Costa & Weems, 2005; Roelofs, Meesters, Huurne, Bamelis & Muris, 2006; van Brakel, Muris, Bogels & Thomasson, 2006; but for exceptions, see Bosquet & Egeland, 2006; Feng, Shaw & Silk, 2008).

There have been several reviews in recent years considering attachment and internalising symptoms such as depression and anxiety. Findings included that attachment security is more consistently related to anxiety and depression over global internalising symptoms in childhood and adolescence (Brumariu & Kerns, 2010). Attachment has been shown to be moderately related to anxiety with effect sizes ranging from $d = .15$ to $d = .62$; which was stronger during adolescence than childhood (Brumariu & Kerns 2010; Colonnesi, Draijer, Stams, Van der Bruggen, Bögels & Noom, 2011). These latter reviews have tended to rely heavily on self-reports of attachment security. Other meta-analyses relying solely on observational measures of attachment have found much smaller effect sizes for the association between attachment and internalising behaviours in childhood (Groh, Roisman, van IJzendoorn, Bakermans-Kranenburg & Fearon, 2012; Madigan, Atkinson, Laurin &

Benoit, 2013). Whilst this research suggests that attachment plays an important role in childhood and adolescent depression and anxiety, the findings are complex because self-report measurements are prone to bias, and observational studies tend to report weaker associations.

Related to this, this study aimed to address some of the inconsistencies within the literature regarding parent-child relationship processes influencing risk for anxiety and depression. In particular, this study considered how the type and quality of measurement of both attachment and parenting were influential to the strength of associations found. For example, McLeod et al's (2007) meta-analysis into parenting and childhood anxiety found that effect sizes were $d = .28$ for observational measures of parenting versus $d = .20$ for questionnaire and $d = .15$ for interview. Observational measures are considered the 'gold standard' for assessing the quality of parenting and attachment (Solomon & George, 1999; van IJzendoorn & Sagi, 1999) where self-report measures of parenting can be more susceptible to bias (Morsbach & Prinz, 2006) and research has suggested that parental report is only weakly correlated with observations of parenting (Sessa, Avenevoli, Steinberg, & Morris, 2001). Therefore, this study used more precise observational measures of both parenting and attachment.

In summary, attachment is a key factor when considering parent-child relationship factors and the effects on child depression and anxiety symptoms. However, other sources of evidence indicate that moderating factors may also play an important role in explaining the extent to which parenting or attachment are associated with later childhood or adolescent depression or anxiety.

Moderators

Parental Depression.

A large number of studies have found that maternal depression is significantly associated with childhood depression (Bureau, Easterbrooks & Lyons-Ruth, 2009), adolescent depression (Brown et al., 2015; Bureau et al., 2009; Murray et al., 2011); adolescent anxiety (Murray et al., 2011) and internalising symptoms (Campbell, Morgan-Lopez, Cox & McLoyd, 2009; Goodman, Rouse, Connell, Broth, Hall & Heyward, 2011).

Halligan, Murray, Martins and Cooper (2007) conducted a 13-year longitudinal study and found that mothers who experienced postnatal depression were at an increased risk to develop a further depression episode and that it was this later depression episode/s that increased the risk of adolescent depression. Halligan et al. (2007) also found that for anxiety disorders, children with mothers who experienced postnatal depression were at increased risk regardless of subsequent depression episodes. However, other studies have found that parental depression has weaker associations with adolescent anxiety than adolescent depression (Brown et al., 2015).

Additional research has looked into the role of other parent-child relationship factors when considering parental depression and child and adolescent depression and anxiety. Adolescent attachment has been shown to moderate the link between parental and child depression and anxiety symptoms where secure attachment played a protective role (Woodhouse, Ramos-Marcuse, Ehrlich, Warner & Cassidy, 2010). In line with this, research has found that maternal depression symptoms predicted higher rates of insecure attachment at age three (Campbell et al., 2004). Campbell and colleagues found that mothers who experienced late, intermittent, or chronic

depression symptoms and who were low in parenting sensitivity were more likely to have children with less secure attachments than symptomatic mothers who were high in sensitivity. This highlights the important moderating relationships between parenting, attachment and parental depression. Whether such moderating relationships might explain the emergence of child anxiety or depression symptoms remains to be rigorously evaluated.

Parental Anxiety.

There is considerable evidence that maternal anxiety is related to child anxiety (Bogels & Brechman-Toussaint, 2006; Burstein, Ginsburg, & Tein, 2010; Costa & Weems, 2005; Shamir-Essakow et al., 2005) and there is some evidence that maternal anxiety disorder alone significantly predicted the presence of anxiety disorders in children regardless of parenting (McClure, Brennan, Hammen & Le Brocque, 2001).

Studies have found that parental anxiety is also significantly associated with child depression (Biederman, Rosenbaum, Bolduc, Faraone & Hirshfeld, 1991; Burstein et al., 2010). However, other studies have shown that parental anxiety is either a stronger predictor of child anxiety than depression or in some studies is not related to child depression at all (Beidel & Turner, 1997). Kerns et al. (2011) found that children who were more anxious at the beginning of middle childhood were more likely to have more anxious mothers and to be in families experiencing more negative life events. Children who became more anxious across middle childhood were less securely attached to their mothers, had mothers with more anxiety symptoms, and were in families experiencing more negative life events. These results suggest that both aspects of the parent-child relationship and qualities of the

family environment explained changes in anxiety symptoms across time. Furthermore, the study found that more anxious children were in turn exposed to a more anxious and stressful family environment, which subsequently was associated with an increase in anxiety symptoms over time.

Negative Life Events.

Negative life events are a well-established risk factor for the development of depression and anxiety in adolescence and adulthood (Brown, 1993; Brown & Harris, 1978; Goodyer, Herbert, Tamplin & Altham, 2000; Hammen, 2005; Kendler, Hettema, Butera, Gardner & Prescott, 2003; Kendler, Karkowski & Prescott, 1999; Kessler, 1997; Larson & Ham, 1993; Lewinsohn, Allen, Seeley & Gotlib, 1999; Ormel, Oldehinkel & Brilman, 2001). Studies have reported both a larger amount of and a larger impact of negative life events experienced by children with anxiety disorders compared with controls (Goodyer, 1990; Tiet et al., 2001). Furthermore, research has found that children with anxiety tend to have experienced more independent life events, suggesting that these events are not simply a result of the children experiencing anxiety (Allen, Rapee & Sandberg, 2008; Eley & Stevenson, 2000). To date, there have been few longitudinal studies that have established these life events occurring prior to the onset of anxiety. However, Allen et al. (2008) found that children with anxiety experienced a greater amount of life events in the year prior to the onset of anxiety compared with an equivalent period among nonclinical controls. Similarly, Williamson, Birmaher, Dahl and Ryan (2005) found that children with depression experienced significantly more life events including those independent of their behaviour compared to children with anxiety and controls. In line with this, Ge, Lorenz, Conger, Elder and Simons (1994) found that for

adolescents, over a four-year period, depression symptoms changed in accordance with changes in negative events.

Research has also investigated how both parent- relationship factors and family context factors may explain this association. Ge et al. (1994) found that girls with less supportive mothers were more likely to experience negative life event changes. Related to this, Bouma, Ormel, Verhulst and Oldehinkel (2008) found that adolescents whose parents had a history of depression reported more depression symptoms after negative life events than adolescents whose parents did not have a history of depression. These studies add to the complex picture of family context factors and anxiety and depression development and highlight the interplay that may exist between parent-child relationship factors, family context factors and childhood and adolescent depression and anxiety symptoms.

Interparental Relationship.

A further important moderator to consider is the interparental relationship and, related to this, family conflict. Research has shown that family conflict (including parental, sibling and parent-child) is associated with increased adolescent anxiety over time (Mechanic & Hansell, 1989) and is a significant risk factor for adolescent anxiety and depression (Murray et al., 2011). Furthermore, research has demonstrated that although marital conflict and divorce were predictive of anxiety six years later, the impact of parental divorce on child anxiety decreased over time, whereas anxiety was maintained for children whose parents did not divorce and remained in high conflict environments (Jekielek, 1998). This supports other research that found at a 10-year follow up that for children who were in families with greater marital problems and poor marital adjustment, there was a fourfold increase

in anxiety disorders, compared to children from families without poor marital adjustment (Nomura, Wickramaratne, Warner, Mufson & Weissman, 2002). In addition, inter-parental relationship functioning has been shown to be associated with the trajectory of children's depression symptoms over the transition to puberty where greater levels of marital conflict related to greater increases in symptoms over time (Papp, 2012).

Other research has considered the potential mechanisms behind this relationship between interparental relationship functioning and the development of depression and anxiety. In particular, the role of parenting and attachment has been explored. Shelton and Harold (2008) found that inter-parental conflict was related to child appraisals of parental rejection. This, in turn, was related to children's internalising symptoms and externalising symptoms. This fits with other studies that have found that children in families experiencing inter-parental conflict view the parent-child relationship as more hostile and insecure (Harold, Fincham, Osborne & Conger, 1997; Harold, Shelton, Goeke- Morey & Cummings, 2004). This supports Belsky (1984) who argues that the marital relationship is a primary emotional context in which parent-child functioning occurs. Taken together these studies suggest that parenting and attachment may play a key role in understanding the transmission of risk via the interparental relationship and anxiety and depression development which the current study aimed to investigate.

Research has also investigated how other family context factors and the interparental relationship may play a part in child adjustment. For example, inter-parental relationship functioning has been shown to moderate the interplay between parental and child depression symptoms specifically regarding marital conflict resolution

(Papp, 2012). Furthermore, Shelton and Harold (2008) found that parental depression predicted increased marital conflict, which in turn influenced adolescent adjustment problems via the impact on parent-child relations. This research highlights the complex interplay between parent-child relationship factors and family context factors and how each may have an effect in turn on the other. The current study aimed to add to this literature by considering the role of each of these factors in the same population.

The Current Study

Although considerable progress has been made in identifying risk factors for the development of depression and anxiety in childhood and adolescence, there are significant limitations to our understanding. For example, despite empirical support for the moderators presented, the current literature indicates that no moderator consistently affects the strength of the association between attachment, parenting and anxiety and depression (Brumiaru & Kerns, 2010).

An important consideration emphasised by a developmental psychopathology framework is whether these factors predict change in both depression and anxiety symptoms over time. Cross-sectional studies have dominated the literature over longitudinal designs (Bogels & Brechman-Toussaint, 2006; McLeod et al., 2007), which has impacted on our ability to understand these risk factors and their impact on depression and anxiety symptoms over time (Weems, 2008). Furthermore, although the current findings are to some extent consistent with Bowlby's (1973, 1980) hypothesis that attachment security is positively associated with anxiety and depression, because mood can influence the recollection of attachment-related events (Roisman, Fortuna & Holland, 2006), by utilising longitudinal studies this allows for

control regarding the initial levels of internalising symptoms. This may help disentangle the possible bidirectional effects between attachment and anxiety and depression.

The present study aimed to extend the literature by using a longitudinal design to focus on the transition from childhood into adolescence. This design allowed for the measurement of both depression and anxiety symptoms at several points through early and middle childhood and adolescence. It further allowed for testing whether the risk factors predicted changes in depression and anxiety symptoms over time. The study drew on a developmental psychopathology framework, in particular, the concept of equifinality, as an overarching theory where multiple pathways can lead to the same outcome. This is also in line with more contemporary models of anxiety and depression development that highlight the importance of multiple factors, among them the role of attachment, parenting, negative life events and parental mental health (DeKlyen & Greenberg, 2008; Goodman & Gotlib, 1999; Rapee, 2001).

The study looked specifically at two proposed questions (see figure 2.1). Firstly, whether parent-child relationship factors, specifically attachment and parenting, predicted both childhood and adolescent depression and anxiety symptoms. Consistent with the literature, it was hypothesised that children with insecure attachments to their parents and who experienced less sensitive parenting would be more likely to experience depression and anxiety symptoms and also experience increases in depression and anxiety symptoms over time (Colonnessi et al., 2011; McLeod et al., 2007).

Secondly, the study examined whether this relationship was moderated by family context factors, specifically; parental depression/anxiety, negative life events and the

interparental relationship. Based on previous research, it was predicted that this would be moderated where children of parents who: experienced more depression and anxiety symptoms (Bureau et al., 2009; Kerns et al., 2011); experienced more negative life events (Allen et al., 2008; Williamson et al., 2005); and had higher levels of family conflict within the interparental relationship (Papp, 2012) would be more likely to experience these increases in depression and anxiety symptoms over time.
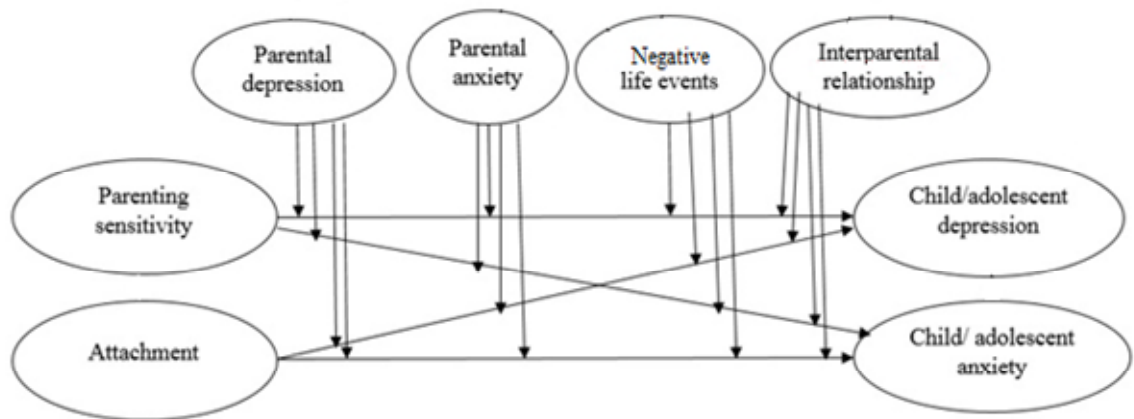
Figure 2.1 Proposed model of variables showing predicted moderator relationships

Method

Participants, Design, Procedure and Ethics

Participants were part of the NICHD Study of Early Child Care and Youth Development, a longitudinal study following children and their families in 10 locations across the USA. Children and their families were recruited from hospital visits shortly following the birth of their child in 1991. A total of 1,364 mothers, who completed a home interview when their infant was one month old, became the study

participants. The final child sample was 48% female and 80.4% Caucasian, 12.9% African American, and 6.7% another ethnicity.

Children were followed from birth until age 15 in four phases of data collection (for more details on procedure and data collection, see NICHD Early Child Care Research Network, 2001). For this study, data were utilised from phase one (one month to 36 months); phase two (54 months through first grade); phase three (second through sixth grades) and phase four (seventh grade, eighth grade and age 15). At age 15, measures of adolescent outcomes were obtained for 958 youth (70% of the original recruitment sample).

The following sections describe the particular measures used in the current analyses and the time points of administration. Further details about psychometric properties of the measures, all data collection procedures, and descriptions of how composites were derived and constructed can be found in the study's Manuals of Operation and Instrument Documentation (http://secc.rti.org). Procedures have been standardised across sites, and interviewers were trained and monitored for consistency.

This project used a publicly accessible dataset which had US ethical clearance. Specifically, the original ethical approval for the NICHD study was granted by each of the data-collecting universities before data collection and for every assessment informed consent was gained from parents and/or teachers. For more details please visit the NICHD study website (https://www.nichd.nih.gov/research/supported/Pages/seccyd.aspx).

Statistical Power

Using GPower, an apriori power analysis was calculated and the minimum target sample size generated was 620. This was based on a linear multiple regression test with five predictors. It was a two-tailed test with 80% power, α = .05 and with an effect size based on the literature of F^2 = .01. However, given that an interaction may be present and that the effect size estimate was based on literature conducted largely on cross-sectional studies which can have larger effect sizes than longitudinal studies (Colonessi et al., 2011), the study aimed to use a sample of 1240. This fitted the sample available for the secondary data analysis.

Measures

Measures of Attachment.

Children's attachment security was assessed on two occasions, at 15 and 36 months, using two different measures. At 15 months of age, quality of attachment was assessed using the Ainsworth and Witting (1969) Strange Situation procedure. As the Strange Situation is designed to measure attachment for infants aged between 12 to 18 months, in order to assess attachment styles in older children, modifications and other procedures have been developed to remedy this discrepancy. Therefore, Cassidy, Marvin and the MacArthur attachment working group's (1992) modified preschool Strange Situation procedure was used at 36 months.

Ainsworth and Wittig (1969) Strange Situation Procedure.

Assessments of attachment security using the Strange Situation occurred in laboratories when children were 15 months old. Standard procedures for conducting and coding the Strange Situation were followed (Ainsworth, Blehar, Waters & Wall,

1978). Coding involves evaluating children's behaviours during a series of departures and reunions between the mother and child in three-minute intervals. These episodes involved both the mother and a stranger being in the room with the child. The episodes are designed to activate the attachment system by creating a stressful situation through separation that produces attachment behaviours that are assessed upon reunification of the child and the mother.

Children's behaviours were recorded and all recordings were sent to one location for coding. Attachment security was assessed by a team of three coders. Each Strange Situation was scored independently by two coders using the standard classifications of secure (B), insecure-avoidant (A), insecure-resistant (C), disorganized (D), and unclassifiable (U). Disagreements were inspected by the group and a code was allocated by consensus. Across all coder pairs, before conferencing, agreement with the five-category classification system was 83% (kappa = .69) and agreement for the two-category system (secure/insecure) was 86% (kappa = .70). The strange situation procedure has demonstrated good construct validity (Ainsworth et al., 1978).

Cassidy, Marvin and the MacArthur Working Group (1992) Modified Strange Situation Procedure.

At 36 months, a modified Strange Situation procedure was conducted in the laboratory, following procedures outlined by Cassidy et al. (1992). In this modification, the procedure has fewer sessions of separation and reunification. Specifically, there were two separations, one lasting three minutes, the other lasting five minutes, and two reunions, both lasting three minutes. After the second reunion, the assessment was finished. The coding system produced the same attachment categories as the Strange Situation at 15 months, in addition to an attachment

security rating (scale of one to nine, where one = very insecure and nine = very secure). The codes used for children's behaviours were modified to be age appropriate as this procedure is designed for children aged between three and five. Intercoder agreement before consensus conferencing on the four-category classification was 76% (kappa = .58).

Measures of Parenting.

Parent-Child Interaction Task.

The Parent-Child Interaction Task (Egeland & Hiester, 1993) took place in the laboratory when the child was 36 months, 54 months and in first (age six to seven), third (age eight to nine) and fifth (age ten to eleven) grades. The task included recorded, 15-minute observations of mother-child interactions. The tasks varied across the time points to be developmentally appropriate but included tasks that were too hard for the child to undertake alone and needed the mother's instruction and assistance and also some tasks involved free play designed to elicit emotional expression. Parental behaviour was coded across all activities into the following scales: supportive presence, respect for autonomy, stimulation of cognitive development, quality of assistance, hostility, and confidence. Each score was coded along a seven-point Likert-type scale ranging from very low to very high. Internal consistency for the maternal sensitivity composite was acceptable across time points (α=.78 at 36 months; α=.78 at 54 months; α=.83 at first grade (age six to seven); α=.72 at third grade (age eight to nine); α=.74 at fifth grade (age ten to eleven)).

Outcome Measures.

Child Depression Symptoms.

The Child Depression Inventory-Short Form (CDI; Kovacs, 1992) is a self-report measure for assessing depression symptoms in children and adolescents that typically generates similar findings as the 27-item form (Kovacs, 1981). Children answer items measuring sadness, self-blame, and interpersonal relationships by choosing which of the three descriptions best suits how they have been feeling during the past two weeks (e.g., ''I do most things O.K., I do many things wrong, I do everything wrong'') for 10 items. It is scored on a zero-to-two scale, with higher total scores indicating more depression symptoms. The CDI was completed at fifth (age ten to eleven) and sixth grades (age 11 to 12) and at age 15. The CDI has been widely used in clinical and research settings and has good internal consistency, test-retest reliability, and discriminant validity (Kovacs, 1992).

Child Anxiety Symptoms.

The child's anxiety symptoms were measured using the Child Behavior Checklist (CBCL; Achenbach, 1991), which mothers completed eight times: at 54 months, kindergarten (age five to six), first (age six to seven), third (age eight to nine), fourth (age nine to ten), fifth (age ten to eleven) and sixth grades (age 11 to 12) and age 15. The CBCL is a commonly used assessment tool that measures problem behaviours and social competence in children and adolescents aged between four and 18. The study utilised the 12-item anxiety scale that has been previously used in research and demonstrated adequate internal consistency (Bosquet & Egeland, 2006; Feng et al., 2008; Kerns et al., 2011). As recommended by Wadsworth, Hudziak, Heath, and Achenbach (2001), this scale includes eight items from the Depression/Anxiety

Scale (Items 31, 32, 34, 45, 50, 71, 89, and 112) and four other items from the CBCL (Items 9, 29, 30, 66). The mean of these items was calculated to form an anxiety score where higher scores represented more anxiety symptoms. Mothers completed the CBCL using a three-point scale ranging from zero (not true of the child) to two (very true of the child). Internal consistency was adequate across time points (α=.68 at 54 months; α=.67 at kindergarten (age five to six); α=.69 at first grade (age six to seven); α=.74 at third grade (age eight to nine); α=.74 at fourth grade (age nine to ten); α=.73 at fifth grade (age ten to eleven) ; α=.75 at sixth grade (age 11 to 12) and α=.76 at age 15).

Moderator Measures.

Parental Depression.

Parental reports of depression were obtained with the Center for Epidemiological Studies–Depression Scale (CES-D; Radloff, 1977) when children were one month, six months, 15 months, 24 months, 36 months, and 54 months and at each assessment from first through sixth grade (i.e., at ages seven years through 12 years) and at age 15, for a total of 11 assessments. The CES-D is a widely used, 20-item self-report assessment tool with well documented psychometric properties (Roberts, Lewinsohn, & Seeley, 1991) that measures depression symptoms within the past two weeks. Parents state how frequently in the last week they have experienced cognitive, somatic, and affective components of depression on a zero (not at all) to four (five–seven days) scale. Examples include ''I felt that everything I did was an effort'' and ''I was bothered by things that usually don't bother me.'' Scores can range from zero to 60; scores of 16 or higher are understood to have clinical significance. Individuals diagnosed with clinical depression score higher on the

CES-D than individuals without depression. Previous research has demonstrated that this measure has adequate psychometric properties (Radloff, 1977).

Parental Anxiety.

Parental anxiety was assessed using the 10 state items from the State–Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), which was adapted for the NICHD study by questioning how the respondent felt over the last week rather than how the respondent felt at that moment. Parents scored their anxiety on a four-point scale, where a score of one = not at all, and a score of four = very much. An example of an item is "I feel nervous and restless." Higher scores represent more anxiety symptoms. Data was collected whilst the child was in first (age six to seven), third (age eight to nine), fifth (age ten to eleven) and sixth (age 11 to 12) grade and at age 15. The scales have good convergent validity with other questionnaire measures of anxiety and has high internal consistency (.89 to .95) (Spielberger et al., 1983).

Negative Life Events.

At 54 months, third grade (age nine to ten) and fifth grade (age 11 to 12), mothers filled out the Life Experiences Survey, a 57-item questionnaire where they specified which major life events had happened during their lives in the past year, and then they rated the impact of these events (Sarason, Johnson & Siegel, 1978). The events ranged from routine events (e.g., child started school) to more major and catastrophic events (e.g., death of a close family member). The impact of each event was rated on a seven-point scale ranging from minus three to positive three where a rating of positive three represented that the event was very positive, zero represented that it was a neutral event, and minus three represented that the event had been very

negative. Items with a negative impact score were used to calculate a score for negative life events. The negative life event scores were reversed and summed at each time point where higher scores indicated a greater number of negative life events. Previous research has demonstrated that this measure has adequate psychometric properties (Sarason et al., 1978).

Interparental Relationship.

Parents completed the six-item intimacy subscale of the Personal Assessment of Intimacy in Relationships (PAIR) (Schaefer & Olson, 1981) at one month, 36 months, 54 months, first (age six to seven), third (age eight to nine), fifth (age ten to eleven), sixth grade (age 11 to 12) and age 15. This subscale includes six items that monitor feelings of relationship emotional closeness and support. Sample items include, "I can state my feelings without him getting defensive" and "I sometimes feel lonely when we're together" (reversed). Items were rated on a five-point Likert response scale (from one = strongly disagree to five = strongly agree). The PAIR Scale has shown good reliability and validity (Schaefer & Olson, 1981). Lower scores represent greater intimacy in the relationship.

Missing Data

The number of participants with valid data varied significantly across time and across measures (see Table 2.1 for a summary). So that participants with missing data were not excluded, which can bias parameter estimates and undermine statistical power (Allison, 2003), the full information maximum likelihood (FIML) method was used. This method uses all of the available data to estimate the parameter estimates of a model and calculates the log-likelihood of the data for each observational unit individually. This approach is superior to mean substitution and

list-wise deletion and is comparable to multiple-imputation (Allison, 2003; Schafer & Graham, 2002).

Data Analysis

Prior to analyses, the data was analysed for normality using univariate and graphical approaches including inspections of skewness and kurtosis. In addition, boxplots were inspected for outliers based on the criteria of one and a half times the interquartile range. Upon inspection, transformations of the data were conducted where appropriate. Specifically, logarithmic, square root and reflective transformations were employed which improved the normality of the data and also resulted in no extreme outliers being present. In addition, by using the (FIML) estimation procedure, this had the advantage of being robust against moderate departures from normality (Joreskog & Sorbom, 1989).

A developmental approach to data analysis was chosen, using structural equation modelling (SEM). SEM was ideal for measuring relationships between variables across time while controlling for earlier influences, and for exploring multiple pathways simultaneously. It also allows for the measurement of overall growth, the estimation of the heterogeneity in the growth curve at the individual level, and for efficient estimation of effects when there is missing data (Curran & Willoughby, 2003). The main hypotheses were tested using longitudinal structural equation models in which growth in anxiety and depression between childhood and adolescence were modelled as latent growth curves (LGCs), and antecedents and moderators entered as predictors of the intercept and slope of the respective latent growth curves. Relevant covariates were also entered as predictors of the intercept and slope so that their effects are adjusted in all analyses. Latent growth curve modelling allowed for the identification of how predictors (parenting sensitivity and

attachment) were related to initial levels of anxiety or depression (i.e., intercept for anxiety or depression) and to increases in anxiety or depression symptoms across childhood such as age-related changes (i.e., slope for anxiety or depression).

The child anxiety models were constructed to estimate the initial level at 54 months (the intercept parameter) and growth from 54 months to age 15. The child depression models were constructed to estimate the initial level at grade five (age 11-12; the intercept parameter) and growth from grade five (age 11-12) to age 15. The overall means and variances in these parameters were estimated using the FIML method. The quality of the model fit was simultaneously tested using the FIML ratio-test statistic and indices of model fit (i.e. the comparative fit index [CFI], and the root-mean-square error of approximation [RMSEA]). CFI values of .90 or above (Hu & Bentler, 1999) and RMSEA values of .08 or lower are considered to indicate an acceptable model fit (Bentler, 1990).

Once the growth models of child anxiety and child depression were completed, the relation to attachment security was explored. Attachment was treated as a binary independent variable where scores of zero represented an insecure attachment style and scores of one represented a secure attachment style. Both the slopes and intercepts of child anxiety symptoms and child depression symptoms were regressed separately on attachment security.

To minimise measurement error, multiple measures of parenting sensitivity were used, creating several indicators of the latent constructs. The measure was constructed using the same variables as used by Kerns et al. (2011), using the Parent-Child Interaction task, ratings from supportive presence and reversed ratings from hostility, both related to parental rejection, and ratings from respect for autonomy,

related to parental control, were summed for each task. A latent variable analysis extracted the common variance between them to create one overall measure of parenting sensitivity. Higher scores on this overall measure of parenting sensitivity represent more insensitive parenting. The overall parenting sensitivity measure was then regressed onto both the slopes and intercepts of child anxiety and child depression separately. Again, the quality of the model-fit for both attachment and parenting sensitivity as predictors was ascertained by inspecting the CFI and RMSEA values.

Finally, moderator analyses were conducted where interaction terms were included in the LGC models for both child depression and anxiety with parenting and attachment used as predictors. To create robust indices of family risk, the average across all time points was used for all moderators. All analyses were conducted using MPlus Version 8 (Muthén & Muthén, 2017).

Results

The results are presented in four subsections: (a) descriptive statistics and the overall LGC models for child depression and child anxiety, respectively, over grades five (age 11-12) to age 15, and 54 months to age 15; (b) the latent variable analysis of parenting sensitivity and its association with the growth curves of child anxiety and depression (c) the association between attachment and the growth curves of child anxiety and depression and (d) the role of maternal depression, maternal anxiety, frequency of negative life events and the interparental relationship in moderating the association between attachment and parenting on the growth curves of child anxiety and depression.

Descriptive Statistics and LGC Modelling of Child Depression and Anxiety

Table 2.1 presents the descriptive statistics for child depression and child anxiety

from grade five (age ten to eleven) to age 15 and 54 months to age 15, respectively

as well as the correlations between these variables. Table 2.2 presents the zero-order

correlations among the main study variables. Table 2.1 shows that, overall,

children's depression levels steadily increased over time; this suggested a general

increase and a linear pattern to the data and therefore a linear growth curve would

best describe the pattern of growth. A latent growth curve model comprising a

random intercept and a linear slope proved a good fit to the data, X^2 (1) =, p= .560;

CFI= 1.000; RMSEA <.01 (95 % CI =.000- .076). Age-related increases in

depression as indicated by the LGC slope, were significant (β = .473, SE= .142,

p=.001). The variance of the slope was not significant (variance =.013, SE = .007, p=

.052) suggesting there were not substantial individual differences in patterns of

change over time. The intercept was also significant (β= 1.338, SE=.106, p<.001)

and showed significant variance (variance =.213, SE =.027, p <.001).

Overall, child anxiety levels were relatively stable over time with a slight decrease

between 54 months and age 15, as shown in table 2.1. This suggested a linear pattern

to the data. A latent growth curve model with intercept and linear slope was thus

chosen as the best fitting model. The chi-square test of overall model fit was

significant, X^2 (31) = 258.747, p <.001, although this largely reflects the large

sample size, as the goodness of fit statistics showed that the model fit the data well

(CFI =.941; RMSEA = .008 (90% CI = .071-.089)). Examination of the slope

estimates showed that anxiety symptoms decreased with age (β = -.264, SE= .049,

p<.001). The intercept was also significant (β= 2.021, SE=.067, p<.001). Significant

individual variation occurred in the intercept and slope variance (intercept variance =

.036, SE = .002; linear slope variance = .000, SE = .000).

Table 2.1
Correlation matrix, means, standard deviations and minimum and maximum Ns for anxiety and depression outcome variables

	1	2	3	4	5	6	7	8	9	10	11
1.Anxiety 54 months		.598***	.553***	.439***	.467***	.437***	.374***	.370***	.035	.051	.009
2.Anxiety Kindergarten (age 5-6)			.586***	.505***	.513***	.466***	.406***	.399***	.014	.022	.035
3.Anxiety Grade 1 (age 6-7)				.564***	.578***	.527***	.492***	.477***	.094**	.093**	.106**
4.Anxiety Grade 3 (age 8-9)					.731***	.656***	.612***	.561***	.130***	.122***	.149***
5.Anxiety Grade 4 (age 9-10)						.701***	.649***	.598***	.150***	.141***	.136***
6.Anxiety Grade 5 (age 10-11)							.697***	.602***	.152***	.133***	.187***
7.Anxiety Grade 6 (age 11-12)								.647***	.193***	.202***	.201***
8.Anxiety Age 15									.096**	.154***	.233***
9.Depression Grade 5 (age 10-11)										.502***	.245***
10.Depression Grade 6 (age 11-12)											.331***
11.Depression Age 15											
Mean	.209	.191	.199	.210	.194	.208	.191	.171	1.281	1.408	2.005
SD	.200	.192	.198	.216	.211	.215	.216	.200	1.951	2.154	2.636
Max N	1057	1046	1009	1007	992	995	987	931	1019	1011	957
Min N	860	881	862	886	888	894	905	860	899	903	870

Note. ***p<.001, ** p<.01, *p<.05

Table 2.2

Correlation matrix of main study variables with means and standard deviations

	1	2	3	4	5	6	7	8	9	10	11
1.Anxiety 54 months		.370***	.035	.009	-.003	-.074*	.145***	.296***	.251***	.137***	.191***
2.Anxiety Age 15			.096**	.233***	.018	-.081*	.047	.308***	.350***	.174***	.219***
3.Depression Grade 5 (age 10-11)				.245***	-.014	.022	.085**	.164***	.107***	.097**	.113***
4.Depression Age 15					-.031	-.045	-.010	.127**	.152**	.148**	.157**
5.Attachment 15 months						.055	-.057	-.018	-.006	.005	-.046
6.Attachment 36 months							-.183***	-.077*	-.030	.054	-.001
7.Parenting								.331***	.200***	-.103***	.095**
8.Parental depression									.770***	.276***	.484***
9.Parental anxiety										.290***	.443***
10.Negative events											.273***
11.Interparental relationship											
Mean	.209	.171	1.281	2.005	.690	.610	.000	9.467	17.563	3.264	4.096
SD	.200	.201	1.951	2.636	.462	.487	.268	6.163	4.124	2.550	.706

Note. ***$p<.001$, ** $p<.01$, *$p<.05$, all values are rounded to three decimal places.

Parenting Sensitivity Latent Variable Analysis

A one-factor latent variable analysis of the sensitivity measures provided a good fit to the data, X^2 (85) = 310.814, $p<.001$; CFI= .931 RMSEA = .062 (90% CI = .055-.070). Table 2.3 presents the standardised model results for overall parenting sensitivity by time point factors and shows that each time point was significant suggesting that each time point was individually contributing similarly to the overall latent variable, parenting sensitivity.

Table 2.3

Confirmatory factor analysis standardised coefficients and standard errors for overall parenting sensitivity by time point factors

Time point	β	SE	*p*
36 months	.702	.045	<.001***
54 months	.696	.040	<.001***
Grade 1 (age 6-7)	.758	.038	<.001***
Grade 3 (age 8-9)	.723	.038	<.001***
Grade 5 (age 10-11)	.676	.041	<.001***

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

Parenting Sensitivity and the Growth of Child Depression and Child Anxiety.

LGC models were estimated in order to investigate the main effects of parenting sensitivity on both the intercept and change parameters for children's depression and anxiety levels.

For child depression symptoms the effect of parenting sensitivity on the intercept was significant (β = .138, SE= .054, $p=.011$). The effect of parenting sensitivity on the slope, however, was not significant, β = -.060, SE= .069, $p=.384$. The results

showed that children who experienced less sensitive parenting had a greater level of depression symptoms at grade five (age ten to eleven) compared to children who experienced more sensitive parenting. However, parenting sensitivity did not significantly explain the changes in depression symptoms over time.

For child anxiety symptoms, the effect of parenting sensitivity on both the slope and intercept was significant, (slope β = -.111, SE= .047, $p<.018$; intercept β = .128, SE= .034, $p<.001$). Figure 2.2 shows that children who experienced less sensitive parenting had higher levels of anxiety compared to those with more sensitive parents at 54 months. Additionally, figure 2.2 shows that children who experienced less sensitive parenting showed increases in anxiety symptoms over time whereas children who experienced more sensitive parenting showed decreases in anxiety symptoms over time.
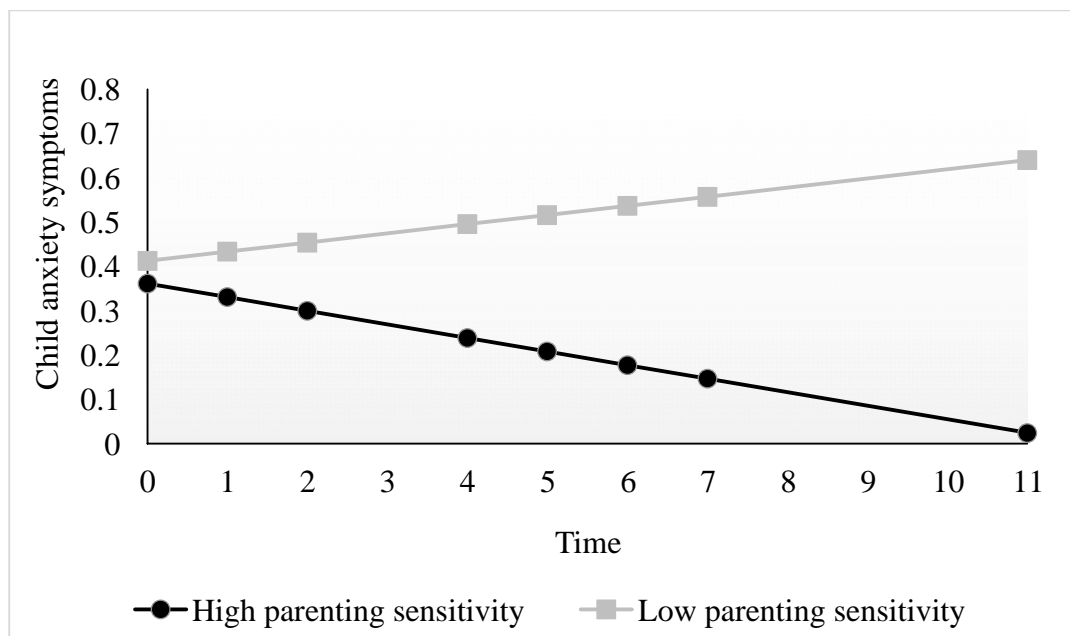

Figure 2.2 A graph showing the main effect of parenting sensitivity for child and adolescent anxiety symptoms. Note. Child anxiety symptoms are logarithmically transformed.

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Attachment Security and the Growth of Child Depression and Child Anxiety

Table 2.4 presents both the sample means and standard deviations of child depression scores and child anxiety scores by attachment group (secure vs. insecure attachment) as measured at both 15 and 36 months of age. Table 2.4 shows that depression slightly increases over time for both insecurely attached and securely attached individuals. However, there are slightly higher scores of depression for adolescents at age 15 who were classified as having insecure attachments versus those with secure attachments. Table 2.4 also shows that anxiety was relatively stable over time for both insecurely and securely attached groups.

Table 2.5 presents the intercept and growth parameter estimates of the child depression symptom model and the child anxiety symptoms model as predicted by attachment at 15 months and 36 months respectively. Table 2.5 shows that attachment at 15 months did not significantly predict child depression or child anxiety symptoms for either model. Table 2.5 also shows that the effect of attachment security at 36 months was not associated with initial child depression scores. However, attachment security at 36 months did predict change over time where children with insecure attachments to their mothers at 36 months had greater growth in depression symptoms over time. In contrast, those with secure attachments to their mothers showed less growth in depression symptoms over time.

Table 2.5 also shows that children with insecure attachments at 36 months had higher levels of anxiety initially at 54 months compared to children with secure attachments. However, attachment security at 36 months did not predict later changes in anxiety over time.

Table 2.4

Sample means and standard deviations of child depression scores and child anxiety scores by attachment group as measured at both 15 and 36 months of age.

	Insecure (15 months) (31%)		Secure (15 months) (69%)		Insecure (36 months) (39%)		Secure (36 months) (61%)	
Time	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)
Anxiety								
54 months	305	.211 (.211)	711	.209 (.198)	388	.227 (.215)	626	.197 (.188)
Kindergarten	307	.203 (.204)	696	.188 (.189)	372	.204 (.192)	624	.180 (.181)
Grade 1	297	.204 (.204)	677	.199 (.197)	360	.216 (.207)	600	.188 (.188)
Grade 3	295	.214 (.227)	662	.205 (.205)	345	.236 (.233)	588	.185 (.191)
Grade 4	290	.201 (.213)	654	.195 (.213)	344	.217 (.240)	578	.179 (.188)
Grade 5	294	.216 (.218)	653	.206 (.216)	341	.232 (.250)	578	.192 (.184)
Grade 6	290	.191 (.209)	646	.193 (.220)	337	.205 (.244)	576	.180 (.192)
Age 15	272	.169 (.185)	611	.177 (.210)	317	.193 (.227)	546	.160 (.181)
Depression								
Grade 5	301	1.340 (1.888)	666	1.281 (1.995)	355	1.231 (2.001)	583	1.321 (1.946)
Grade 6	298	1.435 (2.118)	661	1.400 (2.149)	352	1.314 (2.111)	582	1.458 (2.179)
Age 15	280	2.140 (2.556)	630	1.960 (2.706)	336	2.142 (2.696)	551	1.897 (2.597)

Table 2.5

Latent growth curve analysis standardised coefficients and standard errors for attachment security for both child depression symptoms and child anxiety symptoms

	Attachment 15 months			Attachment 36 months		
	β	S.E.	*p*	β	S.E.	*p*
Child Depression						
Intercept	.034	.057	.552	.063	.058	.281
Slope	-.127	.080	.110	-.219	.094	.020*
Child Anxiety						
Intercept	-.023	.036	.531	-.095	.036	.008**
Slope	.010	.050	.834	-.014	.050	.786

Note. ***p<.001, ** p<.01, *p<.05

Moderators and the Growth of Child Depression and Child Anxiety

Child Depression Symptoms

As presented in table 2.6, maternal depression and negative life events were significant predictors of the intercept of child depression in the model that included parenting. The results showed that children whose mothers were showing more symptoms of depression and had experienced more negative life events were more likely to have depression symptoms at grade five (age ten to eleven). Although the interparental relationship was not directly associated with the intercept or slope for depression it did show a significant interaction with parenting sensitivity in relation to the intercept. As shown in figure 2.3, for children experiencing greater parenting sensitivity those who also had greater intimacy in the interparental relationship had significantly fewer depression symptoms at grade five (age ten to eleven) compared to children whose parents reported less intimacy in the interparental relationship. In contrast, children experiencing lower parenting sensitivity did not show significant differences in depression symptoms according to the intimacy level in the interparental relationship. This finding, however, should be noted with caution as the interaction effect was weak and only just significant at the five percent level.

Table 2.6

A series of latent growth curve analyses standardised coefficients and standard errors for child depression symptoms.

	Intercept			Slope		
Child depression	β	S.E.	*p*	β	S.E.	*p*
Parenting	.176	.181	.329	-.041	.229	.859
Maternal depression	.187	.056	.001***	-.036	.074	.633
Interaction	-.118	.178	.509	.010	.226	.966
Parenting	-.107	.672	.873	.216	.831	.795
Maternal Anxiety	.109	.056	.052	.075	.072	.299
Interaction	.212	.670	.752	-.284	.827	.732
Parenting	.287	.122	.019*	-.137	.168	.414
Life events	.113	.054	.035*	.055	.073	.450
Interaction	-.141	.122	.248	.077	.164	.639
Parenting	.313	.105	.003**	-.094	.120	.437
Interparental relationship	.057	.061	.351	.098	.070	.165
Interaction	-.204	.101	.044*	.014	.115	.906
Attachment 15 months	-.211	.173	.223	-.091	.237	.700
Maternal depression	.106	.091	.241	-.008	.123	.950
Interaction	.299	.185	.106	-.074	.255	.772
Attachment 15 months	-1.008	.681	.139	.451	.916	.623
Maternal Anxiety	.014	.097	.886	.136	.135	.314
Interaction	1.078	.685	.115	-.621	.927	.502
Attachment 15 months	-.056	.131	.668	-.396	.217	.069
Life events	.074	.088	.403	-.101	.129	.434
Interaction	.118	.150	.428	.305	.228	.181
Attachment 15 months	-.080	.119	.502	-.097	.137	.479
Interparental relationship	.017	.114	.883	.099	.130	.449
Interaction	.190	.149	.202	-.030	.170	.861
Attachment 36 months	.324	.179	.071	-.495	.262	.059
Maternal depression	.316	.085	<.001***	-.163	.116	.162
Interaction	-.263	.184	.153	.304	.248	.220
Attachment 36 months	.962	.689	.163	-1.579	.957	.099
Maternal Anxiety	.245	.089	.006**	-.100	.115	.384
Interaction	-.891	.688	.195	1.372	.933	.141
Attachment 36 months	.173	.138	.211	-.453	.227	.046*
Life events	.184	.092	.044*	-.071	.127	.574
Interaction	-.141	.156	.367	.272	.224	.226
Attachment 36 months	.127	.121	.293	-.228	.140	.104
Interparental relationship	.149	.111	.181	.101	.121	.401
Interaction	-.053	.146	.715	.020	.158	.902

Note. ***p<.001, ** p<.01, *p<.05

Table 2.6 shows that no interactions were present for all proposed moderator variables regarding the relationship between attachment and child depression symptoms.
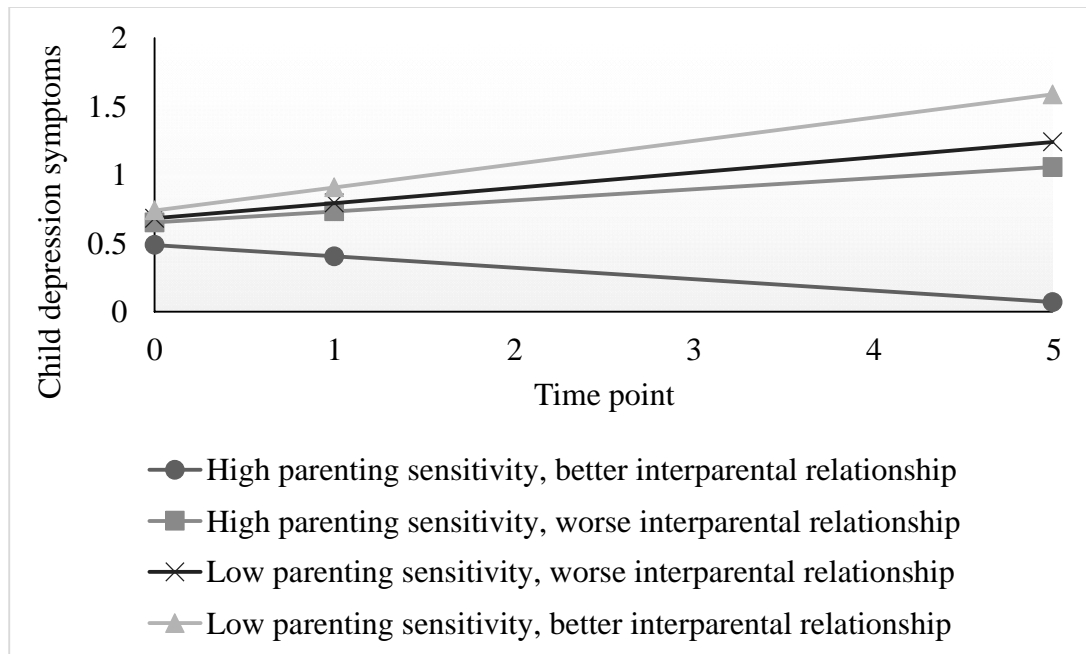

Figure 2.3 A graph showing the interaction between parenting sensitivity and the interparental relationship for child and adolescent depression symptoms

Child Anxiety Symptoms.

As presented in table 2.7, maternal depression, maternal anxiety, negative life events and the interparental relationship were all significant predictors of child anxiety symptoms at both the intercept and the slope in the parenting model. This suggests that children whose mothers were showing more symptoms of depression and anxiety, had experienced more negative life events and whose parents had a poorer quality interparental relationship were more likely to experience anxiety symptoms at 54 months and that these symptoms may grow more rapidly over time. Furthermore, an interaction was present in the parenting model for both the intercept and the slope. As shown in figure 2.4, children experiencing lower parenting

sensitivity and whose mothers had more symptoms of anxiety had more anxiety symptoms at 54 months and greater increases in anxiety symptoms over time. Seemingly, lower sensitive parenting had a greater effect on children whose mothers experienced anxiety symptoms compared to those with greater parenting sensitivity, both in relation to the intercept and the slope. In contrast, for children whose mothers had lower levels of anxiety, parenting sensitivity did not affect growth in anxiety symptoms. Again, caution is noted when considering the significance of the interaction at the slope as this only just reached significance.

Table 2.7 also shows that interactions were not present for all proposed moderators of the relationship between attachment and child anxiety symptoms.
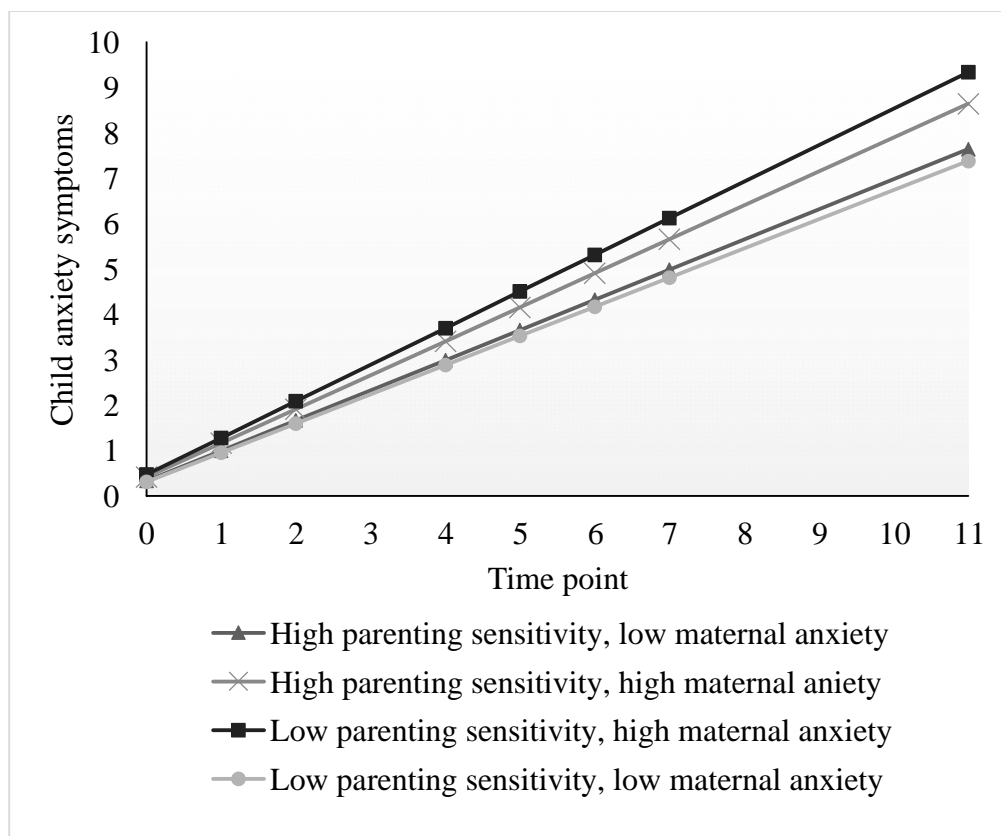

Figure 2.4 A graph showing the interaction between parenting sensitivity and maternal anxiety for child and adolescent anxiety symptoms.

Table 2.7

A series of latent growth curve analyses standardised coefficients and standard errors for

child anxiety symptoms

	Intercept			Slope		
Child anxiety	β	S.E.	*p*	β	S.E.	*p*
Parenting	-.142	.107	.185	.035	.231	.817
Maternal depression	.368	.033	<.001***	.098	1.982	.047*
Interaction	.149	.105	.157	-.178	-1.192	.233
Parenting	-1.179	.438	.007**	1.044	.601	.083
Maternal Anxiety	.324	.034	<.001***	.187	.048	<.001***
Interaction	1.241	.436	.004**	-1.191	.600	.047*
Parenting	.186	.077	.016*	-.206	.105	.051
Life events	.227	.034	<.001***	.116	.048	.015*
Interaction	-.040	.078	.609	.120	.106	.255
Parenting	.112	.066	.089	-.191	.092	.038*
Interparental relationship	.222	.038	<.001***	.147	.053	.006**
Interaction	-.037	.066	.576	.128	.090	.154
Attachment 15 months	-.093	.104	.370	-.002	.151	.990
Maternal depression	.321	.058	<.001***	.059	.086	.493
Interaction	.092	.114	.420	.009	.166	.957
Attachment 15 months	-.086	.446	.847	.209	.612	.732
Maternal Anxiety	.310	.064	<.001***	.207	.088	.019*
Interaction	.065	.448	.884	-.198	.616	.748
Attachment 15 months	.112	.085	.188	-.065	.116	.574
Life events	.311	.060	<.001***	.058	.084	.487
Interaction	-.180	.099	.068	.109	.135	.420
Attachment 15 months	.053	.073	.464	-.096	.100	.340
Interparental relationship	.311	.074	<.001***	-.020	.103	.847
Interaction	-.114	.095	.232	.192	.131	.143
Attachment 36 months	-.029	.107	.788	-.139	.154	.366
Maternal depression	.370	.052	<.001***	-.017	.077	.824
Interaction	-.039	.111	.724	.163	.162	.312
Attachment 36 months	.031	.446	.945	-.550	.618	.373
Maternal Anxiety	.322	.056	<.001***	.107	.079	.176
Interaction	-.099	.447	.825	.562	.619	.364
Attachment 36 months	-.052	.087	.551	-.031	.120	.797
Life events	.256	.059	<.001***	.102	.083	.222
Interaction	-.059	.101	.561	.030	.140	.828
Attachment 36 months	-.094	.068	.168	-.077	.095	.420
Interparental relationship	.222	.063	<.001***	.033	.088	.711
Interaction	.025	.085	.768	.080	.118	.495

Note. ***p<.001, ** p<.01, *p<.05.

Discussion

In this section, the key findings are summarised and linked with previous research. Limitations, as well as strengths of the research, are then discussed. Lastly, both theoretical and clinical implications are explored.

The current study used a longitudinal design and latent growth curve modelling to assess whether parent-child relationship factors, specifically attachment and parenting, predicted childhood and adolescent depression and anxiety. Furthermore, the question of whether this relationship was moderated by family context was addressed, specifically, by examining the moderating role of parental depression and anxiety, negative life events and the interparental relationship.

Longitudinal analyses revealed that children who experienced less sensitive parenting tended to experience higher levels of depression and anxiety at the initial time points of grade five (age ten to eleven) and 54 months respectively. Low parenting sensitivity was also associated with larger increases in anxiety symptoms over time. However, parenting sensitivity did not explain changes in depression symptoms over time. These results therefore partially support the proposed hypothesis that children who experience less sensitive parenting would be more likely to experience depression and anxiety symptoms and also experience increases in depression and anxiety symptoms over time. This relationship between parenting and both depression and anxiety in childhood supports previous literature (Mcleod et al., 2007) but differs from some other studies that although found trends relating parenting sensitivity and anxiety development, did not find significant effects (Kerns et al., 2011). The difference in findings may reflect that Kerns et al. (2011), for

example, examined the unique contribution of variables within one model rather than examining variables in separate analyses as the current study did.

The analyses did not find evidence that attachment at 15 months predicts child depression or anxiety symptoms. However, attachment security at 36 months did predict change over time in depression, with children who were insecure at 36 months showing higher levels of depression over time between grade five (age ten to eleven) and age 15. Interestingly, the reverse pattern was found for childhood anxiety, whereby children with insecure attachments at 36 months had higher levels of anxiety initially at 54 months, but this did not predict later changes in anxiety over time. These results therefore partially support the hypothesis that children who have early insecure attachments to their mothers are more likely to experience depression and anxiety symptoms and also experience increases in depression and anxiety symptoms over time. Furthermore, the results add some support to reviews finding that attachment is associated with childhood and adolescent depression and anxiety symptoms (Brumariu & Kerns, 2010; Colonnesi et al., 2011). It also supports meta-analyses which show a significant association between insecure attachment and internalising symptoms across multiple existing studies, although the effect size was small (Groh et al., 2012).

This difference between attachment measured at different time points may add to the complex picture of the relationship between attachment and anxiety symptoms in childhood. For example, support was provided to some literature in that no predictor effect was found for infancy attachment and childhood anxiety (Feng et al., 2008). However, the findings also supported that attachment measured beyond infancy was associated with children's anxiety symptoms (Kerns et al., 2011).

The difference in findings when attachment was measured at 15 months versus 36 months may represent a time artefact whereby the shorter gap between assessments of attachment and anxiety symptoms may be partly responsible. However, the fact that attachment at 36 months still predicted increases in depression from grade five (age ten to eleven) onwards makes this less suggestion less plausible. It is, therefore, possible that changes in the developmental character of attachment may occur after infancy which in turn may amplify its predictive capacity (Fearon, Bakermans-Kranenburg, Van IJzendoorn, Lapsley & Roisman, 2010). Relatedly, it may be that attachment remains relatively stable across childhood but is open to considerable change between infancy and toddlerhood (Fraley, 2002) and therefore attachment security measured in infancy may be less predictive of long-term outcomes. This indeed is consistent with meta-analytic findings, which show a similar pattern of results (Fearon et al., 2010; Colonessi, et al., 2011).

The analyses suggested that children whose mothers were showing more symptoms of depression and anxiety and had experienced more negative life events experienced more depression symptoms at grade five (age ten to eleven). In general, however, the results did not find consistent support for the notion that family context variables such as these moderate the association between the parent-child relationship and later childhood anxiety and depression. Findings showed that for children experiencing greater parenting sensitivity those who also had greater intimacy in the interparental relationship had significantly fewer depression symptoms at grade five (age ten to eleven) compared to children experiencing less intimacy in the interparental relationship. In contrast, children experiencing less parenting sensitivity did not show significant differences in depression symptoms according to the intimacy level in the interparental relationship.

The moderator analyses also suggested that childhood anxiety was associated with maternal symptoms of depression and anxiety, negative life events and a poorer quality interparental relationship, in relation to both the intercept (at 54 months) and the growth of child anxiety over time. Furthermore, the results showed that maternal anxiety was a moderator of the relationship between parenting sensitivity and childhood anxiety symptoms at 54 months and also over time. It showed that less sensitive parenting had a stronger association with child anxiety for those with mothers also experienced anxiety symptoms. This was the case for both the intercept (at 54 months) and the growth of childhood anxiety over time. In contrast, for children whose mothers had lower levels of anxiety, parenting sensitivity did not seem to affect growth in anxiety symptoms. Interactions were not present for all other proposed moderator variables.

The results also revealed no significant interactions between any of the proposed moderators and attachment in relation to childhood depression or anxiety symptoms. This is quite consistent with meta-analyses of attachment and internalising problems which did not reveal any reliable study-level moderators (Groh et al., 2012).

In summary, only partial support was found for the hypothesis that the effects of the parent-child relationship in childhood and adolescent anxiety and depression might be moderated by maternal mental health, life events or marital distress. Also, when considering the positive findings reported herein, caution is warranted as the interactions were weak and although there was some indication of a difference in slope, the finding is questionable given the number of tests that were run and it is important not to base conclusions on the arbitrary .05 probability level (Palesch, 2014).

Taken together, these findings add support to other research in that maternal depression (Feng et al., 2008); maternal anxiety (Kerns et al., 2011); experiencing more negative life events (Allen et al., 2008; Kerns et al., 2011) and worse interparental relationships (Nomura et al., 2002) were related to childhood anxiety symptoms. The findings also add support to research finding that parental depression symptoms (Bureau et al., 2009); parental anxiety symptoms (Biederman, et al., 1991; Burstein et al., 2010) and experiencing more negative life events (Williamson et al., 2005) were associated with childhood depression symptoms.

Limitations and strengths

A key limitation of the current study was that the anxiety outcome measure was a questionnaire completed by the child's mother. Questionnaires completed by parents alone have performed poorly compared with self-report measures, as parents tend to underestimate children's internalising symptoms (van der Ende & Verhulst, 2005). With this in mind, although the current data was collected at an age where children would have been unable to validly self-report on their anxiety symptoms (54 months), later time points could have used self-report or clinician-directed measures. However, multi-informant and multi-response assessment are recommended (Silverman & Ollendick, 2005). The design of future studies should consider these important measurement issues in order to improve the reliability and validity of findings.

In addition, the anxiety data were created based on questions from the CBCL that clustered different types of anxiety disorders and internalising problems together as a whole. Future research could consider using specific anxiety measures that are able to tease these apart as research has shown that different types of anxiety disorders

tend to occur at different development stages (Weems, 2008). For example, separation anxiety is more dominant between the ages of six and nine whereas social anxiety may be more dominant between the ages of 14 and 17 (Weems, 2008).

A further limitation of the study was that a community sample was used where the incidence of childhood depression and anxiety are likely to be low. It may be that this population will have produced findings that are not generalisable to clinical populations. For example, there may be a difference between experiencing some anxiety or depression symptoms compared to meeting a clinical diagnosis where quality of life may be more severely impacted.

In addition, the current study did not consider the moderators of parenting, attachment and anxiety and depression together. It may be that the amount of risk factors combined with insecure attachment or negative parenting behaviours is more critical than experiencing an insecure attachment or negative parenting behaviours in combination with a particular risk factor. Furthermore, the current study considered the moderators in isolation. Without this integrative research, understanding the comparative extent these different factors have and also whether interactive effects may exist is limited.

A further limitation in the current study was that only mothers' parenting, attachment and mental health was examined and not fathers'. This decision was made as the NICHD study dataset routinely collected many measures from the mother but fewer from the father (such as for the strange situation procedure at 15 months and 36 months). Future research should consider this when designing measure schedules so that the role of fathers in the development of childhood and adolescent depression and anxiety symptoms can also be examined.

A key strength of this study was that both attachment and parenting measures were observational and hence did not rely on self-reports. Such measurements are arguably more robust and certainly less prone to bias and are considered the gold standard when it comes to measuring parenting (Morsbach & Prinz, 2006; Sessa et al., 2001) and attachment (Solomon & George, 1999; van Ijzendoorn & Sagi, 1999).

Theoretical Implications of the Findings

The results of this study highlight the importance of considering the development of depression and anxiety through a developmental psychopathology lens in particular regarding equifinality. Specifically, equifinality refers to there being multiple pathways to the same outcome (e.g., depression symptoms) and that a single risk factor such as attachment, for example, cannot universally account for the development of anxiety or depression symptoms.

With this framework in mind, more recent theoretical models of depression and anxiety have challenged the traditional models which focused on single main effects as these did not explain a lot of the variance associated with anxiety symptoms in children, for example (Vasey & Dadds, 2001).

Instead, contemporary models have considered multiple risk factors and view the development of anxiety and depression as a complex, multi-determined process (e.g., Goodman & Gotlib, 1999; Rapee, 2001; Vasey & Dadds, 2001). For example, Rapee (2001) posits that genetic factors, parenting and family socialisation, parental anxiety, stressful life events and an individual anxious vulnerability (e.g., temperament) may each explain anxiety development. In addition, DeKlyen and Greenberg (2008) posit that attachment, ineffective parenting, typical child characteristics and high family adversity may explain the development of anxiety.

Overall, these models have proposed a set of factors that contribute to the development of depression and anxiety namely, parent-child relationship, family-context and individual child factors, that all play a role and the current study was able to add support specifically to the parent-child and family-context facets.

In sum, these findings offer convergent evidence that parenting and attachment are associated with both child depression and anxiety symptoms and provides support for theoretical models that include the role of attachment and parenting in the development and maintenance of symptoms. However, it is key to note that this association with parenting and attachment should be considered within the context of other influential factors, particularly family context factors such as negative life events, parental mental health and the interparental relationship. Furthermore, the role of parenting and attachment appears to be smaller than what is emphasised in both the academic literature and wider lay population context and it is important that theoretical models, whilst including parenting and attachment, do not let these become the central focus whilst neglecting other factors which also play an important role.

Clinical Implications of the Findings

This research found that different variables led to childhood and adolescent anxiety and depression symptoms and that this was affected differently over time and through different pathways, for example, the family environment (interparental relationship, negative life events, parental mental health) versus the parent-child relationship (attachment and parenting sensitivity). With this in mind, the current study provides support for interventions that are aimed towards supporting parental mental health as well as the child's (Gunlicks & Weissman, 2008; Hirshfeld-Becker,

Micco, Simoes & Henin, 2008), parenting interventions aimed at helping improve parenting sensitivity and also interventions and preventative treatment options aimed at strengthening attachment security between parent and child beyond infant attachment (Pincus, Eyberg & Choate, 2005).

In addition, the finding that preschool attachment was associated with depression and anxiety symptoms but infant attachment was not, has clinical implications. Currently, there are a number of attachment interventions focused on improving the attachment relationship during infancy, however, there are substantially fewer of these interventions aimed at adolescents and older children (Moretti & Obsuth, 2011). Given that along with the findings, attachment has shown to be an important factor in the development of both adolescent depression and anxiety (Brumariu & Kerns, 2010), the provision of attachment interventions beyond infancy aimed at increasing parenting sensitivity is crucial.

The moderator analyses also suggest that where mothers may be experiencing anxiety symptoms providing interventions that also focus on parenting sensitivity would be helpful both when children may present with anxiety symptoms and over time. Additionally, the moderator analyses suggest that for those children experiencing depression symptoms who also experience more sensitive parenting, having an intervention that works on the interparental relationship itself when needed would be useful. In contrast, for those experiencing less sensitive parenting focusing on the parenting element first would be more helpful.

Taken together these findings suggest adopting a family-wide approach and considering multiple domains of the family environment as well as the parent-child relationship when offering support to children and adolescents with depression and

anxiety symptoms. This has important implications for commissioners of Child and Adolescent Mental Health Services (CAMHS) services.

Summary

In summary, this longitudinal study provides additional evidence of the small yet significant link between parenting, attachment and childhood and adolescent depression and anxiety symptoms. In addition, it stresses the importance of other family context factors including maternal depression and anxiety, negative life events and the quality of the interparental relationship. Future research should consider more integrative research, understanding the comparative extent that these different factors may have in the development of depression and anxiety symptoms during childhood and adolescence.

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Part 3: Critical Appraisal

Introduction

This appraisal critically examined the research process involved in studying parent-child relationship factors and family-context factors in the context of childhood and adolescent depression and anxiety and trauma symptoms. This critical appraisal begins by the researcher situating her context in this research area. Then, there is a discussion of the process of conducting secondary data research within a well-researched area and the challenges that came along with this. Next, the arbitrary nature and classification of the p-value will be explored with reflections on the role of this in the current study. The appraisal concludes by discussing further the clinical and theoretical implications of the research. In particular, it stresses how family-context factors can often be treated as secondary or as an add-on to other treatment pathways, namely individual or parenting and attachment interventions, and how this research sheds light on the importance of tackling children and adolescent mental health by including family-context within these interventions.

My Interest in the Area

This area of research appealed to me because of my keen interest in attachment and parenting and their link with mental health. This is something which I really enjoyed exploring as an undergraduate prior to clinical psychology training, where I looked instead at adolescent antisocial behaviour as the outcome variable. In addition, my personal life experiences led to a curiosity in this area which grew into a more general interest in how different parenting and attachment experiences affect all of us. Through experiences on training, I have also been exposed to various situations that have highlighted the importance of other family-context factors and their role within child mental health such as community psychology and multidisciplinary

team (MDT) working on various Child and Adolescent Mental Health Services (CAMHS) placements.

Prior to this research, I did not have any experience of conducting longitudinal SEM research, however, this was something that I always wanted to be a part of. Time and time again I have written and read within the limitations section of many discussions the importance of conducting longitudinal research in addition to cross-sectional research and so it was fantastic to finally be part of a longitudinal research project.

The Process of Doing Secondary Research in a Well-Researched Area

Using secondary data came with many advantages as well as challenges. The NICHD dataset is a very large data set conducted over the space of 15 years from 1991 and followed an initial 1364 children and their families where upwards of 70 instruments were used throughout the process and at multiple time points. By having access to this secondary data, it meant being able to use a longitudinal design where the data needed would not have been possible to collect independently during the doctorate due to the obvious time constraints. Furthermore, the sheer richness and breadth of measures, time points and sample size opened up the possibility of research questions where the power needed for the analysis could be met. In addition, the time that would have been spent on participant recruitment and data collection, for example, could instead be spent learning complex SEM models and packages as well as freeing up the time to focus on conducting a larger scale meta-analysis. This also meant that in some way given how densely researched this subject area was, my time was used in a more impactful way by summarising research for the field and considering a greater breadth of other factors such as the family context factors together in a larger scale way.

However, there were a unique set of challenges to working in this way. Firstly, due to the sheer scale of the project, it was a challenge working with such a large dataset and getting access to all the necessary documentation to really get a sense of the measures used. Related to this, when beginning the research process and looking for a gap in the literature this meant that although I knew I had access to longitudinal datasets much research had already been done in the area. Furthermore, being unable to contribute to the design and data collection process meant that certain variables had not been measured which limited the questions open to investigation and there was also a restriction regarding the type of measurement that had been used. For example, the anxiety measure that was used, although it met important validity and reliability requirements, it did not have separate measures of different types of anxiety which may have been helpful to disentangle the developmental nature of different types of anxiety through childhood and adolescence.

A further challenge for all researchers to be aware of when working with archived data is that alongside being a recipient of the data, you also become a recipient of the researchers' take on the research area including their orientation and principles which will have guided measure selection, study design, etc. This can potentially also impact your way of viewing the data and any subsequent results that you interpret. By being aware of this as a potential background process it enabled me to consider not only what my take as a clinical psychologist in training may be but also how theoretical frameworks whilst guiding subsequent research can also guide future research. Moreover, without being able to step back and reflect on this and notice it as it happens, it can be all too easy to follow narrower theoretical frameworks and not critically examine other possibilities and important theories.

The Arbitrary Classification of the *P*-Value

Significance tests have been designed to offer an objective measure to help inform judgements about validity when generalising results (Figueiredo Filho et al., 2013). Despite these well-meaning intentions for significance tests to support researchers in their academic work, misinterpretation and abuse of statistical tests have been rife in academic science (Figueiredo Filho et al., 2013). This is in contrast to Neyman and Pearson's (1928) original aim for this statistical approach where it was stated that ''the tests themselves give no final verdict, but [are] tools [to] help the worker who is using them to form his final decision'' (p. 206).

Historically, much research in social sciences used null hypothesis testing when determining the statistical significance of findings. However, this has come alongside a very dichotomous and arbitrary classification of the *p*-value, usually around .05, where <.05 is deemed significant and a finding, and in some instances due to publication bias, "publishable" whereas >.05 is deemed not significant, not a finding and therefore "not publishable". It has been argued that not only is this classification unnecessary but can often be damaging to the valid interpretation of data (Greenland et al., 2016). During the analysis, it was important to remember what the *p*-value actually showed where a smaller *p*-value alerts the reader that this data is unusual if all assumptions when calculating the value are correct, however, it may be that other factors are at play (Greenland et al., 2016). In addition, any value for the null hypothesis *p*-value that is less than one reveals that some association may be present within the data but that further interpretation of the statistics is needed to work out what is most compatible with the data using the assumed model (Greenland et al., 2016).

In recent years, important institutions such as the American Statistical Association have issued warnings against how *p*-values are currently being used (Wasserstein & Lazar, 2016) although these warnings have been present for a while, for example, Cohen's famous "the earth is round, *p*<.05" paper (Cohen, 1994). This no doubt relates to similar concerns raised by Hill (1965) who argued that "too often we weaken our capacity to interpret data and to take reasonable decisions whatever the value of *p*. And far too often we deduce 'no difference' from 'no significant difference" (p. 299) and Greenland et al. (2016) who called the "degradation of *p*-values into ''significant'' and ''nonsignificant'' as an especially pernicious statistical practice" (p. 348).

Being reminded of this dichotomous *p*-value classification was important during my data analysis. I was aware when interpreting results that for *p*-values that were just below .05, for example, these were therefore deemed significant and required further inspection whereas others which may have been marginally over .05 were interpreted as no longer significant enough to discuss. This relates to Gill (1999) who argued that "from the current presentation of null hypothesis significance testing in published work it is very easy to confuse statistical significance with theoretical or substantive importance" (p. 669). An example of this in the current research was the fact that none of the moderators from the meta-analysis reached statistical significance and so could potentially be more easily ignored yet this was an important finding in itself.

I also learnt an important lesson regarding looking at the results as a whole. Once the analysis was complete I was keen to see if the results were significant, however, when interpreting results, it is important to consider what key patterns may also be apparent. For example, when many of the family-context factors were added to the

LGC models of depression and anxiety that included parenting sensitivity, parenting sensitivity was no longer significant in several of the tests. This was an important observation regarding the power of the *p*-value culture and how it can narrow the focus of interpretation of results. For example, by being immersed in this *p*-value culture I was initially focusing more on what results were statistically significant rather than looking at the results as a whole. Furthermore, I was also paying less attention to what results were no longer statistically significant when the model changed, when this finding in itself may have actually been more theoretically important.

Moving forward, important elements of good practice can be taken forward by researchers. For example, reporting precise *p*-values not just in relation to a certain threshold and supporting a shift in emphasis from hypothesis testing to estimation where confidence intervals and the size of effect size estimates are promoted (Greenland et al., 2016).

In addition, being aware of the system that this controversy is situated within, where to some publishers, significant findings equal more readers which can create publication bias and the file drawer problem (Rosenthal, 1979). In line with this, the use of open data and analysis, registering research projects, and initiatives, like the Open Science Framework, which aim to improve transparency around the research process are all steps in the right direction to readdress the current system.

In conclusion, through this project, I have gained more insight regarding the *p*-value crisis and its proponents and now hold in mind the question as to whether *p* is above or below .05 means it is the universal arbiter of discovery.

Family Context Factors and Interventions

The results from both the meta-analysis and research project highlighted the important role of family context factors, namely, the interparental relationship including intimate partner violence, parent mental health, specifically depression and anxiety, and also negative life events. Despite this, these family context factors may often be neglected in CAMHS services or treated as an add-on to standard child-centred or parenting or attachment centred interventions. This is likely to reflect the fact that the evidence base for many of these family context factors has been narrow which has no doubt intensified the current lack of provision and cohesion of these services.

Considering the interparental relationship alone, a number of programmes have found that enhancing the interparental relationship offers advantages to children (Cowan, Cowan & Heming, 2005; Pruett, Insabella & Gustaffson, 2005). More recently, initiatives have demonstrated how making small changes such as integrating a relationship component into existing parenting provision can provide similar outcomes (Local Family Offer Network, 2017). In addition, the Local Family Offer initiative has stressed the need to change the culture of viewing the interparental relationship work as merely an add-on to a key integrative part of the treatment package (Local Family Offer Network, 2017). Research has highlighted how this needs to include recognising that the quality of couple and co-parental relationships have a fundamental impact on outcomes for children and their families (Local Family Offer Network, 2017). Furthermore, this may include: skilling up the current workforce; more integrated multi-agency working; more key professionals as a regular fixture within current CAMHS MDTs such as a couples therapists and a move away from a default referral culture outward where interparental relationship

factors are treated separately and instead treating these as part of an overall picture of need for the family. Future research could study the outcome of this.

Acri and Hoagwood (2015) conducted a review investigating an additional influential family context factor, parental mental health, and its involvement in child mental health interventions. Results showed that despite there being a large literature concerning children's mental health interventions (200 studies), only 20 of these included a parental component and measured parental mental health. The majority of studies which included a parental component focused on parenting skills, not parental mental health (Silverman, Kurtines, Jaccard & Pina, 2009). Six out of the 20 studies included a parental mental health component, all of which showed some benefit to children's mental health. Given the findings from the current study, future research should examine the evidence of parental mental health interventions in the context of children's mental health.

Parental mental health is an important factor to consider not only given its involvement in the development of childhood and adolescent anxiety and depression but also the finding that when parental mental health is not supported, children's mental health outcomes are affected including poorer treatment outcomes and less therapeutic effectiveness (Beauchaine, Webster-Stratton & Reid, 2005; Pilowsky et al., 2008; Rishel et al., 2006).

Considering this alongside the current study's findings, it is likely interventions aimed at including a parental mental health component alongside child and family-focused interventions would be beneficial. In addition, by combining and integrating parental and child mental health interventions this can help reduce service fragmentation or duplication, make interventions more accessible and improve the effectiveness of outcomes. This may improve the current outlook which has been

described as a fragmented and uncoordinated set of systems for children and their parents (Acri & Hoagwood, 2015).

Finally, given the current financial and political climate, negative life events are increasing; for example, we are seeing rises in homelessness (Fitzpatrick, Pawson, Bramley, Wilcox & Watts, 2017) and poverty (Barnard et al., 2017) and cuts are being made to essential services such as intimate partner violence shelters (Bureau of Investigative Journalism, 2017). It is important when considering interventions aimed at childhood and adolescent depression and anxiety that formulation includes negative life events and alongside this signposting to community resources in order to help minimise the impact alongside clinical interventions. These are also important to consider when working with families especially those in poverty where lack of transportation or childcare can impact on attendance, for example (Miranda & Bruce, 2002; Miranda & Green, 1999; Rosen, Tolman & Warner, 2004).

In conclusion, embedding parental relationship support, parental mental health components and awareness of negative life events within the heart of services aimed at preventing and treating child and adolescent depression and anxiety should be a priority.

Conclusions

This appraisal critically examined the research process involved in studying parent-child relationship factors and family-context factors in the context of childhood and adolescent depression and anxiety and trauma symptoms. The discussion presented the researcher's context, some of the benefits and complexities of working with archived data, the role of the *p*-value's arbitrary significance classification when interpreting results and further clinical and theoretical implications of the project.

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