

Testing the impact of gene-environment interplay on the development of children's
internalising problems

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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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Overview

This thesis aims to further understanding of parenting in infancy. The first part of the thesis aims to build on existing reviews of genetically informed parenting studies across the lifespan by focussing specifically on twin infant studies. This is the first systematic review and meta-analysis of infant twin studies of parenting. A range of phenotypes were identified across types of parenting, informant and age. The meta-analysis showed a high concordance between twin pairs on parenting, the bulk of which was driven by shared environmental factors. There was some evidence of the heritability of parenting but this was not conclusive due to significant heterogeneity in studies. Limitations, including the number and quality of included studies, are discussed.

The empirical paper, which forms the second part of the thesis, continues the exploration of parenting. However, in this case, it explores evocative gene-environment correlation (rGE) in parenting by examining transactional effects between child anxiety and parenting in an adoptive cohort sample. This study is the first to utilise an adoptive cohort to explore the reciprocal effects of child anxiety and parenting over this time period. Findings indicate the bidirectionality of this relationship but did not show genetic influences on evoked pathways.

Finally, the third part of the thesis contains a critical appraisal of the research process. It provides a reflective account of the methodological and ethical considerations of behavioural genetics research, the research implications of secondary analyses and the clinical relevance of findings from behavioural genetics research and its implications for interventions.

Impact Statement

The current study has a number of implications for future academic research as well as in the realm of clinical psychological interventions. In academic terms, this project represents a forward step in the understanding of parenting in early childhood. We have synthesized the existing literature on infant twin studies and identified the gaps for future exploration. Furthermore, the empirical paper demonstrates the bidirectionality of the relationship between child behaviour and parenting in the first seven years of life. The identification of such transactional processes between parenting and child behaviour offer evidence of evoked parenting that build on existing transactional processes between parent and child anxiety. This is an important contribution to the literature trying to understand the aetiology of parenting. Given the significance of parenting on a variety of physical and mental health outcomes, this presents an important direction of study. Future research could build on the work presented here by examining whether the transactional effects between child anxiety and parenting are mediated by parental anxiety over the same time period. It could also explore the origins of the child internalizing behaviour shown to evoke parental overreactivity by exploring the perinatal environment, as well as other models of genetic risk or candidate gene mapping. These research aims would further our understanding of parenting in early life and its determinants.

There are also a number of implications of this research for clinical practice. This work builds on previous research that has explored gene-environment correlation. Here, we have demonstrated that children do influence the parenting they receive and that this influence begins in the first few years of life. It is yet to be shown that this evoked parenting is influenced by the child's genes, however it does lay the groundwork for future studies that may find evidence of this, or identify other aetiological pathways to child-driven effects on parenting. To date, the structure of

parenting interventions has been based on the assumptions that children's behavioural difficulties are a result of their environment and, more specifically, of the parenting they receive. An understanding of the fact that some parenting can be evoked through child behaviour suggests that the picture is more complex. Clinical interventions could thus be adapted to normalize the experience of parents whose parenting has changed in response to their child's behaviour. Furthermore, interventions could offer guidance on the specific interaction between a child's tendencies and the efficacy of certain parenting styles, tailored to the behaviours of the child. This could be particularly important when genetic similarity between parent and child cannot be assumed, such as is the case in adoptive families and for children in care more broadly.

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Part 1: Literature Review

Who's raising whom? A systematic review and meta-analysis of the heritability of parenting behaviours in infant twin studies.

Abstract

AIM: This review aimed to explore the heritability of parenting in early childhood through a systematic review and meta-analysis of twin infant studies.

METHOD: A systematic review and meta-analysis of all twin studies on parenting children age two years and under was conducted. PsychINFO and PubMed were searched up to February 2020. The Cochrane Quality Assessment tool was used to assess the risk of bias of included studies.

RESULTS: Six studies were included in the review, resulting in nine papers and twenty-eight parenting phenotypes included in the meta-analyses. A range of phenotypes were identified across types of parenting, informant and age. The meta-analysis showed a high concordance between twin pairs on parenting, the bulk of which was driven by shared environmental factors. There was some evidence of the heritability of parenting, but this was not conclusive. The quality of studies was a significant moderator of these findings, with higher quality associated with greater twin concordance.

CONCLUSION: Due to the heterogeneity of the included studies and limited sample, the findings offered only mixed support for the existence of evocative rGE in parenting. Further research in this area is warranted.

Introduction

In the first few years of life, parenting represents the predominant aspect of a child's environment and continues to be one of the most consistent influences throughout childhood and adolescence. As such, it is unsurprisingly one of the key areas of study in research on child development. Parenting has been shown to be associated with a number of child outcomes, including externalizing and internalizing problems (e.g. Barber, Olsen, & Shagle, 1994; Piko & Balázs, 2012; Steinberg, Mounts, Lamborn, & Dornbusch, 1991), cognitive development (e.g. Berlin, Brooks-Gunn, Spiker, & Zaslow, 1995; Hubbs-Tait, Culp, Culp, & Miller, 2002) and social competence (e.g. Cassidy et al., 1996; Moss et al., 1998; Sroufe et al., 1999). Parenting has also been associated with outcomes well into adulthood, including cognitive ability, socioeconomic achievement. (e.g. Singh-Manoux, Fonagy, & Marmot, 2006), physical health outcomes (e.g. Dube et al., 2009; Wegman and Stetler, 2009) and mental health outcomes (e.g. Morgan, Brugha, Fryers, & Stewart-Brown, 2012).

However, although these associations are extensive, they are predominantly from observational studies and therefore reverse causation cannot be ruled out – that is to say, it is possible that children may shape the parenting they receive. Indeed, a growing body of research indicates that children can and do influence the parenting they receive (e.g. Bell, 1968; Anderson, Lytton, and Romney, 1986; Pinguart, 2016). This idea dates back to the “child effects model” proposed by Anderson, Lytton, and Romney (1986), which suggested that rather than just passively receiving certain parenting behaviours, children also evoke their parent's behaviour through their actions and interactions.

Gene-Environment Correlation

Prior behavioural genetic research has been crucial in demonstrating the role of child-driven effects on parenting (Bell, 1968). Child-driven effects on

parenting may result from what is known as evocative gene environment-correlation (*rGE*)—the process by which children evoke behaviour from others in their environment that is consistent with their genetic predispositions, through their actions or interactions with those others (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). A growing body of research supports the notion that child-driven influences, including genetic influences and particularly evocative *rGE* processes, partially underlie the parent–child relationship.

The behavioural genetic literature on this topic is best summarized in two recent meta-analyses, one conducted by Klahr & Burt (2014) and the latter by Avinun & Knafo (2013). Klahr & Burt looked at 56 twin and adoption studies that examined the heritability—which is a statistic that estimates the proportion of variance in a trait that can be explained by genetic variance—of parenting, using data from individuals ranging from 5 months to 45 years old. This included examination of the proportion of variance in parenting explained by genetic differences in their children (i.e. child-to-parent effects). The authors estimated that genetic variation in children accounted for approximately 25-40% of the variance in different aspects of parenting. Similarly, Avinun & Knafo conducted a meta-analysis of 32 twin studies examining evocative effects of children’s genes on parenting, which revealed a heritability estimate of 23%, thus offering further evidence that genetically influenced behaviours of the child may affect parental behaviour.

However, most studies included in both meta-analyses focused on adolescence and adulthood, with a much smaller number examining parenting in early childhood. Specifically, Avinun & Knafo included three twin studies involving young children or infants, while Klahr & Burt included three twin studies, two of which were distinct from those included in Klahr & Burt’s review, and one non-twin sibling study. Given the critical importance of the earliest stages of development and the assumed importance of caregiving, it is crucial to establish the magnitude of

child-to-parent effects and parent-to-child effects in infancy. This is particularly important as existing literature indicates that the impact of environmental effects (e.g. parenting) on children's behaviour decrease with age (Bergen, Gardner, & Kendler, 2007; Haworth et al., 2009; Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008), and that parenting in infancy differs from toddlerhood in its interaction with child characteristics (e.g. child sex; Keenan and Shaw, 1997).

Possible Moderators of the Genetic and Environmental Effects on Parenting

Research points to the possibility that several features may moderate *rGE* effects and a number of these were explored in the Klahr and Burt (2014) meta-analysis. One such possible moderator is the age of the child at the point of study. Age is a common moderator of the heritability of a variety of traits (e.g. Haworth et al., 2009; Bergen, Gardner, & Kendler, 2007). One explanation for estimates of heritability of personality characteristics increasing with age is that children's environments are more a function of their own choices as they become older and more independent and they may choose environments more correlated with their genetic predisposition (Scarr & McCartney, 1983). Similarly, it has been suggested that evocative *rGE* could increase with age as a result of the child becoming more active in communicating their preferences and desires in the family environment, thus evoking more concordant behaviour. Evidence for this is found in research showing that heritability estimates of perceived parenting increase with children's age (Elkins, McGue, & Iacono, 1997). However, it is not clear if this effect will be replicated in the earliest year of life.

Another possible moderator is the method of assessment of parenting. Parental behaviour is typically evaluated by self-reports or observations, and initial evidence suggests that heritability estimates might differ based on the assessment method used. For example, Deater-Deckard (2000) found that the heritability estimate of parenting behaviour was 55%, as measured by self-report, and 6%, as

measured by observation. A final potential moderator is the type of parenting being assessed. Heritability estimates of parental positivity (e.g., warmth, sensitivity) and parental negativity (e.g., harsh discipline, overreactivity) appear to differ substantially in some studies (e.g. Knafo, 2011, Deater-Deckard, 2000), suggesting possible moderation of genetic and environmental effects by type of parenting.

The Current Meta-Analysis

The goal of the present meta-analysis is to explore the heritability of parenting in children aged two years and under, thus extending our understanding of the impact of a child's genes on the early parenting that they receive. This will have important implications for our understanding of bidirectional effects (child-to-parent and parent-to-child) in infancy, and may point towards mechanisms that are also important beyond infancy.

This project explores this question by reviewing classical twin studies of infants that estimate the heritability of parenting. Specifically, we focus on twin studies that examine evocative rGE by estimating the proportion of variance in parenting that can be explained by child genetic variance. Classical twin studies rely on differences in genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twins to estimate genetic effects on behaviour. Specifically, the classical twin design utilizes the fact that MZ twins, share 100% of their genes, while DZ twins share approximately 50% of their genes (Plomin et al., 2008) to establish estimates of heritability and environmental influence. The degree to which MZ twins are more similar than DZ twins is assumed to be a result of their increased genetic similarity and can be used to estimate heritability. Comparisons of MZ and DZ correlations can also be used to estimate the contributions to phenotypic variance of environments that are shared and non-shared between twins. Thus, meta-analysing the results of twin studies of evocative rGE , allows us to estimate the contribution of

child genetic factors and the shared and non-shared environment to parenting across all existing studies and thereby build on the previous reviews (Kendler & Baker, 2007; Plomin & Bergeman, 1991) and meta-analyses of *rGE* (Klahr & Burt, 2014; Avinun & Knafo, 2013). In particular, we extend previous studies—that explore this question across the lifespan—through focusing, for the first time, on infancy exclusively, thus broadening our knowledge of this critical period of development. We also build on the previous literature by incorporating studies that have been published on evocative *rGE* effects on parenting since the previous reviews were conducted.

Methods

Search Strategy

This systematic review and meta-analysis was conducted in line with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Moher et al., 2009) recommendations. We developed a review protocol, which was registered with the international prospective register of systematic reviews; PROSPERO (ID: CRD42019151532) for a review of infant twin studies and this systematic review was constructed as a subset of this larger project. Searches were conducted up to February 2020 in PsycINFO and PubMed databases for published studies. Relevant database specific subject headings and text word fields were searched (see Table 1 for exact search terms). Once studies were identified for inclusion, we then manually searched reference lists of eligible studies and reviewed them for inclusion.

Table 1**Search Strategy**

Date	Database	Search terms	N	N (after duplicates removed)
30/11/2018	PubMed	(twin*[Title/Abstract] AND (gene[Title/Abstract] OR genome[Title/Abstract] OR genetic* [Title/Abstract] OR herita* [Title/Abstract] OR environment*[Title/Abstract]) AND (infan*[Title/Abstract] OR early[Title/Abstract]))	2735	2727
30/11/2018	PsychINFO	(twin* and (gene or genome or genetic* or herita* or environment*)) and (infan* or early).ab.	1291	504
05/02/2020	PubMed (from 1/10/18 onwards)	(twin*[Title/Abstract] AND (gene[Title/Abstract] OR genome[Title/Abstract] OR genetic* [Title/Abstract] OR herita* [Title/Abstract] OR environment*[Title/Abstract]) AND (infan*[Title/Abstract] OR early[Title/Abstract]))	120	28
05/02/2020	PsychINFO (From 1/10/18 onwards)	(twin* and (gene or genome or genetic* or herita* or environment*)) and (infan* or early).ab.	315	231
	Reference list searching	(twin*) and (gene or genome or genetic* or herita* or environment*) and (infan* or early or preschool)	358	93
			4819	3583

Study Inclusion and Exclusion Criteria

To identify studies meeting inclusion criteria, titles and abstracts identified in the search strategy were reviewed. Initial inclusion criteria were as follows: (1) study sampled monozygotic and dizygotic twins; (2) mean age of infants when outcomes were collected was less than or equal to 2 years; (3) the study results included twin correlation or concordance data or heritability estimates based only on twins; and (4) the full-text article was available and written in English. Studies were excluded if they (1) only contained data reported elsewhere (e.g. reviews, meta-analyses, etc.) and (2) the paper involved a multivariate analysis or gene-environment interaction analyses and univariate estimates could not be extracted. Studies were then independently coded by two raters to identify studies where one of the outcomes examined was parenting. Disagreement between these raters was resolved by consensus coding.

Data Extraction and Quality Assessment

Studies meeting inclusion criteria were coded using a standard data extraction form. For outcomes of interest, summary estimates (e.g. twin correlations or heritability estimates) and sample size was extracted. Where only heritability estimates were available, Falconer's (1960) formula was used to convert to twin correlations. Potential moderators were also extracted, including the following: type of parenting domain assessed (positive, negative), method of assessing parenting (parent self-report, observation) and child age at the outcome assessment. Additional extracted data included study design, country, and demographics.

At this stage, if duplication of reporting was found (i.e. if two papers reported the same parenting phenotype as the same age within the same study), the paper with the larger sample size was included. If sample sizes were identical, the phenotype from the more recently published paper was included. If, after this process was completed, a paper was identified as containing no novel data (i.e. no data that was not reported elsewhere), it was excluded from the analyses.

Approximately 15% of all included studies in the broader meta-analysis were double coded for the purpose of establishing reliability of data extraction. For all moderators, the percent agreement for all moderators was over 90%. Discrepancies were resolved by consensus coding.

In addition, an assessment of study quality was conducted based on a 14-item quality assessment tool adapted from the Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields for Quantitative Studies (Kmet, Cook & Lee, 2004; see Table 2 for a summary). This assessment-rating tool evaluates elements of study quality endorsed by the Cochrane collaboration. Each item was scored on a scale of 0-2, where 0 indicated non-adherence to that criterion and 2 indicated complete adherence. Each study's score was calculated by summing the total of these scores and then dividing it by the total possible score (i.e. the number of applicable items multiplied by two).

Finally, the degree of publication bias (i.e. the extent to which studies with more significant effects were preferentially published) was examined. Previous twin-based meta-analyses (McCartney, Harris, & Bernieri, 1990; Taylor, 2011) have suggested that concerns about this bias are of less relevance when examining twin studies because they are not typically based on significance testing but rather estimate genetic and environmental components of a given phenotype. However, publication bias was examined using two approaches: first, by calculating Rosenthal's Fail-safe N (Rosenthal, 1979), which estimates the number of additional studies, which, if added to the analysis, would result in the overall effect being statistically nonsignificant, and secondly using the Egger test (Egger, Smith, Schneider, and Minder, 1997). If publication bias was indicated, the trim and fill procedure was used to correct for publication bias. Funnel plots were used to visualise the degree of publication bias on the mean effects and, if the trim and fill procedure was used, to visually represent the imputed studies.

Table 2

Study Quality Assessment

Paper	Boivin (2005)	Boutwell (2012)	DiLalla (1996)	Flom (2020)	Forget-Dubois (2007)	Roisman (2008)	Roisman (2006)	Woodward (2018)	Fearon (2006)	Fearon (2006)
Study	QNTS	ECLS-B	CTR	BUTP	QNTS	ECLS-B	ECLS-B	CTR	NTR	LTR
1. Question or objective sufficiently described?	Yes	Yes	yes	yes	yes	yes	yes	yes	yes	yes
2. Design evident and appropriate?	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
3. Selection method of or source of info appropriate?	yes	yes	in part	in part	yes	yes	yes	in part	yes	yes
4. Subject or input variables sufficiently described?	in part	in part	in part	yes	yes	yes	yes	yes	in part	in part
5. Random allocation to treatment group?	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
6. Investigator blinding?	n/a	no	yes	n/a	n/a	yes	no	no	yes	in part
7. Subject blinding?	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
8. Outcome well defined and robust to bias?	yes	in part	in part	yes	yes	yes	in part	in part	yes	yes
9. Sample size appropriate?	yes	yes	in part	yes	yes	yes	yes	yes	in part	in part
10. Analysis described and appropriate?	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
11. Some estimate of variance reported?	in part	yes	no	yes	yes	yes	yes	in part	yes	yes
12. Controlled for confounding?	yes	yes	in part	yes	yes	yes	yes	yes	yes	yes
13. Results reported in sufficient detail?	yes	in part	yes	yes	yes	yes	yes	yes	yes	yes
14. Results support conclusions?	yes	yes	yes	yes	yes	yes	yes	yes	yes	yes
Score	91%	79%	71%	95%	100%	100%	88%	79%	92%	88%

Quality adherence to each of the criteria was indicated using a four colour coding system (green = completely meets criteria, yellow = partially meets criteria, red = does not meet criteria and grey = criteria are not applicable to this study). Overall scores were the ratio of total points (yes = 1 points, in part = 0.5 points, no = 0 points for each criteria) to total possible points (i.e. number of applicable criteria) as a percentage.

Data Analysis

First, effect sizes from individual studies were converted from Pearson's intra-class correlations to Fisher's Z effect sizes before use. Then, as several studies had multiple assessments of parenting, we initially conducted a metaregression test of dependent effect sizes using the robust variance approach methods developed by Fisher and Tipton and the R package robumeta. Unlike standard meta-analytic techniques, this approach allows the simultaneous estimation of multiple dependent effect sizes (i.e., more than one per study), without under-estimating standard errors and increasing type I error. Following Borenstein, Hedges, Higgins, and Rothstein's (2005) recommendations, we converted intraclass correlation coefficients to Fisher's Z scores and performed all analyses using the transformed values. We first estimated only the overall weighted mean effect size between twins of parenting. We also examined heterogeneity between studies using the I^2 statistic, which examines the rate of variability across studies due to heterogeneity rather than chance. A value of 0% indicates no observed heterogeneity, 25% low heterogeneity, 50% moderate heterogeneity, and 75% high heterogeneity. We also did sensitivity analyses to test a priori assumptions that between-study covariance was considerably smaller than within-study covariances.

As well as establishing the overall mean effect size for parenting, we also explored whether moderators could explain variability across studies using a metaregressions model specifically, age, parenting domain, assessment method and study quality were used as moderators. Each moderator was examined with other moderators controlled for in one metaregression model. Zygoty was also included in the regression model. Again, heterogeneity between studies was examined using the I^2 statistic. Where these moderators did not meet the standard established by Fisher and Tipton (2015) for trustworthiness (i.e. degrees of freedom of estimate equal to or greater than 4), a secondary analysis was constructed using

a random-effects model implemented using the R package metafor to offer a preliminary estimate of the significance and magnitude of effect of that moderator.

Finally, mean effect sizes for MZ and DZ twin pairs were transformed to Pearson correlations and these were used to construct an estimate of child genetic and environmental contributions to parenting.

Results

Studies Selected

The PRISMA flow diagram detailing search strategy and resulting outcomes can be found in Figure 1. Our electronic search of two databases yielded 3583 articles after duplicates were removed. Upon review of the titles and abstracts, 345 articles were identified as potentially meeting study inclusion criteria. After further review of full text articles, 143 were identified for the broader review described above. Of those, 11 met full inclusion criteria for the present study. Two papers contained only data reported elsewhere and were thus excluded (Micalizzi, Wang & Sauldino, 2017; Boeldt et al., 2011) and one paper contained data partially reported elsewhere and as such, only the novel data was included (Forget-Dubois et al., 2007). The 9 papers included in the systematic review, presented below, reported data on 28 phenotypes collected as part of 6 twin studies, from a total of 3031 twin pair-parent triads (2180 parent-DZ twin triads and 851 parent-MZ twin triads). A summary of the phenotypes, papers and studies included is presented in Table 3.

Figure 1

PRISMA Flowchart

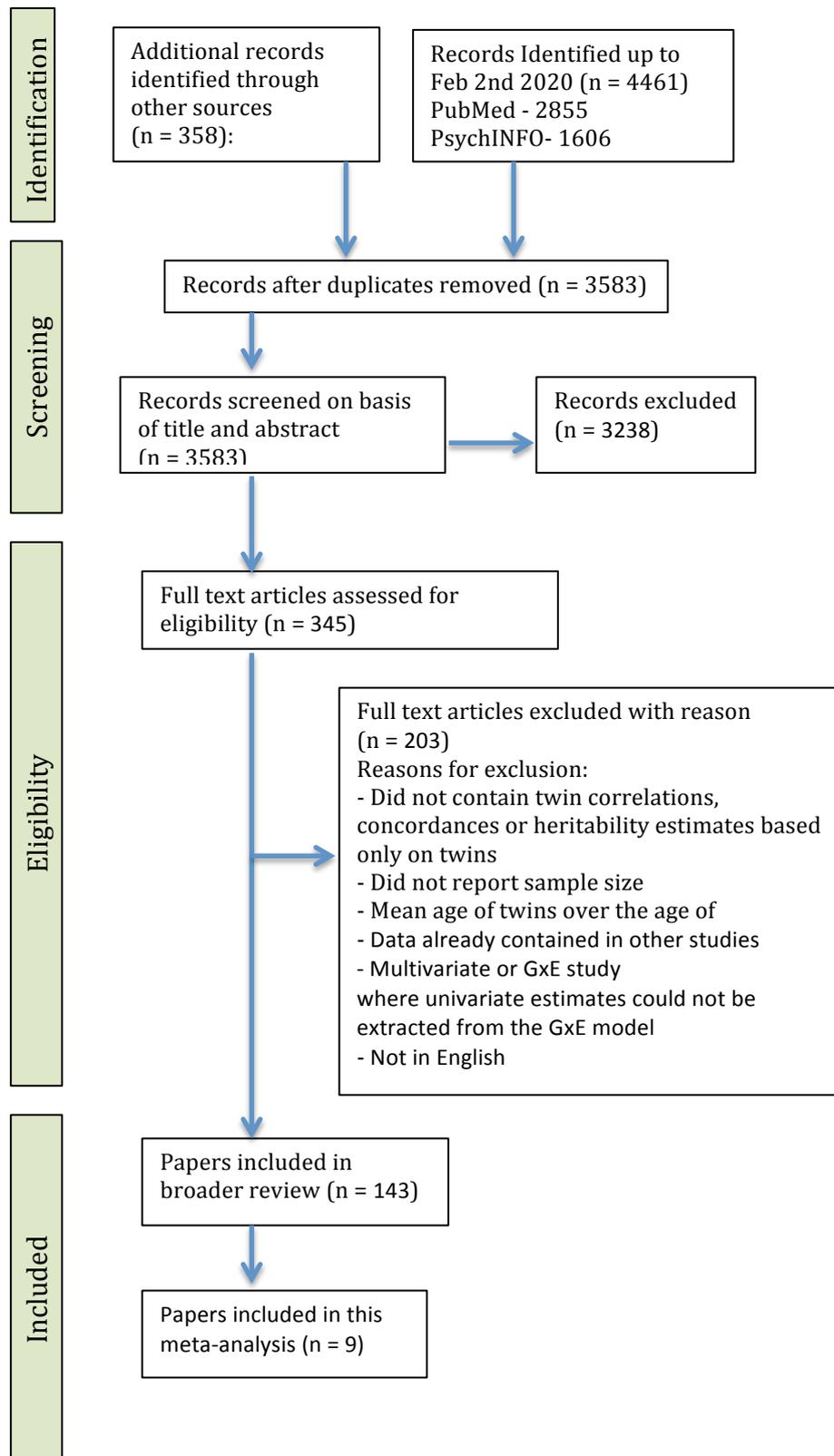


Table 3**Included Studies**

Study	First Author, Date	Country	Race/ Ethnicity	n (twin pairs)	Sex	Age (mo)	No. of Pheno types
BUTP ¹	Flom (2020)	US	85.4% Caucasian	314	M, F	24	1
CTR ²	DiLalla (1996)	US	95% Caucasian	168	M, F	7	8
	DiLalla (1996)	US	95% Caucasian	168	M, F	9	8
	Woodward (2018)	US	86% Caucasian.	485	M, F	18.3 3	1
ECLS-B ³	Boutwell (2012)	UK	-	1600	M, F	24	1
	Roisman (2008)	UK	57.8% Caucasian	485	M, F	24	1
	Roisman (2006)	UK	57.8% Caucasian	505	M, F	9	1
NTR ⁴	Fearon (2006)	Netherlands	-	76	M, F	12.5	1
LTR ⁵	Fearon (2006)	UK	-	81	M, F	12.5	1
	Boivin (2005)	Canada	-	475	M, F	5	4
QNTS ⁶	Forget-Dubois (2007)	Canada	84% Caucasian	393	M, F	18	1

¹ BUTP – Boston University Twin Project; ² CTR – Colorado Twin Registry; ³ ECLS-B – Early Childhood Longitudinal Study, birth cohort; ⁴ NTR – Netherlands Twin Registry; ⁵ LTR – London Twin Registry; ⁶ QNTS – Quebec Newborn Twins Study

Study Characteristics and Quality

Study sample demographics and characteristics are reported in Table 4. The sample sizes of studies ranged from 27 to 264 parent-monozygotic twin pair triads and 49 to 1400 parent-dizygotic twin pair triads. The average twin age at the point of observation of report was 18.23 months. A total of 2 studies were from the US (33.33%), 2 from the UK (33.33%), 1 from the Netherlands (16.67%) and 1 from Canada (16.67%). Of the studies included, 3 only reported on observed parenting (50%), 2 only reported on self-reported parenting (33.33%) and 1 reported on both (16.67%). For study quality, the mean score across all studies was 88.22% (SD 9.52%; range 29.17%) (See Table 2).

Table 4

Study Phenotypes

Study	First Author, Date	Country	Study Type	n twin pairs (MZ /DZ)	Age (mos)	Phenotype (specific)	Measure	Parenting Type
BUTP ¹	Flom (2020)	US	PR	314 (145/169)	24	Negative Parenting	PFQ and Interview	NEG
		US	OBS	168 (76/92)	7	Show Toy	Coded Interaction	POS
CTR ²	DiLalla (1996)	US	OBS	168 (76/92)	7	Hold Touch	Coded Interaction	POS
		US	OBS	168 (76/92)	7	Acknowledge	Coded Interaction	POS
		US	OBS	168 (76/92)	7	Verbal Attempt	Coded Interaction	POS
		US	OBS	168 (76/92)	7	Respect for Autonomy	Coded Interaction	POS
		US	OBS	168 (76/92)	7	Quality of Instruction	Coded Interaction	POS
		US	OBS	168 (76/92)	7	Sensitivity	Coded Interaction	POS
		US	OBS	168 (76/92)	7	Warmth	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Show Toy	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Hold Touch	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Acknowledge	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Verbal Attempt	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Respect for Autonomy	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Quality of Instruction	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Sensitivity	Coded Interaction	POS
		US	OBS	156 (70/86)	9	Warmth	Coded Interaction	POS
	Woodward (2018)	US	OBS	485 (264/221)	18.33	Positive parenting	Coded Interactions	POS

ECLS-B ³	Boutwell (2012)	UK	OBS	1600 (200/1400)	24	Disengagement	TAS-45	NEG
	Roisman (2008)	UK	OBS	485 (120/365)	24	Parenting quality	Two-Bags Task	POS
	Roisman (2006)	UK	OBS	505 (172/333)	9	Parenting quality	NCATS	POS
NTR ⁴	Fearon (2006)	Netherlands	OBS	76 (27/49)	12.5	Sensitivity	Home Observation	POS
LTR ⁵	Fearon (2006)	UK	OBS	81 (30/51)	12.5	Sensitivity	Home Observation	POS
QNTS ⁶	Boivin (2005)	Canada	PR	475 (290/185)	5	Self-Efficacy	PACOTIS	POS
		Canada	PR	475 (290/185)	5	Parental Impact	PACOTIS	POS
		Canada	PR	475 (290/185)	5	Reactive Hostility	PACOTIS	NEG
		Canada	PR	475 (290/185)	5	Overprotection	PACOTIS	NEG
	Forget-Dubois (2007)	Canada	PR	276 (109/167)	18	Reactive Hostility	PACOTIS	NEG

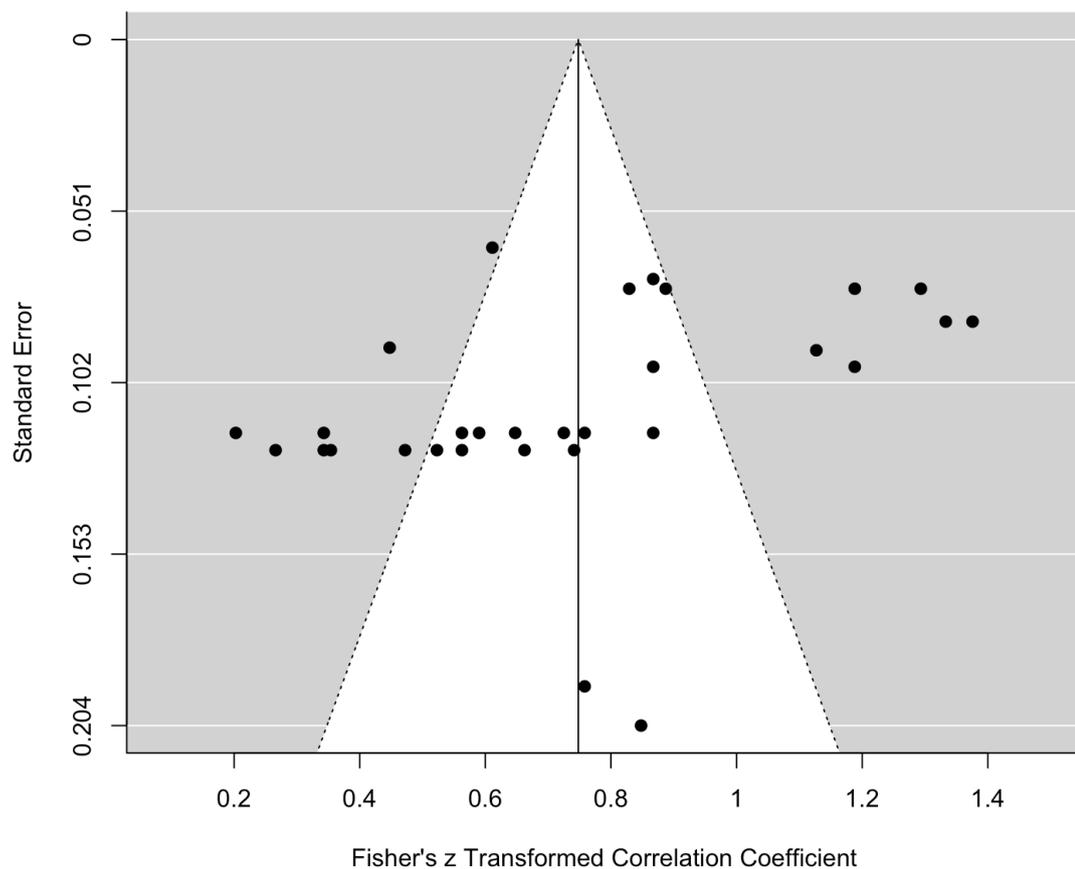
OBS was used to indicate that the phenotype was measured by observation whereas PR indicated parental self-report was used. POS indicated a positive parenting phenotype and NEG indicated a negative parenting phenotype. Phenotypes were labelled as described in the original studies.

¹ BUTP – Boston University Twin Project; ² CTR – Colorado Twin Registry; ³ ECLS-B – Early Childhood Longitudinal Study, birth cohort; ⁴ NTR – Netherlands Twin Registry; ⁵ LTR – London Twin Registry; ⁶ QNTS – Quebec Newborn Twins Study

Fail-safe N for the parenting phenotype was 67,338 indicating that 67,338 unpublished studies averaging a parenting correlation in twin pairs of zero would need to exist to make the summary parenting effect size nonsignificant, indicating that the results presented are unlikely to have been affected by publication bias. This was confirmed by the result of the Egger's test of publication bias, which was not significant when the sampling variance ($z = -1.87, p = 0.06$) and sample size were used as predictors ($z = 0.86, p = 0.39$). This is visually represented as a funnel plot in Figure 2.

Figure 2

Funnel Plot



Metaregression Testing of Dependent Effect Size of Parenting

Studies with an effect size value larger or smaller than 3 SDs from the mean were considered outliers. No studies met these criteria and therefore none were excluded. A total of 6 nonoverlapping studies were available to estimate the mean effect size for parenting similarity in twin pairs. All results for metaregression testing are summarised in Table 5 and a forest plot is presented as Figure 3.

Table 5

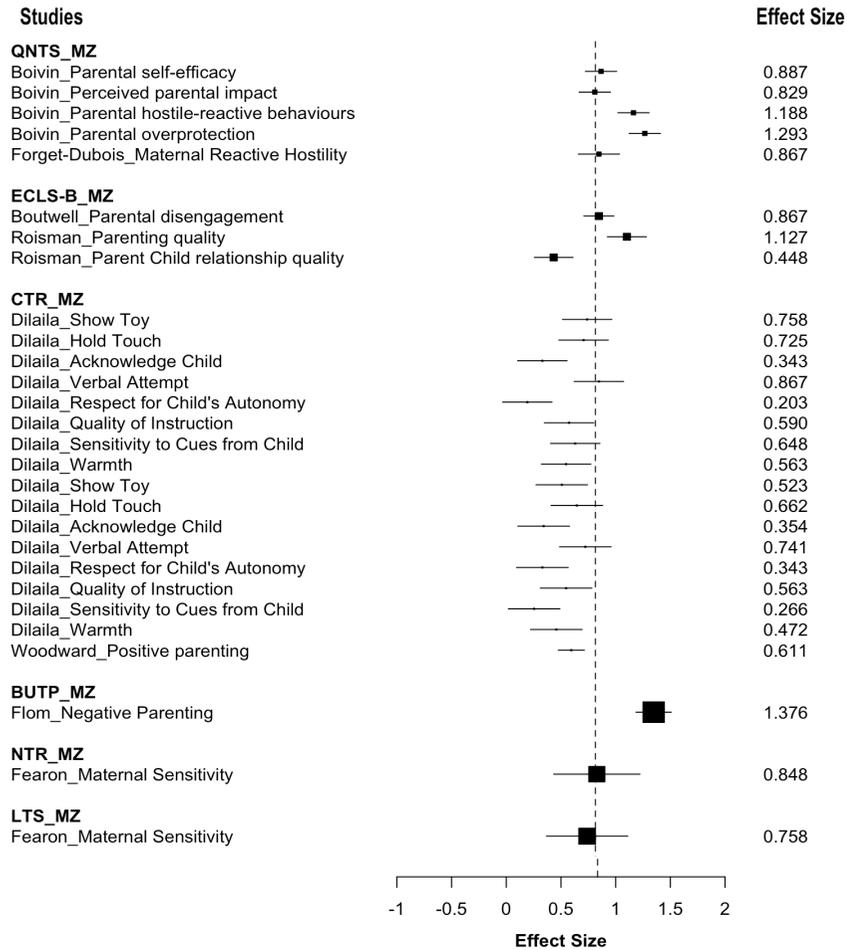
Metaregression Models Results

		β (95% CI)	S.E.	t	dfs	p	I ²
Model with Moderators	Intercept	0.60 (0.15, 1.05)	0.15	3.92	3.51 ⁺	0.02*	85.54
	Zygoty (DZ = 0, MZ = 1)	0.13 (-0.06, 0.32)	0.08	1.64	7.50	0.14	
	Age	0.00 (-0.03, 0.03)	0.01	0.22	3.88 ⁺	0.83	
	Parenting Type (NEG=0, POS= 1)	-0.05 (-0.46, 0.36)	0.13	-0.38	2.96 ⁺	0.73	
	Assessment method (OBS = 0, PR = 1)	0.16 (-0.27, 0.60)	0.17	0.94	5.52	0.38	
	Quality	1.51 (0.52, 2.50)	0.37	4.03	4.55	0.01*	
Model estimating Zygoty	Intercept	0.77 (0.58, 0.97)	0.08	10.2	4.97	<0.001	92.04
	Zygoty (DZ = 0, MZ = 1)	0.13 (-0.19, 0.45)	0.14	0.92	9.89	0.38	
Intercept Only Model	Intercept	0.84 (0.68, 0.99)	0.07	11.8	10.9	<0.001	92.16
					0	***	

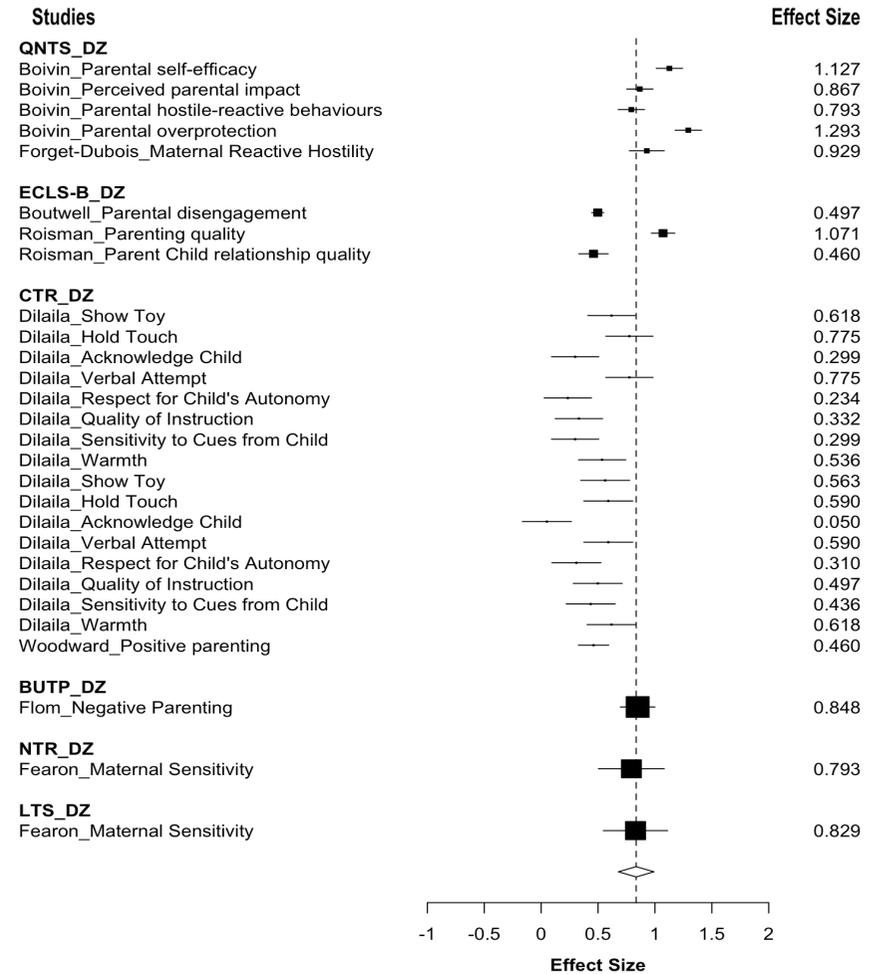
* $p < .05$. ** $p < .01$. *** $p < .001$

Figure 3

Forest Plot (MZ Studies)



Forest Plot (DZ Studies)



Across all parenting phenotypes, the weighted average effect size was $\beta = 0.84$ ($SE = 0.07$, $t = 11.80$, $p < 0.01$). I^2 statistic was 92.1% indicating a high degree of heterogeneity between studies. Sensitivity analyses showed that assuming differing levels of within-study covariance only minimally affected model estimates (test values ranged from 0 to 1 in 0.20 intervals, difference between coefficient values was less than 0.0001). The effect size for MZ and DZ twins (0.90 and 0.77 respectively) were then converted to Pearson's correlations: $r = 0.72$ and $r = 0.65$ respectively. We then used Falconer's (1960) formula to convert these correlations to additive genetic component, h^2 , as 0.14, shared environmental component, c^2 , as 0.58 and nonshared environmental component, e^2 , as 0.28.

Moderators

A further metaregression was run including age, zygosity (MZ/DZ), parenting type (positive/ negative), assessment method (observation/ parent report) and study quality as moderators. The full results of these meta analyses are reported in Table 5. The only significant moderator was study quality ($\beta = 1.51$, $SE = 0.37$, $t = 4.55$, $p = 0.01$), indicating that higher study quality was associated with higher twin correlations for parenting. The assessment method (observation versus parent report) was not a significant moderator ($\beta = 0.16$, $SE 0.17$, $t = 0.94$, $p = 0.38$).

Crucially, when zygosity was considered as a moderator, it did not have a significant effect ($\beta = 0.13$, $z = 0.08$, $t = 1.64$, $p = 0.14$), indicating that there was not a significant difference in the effect size of parenting concordance between MZ twins and DZ twins. This suggests that there is not sufficient evidence on the basis of this meta-analysis to affirm that parenting is heritable for children.

Although both age and parenting type were not significant moderators ($p = 0.83$ and $p = 0.73$ respectively), neither met the standard established by Tipton and Fisher

(i.e. $dfs > 4$) for results to be interpretable. As such, these moderators were re-examined using a mixed random effects meta-analysis to offer some indication as to whether they might have a significant effect. Parenting type (positive/negative) was found to be a significant moderator ($\beta = -0.39$ (-0.58, -0.20), $z = -4.03$, $p < 0.0001$), indicating that positive parenting was associated with lower correlations between twins in parenting than negative parenting, but age was not ($\beta = 0.00$ (-0.01, 0.01), $z = 0.21$, $p = 0.84$). Heterogeneity of studies remained high, as the I^2 statistic was 88.08%. A follow-up analysis exploring just the parenting type effect and its possible interaction with zygosity found no significant evidence for such an interaction ($z = 0.22$) and a consistent effect of parenting type in the same direction, even when zygosity was controlled for ($\beta = -0.29$ (-0.53, -0.05), $z = -2.40$, $p = 0.02$). The full results of these meta-analyses are reported in Table 6.

Table 6

Mixed Effects Model

		β (95% CI)	S.E.	z	p	I^2	R^2
Mixed Effects Mediation Model	Intercept	0.99 (0.82, 1.15)	0.09	11.51	<.0001 ***	88.02	27.02
	Parenting Type (NEG=0, POS=1)	-0.39 (-0.58, -0.20)	0.10	-4.03	<.0001 ***		
	Age	0.00 (-0.01, 0.01)	0.01	0.21	0.84		
Mixed Effects Interaction Model	Intercept	0.87 (0.65, 1.08)	0.11	7.93	<.0001 ***	87.64	30.18
	Parenting Type (NEG=0, POS= 1)	-0.29 (-0.53, -0.05)	0.12	-2.40	0.02 *		
	Zygosity (DZ = 0, MZ = 1)	0.25 (-0.05, 0.56)	0.16	1.61	0.11		
	Parenting type * Zygosity	-0.21 (-0.55, 0.13)	0.17	-1.22	0.22		

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

Discussion

The present review examined genetic, shared and non-shared environmental influences on early parenting. Specifically, we identified, summarized and quantitatively synthesized twin studies of parenting in children aged two years and under and used the differences in MZ and DZ twin correlations to establish estimates of the contributions of child genetic influences and child shared and non-shared environmental influences on parenting. We examined 9 papers spanning 6 twin studies of the aetiology of parenting. We then conducted a series of meta-analyses of parenting phenotypes using a metaregression model to account for the nonindependence of phenotypes reported from the same study. Results offered clear evidence supporting the role of shared environmental influences on parenting behaviour and some more mixed evidence for the role of child genetic effects. We also explored which variables might moderate this relationship and the results suggested that study quality was a significant moderator, with higher quality studies showing less differential parenting of twins. We found no evidence for the effect of age or assessment method on this effect. Results did offer preliminary indication that positive parenting was more affected by nonshared environmental effects than negative parenting.

First, we examined the correlation between twin pairs in parenting. Findings indicated a high degree of correlation between twin pairs, regardless of zygosity, indicating a significant contribution of shared environmental factors, c^2 , on parenting. This is consistent with literature indicating that parenting is influenced by factors of the parent's personality (e.g. Prinzie, Stams, Deković, Reijntjes, & Belsky, 2009) which form a part of the twin children's shared environment as well as factors that would be shared amongst all members of the family such as broader societal and cultural factors (e.g. Kendler, Sham, & MacLean, 1997). Given the increased recognition of the importance

of the shared environment (Burt, 2009), future work establishing the magnitude of specific shared environmental factors might shed further light on this. However, the estimate of c^2 in this paper was notably higher than previous meta-analyses of parenting across the lifeline (Klahr & Burt, 2014; Avinun & Knafo, 2013). It is unclear whether, given the relatively fewer studies of parenting in early childhood, this difference is indicative of a true difference in the influence of shared environment at this stage or just an artefact of random variation. Further behavioural genetics studies of parenting in early childhood might offer some clarity on this issue but also further information might be garnered using more longitudinal behavioural genetics designs to establish more concretely whether differential parenting between twins or child genetic effects on parenting increase in magnitude later in childhood.

Next, we examined the effect of zygosity on twin similarity of parenting in an attempt to establish the heritability of parenting. Monozygotic twins were more similar than dizygotic twins and an initial heritability estimate of 14% was established based on these values. However, this estimate was lower than those found in previous meta-analyses (e.g. Klahr & Burt, 2014) and zygosity was not found to be a significant moderator, indicating that the true heritability of parenting could be as low as zero. If so, this is consistent with research indicating that evocative rGE may become larger as people get older (Knafo & Jaffee, 2013) as well as evidence that children's genotypes do not affect parenting. For example, findings reported by Avignun and Knafo (2013) and Kendler & Baker (2007) suggest that observed positive and negative parenting are not heritable. Genetically informed research on the parenting of infants is needed for clarification. In particular, longitudinal designs could offer further clarity on whether genetic influences on parenting become more pronounced beyond infancy and across middle childhood. Such research could offer further elucidation of the role of evocative rGE in the early years.

Although this ratio of genetic and environmental effects was different than the summary statistics offered in previous meta-analyses, the differences were consistent with the patterns noted in Klahr and Burt (2014). Specifically, c^2 was higher at the early stage of development that is the focus of this report, than at later stages of the lifespan. Furthermore, the lower genetic influence was consistent with the pattern of increasing genetic influences with age (Plomin, DeFries, Knopik, & Neiderhiser, 2012).

Finally, we examined possible moderators of the strength of correlation between twin pairs of parenting. Results from the metaregression model suggested that study quality was a significant moderator and that higher quality was associated with increased similarity between twin pairs, offering further support to the findings described above. Results of moderation analysis also indicated that age was not a significant moderator. This is perhaps unsurprising given the limited range but suggests that infancy can be considered as a homogenous period with relatively stable parenting dimensions in future research.

This analysis also examined assessment method as a possible moderator but did not offer evidence of a significant effect of assessment method on differential parenting within twin pairs, in contrast to the findings by Avinun and Knafo (2013) and Klahr and Burt (2014), whose meta-analyses suggested differential parenting is less evident in parent reports. This difference in findings may be related to the age range considered in this study as some literature has suggested that differential parenting associated with a host of factors may begin in toddlerhood or later (citation). As such, observational studies in early childhood may differ from observational studies across the lifespan in their findings of nonshared environmental influences. This meta-analyses offers some indication that early childhood parenting may be less sensitive to assessment methods, which, if true, could suggest two hypotheses: 1) that parents may be more accurate in early childhood or that 2) parents are consistently insensitive to

their own differential parenting but that there is less differential parenting occurring in early childhood. Further research using both methods concurrently at this stage of development could offer insights into which of these two hypotheses would be the case. These findings may in turn shape the assessments used in parenting interventions or the interventions offered (i.e. using self-reports in early childhood, as these tend to be less labour intensive, or increasing parent sensitivity to differential parenting in middle to late childhood).

Parenting type—positive versus negative—was also considered as a possible moderator. Although the metaregression model did not offer support for this variable having a significant impact, the potential moderator did not meet the standard advised for interpretability. As a result, secondary analyses were conducted without considering covariance within studies. These analyses indicated a significant effect of parenting type and offered evidence that this effect did not interact with zygosity—that is, positive parenting was more differentiated between twins than negative parenting but this effect was not genetically driven. These findings suggest that children may evoke positive parenting through behaviour that is not genetically determined, as has been previously indicated by research on MZ twin differences (e.g. Caspi et al., 2004). Further research on non-genetically determined differences in twin behaviour and their aetiology could offer useful insights into early parenting.

Limitations

One limitation of this meta-analysis was that it reviewed a relatively small number of studies. Because of the limited number of examinations of the child-based aetiology of parenting in infancy, our findings were less certain that they might have been otherwise. Furthermore, the number of studies limited our ability to conduct and

interpret some moderation analyses that might otherwise have further elucidated these effects. In particular, the uncertainty around the impact of parenting type is a matter of importance and requires further study. While concerns about sample size are important to hold in mind, this study makes an important contribution in being the first and largest synthesis of genetically informative studies on evocative *rGE* specifically in infancy.

Our rationale for inclusion of classical twin designs only was that it allowed for methodological consistency and more homogeneity between studies. However, inclusion of twin designs exclusively means that the strength of our findings depends on important assumptions about the twin design being met. First, the assumption of equal environments. The twin design assumes that MZ and DZ twins have equally similar environments and that differences between MZ and DZ twins are solely related to their genetic differences. There is mixed evidence on the validity of this assumption as some researchers have challenged it (e.g. Richardson & Norgate, 2005) but research based on DZ twins incorrectly identified as MZ twins suggested that actual—and not perceived—zygosity was more predictive of ratings of similarity between twins (Goodman & Stevenson, 1991; Scarr & Carter-Saltzman, 1979) offering support for this assumption. Second, there are concerns that the parenting of twins is different to the parenting of non-twins, in ways that limit the generalizability of findings based on twin studies (e.g. Kendler et al., 1995). For these reasons, it has been suggested that examining multiple behavioural genetics designs together might minimize the impact of such assumptions.

An additional limitation of our design is that we only considered the effects of child genetic variance. While this is an important contribution to our understanding of evocative *rGE* in infancy, a design that examined both parties concurrently—i.e. also considered the effects of genetic variance of the parents—and longitudinally would

provide a more complete picture and might offer further insight into the presence and onset of evocative *rGE* in early childhood.

More broadly, behavioural genetics has recently come under criticism (Charney, 2008; Charney, 2012) for producing heritability estimates that are not replicated by molecular genetic research (often referred to as the “missing heritability” problem; Maher, 2008; Turkheimer, 2011). Additionally, there are known limitations of quantitative genetic approaches, including twin studies. For example, phenotypic twin studies do not directly index genetic or epigenetic processes (Wolffe & Matzke, 1999) or gene–environment interactions (Cicchetti, 2007). Nonetheless, research has indicated that twin findings converge with findings from other behavioural genetics methods with different assumptions (e.g. adoption studies), adding strength to the findings. Furthermore, they do offer some insight into the child’s role in their own parenting and more specifically, into the role of the child’s genes. As such, these studies offer a meaningful contribution to the literature on parenting.

Clinical Implications

Given that the findings of this meta analysis indicate that shared environmental factors are strongly determinant of parenting in infancy, this has a number of consequences for clinical practice. This finding is consistent with existing literature on the importance of home environment on parenting and subsequently child development (e.g. Duncan, Ziol-Guest, & Kalil, 2010) as well as on how parenting is influenced by the parent based factors (e.g. Dix & Meunier, 2009) and broader societal and cultural factors (e.g. Kendler, Sham, & MacLean, 1997). As such, interventions for parents would be strengthened through a thorough assessment and consideration of these factors in play a part in shaping and maintaining unhelpful parenting strategies.

Future directions

Future research should include studies of parenting that are longitudinal and ideally measure parent- and child-based effects concurrently to get a better understanding of when different factors become important and how they interact. Including molecular genetics designs into future meta-analyses as well as a broader range of behavioural genetic study designs could also allow for a more thorough examination of evocative *rGE* in parenting. Despite the limitations of this meta-analysis, it offers an initial estimation of the child-based factors that might influence the parenting children receive. Now that genetic and environmental contributions to parenting in infancy are better understood, we can move our attention to identifying the specific environmental processes and factors that underlie the consistency of parenting between twins and to understanding when in development, gene environment correlation becomes more prominent.

Conclusion

This is the first meta-analysis of twin studies of parenting in infancy. It aimed to try and establish the existence and magnitude of evocative *rGE* in this context and explore moderators of the heritability of parenting at this early stage of development. The results of the current meta-analysis have several implications.

Results suggest that infant twins experience very similar parenting, which appears to be a result of predominantly shared environmental factors. Study quality was a significant mediator and this effect strengthened with quality. This effect does not appear to be mediated by age, suggesting that within infancy, there is not significant differentiation in parenting. Interestingly, zygosity was also not a significant mediator,

leaving open the question of whether there is a genetic component to parenting at this early stage and therefore not resolving the question of the presence of gene-environment correlation in parenting. This stands in contrast to meta-analyses conducted across the lifespan of studies of parenting, indicating that evocative rGE in parenting may become stronger when a child is older, more independent and thus more able to influence the environment to evoke the parenting that they receive. The predominance of shared environmental effect offers some further insight into the factors that influence early parenting as being more environmental than genetic.

Collectively our findings provide some evidence that early parenting is predominantly determined by parent-based factors or factors shared across the family and not differentiated by children. Given the crucial importance of parenting in early childhood for early development and in establishing trajectories for children's attachment and wellbeing throughout the lifespan, the importance of shared environmental factors such as parent-based factors and social and cultural factors offers guidance for increasing the effectiveness of early parenting intervention for clinicians and politicians.

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Part 2: Empirical Paper

Testing the impact of gene-environment interplay on the development of children's
internalising problems

Abstract

AIM: To test for the impact of parenting on child anxiety and reciprocal influences of child anxiety on parenting in a cross-lagged longitudinal framework from nine months to seven years. Further, to test whether evoked over-reactive parenting is linked to genetic risk for internalizing.

METHOD: Using longitudinal data from the EGDS, transactional relationships between adopted child anxiety and adoptive parental overreactivity from nine months to seven years were explored in a cross-lagged structural equation model. Genetic risk was modelled using a composite of birth mother internalizing problems and family history as measured using the CIDI and the model also explored whether any identified transactional associations were associated with genetic risk.

RESULTS: There was evidence of child anxiety at 54 months evoking increased over-reactive parenting at 72 months and parenting at 72 months prospectively predicting increased child anxiety at 84 months. Although the timing of this effect varied with informants, there was a consistent pathway from child anxiety to parental overreactivity. However, internalizing genetic risk was not associated with these transactional processes or to child anxiety or parenting at any point during this time frame.

CONCLUSION: The current findings suggest there is a bidirectional relationship between child behaviour and parenting behaviour and, thus, that children partially evoke the parenting they receive. However, there was no evidence found for this evocative process being genetically based.

Introduction

Internalising disorders in early childhood show a prevalence of around 12.8% in Western countries (Sawyer et al., 2016), with symptoms being fairly stable over time (Bayer, Sanson, & Hemphill, 2006) and predictive of later depression and adjustment problems (e.g., Bittner et al., 2007). Increasing recognition of the impact of childhood emotional problems on long-term physical and mental health outcomes (Jokela, Ferrie, & Kivima, 2009) has focused attention on the early causal origins of these difficulties in order to support effective prevention (Bayer & Beatson, 2013). Four factors have been commonly hypothesized to be implicated in the development, maintenance, and transmission of internalizing symptomatology: genetic factors, cognitive factors, family factors, and sociocultural factors (e.g. Manassis et al, 2005; Merikangas, 2005). A considerable volume of research has been directed towards genetic and family factors, the focus of the current study.

Parenting

One particular family factor, parenting, has long been hypothesized to affect risk for the development of child internalizing problems (Vasey & Dadds, 2001). Meta-analyses have found consistent, if small to moderate, associations between parenting quality and a range of internalizing problems (McLeod, Weisz, et al., 2007; McLeod, Wood, et al., 2007; Brook & Schmidt, 2008; Yap & Jorm, 2015). Specifically, notable associations between negative strategies such as hostility and overcontrol and internalizing problems in childhood (Pinquart, 2016).

Further evidence for the importance of parents in the development and maintenance of child internalizing problems is provided by the literature on interventions for child anxiety, which show that parent led interventions are as successful as clinician

delivered CBT (McKinnon et al, 2018; Lebowitz et al. 2019) in reducing child anxiety. A meta-analysis of RCTs comparing child focussed interventions and child-parent interventions (Brendel & Maynard, 2013) found that involving parents led to a higher effect size of treatment. Similarly, a review of family CBT (Creswell & Cartwright-Hatton, 2007) found that it was superior to no treatment and, for some outcome measures, including diagnosis, also superior to Child CBT. Furthermore, the authors found that this effect was more profound for children of anxious parents. This literature provides further evidence for the causal role of parental behaviour in child anxiety.

Child to Parent Effects

In general, developmental scientists and interventionists have tended to assume that associations such as these reflect causal effects of parenting on child development. However, increasingly it is becoming recognised that child characteristics—some of which are likely genetic in origin—may also influence parenting (Pinquart, 2016). Genetically sensitive studies are crucial in this context, as they can be used to parse genetic from environmental effects and, when combined with longitudinal data, examine bidirectional influences of genes and environments on child development, as they unfold over time.

Behavioural Genetics Studies of Directionality

Genetically informative studies can yield strong evidence of environmental pathways of influence in child development by ruling out alternative genetic explanations. Children-of-twins studies have been used, for example, to show the presence of environmental mechanisms underpinning parent-to-child transmission of psychopathology. In one such study, Eley et al. (2015) used a cross - sectional children - of - twins design to directly examine genetic mechanisms influencing

intergenerational associations between parental and offspring anxiety. They found that the correlation between parent and adolescent anxiety was not accounted for by genetic factors, indicating that the familial association is attributable to environmental factors, such as exposure to an anxious relative, although the possibility of environmentally-based child-to-parent effects (i.e., reverse causation) could not be unequivocally ruled out due to the cross-sectional nature of the data. Similarly, Silberg et al. (2010) found that the relationship between parental depression and child depression was driven by direct environmental transmission and not genetic factors. Singh et al. (2011) provide further evidence using the children-of-twins approach that the association between depression in parents and children is primarily environmental. These studies suggest that for internalizing symptoms parent-child associations may be largely environmental in nature. However, such studies do not directly address parenting processes, and by focusing on the inter-generational similarity between parents and children, they do not address familial influences on child internalizing problems that are independent of the parent's internalizing problems.

Gene-Environment Correlation

Child-to parents-effects, typically captured by the presence of gene-environment correlation (r_{GE}) in genetic studies, have been shown to play a role as well (Hayden et al., 2010; Lau et al., 2007). r_{GE} , a key focus of this study, can be defined as a correlation between an individual's genome and the environment they inhabit. There are three forms of r_{GE} : passive, active, and evocative (Plomin, Defries, & Loehlin, 1977; Scarr & McCartney, 1983) but our focus in this report will be evocative r_{GE} , which occurs when genetically influenced behaviour of the child evokes a particular response from the environment, such as parenting. For example, data from genetically sensitive studies have indicated that children at genetic risk for anxiety problems may evoke

more negative parenting (Hayden et al., 2010). Although quite a number of studies examining *rGE* and childhood internalizing problems, such as Hayden et al., (2010) have relied on candidate gene associations, the replicability of which are open to question, twin data also provides evidence of gene-environment correlation in relation to child or adolescent internalizing problems. For example, Pike et al. (1996) used a sample of twins and siblings, aged nine to eighteen months, to explore the genetic and environmental contributions to relationships between parent negativity and adolescent child depressive symptoms. They found that there was a significant effect of the child's genes on the association between parental negativity and adolescent depression, indicating evocative *rGE*. Another twin study, Eley et al. (2010), used a sample of 8-year-old twins to examine the relationship between maternal controlling behaviour and childhood anxiety. They found that individual differences in maternal control were highly related to child genes, and that the overlap between high child anxiety and maternal control was primarily due to shared genetic factors within children between the two phenotypes. These results suggest that maternal control is likely to have been elicited by children with high levels of anxiety, further evidence of evocative *rGE*. Furthermore, an extended children-of-twins model was used by Narusyte et al. (2008), using both children of twins, aged 11-20 years, and twin children of non-twin parents, aged 16-17 years, to test for different forms of *rGE* in the association between maternal emotional overinvolvement and child internalizing problems. They found evidence of evocative *rGE*, that is that the child's internalizing problems evoked emotional over involvement on the part of the mother.

Limitations of Existing Research

Two important limitations of existing behavioural genetic research are evident from a review of the literature. First, the bulk of the evidence for *rGE* is derived from

studies of children aged 8 and above. This is a significant limitation because child internalizing symptoms are apparent from late infancy and increase in prevalence across preschool age and childhood (Bongers et al., 2003). Furthermore, internalizing symptoms appear to be heritable as early as 24 months (Saudino et al. 2008), so it is plausible that these genetically influenced symptoms might evoke responses in caregivers. Prospective studies beginning in early development are important for understanding the emergence of *rGE* and characterising the extent to which the balance between parent-to-child and child-to-parent effects changes with development, as many developmentalists would expect (Möller et al., 2016). Secondly, the majority of genetically informative studies of child internalizing problems, including the ones referred to above, are cross-sectional, which means that direct evidence of predictive and directional effects of parenting on child internalizing problems or child effects on parenting is mostly lacking. Longitudinal data is particularly critical when exploring *rGE*, where evoked parenting is sometimes assumed to mean that parenting does not directly affect child outcomes, but rather is a correlate of genetic risk. The possibility that such evoked parental responses do in fact contribute directly to future child internalizing symptoms remains to be thoroughly tested and doing so requires longitudinal data.

Early Growth and Development Study

The Early Growth and Development Study (EGDS) is a valuable resource for examining these forms of longitudinal genetic and environmental questions, and it has produced important evidence of both environmental influences on child internalizing problems and evidence of genetic effects and gene-environment interplay, including gene-environment correlation and gene-environment interaction (GxE). For example, Brooker et al. (2011) found, in the EGDS sample that infants were more prone to social

inhibition in a live observation of interaction when three conditions were met: genetic risk was elevated, as indicated by birth mother social phobia diagnosis; the child had difficulties with attentional regulation; and the adoptive parent had elevated symptoms of anxiety. When the cohort was followed up at age 18- and 27-months of age, a similar pattern was observed: heightened anxiety symptoms in the child were observed when the child's birth mother had higher anxiety symptoms, the child had poorer attentional control at age 9 months and an adoptive parent (adoptive mother or father) had elevated anxiety symptoms (Brooker et al., 2014). Although adoptive parental behaviour was not directly measured, the adoption design strongly suggests that—since the adoptive parent and adopted child do not share genes—the adoptive parent's contribution to this moderated effect is environmental in nature and, presumably, mediated by parenting or other family environment factors. Natsuaki et al. (2013) found corroborating evidence for this, showing that, given genetic risk for social inhibition, adopted children were likely to show elevated levels of behavioural inhibition only in the context of lower maternal responsiveness.

In addition to these GxE processes, the possibility that evoked maladaptive parental responses (i.e., *rGE*) may also play a role in child anxiety or internalizing problems was suggested by a further analysis of EGDS data reported in Brooker et al. (2015). This report used cross-lagged longitudinal modelling to demonstrate not only that adoptive parent anxiety symptoms predicted future child negative affect, but also that the reverse was true: child negative affect at 9 months predicted parental anxiety symptoms at 27 months. In this latter analysis, birth parent data was not used to test the hypothesis that such an evocative effect could be linked to genes involved in internalizing disorders, but this remains a plausible possibility. Nevertheless, the results suggest both parent-to-child environmental influence and child-effects on the parent.

Interestingly, more recent analysis by Ahmadzadeh and colleagues (2019) using the same EGDS sample from ages 6 to 8 years, found no effects of genetic risk on child internalizing problems but evidence emerged of a bidirectional relationship between child internalizing problems and adoptive parent anxiety symptoms. The study found that earlier child internalizing problems, at age 7, predicted later adoptive mother anxiety symptoms, at age 8. However, the data also indicated parent-to-child longitudinal prediction, with earlier paternal anxiety symptoms, at age 6, predicting later increased child internalizing problems at age 7. As with the Brooker (2011) paper, the adoption design would lead us to suspect that the parent-to-child effect would be a result of environmental factors such as parenting quality, but this was not explored in that report. Furthermore, the existing analyses have focussed only on parental internalizing phenotypes, and therefore do not address the broader influence of parenting on child internalizing problems, which may show a different pattern of parent-to-child and child-to-parent effects than those effects that are specifically linked to the parental internalizing phenotype.

The present study

The present study built on the work described above from EGDS by:

- 1) Testing for parenting influences (as measured by adoptive parent negative/over-reactive parenting) on child anxiety symptoms in a cross-lagged longitudinal framework from 9 months to 7 years (see Figure 1).
- 2) Testing for reciprocal influences of child anxiety on over-reactive parenting in the same cross-lagged model from 9 months to 7 years (see Figure 1).
- 3) Test whether evoked over-reactive parenting is linked to genetic risk for adult internalizing disorders, as indicated by birth parent diagnoses and family history (see Figure 2).

Figure 1.

Cross-lagged longitudinal SEM model testing bidirectional transactions between child anxiety problems (Anx) and overactive parenting (OR).

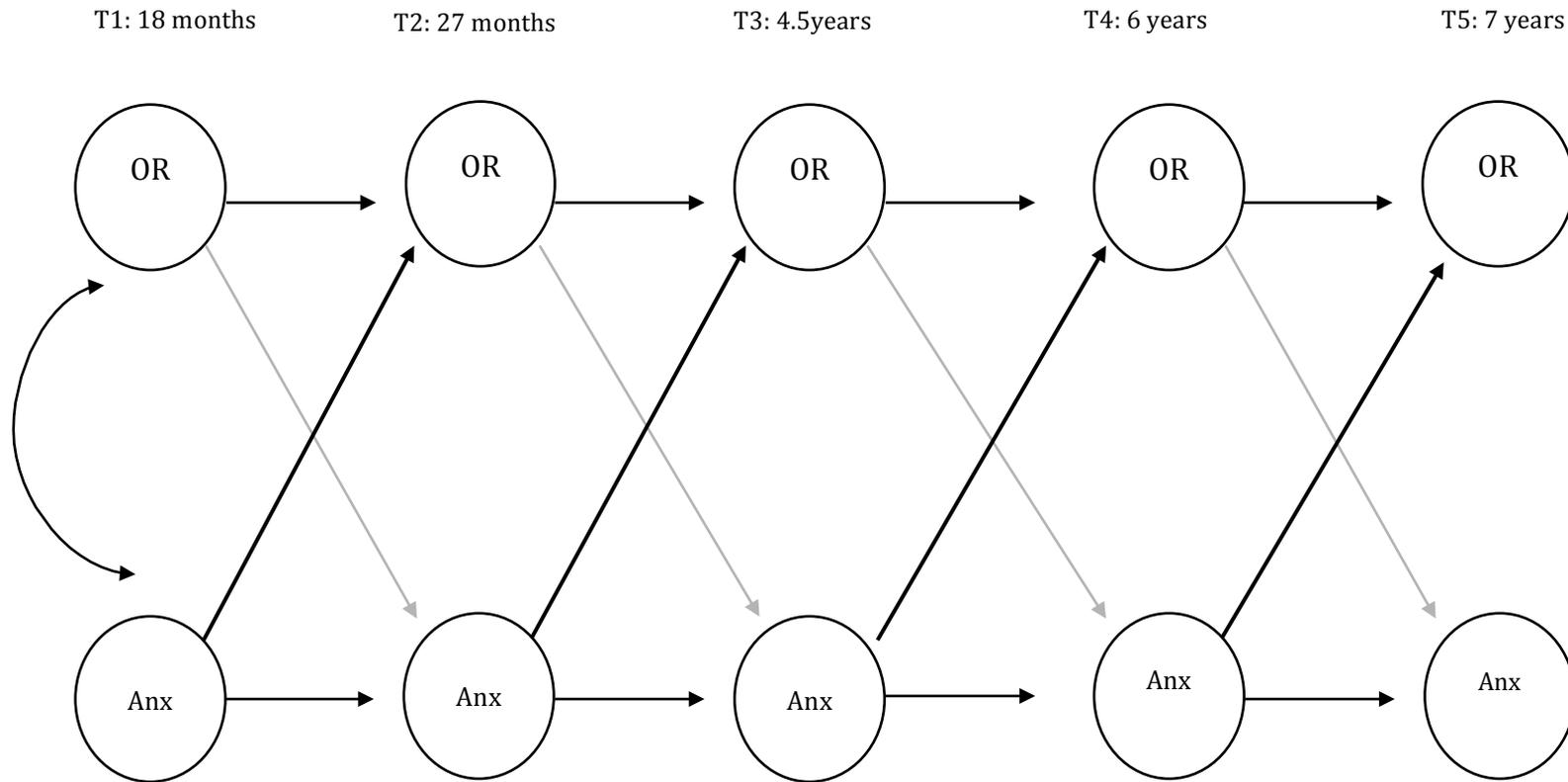
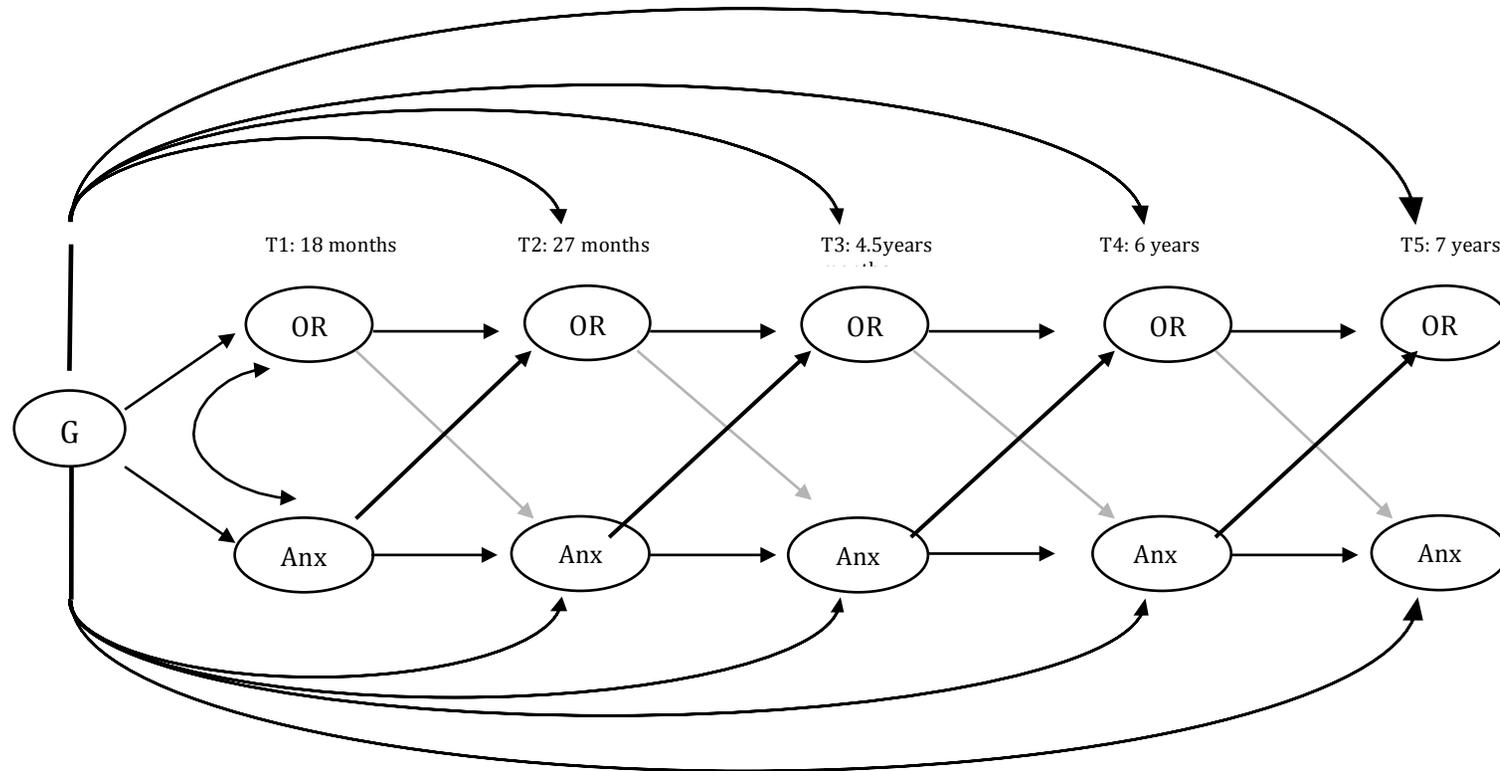


Figure 2.

Cross-lagged longitudinal SEM model testing mediated rGE by child anxiety



G- Internalizing Genetic Risk; ANX - Child Anxiety; OR - Parental Overreactivity

Our hypotheses were as follows: First, we hypothesised that more overreactive parenting would be associated with subsequent higher child anxiety symptoms. Second, we hypothesized that higher child anxiety symptoms would be associated with more overreactive parenting later. Third and finally, we hypothesized that higher genetic internalizing risk would be associated with more overreactive parenting and specifically, with evoked over-reactive parenting. We hypothesized that these associations would be maintained when we controlled for child sex, prenatal risk and adoption openness.

Methods

Participants & Procedures

The study used data from participants of the Early Growth and Development Study (EGDS), an on-going prospective cohort study of adoption across the United States (Leve et al 2007; 2013; 2019). Families were recruited from adoption agencies if they met the following criteria: 1) the adoption was domestic, 2) the baby was placed with a non-relative, 3) the baby had no known medical conditions, 4) the adoption took place within 3 months post-partum, and 5) all parents were able to read and understand English at an eighth-grade level.

The first two cohorts of the study were used for this project and were composed of 561 linked triads of adopted children, adoptive parents and birth parents, recruited from 2003 with the help of 45 US adoption agencies across 15 states. At adoption placement, the mean child age was 6.2 days ($SD = 12.4$ days) and the sample was slightly more male than female (57.2%). Birth parents were typically in their mid-twenties, birth mothers' mean age was 24.4 ($SD = 6.0$) and birth fathers' mean age was 26.1 ($SD = 7.8$), whereas adoptive parents were typically in their late-thirties, adoptive

mothers' mean age was 37.4 ($SD = 5.6$) and adoptive fathers' mean age was 38.3 ($SD = 5.8$). Both sets of parents were typically Caucasian but adoptive parents were more likely to be (mothers: 91.8%; fathers: 90.4%) than birth parents (mothers: 70.1%; fathers: 69.9%). Adoptive parents were likely to be middle to upper-middle class (college educated and with a household income above \$100,000) whereas birth parents were more likely to be working class (less than a college education and had household annual incomes less than \$25,000). Detailed information regarding the demographic composition of the full sample as well as more information about recruitment and engagement has been reported elsewhere (Leve et al., 2013; 2019). To enable comparison and separate consideration of adoptive mothers and fathers, all triads with same-sex adoptive parents were removed, leaving a final sample of 521 families.

Ethical Considerations

Adult participants received full information about the study and gave written consent. Adoptive parents gave consent on behalf of their children. The present study was covered under the ethical approval obtained for the EGDS project from institutional review boards at the University of Oregon and Pennsylvania State University, as these were the institutions leading on data collection.

Measures

Adoptive Maternal and Paternal Negative Parenting: The Parenting Scale (Arnold, O'Leary, Wolff, & Acker, 1993). Negative parenting was measured by adoptive parent self-report using the over-reactive parenting subscale from The Parenting Scale (Arnold et al., 1993). Assessments of over-reactive parenting from 9 months to 8 years were used, at 9, 18, 27, 54, 72, 84 and 96 months respectively. At 9 months, a reduced version of this subscale was used, not including three items that were deemed to be not

age-appropriate (e.g. items with the framing “when my child misbehaves...”). To maximise consistency across time-points, this reduced 7-item scale was used throughout. To confirm that the reduced subscale was a reliable measure, correlations between the reduced subscale and the full subscale were calculated for all time points where both were available and these correlations were 0.95 or above for all time points, ($p < 0.001$), offering support for the reliability of this reduced subscale.

The primary analysis was run using the average of both adoptive parents’ self-reports of over reactivity, where available. This decision was driven by the importance of the contributions of both parents at different points across early and middle childhood (Majdandžić et al., 2018) and the fact that over this time period, a significant number of adoptive parents’ separated and averaging allowed us to account for the role of both households in the child’s experience of parenting. However, given small correlations between mothers’ and fathers’ ratings (r range: 0.25-0.35) and research indicating differences in evocation (Marceau et al., 2013) and stability across time (e.g. Connell & Goodman, 2002; Hudson et al., 2008) of maternal and paternal parenting strategies, separate analyses were then run for mothers’ and fathers’ over-reactive parenting self reports and sensitivity analyses were conducted to examine whether there were differences in the pattern of results by rater.

Child Anxiety: The Child Behaviour Checklist (CBCL; Achenbach & Rescorla, 2001).

The CBCL was used as a measure of anxiety problems. Assessments of child anxiety from the CBCL were collected at 18, 27, 54, 72, 84 and 96 months respectively. This study used a harmonized version of the DSM-5 Anxiety Problems subscale from the 1.5-5yr and 4-18yr versions of the CBCL as both versions were used in the EGDS database at age appropriate time points. This subscale was chosen because it had the highest proportion of items that were present in both versions of the CBCL of all subscales measuring child anxiety. We also felt it was important to disaggregate anxiety

and depression as these have been shown to be differentially affected by different parenting strategies (Yap & Jorm, 2015) so the broader internalizing subscale and the anxious/depressed subscale were not appropriate. This reduced subscale contained the 6 items that are consistent across the two versions for this measure (items 11, 29, 45, 47, 50, 71 and 112 from the CBCL 4-18 version). Scores were only used if all 6 items had been completed (this resulted in the exclusion of less than 5% of available data at all time points). This reduced scale was highly correlated with the full anxiety problems scale ($r > 0.85$ at all time points, $p < 0.001$). Furthermore, due to an idiosyncrasy in the data collection, the two cohorts of the EGDS data study were given different versions of the CBCL at 72 months. Due to the fact that the same wording was used for the 6 items making up the reduced subscale in both measures, this allowed us to compare whether responses to the 6 items in the context of different versions was significantly different, assuming that the cohorts were not significantly different for any other reason. Looking at birth father and birth mother data separately, we found that the scores were not significantly different between the two groups ($t = 0.58$, $p = 0.56$ and $t = 1.01$, $p = 0.31$ respectively), offering evidence that this reduced subscale showed consistency between versions.

The primary analysis was run using the average of both adoptive parents' self-reports of child anxiety, where available. However, given the moderate correlations between mothers' and fathers' ratings (r range: 0.32-0.52), separate analyses were then run for mothers' and fathers' reports of child anxiety and sensitivity analyses were conducted to examine which models was the best fitting.

Internalizing Genetic Risk: The Composite International Diagnostic Interview (CIDI). To assess genetic risk for internalizing disorders, we followed previous analyses (e.g., Marceau et al., 2016) and used birth parents' reports in the CIDI on symptom counts,

diagnosis counts, and age of onset for 11 internalizing disorders as well as the number of their first-degree relatives with internalizing problems to create a composite score was created from these variables. This composite score acted as a proxy-measure for offspring genetic risk. Analyses were all run with birth mother risk scores because, as is the case with many adoption datasets, birth father involvement was less consistent than birth mother involvement and therefore the data was sparser, with over 64% of birth father data being imputed to calculate birth father risk scores (Ahmadzadeh et al., 2019).

Covariates

In keeping with previous EGDS analyses (e.g. Ahmadzadeh et al., 2019), we controlled for a number of key covariates: adoption openness (measured across the duration of the study), obstetric and prenatal risk, adoptive family socio-economic status and child sex. As well as allowing for more direct comparison to other reports from the EGDS study, these covariates allowed us to account for birth parent contributions outside of genetic effects (i.e. continued contact post-adoption) as well as factors commonly known to impact parenting (e.g. socioeconomic status; Bornstein et al., 2003).

Adoption Openness: Adoption openness was measured using a mean standardised composite (Ge et al., 2008). of birth mother and adoptive parent ratings of (1) the degree of information shared, (2) frequency of communication and (3) contact between birth parents and adoptive families. Ratings were given for each of the three scales at each time point of assessment (i.e. 9mo, 18mo, 27mo, 4.5yr, 6yr and 7 year for adoptive parents and 4mo, 18mo and 5yr for birth mothers) on a 7-point scale from very closed, where neither sets of parents had any information about or contact with the

other, to very open, which involved frequent and regular visits and communication. To arrive at a single construct, we standardized each item, averaged within subscales, and then summed the results. Ratings were highly stable over time for all reporters with 68-72% of variance stable over time and there was a high correlation between reporters at all time points ($r = .71-.84$, $ps < 0.001$) suggesting adoptive parents and birth mothers agreed strongly about the level of openness (Ge et al., 2008). For this study, the adoption openness score used at each time point was an average of all available scores collected up to and including that point.

Obstetric and prenatal risk: Obstetric and prenatal risk were measured using a weighted total risk score (Marceau et al., 2016) of birth mother reports at 5 months postpartum. The risks assessed in this report included assessment of complications during pregnancy (e.g. substance use), during labour and delivery (e.g. induction) and neonatally (e.g. prematurity).

Adoptive family socio-economic status: Socio-economic status was measured using adoptive parents' self-reported household income at the start of study. Income was reported on a categorical scale from 1 to 11 (i.e. 1=less than \$15,000, 2=\$15,001 to 25,000, 3=\$25,001, to 40,000, 4=\$40,001 to 55,000, 5=\$55,001 to 70,000, 6=\$70,001 to 100,000, 7=\$100,001 to 125,000, 8=\$125,001 to 150,000, 9=\$150,001 to 200,000, 10=\$200,001 to 300,000 and 11=more than \$300,000).

Child sex: Child sex was collected at recruitment and scored on a binary scale (0 = male, 1 = female) in line with previous EGDS research (Leve et al., 2013).

Data Preparation

Prior to running the main analyses, each of the included variables and covariates was examined to check if it met normality assumptions for Full Information Maximum Likelihood (FIML) imputation method for dealing with missing data and appropriate transformations were applied if not. All variables were within acceptable thresholds for skewness and kurtosis and therefore, none were transformed. Furthermore, the distribution for each variable was examined to identify outliers (i.e. values more than 2 SD from the mean) and to identify any outliers through the use of visual examination of a box plot. Outliers that were identified were then addressed through winsorization. One outlier was identified in the obstetric risk variable (represented in Figure 3) and this outlier was assigned a value 0.1 higher than the next largest value. Similarly, six outliers were identified in the birth mother internalizing genetic risk variable (represented in Figure 4) and these outliers were assigned a value 0.01 higher than the next largest value.

Figure 3

Obstetric Risk Weighted Score Box Plot

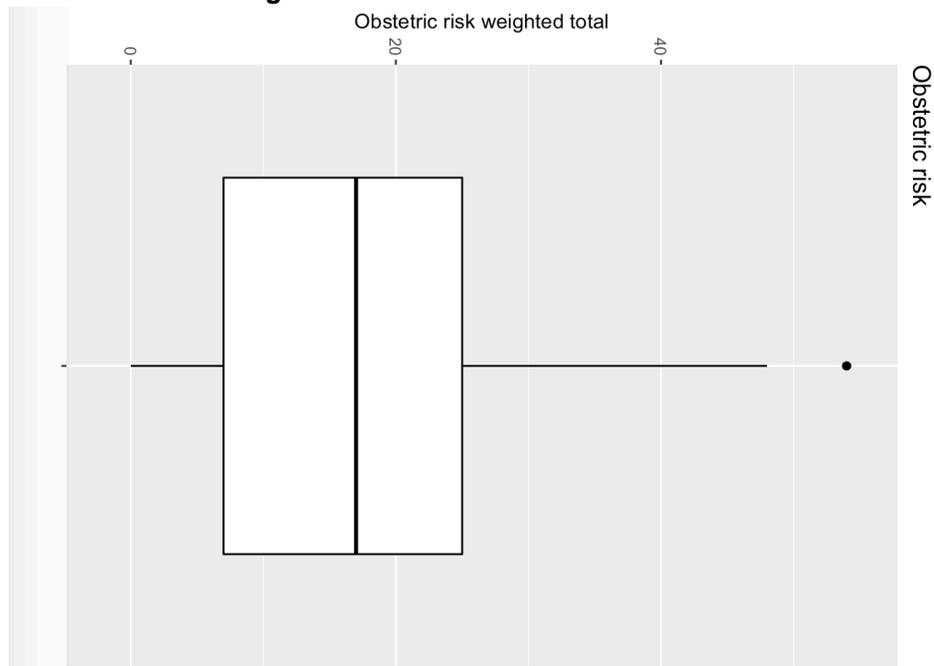
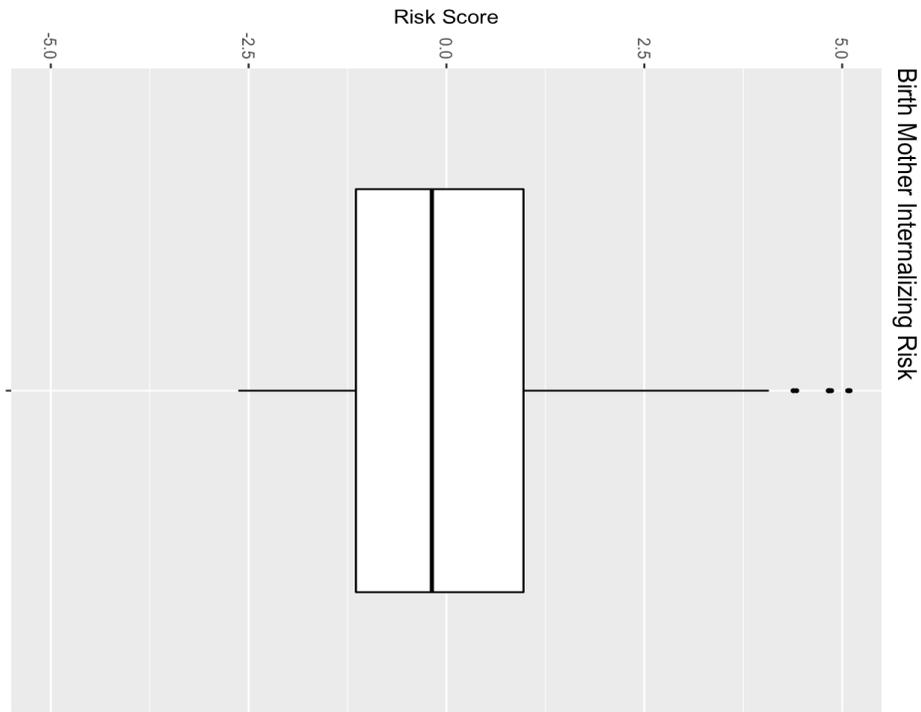


Figure 4

Birth Mother Internalizing Genetic Risk Score Box Plot



Data Analysis

The primary analyses utilised longitudinal cross-lagged modelling using the structural equation-modelling (SEM) framework. Maximum likelihood SEM techniques were used to test for cross-lagged effects and longitudinal mediation by constraining the relevant paths to be equal and assessing the significance in the change in the model likelihood ratio statistic. Using SEM with longitudinal data allowed us to examine whether new variance in a mediator or outcome was predicted by variance earlier in a predictor or mediator, respectively, after accounting for stability of and prior associations between the variables. We followed Cole and Maxwell's (2003) guidelines for testing mediational hypotheses with longitudinal data, using the statistical program R 3.5.2 using the package lavaan (Rosseel, 2012). Model fit was assessed using chi-square tests of difference and was considered acceptable if root-mean square error of

approximation (RMSEA), comparative fit index (CFI) and Tucker–Lewis index (TLI) fell within recommended ranges (Hu & Bentler, 1999). Post-hoc sensitivity analyses were compared using the AIC and BIC, to establish which reports for parent overreactivity and child anxiety respectively resulted in the best fitting model.

Because of the number of assessments and data collected at each time point, a significant number of participants did not provide data for each assessed variable here. Previous research has indicated that excluding such cases can not only undermines statistical power but also can bias parameter estimates (Allison, 2003). As such, missing data was accounted for using the FIML methodology, which utilizes all available data to estimate the parameter estimates of a model. The primary source of missing data was missing adoptive parents' self-reports of overreactivity at 4.5 years and 6 months.

Results

Descriptive statistics for all variables used in the models below are presented in

Table 1.

Table 1

Descriptive Statistics of Included Variables

		N	Mean	SD
	Obstetric Risk Score	521	16.68	11.76
	Household Income	506	6.81	1.91
	Internalizing Genetic Risk	511	-0.04	1.43
	9M	488	1.77	0.52
	18M	485	2.04	0.58
	27M	463	2.2	0.58
	54M	249	2.48	0.57
Parent	72M	252	2.43	0.57
Overreactivity	84M	291	2.47	0.6
	18M	474	7.03	0.89
	27M	417	7.2	1.08
	54M	336	7.61	1.4
	72M	391	7.42	1.35
Child Anxiety	84M	313	7.46	1.46
	9M	518	0.02	0.92
	18M	520	0	0.9
	27M	521	-0.02	0.9
	54M	521	-0.03	0.89
Adoption	72M	521	-0.03	0.88
Openness	84M	521	-0.03	0.88

Bivariate correlations between all variables are presented in Table 2.

Table 2

Bivariate Correlations of Variables Included

	SEX	Obs Risk	Hhold Inc	BM Int Risk	Parent Overreactivity					Child Anxiety					Adoption Openness					
					9M	18M	27M	54M	72M	84M	18M	27M	54M	72M	84M	9M	18M	27M	54M	72M
Obs Risk	0.01																			
Hhold Inc	0.02	0.01																		
BM Int Risk	-0.04	0.05	0.01																	
Parent Overreactivity	9M	-0.12	-0.05	-0.09	-0.02															
	18M	-0.07	-0.01	-0.15	-0.03	0.62														
	27M	-0.03	-0.05	-0.15	-0.02	0.57	0.74													
	54M	-0.07	-0.02	-0.01	-0.06	0.47	0.61	0.65												
	72M	0.02	-0.03	-0.1	-0.02	0.44	0.52	0.57	0.67											
	84M	0.03	0.04	-0.09	-0.01	0.44	0.58	0.62	0.61	0.71										
Child Anxiety	18M	-0.02	0.01	0.03	0.01	0.06	0.1	0.12	0.1	0.14	0.05									
	27M	0.04	0	-0.02	0	0.04	0.09	0.2	0.1	0.06	0.07	0.6								
	54M	0.04	-0.01	-0.04	0.04	-0.01	0	0.08	0.19	0.2	0.05	0.28	0.37							
	72M	0.05	0.08	0	-0.01	-0.05	-0.03	0.02	0.08	0.09	0	0.18	0.28	0.66						
84M	0.06	0.09	0.03	0.05	0.02	0.09	0.15	0.13	0.05	0.11	0.21	0.31	0.56	0.62						
Adoption Openness	9M	-0.06	0.06	0	0.1	-0.08	-0.12	-0.13	-0.13	-0.1	-0.09	0	-0.03	-0.05	-0.04	-0.01				
	18M	-0.06	0.06	-0.01	0.09	-0.09	-0.11	-0.13	-0.14	-0.1	-0.07	-0.01	-0.02	-0.07	-0.04	-0.02	0.97			
	27M	-0.06	0.06	0	0.08	-0.08	-0.12	-0.13	-0.13	-0.1	-0.05	-0.01	-0.01	-0.06	-0.03	-0.01	0.94	0.99		
	54M	-0.06	0.06	0.01	0.08	-0.09	-0.13	-0.13	-0.13	-0.1	-0.05	-0.01	-0.02	-0.06	-0.04	-0.02	0.93	0.98	0.99	
	72M	-0.05	0.05	0.01	0.07	-0.1	-0.13	-0.14	-0.13	-0.1	-0.05	-0.01	-0.01	-0.05	-0.04	-0.01	0.91	0.97	0.98	0.99
	84M	-0.05	0.05	0.01	0.07	-0.1	-0.13	-0.14	-0.13	-0.1	-0.06	-0.01	-0.02	-0.05	-0.03	0	0.91	0.96	0.98	0.99

Obs Risk – Obstetric and Prenatal Weighted Risk Score. Hhold Inc – Adoptive household income. BM Int Risk – Genetic risk for internalizing using birth mother data.

Significance values are as follows: red = $p < 0.001$, pink = $p < 0.01$, peach = $p < 0.05$, cream = $p < 0.1$.

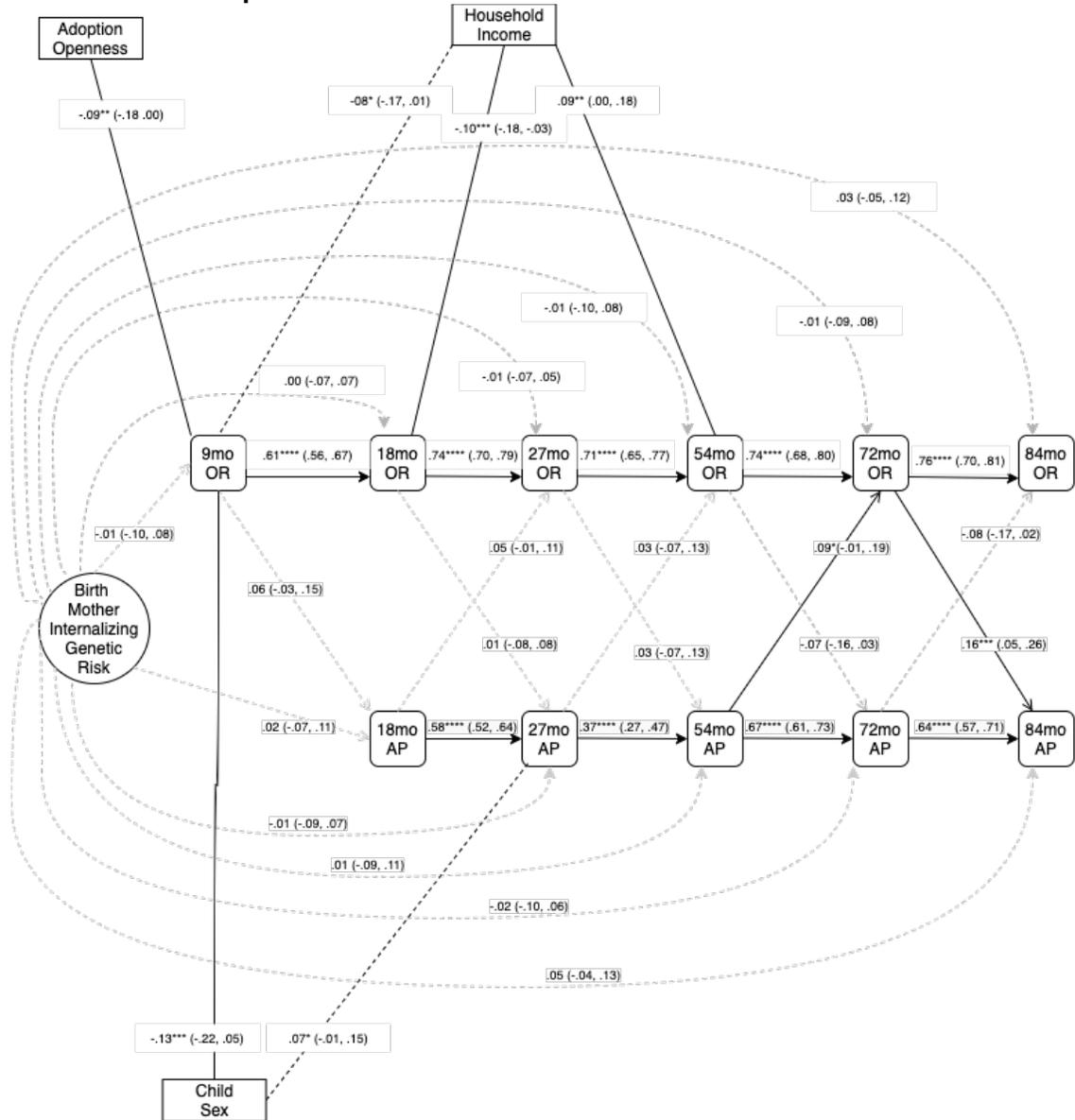
Figure 5 presents the path diagram for adoptive parent overreactivity and child anxiety symptoms from 9 months to 84 months. Adoptive parent overreactivity was stable across this time frame (β s ranging from 0.61 to 0.75 $ps < .001$) and effects were in the expected direction – higher parental overreactivity predicted subsequent higher parental overreactivity. Similarly, adopted child anxiety was stable across this time frame (β s ranging from 0.37 to 0.67 $ps < .001$) and effects were in the expected direction – higher anxiety predicted subsequent higher anxiety.

Adopted child to adoptive parent paths showed that that child anxiety at 54 months prospectively predicted parental overreactivity at 72 months ($\beta = 0.16$, $SE = 0.05$, $p = 0.003$). Adoptive parent to adopted child paths showed that parental overreactivity at 72 months marginally prospectively predicted child anxiety at 84 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.09$). Birth mother based genetic risk of internalizing was not a significant predictor of adoptive parent overreactivity at any time point ($ps > 0.4$) or child anxiety at any time point ($ps > 0.2$).

As reported earlier, covariates were also included in every association in the model. Income was found to be positively associated with parental overreactivity at 54 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.046$) and negatively associated with parental overreactivity at 18 months ($\beta = -0.10$, $SE = 0.04$, $p = 0.004$) and 9 months ($\beta = -0.08$, $SE = 0.05$, $p = 0.07$). We also found that child sex was associated with parental overreactivity at 9 months ($\beta = -0.13$, $SE = 0.04$, $p = 0.003$), indicating that males received more overreactive parenting than female adopted children at this age and child anxiety at 27 months ($\beta = 0.07$, $SE = 0.04$, $p = 0.086$), indicating that males were less anxious than female adopted children at this age. Finally, adoption openness was negatively associated with parental overreactivity at 9 months ($\beta = -0.09$, $SE = 0.05$, $p = 0.045$)

Figure 5:

Full Structured Equation Model



Significant paths on this diagram are represented by solid lines whereas non-significant paths are shown using dotted lines.

Significance levels: * = $p < 0.1$, ** = $p < 0.05$, *** = $p < 0.01$, **** = $p < 0.001$

The model accounted for 41% of the variance in parental overreactivity at 18 months old, 57% of the variance at 27 months, 52% at 54 months, 56% at 72 months and 58% at 84 months. It also accounted for 34% of the variance in child anxiety at 27 months, 15% at 54 months, 45% at 72 months and 45% at 84 months of age. Fit indices indicated acceptable to good fit for the specified model ($\chi^2(91) = 206.72$, $p < .001$, CFI = .93, RMSEA = .05).

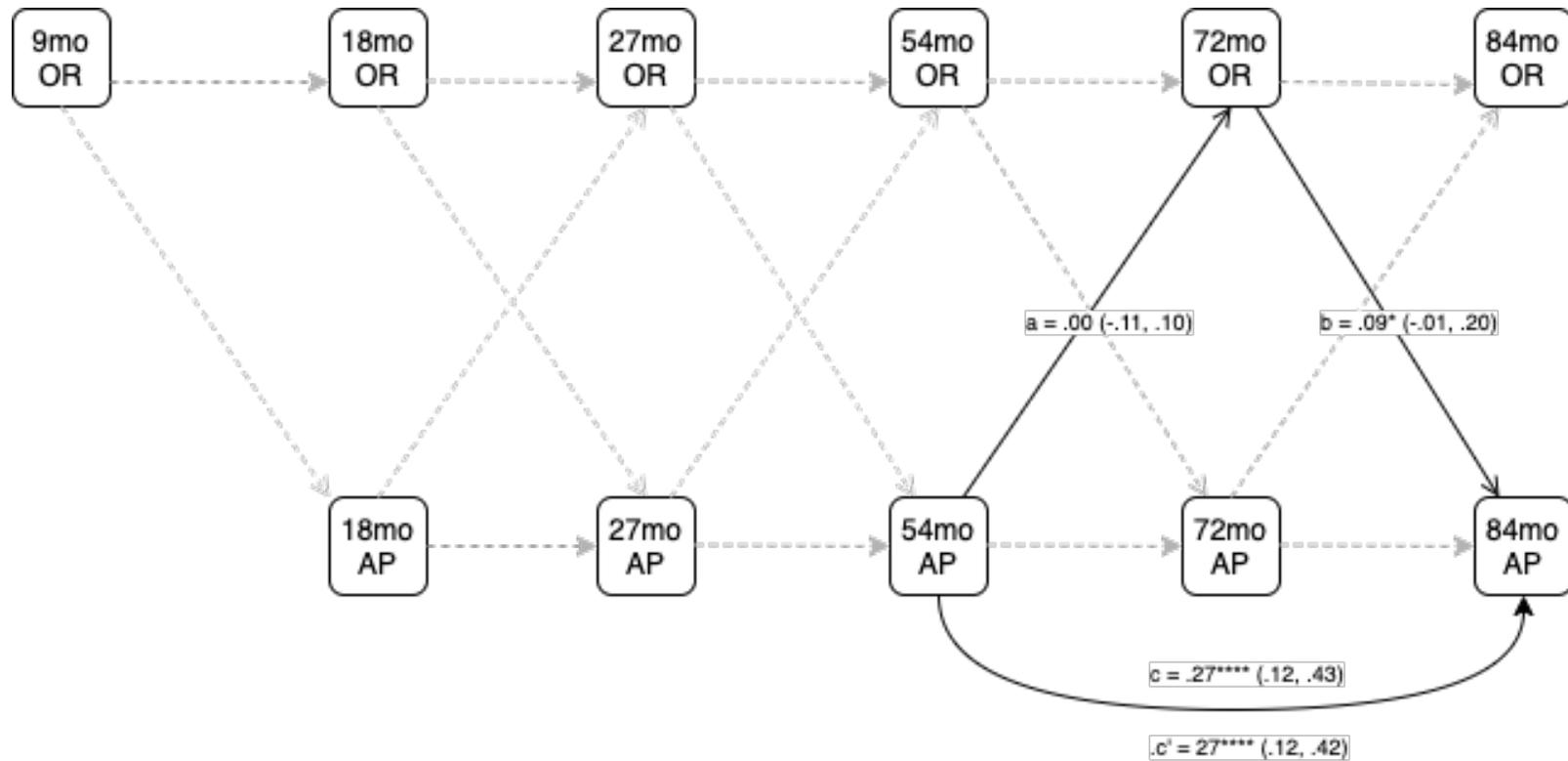
Post-hoc analyses exploring associations between internalizing genetic risk and child anxiety at each time point found no significant associations at any time point ($ps > 0.3$, $\beta s < 0.05$) and found that internalizing genetic risk accounted for less than 0.01% of the variation in child anxiety at any time point. Similarly, post-hoc analyses exploring associations solely between internalizing genetic risk and parent overreactivity found no significant associations at any time point ($ps > 0.3$, $\beta s < 0.05$) and found that internalizing genetic risk accounted for less than 0.01% of the variation in child anxiety at any time point.

Mediation Analyses

As Figure 6 illustrates, the direct effect of child anxiety at 54 months on child anxiety at 7 years old was significant ($\beta = .27$, $p < 0.001$) but the indirect effect, mediated through parental overreactivity at 6 years was not statistically significant ($\beta = .00$, $p = .94$).

Figure 6

Mediation Model



AP: Child Anxiety Problems; OR: Parental Overreactivity

Sensitivity Analyses

Regardless of the informant (i.e. adoptive father, adoptive mother or average of both adoptive parents) for either child anxiety or parental overreactivity, genetic risk was not significantly associated with either variable, and both were stable across all time points. However, parent-child and child-parent paths did vary depending on the informants and covariates were differentially associated with child anxiety and parental overreactivity. The most parsimonious model, using the AIC and BIC, indicators was the model with the average of both parents report on both child anxiety and parent overreactivity ($AIC = 8097.06$, $BIC = 8500.51$). Significant pathways for each of these models are outlined below and presented diagrammatically in Figures 7-10.

Adoptive Mother Reported Anxiety – Adoptive Mother Overreactivity Model

This model is presented in Figure 7. When adoptive mothers reported both child anxiety and parental overreactivity, a parent to child pathway was found. Specifically, overreactivity at 9 months marginally predicted child anxiety at 18 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.07$). There were no significant child to parent pathways. In terms of covariates, child sex was marginally associated with overreactivity at 9 months ($\beta = -0.08$, $SE = 0.05$, $p = 0.10$), with females receiving less overreactive parenting, and at 72 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.05$), with males receiving less overreactive parenting. Obstetric risk was negatively associated with parental overreactivity at 54 months ($\beta = -0.11$, $SE = 0.04$, $p = 0.01$) and positively associated with child anxiety at 72 months ($\beta = 0.10$, $SE = 0.05$, $p = 0.03$). Income was marginally associated with parental overreactivity at 18 months ($\beta = -0.06$, $SE = 0.04$, $p = 0.10$). Finally, adoption openness was negatively associated with parental overreactivity at 18 months ($\beta = -$

0.09, $SE = 0.04$, $p = 0.02$) and child anxiety at 54 months ($\beta = -0.10$, $SE = 0.05$, $p = 0.04$) but was positively associated with child anxiety at 27 months ($\beta = 0.08$, $SE = 0.04$, $p = 0.04$).

Adoptive Mother Reported Anxiety – Adoptive Father Overreactivity Model

This model is presented in Figure 8. When adoptive mothers reported both child anxiety and adoptive fathers reported parental overreactivity, a marginal negative association was found between child anxiety at 72 months and parental overreactivity at 84 months ($\beta = -0.09$, $SE = 0.05$, $p = 0.05$). No significant pathway was found from parental overreactivity to child anxiety at any timepoint. With respect to covariates, child sex was marginally associated with overreactivity at 9 months ($\beta = -0.08$, $SE = 0.05$, $p = 0.10$), with females receiving less overreactive parenting, and at 72 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.04$), with males receiving less overreactive parenting. Obstetric risk was negatively associated with parental overreactivity at 54 months ($\beta = -0.11$, $SE = 0.04$, $p = 0.01$). Income was marginally associated with parental overreactivity at 18 months ($\beta = -0.06$, $SE = 0.04$, $p = 0.10$). Finally, adoption openness was negatively associated with parental overreactivity at 18 months ($\beta = -0.09$, $SE = 0.04$, $p = 0.02$) and marginally with child anxiety at 27 months ($\beta = -0.08$, $SE = 0.05$, $p = 0.08$).

Adoptive Father Reported Anxiety – Adoptive Mother Overreactivity Model

This model is presented in Figure 9. When adoptive fathers reported both child anxiety and adoptive mothers reported parental overreactivity, there were no significant child to parent pathways. Parental overreactivity at 72 months prospectively predicted child anxiety at 84 months ($\beta = 0.24$, $SE = 0.05$, $p < 0.01$). Obstetric risk was positively associated with parental overreactivity at 84 months ($\beta = 0.08$, $SE = 0.05$, $p = 0.06$) and

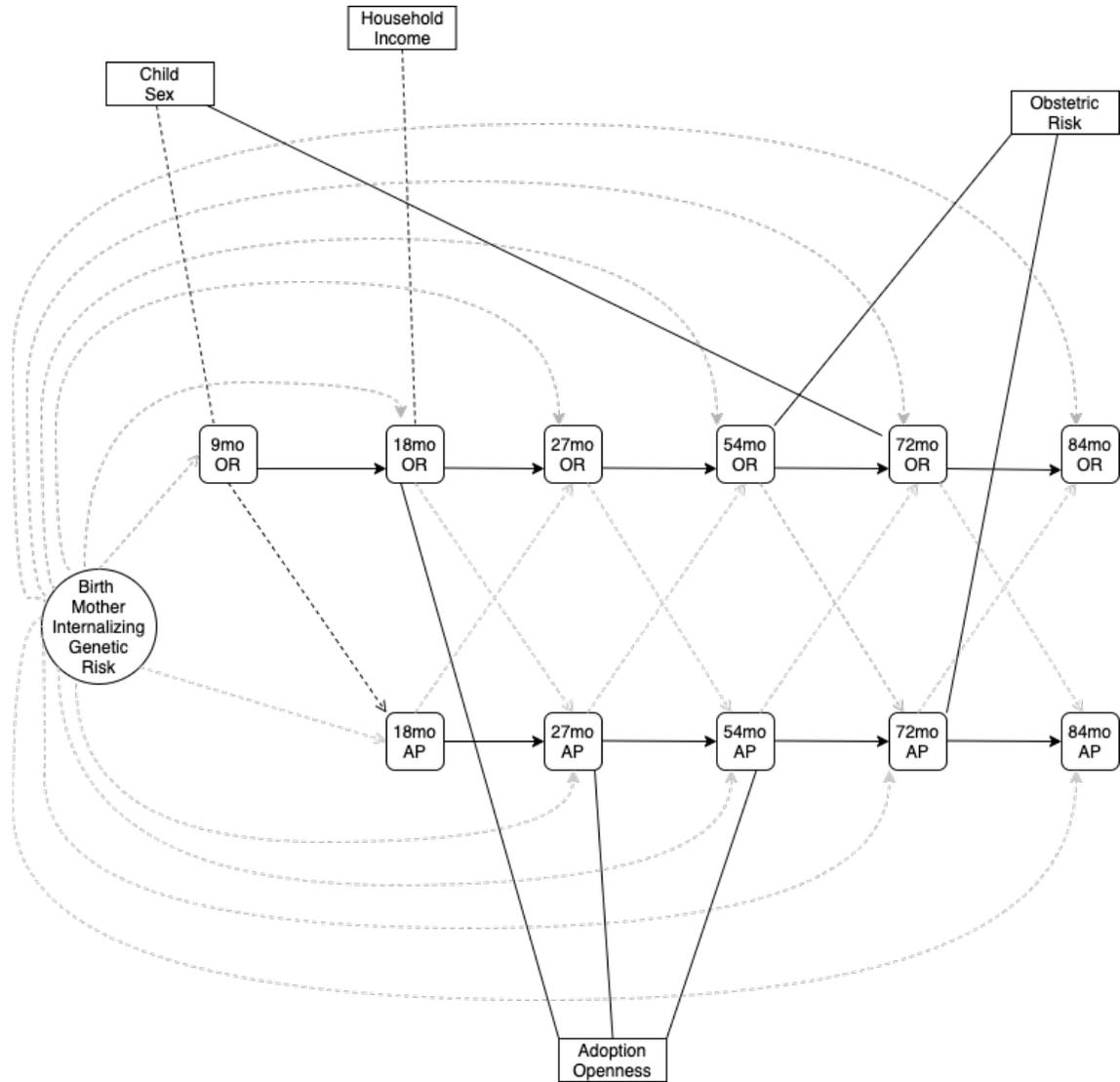
child anxiety at 72 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.09$). Adoption openness was negatively associated with child anxiety at 54 months ($\beta = -0.11$, $SE = 0.05$, $p = 0.02$) but was positively associated with child anxiety at 27 months ($\beta = 0.08$, $SE = 0.04$, $p = 0.04$). Child sex was also associated with parental overreactivity at 9 months and 54 months such that females received less overreactive parenting ($\beta = -0.12$, $SE = 0.05$, $p < 0.01$ and $\beta = -0.08$, $SE = 0.05$, $p = 0.06$ respectively). Finally, income was associated with parental overreactivity at 9 months ($\beta = -0.09$, $SE = 0.05$, $p = 0.05$) and 18 months ($\beta = -0.12$, $SE = 0.04$, $p < 0.01$).

Adoptive Father Reported Anxiety – Adoptive Father Overreactivity Model

This model is presented in Figure 10. When adoptive fathers reported both child anxiety and parental overreactivity, child anxiety at 27 months marginally predicted parental overreactivity at 54 months ($\beta = 0.09$, $SE = 0.05$, $p = 0.06$) and parental overreactivity at 72 months prospectively predicted child anxiety at 84 months ($\beta = 0.17$, $SE = 0.06$, $p < 0.01$). The only covariate associated with adopted fathers' reported child anxiety was adoption openness, which was marginally negatively associated with child anxiety at 27 months ($\beta = -0.08$, $SE = 0.05$, $p = 0.08$). A number of covariates were associated with adopted mothers' reported parental overreactivity throughout this time period. Child sex was associated with adoptive father reported parental overreactivity at 9 months and 54 months such that females received less overreactive parenting from adoptive fathers ($\beta = -0.12$, $SE = 0.05$, $p < 0.01$ and $\beta = -0.08$, $SE = 0.05$, $p = 0.06$ respectively). Income was associated with parental overreactivity at 9 months ($\beta = -0.09$, $SE = 0.05$, $p = 0.05$) and 18 months ($\beta = -0.12$, $SE = 0.04$, $p < 0.01$). Higher obstetric risk was also marginally associated with parental overreactivity at 84 months ($\beta = 0.08$, $SE = 0.05$, $p = 0.08$).

Figure 7

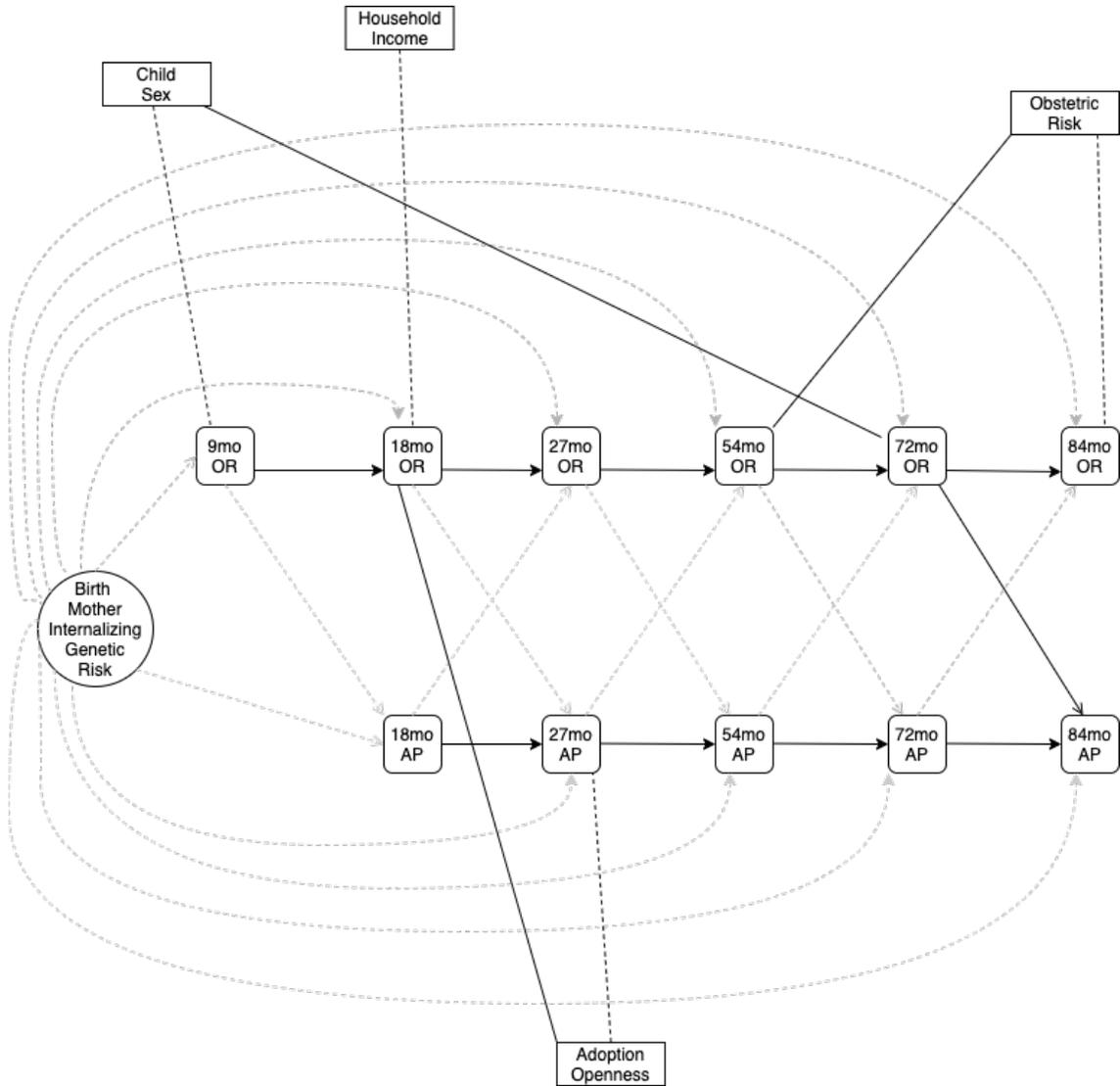
Structured Equation Model with Adoptive Mother Report of Child Anxiety and Adoptive Mother Overreactivity



Significant paths on this diagram are represented by filled lines. Marginal pathways are represented by black dotted lines. Non-significant paths are shown using grey dotted lines.

Figure 8

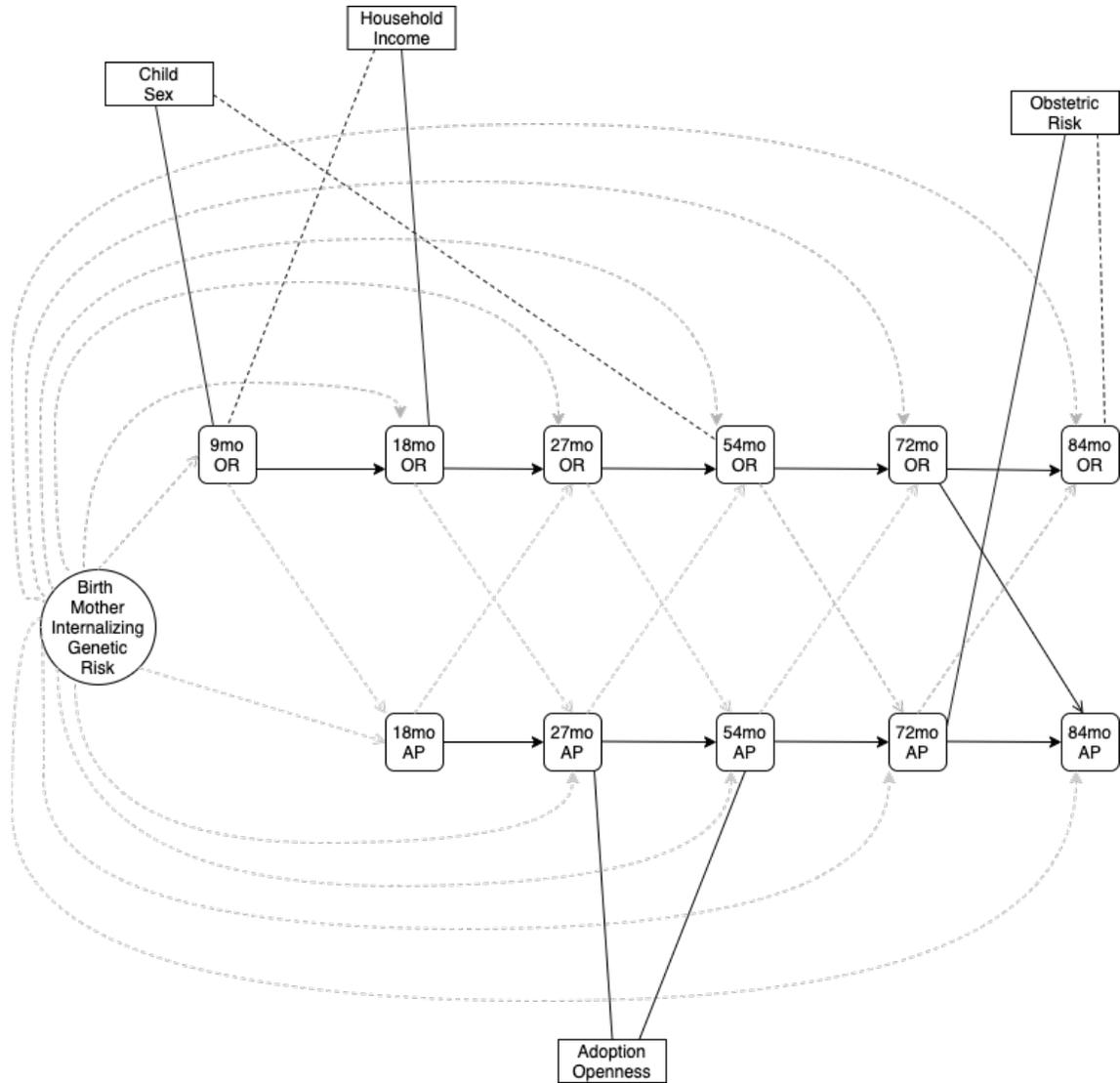
Structured Equation Model with Adoptive Mother Report of Child Anxiety and Adoptive Father Overreactivity



Significant paths on this diagram are represented by filled lines. Marginal pathways are represented by black dotted lines. Non significant paths are shown using grey dotted lines.

Figure 9

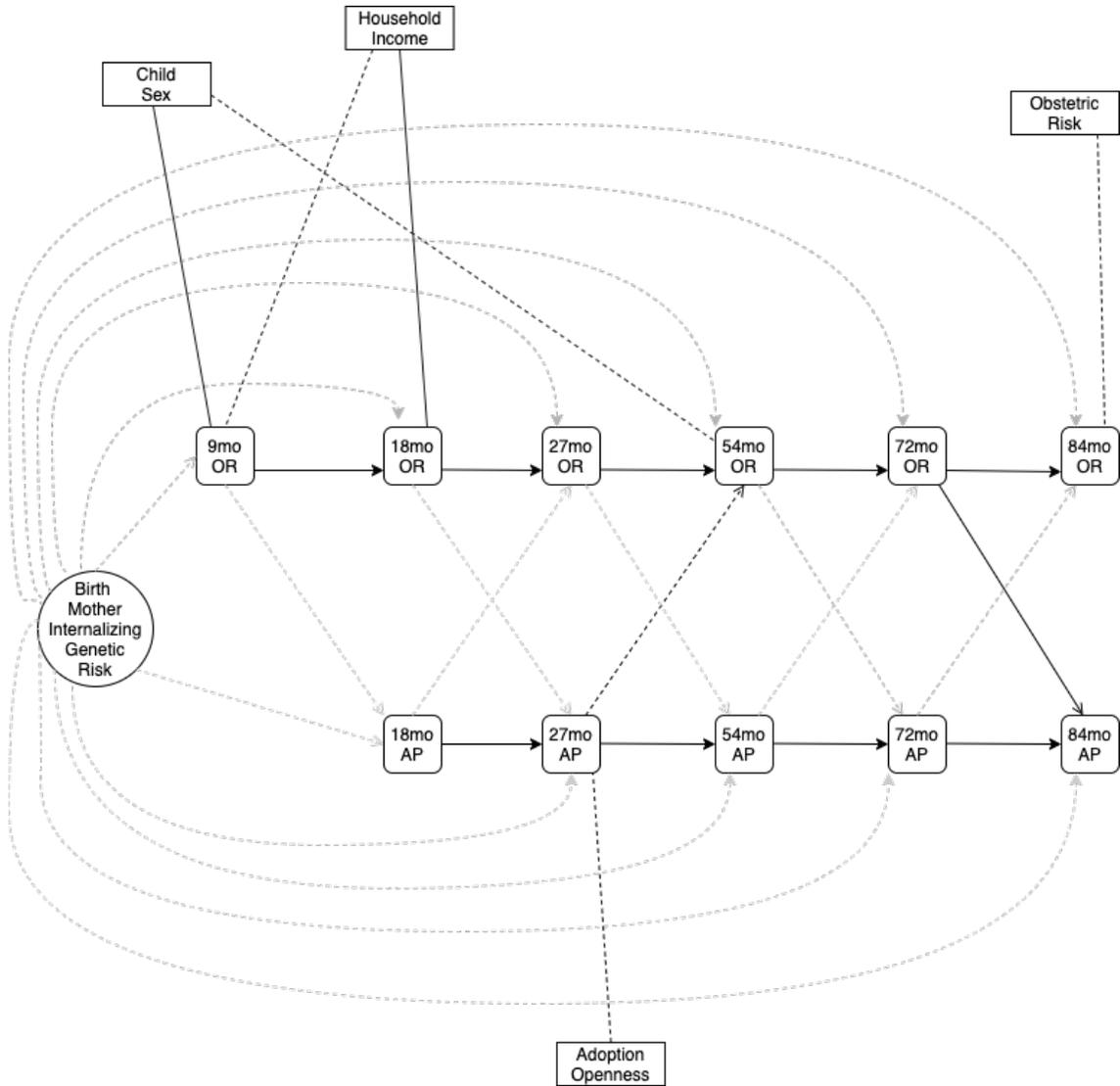
Structured Equation Model with Adoptive Father Report of Child Anxiety and Adoptive Mother Overreactivity



Significant paths on this diagram are represented by filled lines. Marginal pathways are represented by black dotted lines. Non-significant paths are shown using grey dotted lines.

Figure 10

Structured Equation Model with Adoptive Father Report of Child Anxiety and Adoptive Father Overreactivity



Significant paths on this diagram are represented by filled lines. Marginal pathways are represented by black dotted lines. Non-significant paths are shown using grey dotted lines.

Discussion

The present study examined the bidirectional relationship between child anxiety and parenting. Specifically, using the EGDS dataset from 9 to 84 months, we looked for overreactive parenting influences on child anxiety symptoms and reciprocal influences of child anxiety on overreactive parenting over this timeframe. Furthermore, we tested whether either parenting or anxiety was linked to internalizing genetic risk over this time frame and, specifically, whether any evoked overreactive parenting was associated with genetic risk to identify the presence of evocative gene-environment correlation (*rGE*).

First, we examined the role of early child anxiety in predicting later child anxiety and similarly, the role of early parental overreactivity on later parental overreactivity. Unsurprisingly, these two traits were highly stable across the time frame explored. We then examined the influence of parental overreactivity on later child anxiety. Findings offered preliminary support for our first hypothesis at only one time point in middle childhood — there was a marginally significant association between parental overreactivity at 72 months and subsequent child anxiety at 84 months. Sensitivity analyses replicated this finding in all situations except when adopted mothers reported anxiety. Given that mothers continue to be over-represented as primary caregivers, it is possible that they were more sensitive to offspring anxiety symptoms (e.g. Hudson et al., 2018) at an earlier stage, leading to a more stable report of child anxiety. In contrast, adoptive fathers may be more involved in parenting later in childhood, leading to a change in their perception of the child's anxiety and a reciprocal change in their overreactivity. Further research could include more objective measures of child anxiety to disentangle these possibilities.

Findings more clearly offered support for our second hypothesis of child anxiety influencing parental overreactivity. This positive association was found between child anxiety at 54 months and parental overreactivity at 72 months. Furthermore, sensitivity analyses offered further support for this as, when adoptive fathers reported, both anxiety and overreactivity, this evoked parenting effect occurred earlier in childhood, with child anxiety at 27 months predicting parental overreactivity at 54 months. This offers considerable support to the bidirectional effects of parenting and, specifically, the notion that children influence the parenting they receive (Bell, 1968). Further research could extend this later into childhood and adolescence and explore the differential time frame upon which these evocative effects occur for mothers and fathers.

The third and final hypothesis explored in this study looked for evidence of a genetic contribution to the parenting evoked by child anxiety. However, findings did not offer support for this hypothesis, with no significant association found between genetic risk and child anxiety or parental overreactivity at any time point. This is consistent with other papers from the EGDS study that have looked at child anxiety (e.g. Ahmadzadeh et al., 2019) but extends this finding to a broader time frame and to include parental overreactivity. As previously discussed, it may be that the genes associated with adult internalizing are distinct from those in childhood anxiety. It may also be the case that *rGE* does occur, but later still in adulthood. There is also the possibility that the child anxiety evoking parental overreactivity is a result of something other than genetics, for example perinatal environment. Obstetric risk scores in this study were not found to be related to child anxiety or parental overreactivity in the main analyses but were when adoptive parents were considered separately, offering some preliminary support for this hypothesis. As such, further genetically informed research which considers perinatal

environment as well as other environmental factors is required to further this area of study.

Although evidence of evocative *rGE* was not found in this study, there was evidence of bidirectional influences between child anxiety and parental overreactivity. As such, our results offer support for internalizing problems interventions that target parenting in addition to or instead of working directly with children. It also suggests that earlier intervention might be of benefit before cascading effects take place. Finally, it indicates that it may be important to offer support to parents who are perceiving their children as being anxious at a younger age to offer more adaptive responses to anxious behaviour.

Clinical Implications

The research presented in this study has a number of implications for clinical practice. For example, parenting interventions that are currently offered often centre the environment and, more specifically, parenting in explaining children's behavioural difficulties. Evidence of evoked parenting, as is presented here, suggests that such interventions could be improved by normalizing the experience of parents who feel that their parenting is responsive to their perception of their child's needs based on their child's behaviour. Furthermore, interventions could offer targeted guidance on helpful and unhelpful way to respond to specific tendencies and behaviours in children. This would be of particular use when parents and children are not genetically related, such as in adoptive and foster families as well as in blended families. Although it is important not to blame or locates problems in children, it is possible that parenting interventions could be improved by endorsing the idea that children play an active role in shaping their environment.

Strengths, limitations and future directions

There are a number of strengths to this study. In particular, the use of an adoption data set allowed us to separately consider genetic and environmental contributions to parenting and child anxiety, controlling for prenatal risk and adoption openness. The longitudinal nature of the dataset also allowed us to examine a significant span of childhood, beginning in infancy. Furthermore, although the assumption of independence of environment and genetics contributions in adoption designs can be threatened by selective placement, there is no evidence of this in the EGDS sample (Leve, Neiderhiser, et al., 2013).

However, although the adoption design has many strengths, critics of it have noted that the experience of adoptive families is a unique one and this may limit generalizability. In particular, children adopted at birth are likely to have higher inherited risk of psychopathology and more prenatal risk factors than non-adopted children (Cadoret, 1990). Furthermore, the generalizability is limited by the representativeness of any member of the adoptive triad – that is, if the sample of biological parents, adoptive parents or adopted children are not representative of the rest of the population, this would limit the extent to which results could be generalized. In the EGDS sample, adoptive parents were more educated and had higher incomes than the average non-adoptive American parents with young children (Leve, Neiderhiser, et al., 2013). Nevertheless, the effects explored in this study offer some insight into the transactional effects that might occur between parents without the impact of the life stressors associated with low socioeconomic status.

Furthermore, one limitation of this study is that it does not explore the types of environmental mechanisms outside of the parent-child relationship that may influence

parent-child associations. It is plausible that exposure to environmental factors may be at play within this relationship or that might affect both parent and child over a period of time, resulting in an apparent transactional effect.

Finally, this study was limited by only considering birth mother genetic contributions. This decision was made due to the considerable difference in data completeness between birth parents, with 64% of birth father genetic risk scores being imputed due to missing data. However, given that birth fathers contribute 50% of genetic material, their absence in this analysis is notable. Interestingly, new analyses from the EGDS study suggest that birth father attrition in EGDS was unrelated to all of the measured demographic variables but was related to adoption openness (Marceau et al., 2019). Furthermore, total attrition at 8 years was also found to not be related to any demographic variables. Together, these findings suggest that replicating the results presented here using the subset of available birth father data might provide an important opportunity for replication that would not affect the generalizability of the findings.

Conclusion

This is the first genetically informed study examining transactional associations between parental overreactivity and child anxiety symptoms from infancy into middle childhood. Using data from the EGDS between child ages 9– 84 months, we found positive effects in middle childhood from parental overreactivity to subsequent child anxiety and from child anxiety to subsequent parental overreactivity, offering support for Hypotheses 1 and 2. However, we found no evidence for Hypothesis 3, as genetic risk modelled using birth mother internalizing symptoms and family history, was not significantly associated with child anxiety or parental overreactivity at any point in this

age range. Taken together, these findings offer an important step in understanding the bidirectional relationship between parenting and child psychopathology and offer insight into how interventions targeting either of these might impact or be limited by the other. Further research is needed to explore the possibility of other genetic contributions to this process and to extend it into late childhood and adolescence.

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Part 3: Critical Appraisal

Introduction

As someone with a background in mathematics and computer science, I have always been interested in “big data” projects and in utilising statistics to increase and expand our understanding of human behaviour and relationships. While I also have a deep passion for clinical psychology, my research training was in a basic science oriented psychology lab and I found that an understanding of normative development, emotion and social behaviour only enhanced my clinical work as it helped me see the diversity of experiences that are contained within the “normal” as well as allowing me to take a critical stance towards the pathologising of certain patterns of behaviour without a more complex understanding of context. However, as with any macro-level process, statistical analyses of large samples have a number of assumptions embedded in them and are abstracted from certain realities. As such, I have chosen to focus on these assumptions and their consequences in this critical appraisal. More specifically, I will outline methodological considerations within behavioural genetics research, ethical considerations of common data cleaning practices, the research implications of secondary data analyses and the clinical relevance of such research.

Methodological Concerns of Behavioural Genetics

Behavioural genetics research and, in particular, adoption studies, are cohort-based designs. In such research, any associations found between variables may be a result of bias, confounding, change or a causal relationship between those variables (Hulley, 2007). As confidence intervals and significance testing are already consistently used to try and differentiate between chance and causal associations, the methodological concerns addressed here are focussed on bias.

There are two key types of bias considered here – selection bias and information bias. Selection bias in cohort studies occurs when the groups differ systematically on something other than the criteria upon which they are categorized and that difference is associated with the outcome of interest. Selection bias, when it exists, limits the interpretability and/or generalizability of the research findings.

One such consideration in the research presented here is the assumption that adoptive families are similar to non-adoptive families. While there is some research showing this to be true on individual variables, the questions of how the particularities of adoption affect subsequent parenting behaviour is still an open one. Given the substantial financial investment involved in adoption and the years adoptive parents might wait for a healthy infant in the US (Adoption Center, n.d.), it is plausible that the attachment of parent to child might manifest differently than for parents who have experienced less hardship in the process of becoming a family. While we know that adoptive parents, probably for the reasons above, are both older and of higher socioeconomic status than the average parent, we don't yet know in sufficient detail how this process affects parenting behaviour. One might expect that this considerable investment could lead to heightened pressure on adoptive parents to be "perfect", thus resulting in more overreactive and intrusive parenting behaviours. It could just as plausibly be claimed that the commitment that adoptive parents have shown to this process is indicative of a steady and not easily shaken character. Further research is needed to lend plausibility to either of the hypotheses described above, or possibly to offer further credence to the idea that adoptive parents are otherwise indistinguishable from biological parents.

Similarly, research on adopted children would be much enhanced by considering when adopted children are made aware of their adoption, and how, if at all, this disclosure impacts the relationship they have with their adoptive parents and the parenting they

evoke. As adoption openness has been considered as a covariant in this research, the changing amount of information a child might have about their own adoption and family history could be an equally informative mediator in some of the examined relationships. Arguably, some of this is captured in the concept of adoption openness, since children having regular contact with their birth parents must necessarily be aware of their existence, but the lack of direct examination of this variable should still be redressed.

Similarly information bias, which describes an error in the measurement of the outcome, impacts the strength of the findings. One particular example of this in the research presented in this thesis is the methodological consideration of the impact of being observed on the participants of the studies examined here. While there is not a clear consensus on this in the literature, there are at least some indications that observation can impact the behaviour of those observed (McCambridge, Witton & Elbourne, 2014). In particular, recall bias is a well-established effect that can distort findings (Coughlin, 1990).

Ethical considerations of common data cleaning practices

In trying to reduce the number of potential confounds in the data through restricting the sample, it is possible to introduce a number of ethical concerns. Throughout the process of completing this research, I have had to make a number of decisions that have elucidated some of the limitations of large sample analysis for the purpose of behavioural genetics. For example, due to the relatively small size of this subsample, same sex couples were excluded from the analysis. Since this project required looking at the data for adoptive fathers and mothers separately, the decision was made that data from same sex couples might be difficult to interpret within this structure. While a reasonable cause for that exclusion in this particular case, when this logic is generalized to research that, even if it mirrors the general population, will as a result have relatively few same sex

couples as opposed to opposite sex couples, it is conceivable that this exclusion might be repeated again in such research projects, as was the case in Ahmadzadeh et al.'s 2019 paper from the same dataset. While the concern I have described here is specific to one minority identity, we can imagine the same logic might be used to exclude other non-traditional family structures. For example, given that transracial adoptions are known to have additional complexity that differentiate them from interracial adoptions, the same rationale could be applied to exclude them from research to the detriment of those trying to better understand the similarities and differences between these forms of adoption. Given the history of research, excluding minoritized identities on the basis of them adding "unnecessary complications" that persists to this day (e.g. Criado-Perez, 2019), it is particularly important that we reflect on the impact of such exclusions and, where possible, use over-sampling and inferential statistical techniques to correct for this. However, even these techniques must be used with caution as inferences made on limited data sets can prove to be unsuitable, inaccurate and even damaging. One recent example of this can be found in the example of a paper recently published and subsequently retracted for drawing inferences about global IQ levels based on insufficient, and unrepresentative, data (Bauer, 2020).

It's one thing to reflect on these exclusions in the abstract and quite another to be conducting the reflection in a time of mass global protests, and a political context of a pandemic that disproportionately affects minorities. It also particularly notable that while conducting this research project I have become more involved in cultural competency work in other parts of my psychological working and have even found an organisation with a focus on increasing representation in all aspects of our profession. In thinking more deeply about inclusive practice, I've often reflected on the potential for harm when

majority groups are consistently represented in research to the exclusion of others. One of the key realities that I have come to understand through the process of doing this research project is that often these exclusions are done for the purposes of making research more interpretable or feasible. However, through my reflections as a culturally sensitive practitioner, I've become aware of how these gaps or under-representations in literature leave many minority groups with models that do not adequately describe their experience and sometimes, models and theories that devalue, contradict and/or undermine their experiences. Balancing this tension is an important point of learning for me going forward.

Research implications of conducting secondary data analyses

One of the considerations in deciding what project to pursue for my thesis was the decision whether to gather data or conduct an analysis on an existing data set. There are a number of considerations in making this decision. Although multiple analyses of an expansive dataset are common as demonstrated by the fact that multiple analyses from the same dataset are common is offered by the fact that 11 papers reporting on parenting were identified from 6 studies in the meta-analyses reported here. Many of these papers also reported on phenotypes other than parenting (e.g. Forget-Dubois et al. 2007) and many other papers reported on other phenotypes from the same studies (e.g. Saudino, Ronald & Plomin, 2005). As such, although I could identify a number of papers on the area of parenting and infancy from a twin sample, there were only 6 samples, which could be considered independent. Although I chose to use an analysis strategy that treated phenotypes from the same sample as being non-independent, other meta-analyses have continued to treat them as independent (e.g. Polderman et al., 2015) possibly leading to an overestimation of the strength of findings. Where non-quantitative reviews are conducted, this problem could be even more significant as qualitative/narrative synthesis of these

publications could mistakenly conclude that a significantly larger evidence base exists than is the case. As such, the question remains whether the likelihood of a false positive (i.e. a finding that is significant due to chance and which is not indicative of a true relationship) is enhanced by such repeated exploration of the same data sets.

On the other hand, we know that some datasets are more difficult to collect than others and given the relatively low percentage of adoptive families or families with twins relative to families with non-twin or biological children, it is particularly important that data gathered from these samples is thoroughly investigated. This is particularly important given the resources required to recruit and maintain involvement over a period over a decade of these participants as well as because families of this nature allow us a unique and rare opportunity to explore behavioural genetics questions. Although behavioural genetics has made advancements in identifying and mapping genes associated with some behavioural phenotypes, we are still far from an exhaustive list and it is not clear that it will ever be possible to identify the genes associated with every behavioural phenotype, meaning that studies directly mapping DNA to complete genetically informed analyses may often be impossible. As such, genetically informed analysis of certain crucial variables such as parenting require indirectly exploring genetic influences, through natural experiments such as adoptive families or those with twins.

Secondary data analyses in the current climate

Another aspect to the advantage of this research project was that, in the current global pandemic, my research was unaffected by changes in rules about in-person interactions as the data had already been collected. Researchers working in this uncertain time may well choose to delve into data already collected to manage the current limitations on data collection and lack of opportunities for direct interview or observation.

Reconsidering older datasets with current understandings of literature in mind may allow people to bring fresh perspectives to existing datasets. Furthermore, as numerous longitudinal or epidemiological studies are conducted in a specific area, although each of them might be underpowered to conduct analyses on specific subgroups, aggregating them might allow for this. For example, combining data from a few adoption studies might allow us to have explored transracial adoptions or adoptions by same-sex parents in particular. Although this was not conducted in this thesis, the data in the empirical chapter presented here was able to aggregate data across a larger geographic area and with more participants than could feasibly have been collected. Additionally, the longitudinal nature of this dataset would definitely not have been feasible to collect during the doctorate and as such, secondary analyses offered a unique opportunity to look across a large segment of childhood and examine transactional processes. As well as not being feasible for a doctoral project, it is probable that such longitudinal data collection would be a challenge in any research setting, suggesting that, when it is conducted, secondary analyses are an important way to tap into this rich dataset.

However it is important to note that the dataset I relied on had required regular in person contact to collect the data I analysed in my empirical chapter. As such, some consideration will need to be given by projects (including EGDS) that have on-going data collection for how to transition their collection to be conducted remotely. On-going projects could use this opportunity as an additional natural experiment to see how the current context is impacting their participants. As such, one other reason to conduct secondary analyses might be that unpredicted changes in circumstances might allow for analyses of datasets that were not within their initial plans. It may be that remote data collection becomes the norm going forward if, as one might imagine, it results in improved engagement from participants and thus reduced sample attrition. As such, secondary

analyses on on-going data projects might also be used to see the impact of different data collection strategies within the same sample and whether remote data collection improves engagement and shows more equal engagement across demographics.

Clinical relevance of behavioural genetics research

While research, presented here and otherwise (e.g. Ahmadzadeh et al., 2009) has indicated that children influence their environment through evoking certain parenting, this understanding is often absent from parenting interventions. In fact, many of these interventions implicitly locate the behavioural difficulties experienced by children in their environment and, specifically, their parents. Working in a service for children with disabilities, embedded in social care, I encountered the power of simply educating parents about a child's genetically or biologically based needs in changing parental behaviour. Many of these parents felt blamed by services but also had an insufficient understanding of the nature of their child's differences and/or disabilities to respond appropriately. Validating their experience and educating them as well as inviting them to groups where they were able to access social support and connection were incredibly powerful interventions. At the time, it struck me that this type of intervention might also be powerful for parents of neurotypical children without disabilities who nonetheless show great variation in their genetic predisposition towards internalizing or externalising behaviour.

The research explored in this thesis also offers an indication that this type of intervention might be especially powerful at an early age, even as soon as infancy but definitely within the first few years of parenting. This seems particularly important as the research contained here as well as other research referenced within it (e.g. Ahmadzadeh et al. 2019) indicates that in school aged children, parenting behaviour and parental anxiety,

both of which might have been evoked in part by child internalizing behaviour earlier, begin to prospectively predict increased child internalizing behaviour later, forming a cascade that may lead to more significant and less tractable anxiety problems for both parent and child later on. In particular, targeted interventions that teach parents how to respond to specific child behaviours or to children who tend towards internalizing or externalizing behaviours might be especially important when parents are not biologically related to their children as this increases the likelihood that children will have different genetically-determined tendencies than their parents and therefore the usual sources of parenting strategies in older family members or self-regulation strategies may be less useful. For example, a parent without personal experience of internalizing behaviours might be more likely to think that an anxious or withdrawn child needs more attention and may end up becoming overreactive instead of building the young person's confidence and sense of their own ability.

Conclusion

In conclusion, there are a number of trade-offs to large-scale quantitative analyses, particularly those that are secondary analyses of pre-existing datasets. When a degree of abstraction is introduced to a project, simplification and exclusion inevitably occur and these can impact the representativeness or generalizability of the findings. Furthermore, broad practices towards exclusion of subsamples to increase generalizability can lead to implicit discrimination and underrepresentation of minorities in research. Additionally, analyses on pre-existing data can lead to an overestimation of the strength of an evidence base as they result in a number of publications from the same sample, creating an impression that more data has been collected than is accurate. However, even with all of these considerations, the findings of such studies offer us important insight into the

relationships between parenting and child behaviour. These insights could be embedded into the interventions offered to parents to great effect and should be considered in the creation of parenting programmes going forward. As such, an important take away from this research project for me was that the learning, though limited and possibly imperfect, was worth doing.

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