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A Systematic Review and Meta-Analysis of the Associations Between Father and Child Anxiety and Depression

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With the aim of providing an estimate of the association between father and child internalizing symptoms, the present study systematically reviewed and meta-analyzed associations between father and child anxiety and depression symptoms from existing research (mostly from North America and Europe, conducted between 1981 and 2021). Based on 70 samples (158 effect sizes, $N = 58,255$), the meta-analysis with robust variance estimation identified a positive and significant association between depression in fathers and children ($r = .15$, 95% CI [.12, .17]). Similarly, a positive and significant association between father and child anxiety was observed ($r = .13$, 95% CI [.07, .18]) based on 39 samples (72 effect sizes, $N = 11,683$). The effect sizes for depression and anxiety did not significantly differ from one another or as a function of sample characteristics and methodological moderators. Findings show the importance of fathers in the development of child psychopathology. Directions for future research from a family systems perspective are proposed.

Public Significance Statement

Depression and anxiety are prevalent mental health disorders among children and parents. The current meta-analysis examines if depression and anxiety in fathers are associated with the development of such symptoms in children. The results suggest a small-to-moderate association between father and child depression and anxiety.

Keywords: anxiety, depression, fathers, children

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Depression and anxiety are prevalent mental health disorders among families worldwide (World Health Organization, 2017). Prior to the COVID-19 pandemic, estimated prevalence rates of depression and anxiety in youth were 3.2% and 7.1%, respectively (Ghandour et al., 2019). A meta-analysis including over 80,000 youth showed that the prevalence of these symptoms has doubled over the ongoing COVID-19 pandemic, with now 1 in 4 youth experiencing clinically elevated depressive symptoms and 1 in 5 youth experiencing clinically elevated anxiety symptoms (Racine, McArthur, et al., 2021). While numerous factors interact to influence the onset and maintenance of anxiety and depression among children and youth, parental mental health is well known to play an important role in the development of such symptoms.

Historically, there has been a prominent focus on documenting the prevalence of anxiety and depression difficulties in mothers (e.g., Ertel et al., 2011; Shorey et al., 2018; Wang et al., 2021), with comparatively less attention paid to paternal mental health. There is,

however, evidence that fathers also experience significant depression and anxiety symptoms and that these symptoms may contribute to the development of internalizing difficulties in children. It is estimated that between 1.2% and 3.6% of fathers (Nath et al., 2016) and approximately, 7% of new fathers (Leiferman et al., 2021) experience depressive symptoms. Similarly, 11% of new fathers (Leiferman et al., 2021) and between 15% and 20.6% of fathers with older children suffer from anxiety disorders (Cooper et al., 2006; Hughes et al., 2009). Fathers have also suffered increased rates of depression and anxiety during the COVID-19 pandemic, with an estimated prevalence of 37.1% and 22.9%, respectively (Cameron et al., 2020).

Research has shown that parental depression and anxiety are negatively associated with various aspects of children's well-being. For example, children with parents who experience depression or anxiety symptoms are more likely to form an insecure attachment to that parent (Badovinac et al., 2018; Barnes & Theule, 2019), have poorer health outcomes (Surkan et al., 2011), and worse

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psychobiological and socioemotional outcomes (Hayden et al., 2014; Madigan, Oatley, et al., 2018). Most notably, meta-analyses have shown that anxiety, depression, and internalizing symptoms in mothers are related to an increased risk of such symptoms developing in offspring, suggesting a potential for parent–child intergenerational symptom transmission (Connell & Goodman, 2002; Goodman et al., 2011). Seeing as the large majority of the extant, meta-analytic evidence has focused on studies of mothers, however, scarce information exists about the relative influence of fathers in the transmission of internalizing psychopathology. Efforts to amalgamate these father-specific effects by means of meta-analysis are urgently needed in order to arrive at a more balanced and representative estimate of the association between parent and child internalizing psychopathology in family systems. Such estimates can also help to drive further prevention and intervention efforts in at-risk families to better support fathers and their children who experience depression and anxiety. The present study aimed to address this gap in research by systematically reviewing and meta-analyzing concurrent and longitudinal associations between father and child depression, anxiety, and internalizing symptoms.

Fathers in Child Psychopathology Research

Developmental psychology as a field has tended to focus on the role that mothers play in shaping child developmental outcomes, at the expense of the contributions of other important caregivers such as fathers (see Volling & Palkovitz, 2021). In the case of child psychopathology research specifically, the focus on mothers is striking. A systematic review of child psychopathology research ($n = 702$) that included a parental variable showed that only 28% of studies examined the specific contributions of fathers (Cassano et al., 2006). In contrast, the rest of studies consisted of mostly (17%) or solely (55%) mothers. Only 1% of articles included solely fathers. This trend in individual studies is echoed within the realm of research syntheses, which are generally focused on mothers (see also Parent et al., 2017).

Yet, understanding the role of fathers in the development of child psychopathology is a particularly timely research question. Fathers' roles in families have shifted in important ways following societal trends such as women's increased participation in the workforce (Cabrera et al., 2000). In many families, fathers are now co-parents who take on similar parenting responsibilities as mothers, notably participating in daily chores and caregiving (Cabrera et al., 2018). Accordingly, fathers and father–child relationships can play important roles in shaping child developmental outcomes, in ways that can be similar or different from mothers. Fathers' contributions to the development of depression and anxiety may thus be different from that of mothers and are worthwhile to explore in their own right. Such an exploration can be carried out through a systematic and quantitative synthesis of effect sizes from existing bodies of research, with a focus on the association between such difficulties in fathers and children.

The Association Between Parents' and Children's Internalizing Problems

An important negative consequence of internalizing psychopathology is the potential transmission across generations. Internalizing problems include a number of symptoms that are directed inward, the most common examples being anxiety and depression (Achenbach &

Rescorla, 2000). While a complex interaction of factors accounts for the development of anxiety and depression in children, these problems can be transmitted intergenerationally within the family system. Specifically, certain parental characteristics (e.g., depression, anxiety) from one generation increase the likelihood that similar problems will reoccur in the next generation (Branje et al., 2020).

Researchers have proposed specific models to explain the intergenerational transmission of depression, most of which have been mother-centric (see, Garber & Cole, 2010; Goodman & Gotlib, 1999; Hammen et al., 2004). Furthermore, these models emphasize that depressive symptoms may contribute to the development of such symptoms in children through both biological (e.g., genetic heritability, defective neuroregulatory mechanisms, etc.) and environmental factors (e.g., exposure to negative cognitions, behaviors and affect, and stressful environments, etc.). The intergenerational transmission of anxiety has comparatively received less attention, with models suggesting that an interaction between cognitions in anxious parents and their parenting behaviors may influence the development of anxious information processing biases in children. (Hudson & Rapee, 2004; Murray et al., 2009). For instance, parents who experience anxiety may contribute to child anxiety by reinforcing child cognitions that the world is threatening and that the child is not capable of coping or controlling this threat (Murray et al., 2009). Although theoretical and empirical literature emphasizes the mother's influence on child internalizing outcomes, the steady increase in father-focused research has brought to light several key mechanisms of symptom transmission in father–child interactions (Barker et al., 2017). Much like depressed mothers, fathers who experience depression may exhibit more hostile, insensitive, and intrusive parental behavior toward their children (Wilson & Durbin, 2010) and may expose their children to depressotypic cognitions, behaviors, and affect (Jacob & Johnson, 2001; Sethna et al., 2012). Likewise, fathers who experience anxiety may exhibit more controlling and rigid behavior with their children (Bögels & Phares, 2008; Teetsel et al., 2014), display less warmth and positive affect (Hudson et al., 2008), and may engage in more behavioral control (Epkins & Harper, 2016). Several studies have found that these negative parenting behaviors are associated with a greater risk for child anxiety and depression (Bögels & Phares, 2008; Reeb et al., 2015; van der Bruggen et al., 2010). Father-specific influences on children's psychological outcomes are especially important to consider in present times, seeing as fathers have assumed increasingly more involved roles in caregiving activities (Cabrera et al., 2018; Kuo et al., 2018).

Associations Between Father and Child Internalizing Outcomes

While no correlational research can ascertain the extent of the *actual* transmission of symptoms between fathers and children, an increasingly important body of work aligns with the aforementioned theory and supports an association between father and child psychopathology. To date, there have been two meta-analyses that have found modest associations between paternal depression and children's psychopathology, with the association being stronger for children's internalizing versus externalizing psychopathology (Connell & Goodman, 2002; Kane & Garber, 2004). In their meta-analysis, Connell and Goodman (2002) quantified associations between paternal mental health problems (including depression and

anxiety disorders) and child symptoms and compared them with associations centering on maternal mental health problems. They found that psychopathology in both mothers and fathers appears to have an equal effect on children's externalizing psychopathology, but maternal mental health problems have a slightly stronger association with children's internalizing outcomes. Nevertheless, fathers have been shown to influence children's internalizing symptoms to a significant extent, as evidenced by a different meta-analysis that found that paternal depression was significantly associated with both externalizing and internalizing psychopathology in children and adolescents (Kane & Garber, 2004). Despite setting an important impetus for researchers to consider the role of fathers in psychopathology development, these meta-analyses are dated and do not account for more recent research. Given the increased prominence of paternal caregiving roles in recent years, it is imperative to provide an updated estimate of these father-specific effect sizes. Additionally, the larger body of father-inclusive studies may allow for a more thorough examination of moderators that may play a role in the association between symptoms across generations.

Moderators of the Association of Anxiety and Depression in Fathers and Their Children

Sociodemographic Factors

Previous meta-analyses on mothers suggest that socioeconomic risk, child age, and child gender may influence the transmission of anxiety and depression. For instance, in a meta-analytic review of the associations between maternal depression and child behavior problems, effect sizes were stronger in low-income families for all types of behavior problems, with the exception of children's positive affect/behavior (Goodman et al., 2011). Furthermore, studies have found that (SES) in childhood predicts the lifetime risk for developing depression even after adjusting for adult SES (Kim et al., 2013; McLaughlin et al., 2011). Other indicators of sociodemographic risk considered included minority status, presence of risk, child psychopathology, and geographical region (as a proxy for national), all of which can relate to increased external stressors and pressures that could exacerbate the transmission of depression and anxiety.

In terms of children's age, past meta-analyses have found that the association between depression in mothers and internalizing and externalizing problems in children became weaker as children aged (Connell & Goodman, 2002; Goodman et al., 2011). However, as interpreted by Goodman et al. (2011), these results are tentative since the meta-analytic finding was constrained due to the fact that studies of older children did not specify children's prior exposures to mothers' depression (e.g., prior to the time of the study). In terms of child gender, some studies have found that the association between maternal depression and child internalizing symptoms is stronger for girls relative to boys (Goodman et al., 2011). Girls may carry greater heritability of depression (Kendler et al., 2001) and may be more strongly socialized toward maternal depression and affected by stressors associated with depression in their mothers (Hammen, 2002; Hankin et al., 2007). Research examining paternal depression has suggested that boys may be more vulnerable to their fathers' depression, which could be partly due to the fact that fathers tend to spend more time with their sons (Ramchandani et al., 2005;

Ramchandani & Psychogiou, 2009). That said, the moderating effects of gender have been suggested to differ by the developmental phase of the child.

Parent and child age at the time of measurement of depression and anxiety symptoms should also be given consideration as a moderator. A large-scale meta-analysis of 192 epidemiological studies found that the median onset age for anxiety disorders was 17 years, as compared to 30 years for mood disorders (Solmi et al., 2022), which could indicate different developmental trajectories of anxiety and depression. In light of this, sensitive developmental periods could potentially heighten or diminish the potential for symptom transmission from fathers to children.

Measurement Factors

Psychometric research shows that measures of anxiety and depression, while conceptually consistent as a whole, feature some differences that could influence the magnitude of their effects in psychopathology research (von Glischinski et al., 2021). For example, clinician diagnoses have the benefit of drawing from the clinician's experience and training but are prone to bias and rely on limited information. In contrast, self-reports allow participants to draw from all of their experiences of potential symptoms, yet are prone to respondent bias (e.g., social desirability). Similarly, even measures applied in a similar way (e.g., self-report questionnaires) are far from identical and can tap into different aspects of difficulties (von Glischinski et al., 2021). To examine these factors, we considered three measurement aspects: the measure itself (e.g., Beck Depression Inventory), the informant of the measure (e.g., self-report, parent-report), and the type of measure (e.g., interview, questionnaire).

Study-Level Moderators

We considered whether publication types (i.e., peer-reviewed articles vs. dissertations) present similar effect sizes, given evidence showing that peer-reviewed articles may have a bias for the publication of significant results. The study design (cross-sectional vs. longitudinal) was also considered to examine if cross-sectional studies present stronger effect sizes due to the measurement of all measures taking place at the same point in time. We also considered publication year as a potential moderator given the changes in the fathering role over time (Cabrera et al., 2000). It is possible, for example, that effect sizes become stronger over time as a reflection of fathers' increased role in childrearing.

Objectives and Hypotheses

In an effort to update previous meta-analytic reviews (e.g., Connell & Goodman, 2002; Kane & Garber, 2004), the present study sought to systematically review and meta-analyze the association between father depression and child depression, as well as father anxiety and child anxiety. Based on previous research on paternal depression, we expected to identify significant associations between father and child symptoms, consistent with a trend toward intergenerational transmission of internalizing psychopathology. The study also examined whether the aforementioned moderators pertaining to sample characteristics and methodology moderated these associations.

Method

Operational Definition of Constructs

This meta-analysis assessed studies that examined two aspects of internalizing difficulties in fathers and children: depression and anxiety. Depression included depressive symptoms that were self-reported (e.g., via a questionnaire), assessed via a diagnostic interview, or reported by a parent or teacher in the case of children. Anxiety included anxiety symptoms that were self-reported (e.g., via a questionnaire), assessed via a diagnostic interview, or reported by a parent or teacher in the case of children. There was no time restriction as to when these symptoms had to be experienced (e.g., prenatal period, postnatal period, at the moment of the study, lifetime).

Search Strategy

The meta-analysis followed the standards for systematic reviews and meta-analyses established by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (Moher et al., 2009). The search strategy was developed in collaboration with a psychology librarian at the first author's institution. A systematic search for published and unpublished studies until September 2021 was conducted across four databases: PsycINFO, Embase, MEDLINE, and ProQuest Dissertations and Theses Global. The search used a combination of the Boolean "and" and "or," as well as proximity indicators to combine the search terms. An example of the search strategy is presented in Supplemental Table 1. The search sought to identify studies that assessed depression and anxiety. Different variations (e.g., anxiety and anxious) of terms were included, along with related terms (e.g., internalizing). In order to delimitate the search, proximity indicators were used to ensure that those terms were used near terms pertaining to fathers (e.g., father, paternal, dad, parent) and children of different age groups (e.g., toddler, preschool, child). This allowed for a more targeted effort to identify relevant records within the vast literature on depression and anxiety. Truncated symbols were used to ensure that variant spelling and endings would be included (e.g., child*). The search was limited to human populations and articles published in English, French, or Spanish (i.e., the languages spoken by our team); of note, 241 records were excluded due to these language restrictions. The search returned 9,317 records in total, and Covidence identified 5,619 nonduplicate records (see Figure 1).

Study Inclusion and Exclusion Criteria

The abstracts of the 5,619 records were screened for inclusion in the study, resulting in the exclusion of 4,240 records. We sought to retrieve the full text of the 1,379 records that passed abstract screening; five could not be retrieved even after an interloan library request. Accordingly, the full text of the 1,374 records that were successfully retrieved were assessed for inclusion. For the abstract screening step, 23% of records were double-coded, and interrater agreement was high (89.3%; $\kappa > .70$). For the full-text screening step, 21% of records were double-coded. Interrater agreement was high (94.3%; $\kappa > .75$). Disagreements at both steps were resolved by a third coder who was not one of the original coders.

For the abstract and full-text screenings, our team considered the following inclusion criteria:

1. Studies reported on empirical data.
2. Child depression or anxiety was assessed. Only measures of trait anxiety were included given the focus on the association between anxiety as a trait, and not anxiety in specific situations or moments in time. Given the focus on the transmission of depression and anxiety, more broad measures of internalizing problems or combined anxiety/depression measures were excluded.
3. Father depression or anxiety was assessed. Only measures of trait anxiety were included given the focus on the association between anxiety as a trait, and not anxiety in specific situations or moments in time. Given the focus on the transmission of depression and anxiety, more broad measures of internalizing problems or combined anxiety/depression measures were excluded.
4. Studies were in English, French, or Spanish (e.g., languages spoken by our team).

A total of 122 studies were selected for reporting on the association between fathers' and children's symptoms of depression and anxiety. Some of these studies, however, reported on the same data (see Data Synthesis).

Data Extraction

Four independent coders extracted moderators and effect sizes using a standard data extraction coding protocol. An independent research assistant double-coded 21% of studies. The average reliability was high for continuous variables (intraclass correlation coefficient $> .90$) and categorical variables ($>90\%$ agreement). The last author resolved disagreements between coders.

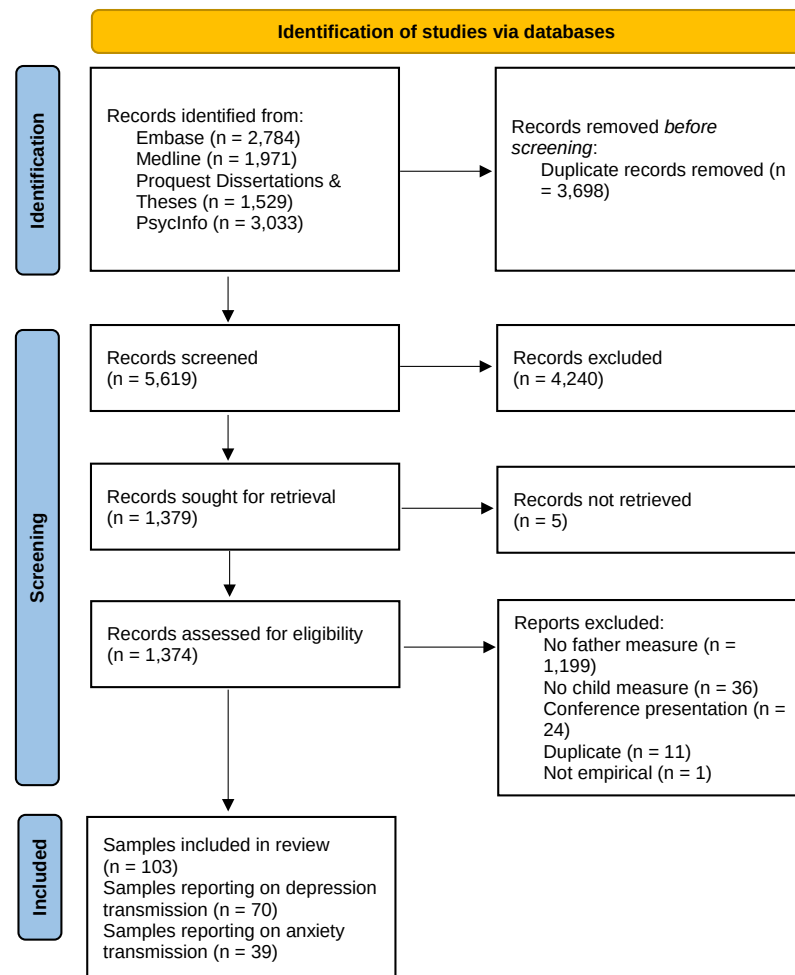
Moderators

The following continuous moderators were coded: (a) father age at the assessment of father depression/anxiety symptoms (in years); (b) child age at the assessment of father and child depression/anxiety symptoms (in months); (c) child gender (% of boys); (d) proportion of the sample that identified as an ethnic or racial minority (% of minority); and (e) publication year. The following categorical moderators were coded: (a) child and father measure of depression and anxiety (e.g., Beck Depression Inventory); (b) informant of child and father depression/anxiety symptoms (coded as child, clinician/specialist, or parent); (c) child psychopathology risk (coded as "yes" if $>80\%$ of the sample presented psychopathology); (d) type of measure to assess child and father depression and anxiety (coded as questionnaire vs. diagnosis/interview); (e) geographical location (continent where the research was conducted); (f) publication source (coded as article, book chapter, or dissertation); (g) risk status of the sample (coded as "yes" if $>80\%$ of the sample were at risk); (h) SES (coded as low SES [$>80\%$ low SES], mid-high SES [$>80\%$ mid-high SES], or mixed [diverse SES]); and (i) study design for the effect size (cross-sectional vs. longitudinal).

Quality Index

Coders also assessed the methodological quality and validity of each article using a 12-point appraisal assessment tool based on past

Figure 1
PRISMA Flowchart of Study Selection



Note. PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses. See the online article for the color version of this figure.

meta-analyses and the National Institutes of Health's National Heart, Blood, and Lungs Institute Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies (e.g., Badovinac et al., 2021; Madigan, Ly, et al., 2018; National Heart, Lungs, and Blood Institute, 2017). The criteria evaluated are presented in Supplemental Table 2. Each item was rated as "yes" (1) or "no" (0). Based on 20% double-coded cases, the average interrater agreement was >85% ($\kappa > .70$). The last author resolved disagreements between coders.

Data Synthesis

Multiple studies used data drawn from the same original sample (e.g., National Institute of Child Health and Human Development data were reported in multiple articles and dissertations). Some effect sizes referred to different time points or measures (e.g., one article measured transmission at 48 months, while a second measured it at 108 months); in such cases, all effect sizes were retained and grouped under the same sample

name for analysis. In doing so, their effect sizes became dependent, and analyses accounted for such dependency. In other cases, articles reported on the same effect sizes measured at the same time point using the same measure; in these cases, the article with the largest N or the most time points reported was selected. When the same effect sizes were reported in a dissertation and an article, we selected the one presented in the article. After applying these synthesis decisions, a total of 103 studies remained for the meta-analysis.

Most samples provided multiple, dependent effect sizes (e.g., transmission was measured at different time points in the same sample). In order to avoid the loss of information relating to the multiple effect sizes, this meta-analysis relied on a robust variance estimation technique (RVE; Hedges et al., 2010). This technique allows for the inclusion of all available effect sizes by accounting for the dependency between dependent effect sizes. Studies reported on a total of 230 effect sizes (studies had a potential of 12 additional effect sizes that were not available in the articles; these studies are included and specified in Table 1).

Data Analysis

Effect sizes were reported in different formats, such as correlations, odd ratios, and regression coefficients. Some studies reported a nonsignificant finding without an exact effect size ($k = 8$). In such cases, an effect size of p value = .50 was applied consistent with Rosenthal's (1995) recommendations and current meta-analytical practices (e.g., Anderson et al., 2021; de Bondt et al., 2020; Eirich et al., 2022; Madigan et al., 2020; Racine, Devereaux, et al., 2021; Sanjuan et al., 2021). Most effect sizes were reported as correlations; other effect sizes were thus transformed into a correlation coefficient using the R package *esc* (Lüdtke, 2019). Correlations were converted to Fisher's z for analyses in order to reduce the dependence of the effect size and variance estimate (Borenstein & Hedges, 2019). Values reported in the text were converted back to correlations for ease of interpretation. Effect sizes were interpreted based on calibrated guidelines specific to psychological research (Funder & Ozer, 2019). Accordingly, an effect size of .10 represented "an effect that is still *small* at the level of single events but potentially more ultimately consequential"; an effect size of .20 represented "an effect of *medium* size that is of some explanatory and practical use even in the short run and therefore even more important"; and an effect size of .30 represented "an effect that is *large* and potentially powerful in both the short and long run" (Funder & Ozer, 2019; p. 166). It should be noted that such guidelines merely provide a frame of reference to anchor and facilitate result interpretation—that is to say, the meaningfulness of any given effect has to be considered not exclusively based on such guidelines but also in light of past research, the nature of the research question, the phenomenon under study (e.g., base rates), potential ramifications of the phenomenon, and other factors.

Effect sizes and their variance were derived as Fisher's z using the *metafor* package (Viechtbauer, 2010). The meta-analyses with RVE were tested using *robumeta* (Fisher & Tipton, 2015), a package that accounts for the dependency between effect sizes. Consistent with norms set to account for such dependency (Tanner-Smith et al., 2016), a value of ρ of .80 was used. Sensitivity analyses testing for different values of ρ (.00, .20, .40, .60, .80, and 1.00) showed negligible differences across ρ levels (e.g., variations on the third decimal). Given that *robumeta* does not provide an option for funnel plots and Egger's tests, tests of publication bias were conducted using *metafor*. Because *metafor* does not account for dependent samples, the funnel plots and Egger's tests were run on a single effect size per study. This effect size was derived by pooling the dependent effect sizes provided by a study (e.g., if a study reported on multiple measures of anxiety, they were pooled to be able to derive a funnel plot and Egger's test). In doing so, each study was represented by one pooled effect size. Of note, we also ran these tests with all effect sizes, treating dependent effect sizes as independent; results were consistent through both approaches.

Heterogeneity level among studies was based on the I^2 index; 50% and 75% indicate medium and high heterogeneity (Higgins et al., 2003), suggesting the need for moderator analysis. The estimated confidence intervals for the true effect sizes, known as prediction intervals, are also reported (Borenstein et al., 2017). Moderators were analyzed using a univariate approach using the RVE technique. The univariate approach (i.e., each moderator tested individually) was preferred to a multilevel approach (i.e., multiple moderators tested in the same model) because the amount of missing data on the study-level moderators made a multivariate approach

untenable. The use of the univariate approach has the benefit of maximizing the available information, but it fails to account for the interrelated nature of some moderators.

Given that the RVE technique does not automatically produce a test of difference for categorical moderators, we used the omnibus test of *clubsandwich* package (Pustejovsky, 2020) to determine if effect sizes varied across levels of moderators. Levels of moderators with fewer than 4 degrees of freedom were not included given that their results are not reliable (Tanner-Smith et al., 2016).

Results

Study Characteristics

A summary of study characteristics is presented in Table 1. A total of 103 studies, drawn from 101 samples, examined the association of depression and anxiety between fathers and their children. The samples included a total of 64,743 unique participants. The median sample size was 187 participants, and sample size ranged from 19 to 9,718 participants. An average of 21% of participants identified as being from a minority ethnic background (range: 0%–100%, median: 13%). With respect to SES, 4% samples were of low SES, 30% of mid-high SES, 23% of mixed SES, and 44% did not report this information. On average, samples included a similar number of sons and daughters (mean: 49.5% boys, median: 50%, range: 0%–100%). Most samples were published in peer-reviewed journals (74%), with the rest being dissertations (25%) or book chapters (2%). Most samples were drawn from a North American (58%) or European population (28%), with the remainder drawn from Asia (7%), Australia/New Zealand (5%), the Middle East (2%), and Africa (1%). Studies were published between 1981 and 2021. The quality of each study on the quality assessment criteria is presented in Supplemental Table 3.

Effect Size Characteristics

Collectively, the 101 samples provided 230 effect sizes. The majority of effect sizes reported on cross-sectional data (168 effect sizes, 73%), and the rest reported on longitudinal data (62 effect sizes, 27%). Fathers were on average 43 years old when their depressive or anxiety symptoms were assessed (range: 28–78 years old). The assessment of internalizing symptoms was conducted via questionnaires (191 effect sizes, 83%), interviews (22 effect sizes, 10%), or formal diagnostic interviews (17 effect sizes, 7%). Measures of paternal depression and anxiety varied considerably, but the most frequent were the Beck Depression Inventory (29 effect sizes, 13%), the Center for Epidemiological Studies Depression Scale (28 effect sizes, 12%), and the State-Trait Anxiety Inventory (22 effect sizes, 10%).

Children were on average 143 months old when paternal symptoms were measured (range: prenatal to 535 months), and 156 months old when their own symptoms were measured (range: 8–535 months). The informant of children's symptoms was the child (142 effect sizes, 62%), the parents (52 effect sizes, 23%), a clinician or specialist (26 effect sizes, 11%), an interviewer (6 effect sizes, 3%), or a combination of informants (e.g., parents, teacher; 4 effect sizes; 1%). The most common measures were the Child Behavior Checklist (31 effect sizes, 14%), the Child Depression Inventory (29 effect sizes, 13%), and the Center for Epidemiological Studies Depression Scale (22 effect sizes, 10%).

Table 1
Summary of Study Characteristics

Study	Meta	N	Location ^b	SES ^c	Minority (%)	Father age	Child gender (% boys)	Child age at father measure (months)	Child age at child measure (months)	Father measure	Child measure
Abate et al. (2018)	Both	62	6	2	2	48	53	190	190	BSI	BAI, BDI
Al-Turkait and Ohaeri (2008)	Both	355	5	—	—	—	51	163	163	HSC	CBI
Allen (2001)	Both	28	4	3	17	46	62	188	188	BDI, STAI	BDI, STAI
Apsley and Padilla-Walker (2020)	Both	338	6	3	40	47	46	160	172, 220	BuAI, CESD	CESD, SCAS
Beato et al. (2016) ^a	Anxiety	390	4	2	—	42	45	—	120	BSI	SCARED
Bergman et al. (2018) ^a	Depression	56	6	3	19	44	50	156	156	BDI	CDI
Bögels and Melick (2004)	Anxiety	75	4	2	—	43	53	124	124	SCARED	SCARED
Bögels et al. (2008)	Anxiety	159	4	—	—	45	40	149	149	SCARED	ADIS
Borelli et al. (2015)	Anxiety	102	6	3	46	41	60	120	120, 152	SCL	STAI
Brennan et al. (2002)	Depression	522	2	1	8	—	50	182	182	SCI	SADS
Capron et al. (2015; ALSPAC)	Anxiety	3,187	4	—	—	—	44	0	216	CCEI	DISC
Carbray (1993)	Depression	40	6	2	9	44	36	175	175	BDI	BDI
Chang and Fu (2020)	Depression	997	3	3	—	—	52	210	210	SCL	SCL
Chubbar et al. (2020)	Depression	247	4	—	—	48	49	181	181	BDI	CDI
Cioffi et al. (2021)	Depression	561	6	2	10	37	—	9, 18, 27, 72	72	BDI, CESD	CBCL, YSR
Cobb (2020)	Depression	626	6	3	9	51	49	108	180	CESD	CBCL, YSR
Cooper et al. (2006)	Anxiety	85	4	—	10	—	51	124	124	SCI	ADIS
Crocker (1994)	Depression	39	6	3	8	—	47	120	120	BDI	CDI
Cunco (1997)	Depression	40	6	2	10	45	35	171	171	BDI	CDI
Curtiss (1999)	Depression	3,372	6	—	9	—	100	535	535	DAC	DISC
Demidenko et al. (2015)	Depression	116	6	—	—	49	0	192	192	BDI	BDI
Dimitrova (2014)	Depression	320	4	3	58	—	48	110	110	CESD	CDI
Du Rocher Schudlich (2004)	Depression	267	6	2	9	40	52	127	127	CESD, SCL	CDI
Esbjörn et al. (2013)	Anxiety	29	4	—	0	—	45	120	120	BAI	SCARED
Fleming (1994)	Depression	307	6	2	4	—	50	18	18	CESD	CESD
Flek et al. (2015)	Anxiety	97	4	—	—	38	58	51	51	STAI	PAS
Freedman-Doan (1994)	Anxiety	194	6	2	—	—	48	108	108, 120	—	—
Gallimore and Kurdek (1992)	Depression	35	6	3	3	41	26	167	167	BDI	CDI
Gere et al. (2013)	Depression	135	4	—	5	—	62	125	125	HSC	CBCL, SMFQ
Gutierrez-Galve et al. (2019; ALSPAC)	Depression	3,176	4	—	—	—	45	5	216	EPDS	DISC
Hanson (1991)	Depression	320	6	3	15	—	44	228	228	BDI	BDI
Harold et al. (2011)	Depression	559	4	—	—	38	47	75	75	HADS	SMFQ
Hastings et al. (2021)	Both	115	6	2	29	46	52	164, 192	164, 192	DAC, STAI	DAC, STAI
Herron et al. (2013)	Anxiety	138	4	2	—	43	47	109	109	BAI	DIMS
Hops (1992), Study 1	Depression	500	6	—	—	—	—	—	—	BDI	CESD
Hops (1992), Study 2	Depression	500	6	—	—	—	—	—	—	BDI	CESD
Hou et al. (2017)	Depression	444	6	3	100	48	46	156, 204	156, 204	CESD	CESD
Hou et al. (2020)	Depression	1,644	3	—	—	42	48	168, 180	168, 180	CESD	CESD
Hudson et al. (2014)	Anxiety	209	2	3	10	44	50	116	116	ADIS	ADIS
Hughes and Gullone (2010)	Both	124	2	3	—	40	40	196	196	DASS	RADS, RCMA5
Johnson (2011)	Both	556	6	2	12	42	48	143	143, 161	SCL	YSR
Kaslow et al. (1988)	Depression	62	6	3	19	37	58	116	116	BDI	DICA
Kassa et al. (2020) ^a	Both	33	4	—	—	—	55	154	154	BAI, BDI	BAI, BDI
Keizer (2012; NICHD)	Both	1,009	6	—	20	—	52	144	144, 180	STAI	CBCL
Kouros (2008)	Depression	248	6	2	13	42	52	133, 145, 157	133, 145, 157	CESD, SCL	CDI
Krain and Kendall (2000)	Anxiety	193	6	3	14	—	58	135	135	STAI	STAI
Kujawa et al. (2019)	Depression	407	6	1	21	—	56	108	108, 144	SCI	CDI
Lachman et al. (2020)	Depression	156	1	—	—	—	59	—	—	CESD	CDI
Landman-Peeters et al. (2008) ^a	Depression	349	6	—	5	—	44	246	246	CIDI, SCI	CIDI

(table continues)

Table 1 (continued)

Study	Meta	N	Location ^b	SES ^c	Minority (%)	Father age	Child gender (% boys)	Child age at father measure (months)	Child age at child measure (months)	Father measure	Child measure
Lee et al. (2020)	Depression	5,315	3	3	—	48	51	202	202	—	—
Lehman et al. (2020)	Both	27	6	—	18	—	51	156	156	BAI, BDI	BASC
Leis and Mendelson (2010)	Depression	1,045	6	3	31	—	51	282	282	FHRDCI	CIDI
Lewis et al. (2017)	Depression	9,261	4	—	—	41	52	96	162	CESD, KPDS	SMFQ
Liber et al. (2008) ^a	Both	108	4	2	—	43	55	121	121	SCI	ADIS
Lim et al. (2011)	Depression	106	6	—	90	42	63	137	137	BDI	CIDI, CDRS
Liskola et al. (2018)	Depression	206	4	2	51	—	44	126	126	GHQ	CIDI
Liu et al. (2017)	Depression	305	3	—	—	—	71	115	115	CESD	CESD
Løchen et al. (2018)	Depression	289	4	—	—	—	42	174, 198	198	HSC	SMFQ
Lubman (2018)	Depression	38	6	2	3	—	63	126	126	MMPI	CBCL
Mackinaw-Koons (2001)	Anxiety	97	6	3	9	—	32	177	177	STAI	RCMAS
Mahedy et al. (2018)	Depression	215	4	—	—	42	57	148	177	PHQ	CAPA
Mason et al. (2017)	Depression	667	6	2	3	—	48	132	132, 216, 252	SCL	CBCL, SCI, YSR
Mayhew (1997)	Depression	398	6	3	—	—	50	149, 161, 173	149, 161, 173	PSSCS	BDI
McClure et al. (2000)	Anxiety	522	2	1	9	—	51	180	180	SCI	SADS
Middelkamp et al. (2016)	Both	671	4	3	—	45	56	144	144	ASR	CBCL
Mitchell et al. (1989)	Depression	132	6	2	12	41	55	—	—	SADS	SADS
Möller et al. (2015)	Anxiety	67	4	—	—	37	49	8	8	SCARED	IBQ
Morris (1998)	Both	32	6	2	18	—	35	168	168	BSI	CBCL, CIDI, RCMAS
Mursaloglu et al. (2021) ^a	Both	48	5	—	—	—	52	190	190	PHQ	PHQ
Ohammession et al. (2005)	Depression	426	6	3	22	—	48	181	181	SSAGA	SSAGA
Olino et al. (2006)	Both	575	6	—	10	—	43	293	293	SCI	LIFE
Pagliaccio et al. (2020)	Depression	9,718	6	2	35	—	53	119	119	—	SADS
Pemble (2005)	Anxiety	50	6	2	16	—	44	84	84	STAI	ADIS
Pilowsky et al. (2014) ^a	Depression	19	6	—	—	—	—	144	144	HAMD	CIDI
Pimentel (2004)	Anxiety	109	6	2	16	—	54	115	115	ADIS	ADIS
Polenick et al. (2018)	Depression	103	6	—	—	78	40	50	50	BSI	BSI
Rasing et al. (2015)	Both	862	4	—	—	—	0	144	144	—	CIDI, SCAS
Reitman and, Asseff (2010)	Anxiety	122	6	2	18	48	46	228	228	STAI	STAI
Ringoot et al. (2015)	Depression	3,178	4	—	—	37	49	0	36, 72	BSI	BePI, CBCL
Rivens (1987)	Anxiety	38	6	—	—	44	57	151	151	STAI	STAI
Sahithya and Vijaya (2020)	Anxiety	84	3	—	—	47	67	117	117	MINI	CBCL
Schick et al. (2013)	Both	153	4	—	—	47	33	172	172	HSC	CIDI, SCAS
Shiner and Marmorstein (1998)	Depression	142	6	3	1	—	43	204	204	SCI	SCI, DICA
Smith (2013)	Anxiety	20	6	2	3	45	61	118	118	AMAS	BASC, MASC
Szkody and McKinney (2020)	Depression	708	6	—	22	—	47	223	223	ABCL	ASR
Telman et al. (2018)	Anxiety	124	4	—	—	45	41	149	149	ADIS	ADIS
Thomassin et al. (2017)	Depression	51	6	3	22	42	51	111	111	SCL	CIDI
Tichovolsky et al. (2018)	Depression	153	6	3	39	—	55	44	44	MCMAI	BASC
Tisher et al. (1994)	Depression	107	2	—	—	—	72	108	108	BDI	CIDI
Tully et al. (2008)	Depression	528	6	—	5	—	46	172	172	SCI	DICA
Turner (2007)	Both	45	6	2	—	40	50	91	91	CIDI	DISC
Turney (2009)	Depression	1989	6	1	79	29	—	12, 30	36	CIDI	CBCL
Tyrell et al. (2019)	Depression	392	6	2	49	—	48	202	202	HSC	CIDI
van Roekel et al. (2011)	Depression	306	4	—	—	—	48	161	161, 173, 185, 197, 209	—	—
van der Giessen et al. (2019) ^a	Anxiety	128	4	—	—	—	41	150	150	ADIS	ADIS
van Gastel et al. (2009)	Anxiety	82	4	—	—	—	44	121	121	CIDI	CBCL
Vami and Setoguchi (1993)	Both	54	6	2	—	39	59	121	121	BDI, STAI	CIDI, STAI
Wang (2018)	Depression	2,282	3	2	0	37	52	89	89	CESD	CBCL

(table continues)

Table 1 (continued)

Study	Meta	N	Location ^b	SES ^c	Minority (%)	Father age	Child gender (% boys)	Child age at father measure (months)	Child age at child measure (months)	Father measure	Child measure
Weller et al. (1994)	Depression	52	6	—	—	—	—	108	108	PDI	DICA
Whiting (1981)	Depression	22	6	—	0	45	58	192	192	BDI	BDI
Woodhouse et al. (2010)	Depression	189	6	2	27	—	38	198	198	CESD	CDI
Yim (1991)	Anxiety	187	6	2	70	—	57	9	9	STAI	RCMAS
Zhou and Sun (2021) ^a	Depression	2,211	3	—	12	34	53	140	140	CESD	CESD
Zoloto et al. (2012)	Depression	764	6	—	8	—	52	159	159	CESD	CESD

Note. SES = socioeconomic status; BSI = Brief Symptom Inventory; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; HSC = Hopkins Symptom Checklist; CBI = Child Behavior Inventory; STAI = Spielberg State-Trait Anxiety Inventory; BuAI = Burns Anxiety Inventory; CESD = Center for Epidemiological Studies Depression Inventory; SCAS = Spence Children's Anxiety Scale; SCARED = Screen for Child Anxiety Related Emotional Disorder; CDI = Child Depression Inventory; ADIS = Anxiety Disorder Interview Schedule; SCL = Symptom Checklist; SCI = Structured Clinical Interview; SADS = Schedule Affective Disorder Schizophrenia; ALSPAC = Avon Longitudinal Study of Parents and Children; CCEI = Crown Crisp Experiential Index; DISC = Diagnostic Interview Schedule for Children; NICHD = National Institute of Child Health and Human Development; CBCL = Child Behavior Checklist; YSR = Youth Self-Report; DAC = Depression Anxiety Checklist; PAS = Preschool Anxiety Scale; SMFQ = Short Mood and Feelings Questionnaire; CDRS = Child Depression Rating Scale; EPDS = Edinburgh Postnatal Depression Scale; HADS = Hospital Anxiety and Depression Scale; DIMS = Diagnostic Interview for Mental Disorders; DASS = Depression Anxiety Stress Scale; RADS = Reynolds Adolescent Depression Scale; RCMAS = Revised Children Manifest Anxiety Scale; DICA = Diagnostic Interview for Children and Adolescents; CIDI = Composite International Diagnostic Interview; BASC = Behavior Assessment System for Children; FHRDCI = Family History Research Diagnostic Criteria Interview; KPDS = Kessler Psychological Distress Scale; GHQ = General Health Questionnaire; MMPI = Minnesota Multiphasic Personality Inventory; PHQ = Patient Health Questionnaire; CAPA = Child and Adolescent Psychiatric Assessment; PSSCS = Pearlin & Schooler's Structure of Coping Scale; ASR = Adult Self Report; IBQ = Infant Behavior Questionnaire; SSAGA = Semi-Structured Assessment for Genetics of Alcoholism; LIFE = Longitudinal Interval Follow-up Evaluation; HAMD = Hamilton Rating Scale for Depression; BePI = Berkeley Puppet Interview; MINI = Mini-International Neuropsychiatric Interview; AMAS = Adult Manifest Anxiety Scale; MASAC = Multidimensional Anxiety Scale for Children; ABCL = Adult Behavior Checklist; MCMAI = Millon Clinical Multiaxial Inventory; PDI = Psychiatric Diagnostic Interview.

^a Effect size not available in the article. ^b 1 = Africa; 2 = Australia/NZ; 3 = Asia; 4 = Europe; 5 = Middle East; 6 = North America. ^c 1 = low; 2 = mid-high; 3 = mixed.

Association Between Paternal and Child Depression

The meta-analysis on paternal depression and child depression drew from 70 samples (58,255 unique participants) including a total of 158 effect sizes. An analysis of outliers ($\pm 3 SD$) identified three outliers that were beyond the range of $-.28-.66$ (Bergman et al., 2018; Morris, 1998 [father depression, child-rated depression]; Ohannessian et al., 2005), which were removed from the analysis, resulting in an analytical sample of 69 samples and 155 effect sizes. The results revealed a significant association between paternal depression and child depression: $r = .15$, 95% CI [.12, .17], with the effect size falling between “potentially ultimately consequential” and “some explanatory and practical use” (Funder & Ozer, 2019). The funnel plot (see Supplemental Figure 1) did not show asymmetry and Egger’s test was not significant ($z = 0.24, p = .81$), suggesting the absence of publication bias. The value of I^2 was 85.06 ($\tau^2 = .009$), suggesting the presence of heterogeneity between studies. The plausible range of true effect size ranged from $r = -.04$ to $r = .33$, suggesting substantial variability in results.

The moderator analysis revealed that child age at the measurement of father depression and child depression were significant moderators (see Table 2). The effect was positive, with the association getting stronger as children become older. It is important to note that this correlation was based on aggregate measures and may not represent the same associations at the individual participant level. However, after observation of the moderation graph (see Supplemental Figure 2), the effect was driven by one extreme value (i.e., which examined depression in elderly fathers and middle-aged fathers). After this value was removed, the moderator was no longer significant. None of the other moderators were significant, suggesting a robust association irrespective of study characteristics and methodological factors. Of note, SES could not be tested as a moderator because too few samples were from a low SES background.

Association Between Paternal and Child Anxiety

The meta-analysis on paternal anxiety and child anxiety drew from 39 samples (11,683 unique participants) including a total of 72 effect sizes. An analysis of outliers ($\pm 3 SD$) did not reveal any outliers. The results revealed a significant association between paternal anxiety and child anxiety: $r = .13$, 95% CI [.07, .18], $p < .001$, with the effect size falling between “potentially ultimately consequential” and “some explanatory and practical use” (Funder & Ozer, 2019). The funnel plot (see Supplemental Figure 4) did not show asymmetry and Egger’s test was not significant ($z = 0.62, p = .53$). The value of I^2 was 84.87 ($\tau^2 = .024$), suggesting the presence of heterogeneity between studies. The plausible range of true effect sizes was quite large, ranging from $r = -.19$ to $r = .42$ and suggesting a great deal of heterogeneity in the true effect sizes.

The analysis of moderators, however, did not identify any significant moderators (see Table 3). Of note, SES could not be tested due to the low number of studies with low SES, and the father measure could not be tested as only the Spielberg State-Trait Anxiety Inventory had sufficient degrees of freedom for the analysis.

Table 2*Moderators of the Paternal Transmission of Depression*

Categorical moderators	<i>k</i>	<i>df</i>	<i>r</i>	95% CI	Omnibus wald test (<i>F</i>)	<i>p</i>
Child measure					0.70	.57
BDI	19	4.3	.16**	[.04, .27]		
CBCL	22	7.1	.16***	[.07, .25]		
CDI	28	15.0	.15***	[.08, .21]		
CESD	22	5.4	.11***	[.06, .16]		
Child measure informant					0.08	.93
Child	103	40.9	.15***	[.11, .18]		
Clinician/specialist	11	5.2	.14**	[.03, .26]		
Parent	34	12.0	.16***	[.10, .22]		
Child psychopathology					2.08	.18
Yes	13	48.6	.11***	[.05, .17]		
No	137	8.7	.16***	[.13, .19]		
Child type of measure					0.03	.86
Diagnosis/interview	22	10.8	.15***	[.09, .22]		
Questionnaire	133	9.6	.15***	[.12, .18]		
Father measure					0.37	.70
BDI	29	12.0	.16***	[.10, .22]		
CESD	28	12.6	.13***	[.08, .17]		
SCL	15	4.1	.12	[−.01, .25]		
Father measure informant					1.42	.26
Clinician/specialist	16	6.9	.09*	[−.001, .18]		
Father	131	46.1	.14***	[.12, .17]		
Father type of measure					0.16	.70
Diagnosis/interview	21	9.0	.13***	[.05, .22]		
Questionnaire	134	49.7	.15***	[.12, .18]		
Geographical location					0.86	.36
Europe	37	13.1	.18***	[.12, .24]		
North America	100	36.7	.15***	[.11, .18]		
Publication source					0.37	.55
Dissertation	44	13.6	.17***	[.09, .24]		
Peer-reviewed article	108	43.7	.14***	[.11, .17]		
Risk status					0.11	.74
Low risk	48	21.0	.13***	[.08, .19]		
High risk	24	13.4	.11	[−.02, .24]		
Study design					1.86	.18
Cross-sectional	104	49.4	.16***	[.13, .19]		
Longitudinal	51	15.9	.12***	[.08, .17]		
Continuous moderators	<i>k</i>	<i>df</i>	<i>b</i>	95% CI	<i>t</i> value	<i>p</i>
Child age (father assessment)	145	64	.0004	[.0001, .0006]	3.33	.001
Without extreme value	144	63	.0003	[−.0001, .0007]	1.68	.10
Child age (child assessment)	145	64	.0004	[.0001, .0006]	3.32	.002
Without extreme value	144	63	.0004	[−.0001, .0008]	1.56	.12
Child gender	139	63	−.001	[−.004, .001]	−1.12	.27
Father age	67	31	.001	[−.004, .006]	0.50	.62
Minority	83	41	−.0001	[−.001, .001]	−0.13	.90
Publication year	155	67	.0003	[−.002, .003]	0.25	.80

Note. BDI = Beck Depression Inventory; CBCL = Child Behavior Checklist; CDI = Child Depression Inventory; CESD = Center for Epidemiological Studies Depression Inventory; SCL = Symptoms Checklist.

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

Comparison of the Effect Sizes for Depression and Anxiety

Using a moderator analysis, we compared whether the magnitude of effect sizes differed between anxiety and depression. Results revealed that the magnitude did not significantly differ ($F = 2.09$, $p = .154$).

Discussion

This study provides a comprehensive meta-analysis of associations between fathers' depression and anxiety and their children's

depression and anxiety. Historically, theory and research have suggested that parental depression and anxiety can negatively affect children's development and increase their offspring's likelihood of developing these internalizing symptoms and disorders. For many years, child psychopathology research has centered on mothers and their influence in transmitting depressive and anxiety symptoms to their offspring (Goodman et al., 2011). By comparison, less attention has been dedicated to the influence of fathers in the intergenerational transmission of internalizing psychopathology. Although previous meta-analyses have demonstrated associations between paternal and child psychopathology broadly (Connell & Goodman, 2002;

Table 3*Moderators of the Paternal Transmission of Anxiety*

Categorical moderators	<i>k</i>	<i>df</i>	<i>r</i>	95% CI	Omnibus wald test (<i>F</i>)	<i>p</i>
Child measure					0.02	.90
ADIS	11	4.9	.11**	[.02, .20]		
STAI	14	5.6	.12	[−.11, .34]		
Child measure informant					2.24	.14
Child	36	14.6	.13***	[.04, .21]		
Clinician/specialist	15	7.4	.10**	[.03, .16]		
Parent	18	5.7	.19***	[.11, .26]		
Child psychopathology					0.34	.57
Yes	24	11.2	.15***	[.07, .22]		
No	47	22.5	.12***	[.04, .20]		
Child type of measure					0.01	.92
Diagnosis/interview	18	10.8	.12***	[.04, .20]		
Questionnaire	54	9.6	.13***	[.05, .20]		
Father measure informant					0.30	.60
Clinician/specialist	13	5.8	.14**	[.004, .28]		
Father	56	26.2	.11***	[.04, .18]		
Father type of measure					1.02	.33
Diagnosis/interview	17	9.0	.17***	[.07, .26]		
Questionnaire	55	49.7	.11***	[.04, .18]		
Geographical location					0.45	.51
Europe	24	11.9	.17***	[.10, .23]		
North America	42	16.9	.13***	[.05, .22]		
Publication source					0.00	.99
Dissertation	26	9.9	.13***	[.05, .20]		
Peer-reviewed article	46	24.9	.13***	[.05, .20]		
Risk status						
Low risk						
High risk						
Study design					4.9	.07
Cross-sectional	61	33.3	.14***	[.07, .20]		
Longitudinal	11	4.4	.06**	[.03, .11]		
Continuous moderators	<i>k</i>	<i>df</i>	<i>b</i>	95% CI	<i>t</i> value	<i>p</i>
Child age (father assessment)	71	37	−.000	[−.001, .001]	−0.01	.99
Child age (child assessment)	72	37	−.000	[−.001, .001]	−0.45	.65
Child gender	72	37	−.001	[−.004, .002]	−0.73	.47
Father age	40	19	−.014	[−.038, .011]	−1.16	.26
Minority	41	19	−.000	[−.004, .003]	−0.05	.96
Publication year	72	37	−.003	[−.011, .005]	−0.76	.45

Note. ADIS = Anxiety Disorder Interview Schedule; STAI = Spielberg State–Trait Anxiety Inventory.

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

Kane & Garber, 2004), these studies did not examine the specific influences of paternal depression and anxiety on these child symptoms. Thus, in an effort to update and expand upon these reviews, we conducted a comprehensive meta-analysis of associations between fathers' and children's depressive and anxiety symptoms and examined the impact of potential moderators on these associations.

Paternal Depression and Child Depression

In the present study, we found a significant positive association of $r = .15$ (95% CI [.12, .17], $N = 58,255$) between paternal and child depression. In terms of magnitude, this effect is comparable to the effect size found in a previous meta-analysis focusing on the association between paternal mental health problems and children's internalizing behaviors ($r = .14$; Connell & Goodman, 2002). Similarly, our effect size was comparable to Kane and Garber's (2004)

meta-analysis on the link between paternal depression and children's internalizing behaviors, given that confidence intervals overlapped. This suggests that the influence of father internalizing symptoms on child internalizing symptoms has remained consistent over time, as further demonstrated by the nonsignificant moderation effect of publication year.

Given that fatherhood has undergone important sociological changes in recent decades, including the increased prominence of paternal caregiving roles, it was anticipated that the strength of the relationship between paternal depression and child depression would be of comparable magnitude to the most recently established effect size between maternal depression and internalizing behaviors ($r = .23$, Goodman et al., 2011). There are several possible explanations for the smaller effect size for fathers. One possible explanation is that the relationship between parent and child internalizing outcomes may vary as a function of the amount and/or quality of parental

involvement in childcare (see Temmen & Crockett, 2021). Despite increases in father involvement, mothers continue to spend more time than fathers with their children. Indeed, while caregiving roles have become increasingly more balanced between mothers and fathers over time, fathers continue to be under considerable pressure to balance work and family responsibilities (Kuo et al., 2018), are more likely to hold full-time jobs than mothers (Catalyst, 2015), and may thus spend less time engaged in childcare than mothers (Parker & Wang, 2013). This could explain why father symptoms appear to have a lesser effect on child depression than mother symptoms. It should be noted, however, that emotional aspects of paternal involvement (e.g., closeness, attachment) may be more powerful than the mere frequency of paternal involvement in predicting child psychopathology outcomes and symptom transmission (Barker et al., 2017). Another explanation for our smaller effect size may relate to the fact that prevalence rates of depression are higher among women than men (Kuehner, 2017) and, as a result, the potential for symptom transmission from mother to child may be heightened. Broader study factors may also offer an additional explanation. For instance, mothers are regularly included as the sole parent participant in studies, whereas fathers are much more likely to participate in research jointly with mothers (Parent et al., 2017). Accordingly, whereas father-specific effects on child symptoms often account for the mother's influence, associations between maternal and child depressive symptoms are more often investigated in a vacuum—that is, in isolation from the father's influence. It is therefore possible that the size of effect characterizing associations between mother and child symptoms is slightly inflated, as compared to that between father and child symptoms. Finally, other unmeasured factors (e.g., parenting differences, nonresident fathers) may influence the strength of the relationship between paternal and child depression. More studies are needed to understand the transmission of depression within the context of multiple caregivers.

Paternal Anxiety and Child Anxiety

In the present study, we found a significant, positive association between paternal and child anxiety ($r = .13$, 95% CI [.07, .18], $N = 11,683$). This result is in line with the aforementioned meta-analysis by Connell and Goodman (2002), which found the association between paternal mental health problems and children's internalizing behaviors to yield an overall effect size of $r = .14$. More generally, this finding is also consistent with previous studies that found that offspring that have parents with anxiety disorders at greater risk for developing anxiety disorders themselves (Lawrence et al., 2019; Micco et al., 2009). Interestingly, the magnitude of the effect sizes for father-child anxiety associations did not differ significantly from father-child depression associations. While neither of these effects approximate a causal mechanism, this could be interpreted to suggest that both paternal depression and anxiety may hold a similar influence on children's development of these disorders. It is important to mention, however, that an overlap in the risk trajectories predicting child anxiety and depression likely exists, seeing as these symptoms are frequently comorbid (Cumplings et al., 2014). Likewise, substantial heterogeneity in effect sizes was found in anxiety results (this was also the case, but to a lesser extent, for depression results). Seeing as this heterogeneity was left largely unaccounted for by moderators, this may indicate that father anxiety is a more unstable predictor of child anxiety; further research into

mediators and moderators of this relationship may thus be especially important to undertake.

This finding on the link between father and child anxiety calls attention to the continued need to consider the relative influences of paternal anxiety separately from the influences of maternal anxiety. Studies examining the intergenerational transmission of anxiety have mainly drawn on theoretical models of childhood etiology that perceive maternal and paternal anxiety as interchangeable (e.g., Hudson & Rapee, 2004). While studies examining the combined contribution of mother and father anxiety are valuable from a clinical perspective, added insight can be gained from examining the independent contributions of paternal anxiety, as well as *joint* contributions of both parents' anxiety. Scholars have emphasized the importance of considering the joint or interactive effect of both parents on child outcomes (e.g., Dagan & Sagi-Schwartz, 2018; Deneault et al., 2022). Indeed, it is possible that children who are raised by two parents who experience anxiety or depression may be especially at risk for internalizing difficulties (additive model). In contrast, it is possible that having one parent without symptoms is sufficient to offset the influence of one parent with such symptoms. Future research should consider both the independent and joint influence of maternal and paternal depression and anxiety on the development of such symptoms in children, such that a more complete picture of children's environments can be reached.

While research examining mediators of the association between paternal anxiety and their children's anxiety is still in emergence, one potential mechanism involved in the father-child transmission of anxiety could be negative parenting behaviors. It has been proposed that paternal anxiety can result in negative parenting behaviors, such as hostility (Gouze et al., 2017), tension (Breinholst et al., 2019), overprotection (Möller et al., 2016), and overinvolvement (Möller et al., 2015), which can increase the risk of child anxiety in early childhood. Furthermore, these parenting behaviors are associated with the development of insecure child-father attachment relationships, which can in turn heighten the risk for emotion regulation difficulties (Esbjörn et al., 2012). Attachment insecurity itself may also be an intervening mechanism, given that children who present an insecure attachment to their father are at heightened risk for internalizing difficulties (Deneault et al., 2021). It is also possible that genetic influences (Franić et al., 2010) and other parent-child processes, such as parental modeling of anxiety (Drake & Ginsburg, 2012) and parent-child communication (Vasey et al., 2014) play a role in the intergenerational transmission of anxiety. Mediators of the father-child anxiety association certainly warrant concentrated attention in future research, as they would inform a more fulsome understanding of the potential mechanisms by which paternal anxiety may be transmitted.

Moderators

The mean child age at the time of measurement of paternal depression and child depression was found to be the only significant moderator of the association between paternal and child depression, whereby studies of older children showed a larger effect. However, after the removal of one extreme value, the moderator became statistically nonsignificant. This result does not align with the previous meta-analysis by Connell and Goodman (2002), who found that the association between paternal psychopathology and internalizing problems became stronger in older children. Since the age of the child at the time of data collection rarely captures the true onset of

depressive symptoms (or the duration of exposure to the parent's depression) in cross-sectional research, it is difficult to determine if child age effects are truly reflective of sensitive developmental periods or if they could be related to other unmeasured factors. Interestingly, some research suggests that intergenerational transmission effects may be particularly strong and may follow a more chronic course when fathers' recurrent depression emerges during their offspring's childhood or adolescence (Jaffee et al., 2021). More prospective research is needed to clarify sensitive developmental periods for potential symptom transmission—namely, periods during which father depression and/or anxiety may be more likely to predict the onset/increase of such symptoms in children, controlling for other relevant factors.

While our meta-analyses on paternal depression and child depression and paternal anxiety and child anxiety indicated between-study heterogeneity, none of the moderators examined could explain the variability in effect sizes. This could be due to the lack of diversity among research on paternal depression and anxiety, reflecting a broader lack of diversity in fathering research. Most research on fathers relies on nonrepresentative samples of fathers who are often homogenous with respect to demographic characteristics, such as SES and ethnicity. Fathers in research are mostly in heterosexual, biparental relationships and tend to be more involved in childcare than the broader population. In our study, there was little variability across studies in SES on account of studies recruiting mostly upper-middle-class fathers, which precluded testing SES as a moderator. It is imperative for future research to recruit more diverse samples of fathers, which would allow developmental science to represent the rich diversity of fathers and their influence on children's development.

In addition, it is likely that moderators that were not included in this meta-analysis (e.g., quality/frequency of parental involvement, parenting sensitivity) may account for the heterogeneity in results. Pursuing such moderators as factors explaining heterogeneity would require methods other than traditional meta-analyses, which are dependent on the inclusion of such information in articles or dissertations. Individual participant meta-analyses (IPD) may be well-suited to such research questions, as researchers share their full data set and can provide the required variables for the analyses. Such IPD techniques may present an interesting way forward for future research, notably by allowing to test whether moderators like paternal involvement matter for the transmission of anxiety and depression.

Limitations

Despite this study's contributions to our understanding of the specific associations of depression and anxiety in fathers and children, it is not without limitations. First, as mentioned previously, meta-analyses are not able to speak to causal links between variables. Therefore, while our interpretation of results was carried from sound theoretical rationale, alternative explanations for our findings are important to mention. It is possible that internalizing difficulties in children could also contribute to such difficulties in fathers in a bidirectional fashion, a possibility that could not be examined here. It is also possible that stressors or vulnerability factors external to the father-child relationship (e.g., traumatic events) may be triggering the risk for depression/anxiety in both the father and child, thus potentially inflating the correlations we found. Explorations of these alternative hypotheses would deserve attention in future research.

Similarly, meta-analyses rely on aggregate measures for each study, and different findings may be identified using individual participant data meta-analyses.

Second, this study was unable to assess the role of SES and culture on the association of depression and anxiety between fathers and children, despite evidence showing that the association may be influenced by such factors (e.g., Aikens et al., 2008; Oliver-Parra et al., 2020; Sullivan et al., 2019). Our inability to dive into the role of these moderators was due to the little diversity in samples included in the meta-analysis. Relatedly, moderators were tested one at a time, and not simultaneously into a meta-regression of all moderators. Many studies fail to report critical information to test moderators, and this incomplete reporting makes it difficult to retain a large enough sample of studies for large meta-regressions. Nonetheless, moderator analyses should be interpreted with caution given that some of the moderator effects may be highly correlated and that our analyses did not adjust for such cofounders. Third, the onset and length of depression and anxiety may play a role in strengthening or weakening the relation between depression and anxiety intergenerationally, but too few studies report this information to allow for testing. Longitudinal study designs with fathers will allow for a better understanding of the various and complex mechanisms involved in the intergenerational transmission of internalizing difficulties. Fourth, our meta-analysis does not account for the overlap (i.e., comorbidity) between child anxiety and depression. Future work would benefit from disentangling independent versus joint risk transmission pathways for child anxiety and depression.

Practical Implications and Future Directions

Children are subjected to a multitude of influences within and across their social environments, and these networks of influence only expand with age. Our results suggest that children exposed to paternal anxiety/depression symptoms may be *slightly* more susceptible to developing their own anxiety and depressive symptoms. It is thus important not to overstate the strength of association between father and child symptoms in an applied sense; indeed, at the level of single events, these associations are quite modest in magnitude and should be interpreted as such as a starting point. Nevertheless, we believe that the magnitude of associations we found between fathers' anxiety/depression and their children's symptoms remains nonnegligible and has applied implications. The presence of internalizing difficulties in a child, let alone in a family system, is almost never a static or isolated problem—when left unaddressed, such symptoms have the potential to become reinforced by the family cycle over time and perpetuate patterns of dysfunction at various levels. Childhood anxiety and depression are, in fact, well known to forecast risk for dysfunction across peer, family, and school environments (e.g., Jaycox et al., 2009), which in turn can generate increased internalizing distress. In addition, anxiety and depression are frequently comorbid, with anxiety symptoms often compounding the risk for developing depressive symptoms later on and vice versa (Cummings et al., 2014). Therefore, even when their effects are estimated to be small, the identification of modifiable risk factors for anxiety/depression in children's immediate and most influential environments—namely, their family environments—is important and can help to inform ways to mitigate potential cascades of developmental risk.

Father psychopathology, much like mother psychopathology, matters for the development of psychopathology in their children. This may prove to be particularly important for clinical interventions, especially given that fathers underutilize mental health services compared to mothers (Phares et al., 2010). Family-centered and/or parent-based interventions for child and adolescent internalizing problems can greatly benefit from taking a systemic approach and engaging fathers more consistently in therapy. In a recent intervention aimed at increasing child development knowledge in mothers and fathers, the most important predictor of participation was the other parent's participation (McKee et al., 2021), highlighting the need for a systemic approach to interventions and therapy to derive benefits in children. Likewise, reducing barriers for fathers to engage in their own individual therapy is an important step toward the prevention of negative child outcomes (Liber et al., 2008). Some research has found that clinical intervention may reduce depressive symptoms in fathers and result in less negative parenting behaviors (Forehand et al., 2012) and that various treatment options for perinatal depression and anxiety for new fathers can be effective (O'Brein et al., 2017). However, there is a lack of data on the effect of paternal psychotherapeutic interventions on child psychopathology (Gunlicks & Weissman, 2008). Therefore, future research would benefit from examining the effectiveness of father-inclusive clinical interventions on child anxiety and depression outcomes.

The results of the present meta-analysis can inform several other suggestions for future developmental and psychopathology research. First, it is important for researchers to continue to examine the effects of fathers' psychopathology on children by including samples of fathers. The recruitment of fathers and mothers is especially recommended to consider independent and joint effects of parental internalizing difficulties. Second, research should make an effort to recruit families from diverse backgrounds, notably in terms of ethnocultural background, SES, family configuration, and sexual orientation. This is especially important given that research has found that the effect of parental depression on children may be influenced by such systemic and sociocultural factors (Aikens et al., 2008; Oliver-Parra et al., 2020; Sullivan et al., 2019). There is also a need to further understand the relationship between child age and the paternal transmission of depression and anxiety, as mentioned previously. Such studies should prioritize prospective designs and consider the role of other factors beyond age, such as duration of exposure, timing, or severity of parental depressive and anxiety symptoms. Third, a large number of studies in this meta-analysis relied on parental report of children's depression. There is a need for studies to assess internalizing symptoms using multiple informants (e.g., parents and children) or formal diagnostic tools, seeing as some evidence suggests that associations between parent and child depression are amplified when symptoms are only reported on by parents (Ringoot et al., 2015). Lastly, research that advances knowledge on the potential mechanisms and mediating variables of the intergenerational transmission of paternal psychopathology is needed. Altogether, such work is essential in improving the treatment and intervention of parents' and children's mental health globally.

Conclusion

In the current meta-analysis, we found small-to-moderate associations between father's anxiety and depression symptoms and

their offspring's own anxiety and depression symptoms, respectively. Results of this meta-analysis provide updated and important evidence for both the clinical and research community to consider, bolstering support to the theory suggesting that fathers' internalizing symptoms can confer risk for the development of depression and anxiety in their children. This meta-analysis is also the first to provide an estimate of the magnitude of associations between fathers' and children's anxiety symptoms. Overall, current results carry important implications for our understanding of developmental psychopathology from a family systems perspective.

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