A Systematic Review and Meta-Analysis: Paternal Anxiety and the Emotional and Behavioural Outcomes in their Offspring

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ABSTRACT

Objective: Anxiety disorders are highly prevalent worldwide; however, the literature lacks a meta-analytic quantification of the risk posed by fathers' anxiety for offspring development. Here, we aimed to provide a comprehensive estimate of the magnitude of the association between paternal anxiety and offspring emotional and behavioral problems.

Method: In this systematic review and meta-analysis, to identify all the quantitative studies that measured anxiety in fathers and emotional and/or behavioral outcomes in offspring, we searched Web of Science, Ovid (Embase, MEDLINE, PsycINFO), the Trip Database and ProQuest in February 2022. We set no limits for offspring age, publication language or year. We extracted summary estimates from the primary studies. We used meta-analytic random-effects three-level models to calculate correlation coefficients. Quality was assessed using the Newcastle-Ottawa Scale. Our study protocol was pre-registered with PROSPERO (CRD42022311501) and adhered to the PRISMA reporting guidelines.

Results: We identified 11,746 records, 98 of which were included in our meta-analysis. We found small but significant associations between paternal anxiety and offspring emotional and behavioral problems overall (r=.16, 95%CI[.13,.19]), behavioral (r=.19, 95%CI[.13,.24]), emotional (r=.15, 95%CI[.12,.18]), anxiety (r=.13, 95%CI[.11,.16]), and depression problems (r=.13, 95%CI[.03,.23]). We identified some significant moderators.

Conclusion: Paternal mental health is associated with offspring development and the offspring of fathers with anxiety symptoms or disorders are at increased risk of negative emotional and behavioral outcomes, in line with the principles of multifinality and pleiotropy. The substantial heterogeneity among studies and the over-representation of White European American groups in this literature highlight the need for further research.

Diversity & Inclusion Statement: While citing references scientifically relevant for this work, we also actively worked to promote inclusion of historically underrepresented racial and/or ethnic groups in science in our reference list.

Data Sharing: All our data and analytic code is openly accessible via the Open Science Framework (https://osf.io/g4kzf/?view_only=7705e0b0362e4471bb4fd74e250fac36).

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Key words: anxiety; fathers; offspring; intergenerational transmission; emotional and behavioral outcomes

INTRODUCTION

Anxiety disorders (ADs) are among the most common psychiatric conditions for men globally[1] (point prevalence in the adult male population: 2.2-3.8%;[2] in new fathers: 6.57-13.54%[3]), representing a significant public health concern. Moreover, ADs prevalence has increased since the outbreak of the coronavirus pandemic.[4]

Children whose parents have ADs, compared to children whose parents do not, are themselves at increased risk of ADs (risk ratio [RR]=1.76, 95%CI[1.58, 1.96])[5], depressive (RR=1.31, 95%CI[1.13, 1.52]),[5] and behavioral disorders.[6] Findings from a meta-analysis by Ahmadzadeh et al.[7] of genetically informed research designs suggest that postnatal anxiety exposure, but not prenatal exposure, may be causally associated with concurrent offspring emotional symptoms (offspring age range=0.75-22 years) via nongenetic mechanisms (r=.13, 95%CI[.04, .21]). These results are in line with evidence supporting the centrality of environmental factors in the intergenerational transmission of psychopathology, even after accounting for genetic risk.[8-10]

However, most of the existing evidence refers to samples comprising only or mainly mothers. Maternal mental health problems during the peri- and postnatal period have been linked to emotional and behavioral difficulties in offspring from infancy through adolescence with small-to-moderate effect sizes.[11] In contrast, the role of fathers' mental health, and their

anxiety specifically, for their children's development have received comparatively little attention.[12,13] Moreover, research has highlighted significant correlations between maternal and paternal mental health problems,[14] including anxiety disorders.[15] Significant correlations have also been reported between maternal and paternal rearing practices which are associated with child anxiety (e.g., overinvolvement).[15] Taken together, when studying the role of environmental factors in the intergenerational transmission of psychopathology, fathers' role must also be considered.

Importantly, fathers and mothers are likely to have different impacts on their children's behavioral and emotional outcomes.[13,16] Theoretical and empirical evidence indicates that fathers' evolutionary-based prominent role in the socialisation and encouragement of children's autonomy makes fathers particularly central in the development of anxiety disorders,[13,16,17] and these historical differences in roles provide a theoretical rationale to consider parent-child associations for fathers and mothers.[18]

Cultural factors, race, ethnicity, and socioeconomic status (SES) are likely to influence the way parents behave across cultures, societies and historical eras.[19,20] The literature suggests that fathers have an active presence in childcare internationally and that their involvement in childcare has increased in recent decades.[21] This trend is partly due to the growing participation of women in the labour force,[22] and partly related to changing cultural expectations for gender equality and fatherhood.[23] However, the ways in which variations due to culture and SES shape individuals' parenting are complex. Fathers' time spent with children has been shown to vary across SES, racial and ethnic groups in American families,[20,22,24] with a mixed pattern of results depending on individual attitudes and family circumstances, as well as measures and constructs used to assess paternal involvement.[20]

Irrespective of the quantity of time fathers spend with their children, others have suggested that it is the quality of time spent that matters for child development and wellbeing.[25]

The literature lacks a meta-analytic quantification of the risk posed by fathers' anxiety for offspring emotional and behavioral disorders. The theoretical principle of multi-finality[26] holds that a single risk, such as paternal AD, could predict many child outcomes (for example, emotional and/or behavioral problems). Importantly, meta-analyses examining parent anxiety and child psychiatric outcomes focus only on offspring emotional disorders and symptoms.[5,7,27,28] For example, in the first meta-analysis of the risk posed to offspring by fathers' trait anxiety, Trepiak et al.[28] reported a positive association between paternal and child trait anxiety (r=.13, 95%CI[.07, .18], k=39, N=11,683). Additionally, most of the existing research looking at the intergenerational transmission of psychopathology has focused on infancy and early childhood, when the impact of parents is theorised to be particularly crucial,[29] although associations were shown to remain significant throughout adolescence.[30,31] Nevertheless, the long-term impact of parental anxiety on offspring development has been understudied.[31]

Previous research has highlighted the potential moderating effect of several sociodemographic (e.g., offspring sex and age, biological relatedness between the parent-offspring dyad, risk level of the parental sample) and study-level variables (e.g., measurement methods used to assess paternal anxiety and offspring outcomes, such as diagnostic interviews administered by professionals vs self-report questionnaires, time lag between the assessments, the country where the study was conducted, and the publication year of the study), which might impact the association between paternal anxiety and offspring psychopathological outcomes.

This study aimed to address two important gaps, namely the relatively poorly understood role of fathers in the intergenerational transmission of psychopathology and the risk posed by paternal anxiety (disorders, trait and state symptoms) for offspring emotional and behavioral outcomes from infancy to adulthood. Importantly, we are writing from a euro-centric, heteronormative and nuclear family perspective.

METHOD

Search Strategy and Selection Criteria

We conducted a systematic review and meta-analysis of the scientific evidence on the associations between paternal anxiety (disorders and symptoms) and their offspring emotional and behavioral outcomes. Our methods, inclusion/exclusion criteria and analyses were pre-registered (PROSPERO, CRD42022311501).

We developed our strategy in accordance with the PRISMA guidelines[32] and refined it with a Psychology Research Engagement Librarian. We searched the Web of Science and Ovid (Embase, MEDLINE, PsycINFO) in February 2022, without time or language limits. To identify relevant grey literature, we searched the Trip Database and ProQuest (see Supplement 1 for the full search strategy).

We used EndNote 20.2 software[33] and the Rayyan web app (https://rayyan.qcri.org)[34] to manage, screen, and review all suitable papers, and Accelerator for the de-duplication process.[35]

We included studies in which there was (a) an assessment of paternal anxiety via a self-report measure or diagnostic interview, in line with the *DSM-5* criteria (this includes trait and/or

state anxiety symptom measures as well as diagnostic assessments; studies focused solely on paternal posttraumatic stress disorder or obsessive compulsive disorder were excluded); (b) an offspring sample; (c) any quantitative measure of offspring emotional and/or behavioral outcomes. Studies were included only if (d) offspring outcomes were measured no earlier than paternal anxiety, because our main interest was to assess the potential impact of paternal anxiety on offspring development. Finally, (e) all quantitative study designs were considered. We excluded records if they (a) were reviews, commentary papers or research protocols; (b) did not assess fathers' anxiety specifically (for clarity, studies that assessed internalising symptoms without reporting a specific measure for anxiety were also excluded); (c) focused on offspring identified in light of specific physical (e.g., cancer, seizures) or mental health (e.g., communication or learning disorders) problems (this criterion was included to prevent the cofounding effects of offspring physical or mental health problems on fathers, given our focus on the risk posed by paternal anxiety for offspring outcomes).

We sought summary estimates from the primary studies.

All titles and abstracts were screened to check whether they met the pre-determined inclusion criteria. Following a calibration exercise on 100 records conducted by FZ and PJL, 52% of all returned records were double-screened by FZ and two research assistants independently (inter-rater agreement=86%). Conflicts were examined and resolved by PJL. FZ reviewed all retained full-texts and 24% of the records were second-assessed by an independent researcher, blind to FZ's decisions; disagreements were resolved by consensus after discussion with PJL. Inter-rater agreement for full-text screening was high (98.9%; κ>.96).

We extracted and recorded (a) authors and publication date; (b) total sample size; (c) demographic characteristics of participants (i.e., age, sex and/or gender, which are different but not always distinguishable in published research, race and/or ethnicity, nationality – if

information on race/ethnicity was unavailable); (d) study design and setting; (e) characteristics of paternal anxiety measure; (f) characteristics of offspring outcomes assessed and measures used.

Our outcome was offspring emotional and behavioral problems. The following continuous moderators were coded: (a) offspring age at the assessment of their behavioral/emotional outcomes (in months); (b) offspring sex (% females); (c) time lag between father and offspring assessment; and (d) publication year of the study. The following categorical moderators were coded: (a) type of assessment of offspring outcomes (coded as diagnostic interview vs non diagnostic interview); (b) informant of offspring outcomes (coded as self-report, clinician/specialist, parent, mother, father, teacher, and combined raters); (c) type of assessment of paternal anxiety (coded as diagnostic interview vs non diagnostic interview); (d) association type (cross-sectional vs prospective longitudinal); (e) geographical location (i.e., continent where the research was conducted); (f) father-offspring biological relatedness (birth fathers only, adoptive fathers only, majority birth fathers, not stated); and (g) risk level of the paternal sample. Positive offspring outcomes (i.e., prosocial behavior, self-esteem, self-efficacy, wellbeing, socio-emotional development) were reverse-coded for the purpose of the meta-analysis.

The methodological quality of the studies included in the meta-analysis was evaluated with the Newcastle-Ottawa Scale (NOS) for case-control and cohort studies,[36] as recommended by the Cochrane collaboration. Some items were irrelevant for some studies so, for comparability, we calculated a mean score for each study. Twenty-five percent of the studies were double-rated, with discrepancies discussed and resolved by consensus, with input from PJL if a consensus could not be reached.

Data Analysis

The meta-analysis was conducted using R-Studio version 4.2.3.[37] Most of the included studies contributed multiple effect sizes, and the same sample was used by multiple studies, thus following a nested structure.[38] To model these dependencies in our data, we used the *Metafor* package[39] to fit meta-analytic random-effects three-level models via restricted maximum likelihood procedures.[40] We used the Akaike information Criterion (AIC), Bayesian Information Criterion (BIC) and likelihood ratio test to assess whether the three-level model was superior (i.e., explained significantly more variance) to a reduced two-level model.

Most effect sizes were reported as Pearson product-moment correlation coefficients (*r*); hence, Pearson's *r* was chosen as the effect size indicator and other effect sizes were transformed into a Pearson's *r*. We did not pool regression coefficients in our meta-analysis. Indeed, regression coefficients extracted from (multiple) regression models are not directly comparable, because studies are likely to have controlled for different co-variates.[38] We interpreted effect sizes referring to calibrated guidelines specific to psychological research that account for the cumulative influence of small effects over time, where r=0.05 is considered very small, r=0.10 is small, r=0.20 is medium, r=0.30 is large, and r>0.40 is considered a very large effect.[41] To prevent the introduction of bias in the estimation of the standard error for studies with a small sample size, we transformed Pearson's *r*s to Fisher's *z* scores for analyses;[42] we reported *z* scores in the forest plots, while values reported in the text were converted back to Pearson's *r* for ease of interpretation.

Meta-regressions were conducted to examine moderating effects if a minimum of 10 effect sizes were available.[43]

The I^2 statistic was used to assess heterogeneity and the values were interpreted in relation to identified thresholds (low=25%, moderate=50%, high=70%).[44] Because we used a three-level meta-analytic model, the heterogeneity variance was split into two parts: one quantifying the percentage of total variation associated with true effect size differences within clusters (i.e., within samples), the other attributable to true effect size differences between cluster (i.e., between samples) variations.[38,40] We conducted leave-one-out sensitivity analyses.[39] Publication bias was assessed via visual inspection of funnel plots and using a proxy for Egger's test[45] by conducting a three-level meta-analysis with each effect size's standard error as a moderator.[46]

RESULTS

We screened k=6,466 abstracts, assessed k=1,137 full-texts and included k=155 papers (see Figure 1 for PRISMA flowchart). We contacted k=103 papers' authors because effect sizes were incalculable from information in the records. Data to run a meta-analysis were unavailable for k=57 studies. Thus, the current meta-analysis included k=98 studies, derived from k=83 independent samples, yielding k=322 effect sizes, comprising 54,998 participants. A list of the records excluded from this meta-analysis, with reasons, is included in Tables S1-S3, available online.

A summary of the characteristics of the included studies is presented in Table 1. Based on the information available in the included studies, we were able to tease apart specific offspring outcomes and create four sub-groups of offspring outcomes, conducting five partially

overlapping meta-analyses examining the associations between paternal anxiety and offspring i) combined emotional and behavioral, ii) behavioral only, iii) emotional only, iv) anxiety only and v) depression only outcomes. We were unable to disaggregate different types of behavioral outcomes due to the relatively small number of studies measuring these and the heterogeneity within this subgroup. See Figure S1, available online, for a visual representation of the outcomes examined in the five meta-analyses.

A summary of quality ratings for the k=98 included studies is in Table 1.

Primary Results of the Meta-Analyses and Moderator Analyses

Meta-Analysis of the Association Between Paternal Anxiety and Combined Emotional and Behavioral Offspring Outcomes. Emotional and behavioral problems were examined in a three-level meta-analysis drawn from 83 samples (322 effect sizes, 54,998 unique participants) and were positively associated with paternal anxiety, r=0.16 (95%CI[.13,.19]; p<0.0001). I^2 was 89.92%, with estimated variance components $\tau^2_{Level\ 3}$=0.01 and $\tau^2_{Level\ 2}$=0.01, meaning that $I^2_{Level\ 3}$=48.80% of the total variation could be attributed to between-cluster, and $I^2_{Level\ 2}$=41.12% to within-cluster heterogeneity. The three-level model provided a significantly better fit compared to a two-level model with level 3 heterogeneity constrained to zero (χ^2_1=46.74; p<.0001).

The moderator analyses showed that the method of assessing offspring outcomes (i.e., diagnostic interview conducted by an expert vs questionnaires), the rater of the offspring outcomes, and the country where the study was conducted were significant moderators of the association between paternal anxiety and offspring general psychopathology (see Table S4, available online). Regarding the assessment methods, effects were significantly larger (*r*=.17) when questionnaires were used to assess offspring outcomes, and significantly smaller

(*r*=.09) when diagnostic interviews were used. Regarding the offspring outcomes informants, associations were significantly stronger for father-rated outcomes (*r*=.27), followed by parent-rated (i.e., both parents/unknown parent) outcomes (*r*=.20); associations were significantly smaller for self-reported outcomes (*r*=.15). Regarding the study location, effects were significantly stronger only for studies conducted in Australia (*r*=.21); an especially large but nonsignificant effect emerged for studies conducted in Asia (*r*=.23).

Meta-Analysis of the Association Between Paternal Anxiety and Offspring Behavioral Outcomes. Offspring behavioral problems, drawn from 27 samples (79 effect sizes and 10,958 unique participants) were significantly associated with paternal anxiety, r=0.19 (95%CI[.13,.24]; p<.0001). I^2 was 90.37%, with estimated variance components $\tau^2_{Level\ 3}$=0.01 and $\tau^2_{Level\ 2}$=0.01, meaning that $I^2_{Level\ 3}$=29% of the total variation could be attributed to between-cluster, and $I^2_{Level\ 2}$=61.37% to within-cluster heterogeneity. The three-level model did not provide a significantly better fit compared to a two-level model with level 3 heterogeneity constrained to zero (χ^2_1=1.11; p=0.29). However, 52/79 of the effect sizes were not drawn from a unique sample; hence, we retained the nested model, because it better represents the way our data were generated. [38]

The analyses of moderators revealed that the rater of offspring outcomes, the country where the study was conducted, and the year of publication of the study had a significant impact on the association between paternal anxiety and offspring behavioral outcomes (see Table S5, available online). Notably, we were unable to run meaningful moderator analyses for the method of assessment of paternal anxiety and offspring outcomes, for the father-offspring biological relatedness, and for the risk-level of the paternal sample. Regarding the informants of offspring outcomes, effects were significantly larger when outcomes were rated by the

father (*r*=.26), followed by parent-rated outcomes (*r*=.23); differences between effect sizes for father-rated and parent-rated outcomes were nonsignificant. Conversely, effects were significantly smaller when the informant of the outcomes was the mother (*r*=.12). Regarding the study location, effects were significantly stronger for studies conducted in Asia (*r*=.27), and significantly weaker for studies conducted in Canada and North America (*r*=.10). Regarding the publication year, effects sizes were found to become stronger over time.

Meta-Analysis of the Association Between Paternal Anxiety and Offspring Emotional Outcomes. Offspring emotional outcomes were drawn from 71 samples (235 effect sizes, 52,327 unique participants) and were positively associated with paternal anxiety, r=0.15 (95%CI[.12,.18]; p<.0001). I^2 was 89.90%, with estimated variance components $\tau^2_{Level\ 3}$=0.01 and $\tau^2_{Level\ 2}$=0.01, meaning that $I^2_{Level\ 3}$=47.91% of the total variation could be attributed to between-cluster, and $I^2_{Level\ 2}$=41.99% to within-cluster heterogeneity. The three-level model provided a significantly better fit compared to a two-level model with level 3 heterogeneity constrained to zero (χ^2_1=27.69; p<.0001).

The method of assessing offspring outcomes (i.e., diagnostic interview conducted by an expert vs questionnaires) and the rater of the offspring outcomes were significant moderators of the association between paternal anxiety and offspring emotional symptoms (see Table S6, available online). Regarding the assessment method, effects were significantly larger (*r*=.16) when questionnaires were used to assess offspring outcomes, and significantly smaller (*r*=.08) when diagnostic interviews were used. Regarding the informants of offspring outcomes, associations were significantly stronger for father-rated outcomes (*r*=.29), followed by parent-rated outcomes (*r*=.20); associations were significantly smaller when outcomes were self-reported (*r*=.14).

Meta-Analysis of the Association Between Paternal Anxiety and Offspring Anxiety Outcomes. Anxiety in fathers and offspring were examined in 52 samples (124 effect sizes, 47,113 unique participants) and were significantly and positively associated with a small effect size, r=0.13 (95%CI[.11,.16]; p<.0001). I^2 was 87.46%, with estimated variance components τ^2_{Level3}=0.001 and $\tau^2_{Level\ 2}$=0.015, meaning that $I^2_{Level\ 3}$=6.77% of the total variation could be attributed to between-cluster, and $I^2_{Level\ 2}$=80.68% to within-cluster heterogeneity. The three-level model did not provide a significantly better fit compared to a two-level model with level 3 heterogeneity constrained to zero (χ^2_1=0.31; p=0.58), but we retained it following the same rationale as in the meta-analysis examining offspring behavioral outcomes.[38]

The analyses of moderators revealed that the method of assessing offspring outcomes (diagnostic interview conducted by an expert vs questionnaires) and the rater of the offspring outcomes had a significant impact on the association between paternal and offspring anxiety (see Table S7, available online). Notably, there were not enough studies to test the effect of the risk-level of the paternal sample. Regarding the impact of the assessment method, effects were significantly larger (*r*=.15) when questionnaires were used to assess offspring outcomes, and significantly smaller (*r*=.06) when diagnostic interviews were used. Regarding the informants of offspring outcomes, associations were significantly stronger for father-rated outcomes (*r*=.25), followed by parent-rated outcomes (*r*=.23); associations were significantly smaller when outcomes were rated by the mother (*r*=.01).

Meta-Analysis of the Association Between Paternal Anxiety and Offspring Depression Outcomes. Offspring depression was examined in 13 samples (21 effect sizes, 4,502 unique participants) and was significantly positively associated with paternal anxiety with a small effect size, r=0.13 (95%CI[.03,.23]; p=0.01). I^2 was 88.55%, suggesting the presence of heterogeneity between studies. The estimated variance components were $\tau^2_{Level\ 3}$=0.02 and $\tau^2_{Level\ 2}$=0.01, meaning that $I^2_{Level\ 3}$=52.65% of the total variation can be attributed to between-cluster, and $I^2_{Level\ 2}$=35.9% to within-cluster heterogeneity. The three-level model did not provide a significantly better fit compared to a two-level model with level 3 heterogeneity constrained to zero (χ^2_1=0.82; p=0.37). However, due to the dependencies in our data, we kept the nested model.[38]

The analyses of moderators showed that only offspring sex had a significant impact in the association between paternal anxiety and offspring depression outcomes, as shown in Table S8, available online, with a stronger association between paternal anxiety and offspring depressive symptoms and disorders for female offspring. Notably, we could not test any categorical variables due to the small number of studies available for each subgroup.

We conducted leave-one-out sensitivity analyses for each meta-analysis. The pattern of significance remained identical, except for depression outcomes, where the lowest value was non-significant (*p*=.05). Results are reported in Table 2, together with a summary of the results of the five meta-analyses and their heterogeneity values (I^2). Table 3 provides a general overview of the results of the moderator analyses for the five meta-analyses. More details on our moderation analyses (with pairwise comparisons) are presented in Tables S4-S8, available online. Figures 2-6 show the forest plots for the five meta-analyses. Publication bias assessments are reported in Figures S2-S6 and Tables S9-S13, available online.

The PRISMA checklist is provided in Supplement 2, available online.

DISCUSSION

To our knowledge, this is the first meta-analysis examining the associations between fathers' anxiety and offspring emotional and behavioral outcomes. We found significant, positive associations between paternal anxiety and offspring emotional and behavioral problems (*r*=.16, 95%CI[.13, .19], k=322, N=54,998), behavioral only (*r*=.19, 95%CI[.13, .24], k=79, n=10,958), emotional only (*r*=.15, 95%CI[.12, .18], k=235, n=52,327), anxiety (*r*=.13, 95%CI[.11, .16], k=124, n=47,113), and depression outcomes (*r*=.13, 95%CI[.03, .23], k=21, n=4,502), with small and small-to-medium effect sizes.[41]

Our results support the importance of paternal anxiety in offspring emotional and behavioral development, and are consistent with findings from previous systematic reviews and meta-analyses examining the associations between parental psychopathology and offspring emotional[5,7,27,28,47] and behavioral[10,47] problems. The magnitude of the effects is comparable with the impact of maternal anxiety, which has been found to be linked to offspring emotional and behavioral problems with small[47] or medium effect sizes, with stronger associations when only emotional outcomes are considered.[11]

The small effect size found in our meta-analysis for anxiety outcomes is consistent with Trepiak et al.;[28] however, our results for offspring anxiety and depression outcomes are distinct from earlier meta-analyses,[5,27] where parental ADs posed greater risk for offspring anxiety, compared to depressive, disorders. Mothers were included in both,[5,27] which might account for the stronger associations for offspring anxiety compared to depression. Indeed, as noted by Lawrence et al.,[5] for those studies included in their meta-analysis where only one parent was included, it was not always possible to determine whether this was a mother or a

father. Notably, our sensitivity analyses showed that the association between paternal anxiety and depression outcomes became nonsignificant when removing the COVID-19 Pandemic Adjustment Survey (CPAS) sample.[48] It is possible that the magnitude of the effect sizes found in the CPAS sample is particularly large because of the Covid-19 pandemic, which has been found to be linked to an increased prevalence and higher severity of mental health problems.[4]

The analyses yielded similar effect sizes regardless of which offspring outcome was assessed. Thus, our findings are consistent with the principles of multifinality[26] and pleiotropy[49] in developmental psychopathology, with paternal anxiety significantly associated with both offspring emotional and behavioral outcomes, though we cannot draw causal inferences. This is a particularly key finding, considering that recent meta-analyses in the field have focused exclusively on emotional problems.[5,7,27,28] The evolutionary-rooted prominent role played by fathers in encouraging children's autonomy and in providing a way into the outside world[50] may make fathers, and their anxiety disorders or symptoms, particularly important in the development of offspring behavioral outcomes.[16]

We also found that some of the associations between paternal anxiety and offspring outcomes were moderated by study-level variables (e.g., method of assessing psychopathology in offspring and rater of offspring outcomes, country in which the study was conducted and year of study publication), as well as by offspring sex. In particular, across all the moderation analyses for which data were analysable, we found weaker associations when the assessment of offspring outcomes relied on a diagnostically-based categorical approach (the magnitude of the effect was particularly small for anxiety outcomes, r=.06), and stronger associations when the assessment relied on symptom ratings. These results are in line with previous meta-

analyses[47] and may reflect an over-estimation of self- or parent-assessed mental health problems, or an under-estimation of expert-assessed psychopathology among offspring. However, a relatively small number of studies used diagnostic interviews to assess offspring outcomes, compared to those studies using only symptom-severity measures, hence these findings should be taken with caution. It should also be noted that questionnaires, compared to diagnostic interviews, provide continuous measures that capture more variance which, in turn, translates into more statistical power to detect differences among participants.[51] This likely contributed to the relatively stronger associations found for questionnaire-assessed outcomes. Furthermore, these differences may be related to the rater of the offspring outcomes. Across all the moderation analyses for which data were analysable, we found the strongest associations when fathers rated their offspring negative outcomes. These findings are consistent with Connell and Goodman[47] and, as noted by them, may have several explanations: parents, and fathers in particular, may be more sensitive than other informants to the presence of emotional or behavioral problems in their offspring, but it is also possible that the presence of anxiety in fathers leads to biased reporting of offspring problems by fathers, or parents in general (e.g., shared rater bias, shared method variance).[47,52] However, there were too few studies to test the effects from other raters, such as teachers, and only in a minority of studies offspring outcomes were assessed by a professional. Overall, differences in the magnitude of effects across raters and methods of assessment highlight the importance of collecting information from multiple informants, adopting different methods, to reduce the problem of rater assessment bias[53] and obtain a more complete picture of offspring's functioning.[47] Effect sizes were the largest for studies conducted in Asia and in the Middle East and the smallest for studies conducted in North America; these differences may reflect an impact of cultural factors, but are also likely to evidence publication bias in countries where there has been less research; indeed, these findings should be taken with caution,

considering the much smaller number of included studies conducted in Asia compared to the ones conducted in North America. The association between paternal anxiety and offspring behavioral problems was moderated by the publication year, with stronger effect sizes over time, potentially reflecting the changing cultural expectations for fatherhood and the increasing levels of father involvement in childrearing, as well as an increasing awareness of symptoms of mental health problems in children.[22] Indeed, the growing attention towards fathers and their impact on children's development is reflected in the literature included in our review, with more studies examining paternal mental health in recent years. Offspring sex significantly moderated only the association between father anxiety and offspring depression outcomes, with stronger associations found for female offspring. These results are in line with previous studies examining the association between mother and offspring mental health,[54,55] but should be taken with caution considering that no significant moderation effect of offspring sex was found for the other offspring outcomes examined in this review.

We pre-registered our protocol and conducted a comprehensive meta-analysis adhering to the PRISMA guidelines[32] and examined the association between paternal anxiety (symptoms and diagnoses) and emotional and behavioral offspring outcomes (symptoms and diagnoses). The k=98 included studies provided some geographical diversity and fathers were drawn from both high-risk (e.g., clinical or vulnerable) and non-high-risk (i.e., community) populations, enhancing the generalisability of our findings. Moreover, we set no limits for offspring age, or publication language or time. Finally, we were able to account for the dependencies in our data fitting meta-analytic three-level models via restricted maximum likelihood procedures.

While our review reflects the current state of available literature, the samples in our meta-analyses comprise mostly White European American participants, hence the results are skewed towards the demographics of those who are more represented in the included studies.

We do not know how well these results would transfer, or generalise, to other populations. Further, in the meta-analytic models, studies with greater statistical power (i.e., larger sample sizes) were given more weight, thereby influencing the pooled effect estimate to a greater extent, leading to findings skewed towards the participant demographics within these larger, better-powered samples. This was particularly true for the depression and anxiety outcomes models, where the greatest samples were more representative of White European American groups (i.e., Hastings et al. (2021), Finsaas and Klein (2022), Flourishing Families Project, Kujawa et al. (2014, 2015) for the depression outcomes model, and Early Growth and Development Study, Flourishing Families Project, Study of Early Child Care and Youth Development for the anxiety outcomes model, respectively). The moderators we examined did not include factors such as adverse experiences, trauma, familial composition, comorbidities, and cultural variables. Most studies did not report results adjusted vs unadjusted for confounding variables, including maternal anxiety. Thus, most of the effect estimates extracted for our analyses were unadjusted for confounders (i.e., Pearson's *r* correlations). Therefore, our reported pooled effect estimates include confounding variables and we could not test the extent to which confounding by mothers' mental health was at play. Furthermore, due to the variability in the way information was reported in the primary research, we could not test some moderators we did include in our protocol (socioeconomic status, ethnicity/race and offspring temperament), despite evidence showing the importance of these factors in the intergenerational transmission of psychopathology.[56] We were unable to examine the associations between paternal anxiety and specific behavioral offspring outcomes due to the relatively small number of studies measuring behavioral outcomes and the heterogeneity within this subgroup. We accounted for statistical heterogeneity in our analyses; however, the interpretation of our meta-analytic results is limited by the high levels of heterogeneity observed across studies and samples (particularly for child anxiety

outcomes) that could not be explained by the moderator analyses. This may reflect the presence of additional confounding variables (such as other environmental stressors and shared genes) that were not accounted for in the present study, but also the substantial level of variability, particularly in the designs and assessment methods, in the existing literature. Conceptually, our findings indicate that paternal mental health is a pivotal factor for offspring mental health and demonstrate that the offspring of fathers with anxiety symptoms/disorders are at increased risk of negative emotional and behavioral outcomes, in line with the principles of multifinality[26] and pleiotropy.[49] Our results also highlight that the methods used to design studies, collect and analyze data matter, pointing to the need of triangulation across methods and approaches. Nevertheless, this study shows that presently many racial and ethnic groups are under-represented and thereby not well served by family mental health research; thus, more research is needed before we can generalise findings across populations globally. Our study assumes heteronormative families and it is key that future research differentiates between sex and gender of parents and offspring. To draw more conclusive results, more studies examining the associations between paternal anxiety and offspring emotional and behavioral problems are needed. The present study did not allow the comparison of maternal and paternal impact on offspring outcomes, but it is important that future research includes both mothers and fathers, to be able to tease apart their relative contributions.

Clinically, our findings underline the importance of considering offspring mental health when working with fathers with ADs and, at the same time, highlight the need to account for fathers' ADs when working with children with emotional and/or behavioral problems and when planning the implementation of intervention or prevention strategies. This is especially important in relation to the body of literature that assigns fathers a unique role in promoting their children's independence and transition to the outside world, which is likely to be negatively impacted if the father suffers from anxiety.[16,50] Because emotional and behavioral

problems are likely to co-occur in offspring, we argue that the findings relating to offspring general psychopathology are the most relevant for clinicians.

The overarching goal of this study was to provide a comprehensive estimate of the magnitude of the association between paternal anxiety and offspring emotional and behavioral problems. We tested the association between fathers' anxiety and offspring general psychopathology, as well as individual behavioral, emotional, anxiety and depression outcomes. Overall, our findings indicate that paternal anxiety is positively associated with a generalised vulnerability to psychopathology, in line with principles of multifinality[26] and pleiotropy.[49] However, the generalisability of the findings is challenged by the substantial heterogeneity among studies that was not explained by the tested moderators and by the sample that comprises mostly White European American groups.

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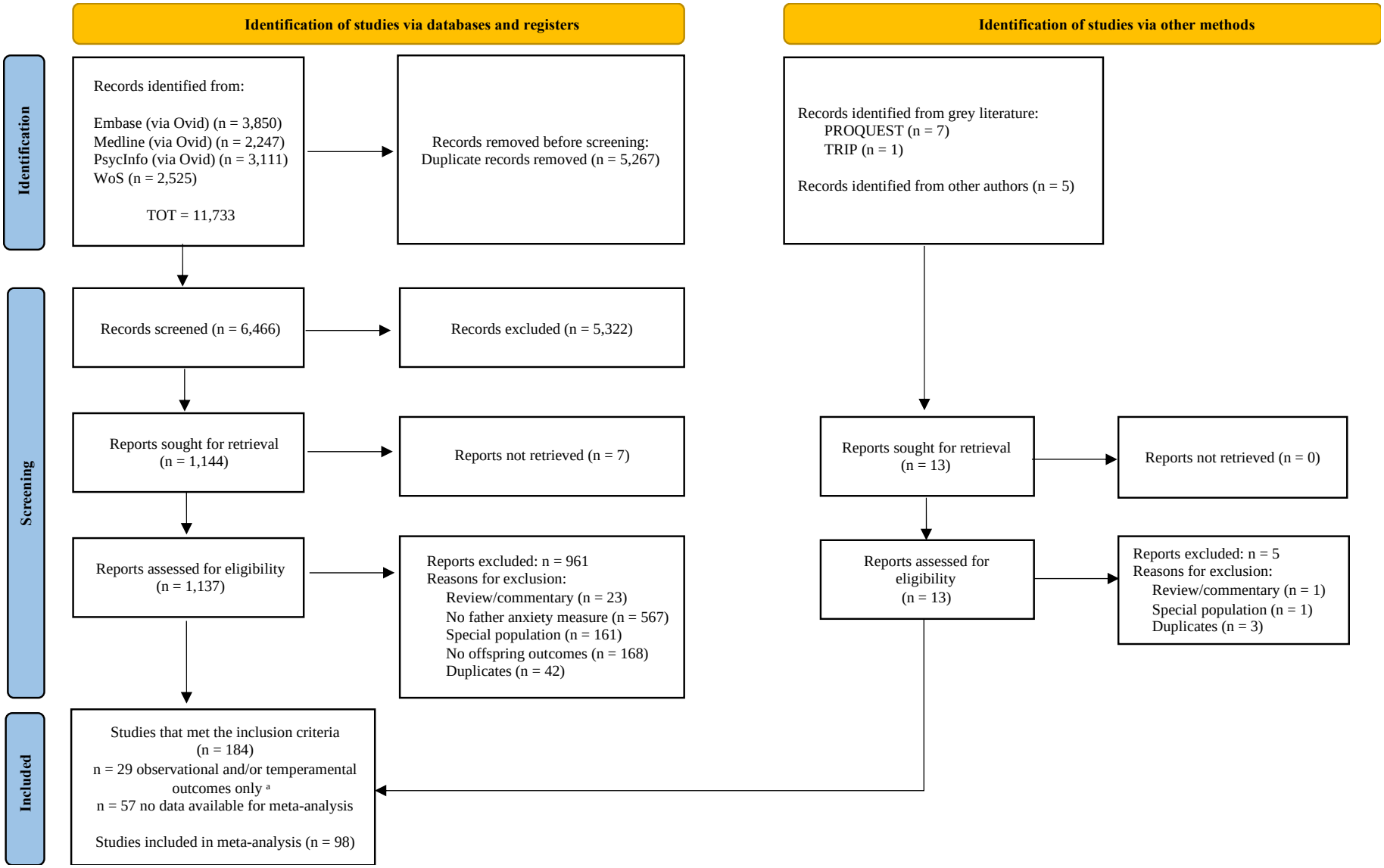


Figure 1: PRISMA Flow Diagram

Note: WoS = Web of Science.

[a] Not included in the present report.

Table 1: Summary of the Characteristics of the Studies Included in the Meta-Analyses

Sample	Study (year)	N	Reported Racial or Ethnic Identities, or Nationalities (%) [a]	Study location	Risk level of father sample [b]	Offspring sex/gender (% females)	Offspring age at assessment (months)	Method of paternal anxiety assessment [c]	Offspring outcome(s) assessed [d]	Method of outcome(s) assessment (measure used) [c]	Outcome rater(s) [e]	Father-offspring biological relatedness	Quality assessment [f]
Adams and Sarason (1963)	Adams and Sarason (1963)[47] [g]	132	Not reported	North America	Non-high risk	45.45	210	Sx	Anx	Sx (TAS, NAS, LPS, MAS)	0	not stated	0.43
EGDS	Ahmadzadeh et al. (2019)[8]	263	Parents: Caucasian (>90)	North America	Non-high risk	-	72; 84; 96	Sx	Anx	Sx (CBCL)	1; 2	no	0.75
	Chen et al. (2020)[48]	561	Fathers: African American (4-5); Caucasian (90-92); Latino (2); multiracial (1); other or unknown or not reported (2)	North America	Non-high risk	43	54; 132	Sx	Ep	Sx (CBCL)	3	no	0.75
	Natsuaki et al. (2013)[49]	269	Parents: Caucasian (>90)	North America	Non-high risk	43	18; 27	Sx	Anx	Sx (CBCL)	1; 2	no	0.88
Aktar et al. (2019)	Aktar et al. (2019)[50]	89	Fathers: Dutch (96.59)	Europe	Non-high risk	52.81	54; 90.24	Sx	Anx	Dx, Sx (ADIS-C, SCARED)	3	not stated	0.75
Alsmeier and Schulz (2020)	Alsmeier and Schulz (2020)[51] [h]	74	Fathers: German (100)	Europe	Non-high risk	-	174	Sx	Ep, Bp	Sx (CBCL)	0; 1; 2	not stated	0.75
Flourishing Families Project	Apsley and Padilla-Walker (2020)[52]	338	Families: African American (11); Asian American (<1); European American (60); combination of two or more ethnicities among family members (18); Hispanic (<1)	North America	Non-high risk	52	171.84; 219.84	Sx	Anx, Dep	Sx (SCAI, CESD)	0	not stated	0.75
	Gibbons (2021)[53]	457	Families: African American (11.52); European American (64.78); other or multi-ethnic (19.78)	North America	Non-high risk	51.89	159.84; 171.48; 183.48; 192; 204	Sx	Anx	Sx (SCAI)	0	not stated	0.75
SECCYD	Bailey and Marker (2021)[54]	707	Fathers: African American (14); American Indian (0.4); Asian/Pacific Islander (1.9); European American	North America	Non-high risk	48.30	72; 96; 120; 132; 180	Sx	Ep, Bp	Sx (CBCL)	1; 2	not stated	0.75

			(81.5); other (2.1)										
	Keizer (2012)[55]	724	Offspring: Asian/Pacific Islander (<2); Black/African American (13); White (80); other (5)	North America	Non-high risk	64.29	144; 180	Sx	Anx	Sx (CBCL)	3	not stated	0.75
	Marker and Bailey (2022)[56]	773	Fathers: African American (14); American Indian (0.4); Asian/Pacific Islander (1.9); European American (81.5); other (2.1)	North America	Non-high risk	48.30	72	Sx	Ep, Bp	Sx (CBCL)	3	not stated	0.75
	Mathews (2021)[57]	635	not reported	North America	Non-high risk	100	100.56; 111.36; 121.44	Sx	Anx	Sx (CBCL)	1	not stated	0.75
	Parrigon and Kerns (2016)[58]	661	Offspring: Caucasian (90.8)	North America	Non-high risk	49.18	72; 180	Sx	Anx	Sx (CBCL, YSR)	0; 2	majority yes	0.75
	Partain et al. (2022)[59]	554	Offspring: African American (10.6); Asian or Pacific Islander (1.3); Caucasian (83.4); other (4.7). Hispanic ethnicity (6.3)	North America	Non-high risk	49.40	85.44	Sx	Bp	Sx (CBCL, TRF)	5	not stated	0.75
	Ranney et al. (2021)[60]	674	Offspring: African American (11.8); Asian or Pacific Islander (1.4); White (81.7); other (4.9); not reported (0.2)	North America	Non-high risk	50.11	84; 180	Sx	Ep	Sx (YSR)	0	majority yes	0.75
Bogels et al. (2011)	Bogels et al. (2011)[61]	99	Fathers: Dutch (92)	Europe	Non-high risk	56.94	124.80	Sx	Anx	Sx (SPAI-C)	0	not stated	0.43
Bogels and van Melick (2004)	Bogels and van Melick (2004)[62]	75	not reported	Europe	Non-high risk	46.67	123.60	Sx	Anx	Sx (SCARED)	0; 1; 2; 5	yes	0.57
Borelli et al. (2015)	Borelli et al. (2015)[63]	102	Offspring: African-American/Black (18.6); Asian/Pacific Islander (7.8);	North America	Non-high risk	40.20	119.40	Sx	Anx	Sx (STAIC trait)	0	not stated	0.75

			Caucasian/White (53.9); other or mixed (19.5). Hispanic/Latino (37.3); Non-Hispanic (62.7)										
3D pregnancy cohort	Caccese et al. (2020)[64]	58	not reported	Canada	Non-high risk	40.98	79.89	Sx	Bp	Sx (SDQ)	3	yes	0.75
ALSPAC	Capron et al. (2015)[30]	3181	Partners (fathers): White (98); other than White (2)	Europe	Non-high risk	56.20	216	Sx	Anx	Dx (CIS-R)	0	not stated	0.71
	van Batenburg-Eddes et al. (2013)[65]	3442	Partners (fathers): Caucasian (99)	Europe	Non-high risk	48	48	Sx	Ep, Bp	Sx (SDQ)	1	not stated	0.71
Generation R	van Batenburg-Eddes et al. (2013)[65]	2280	Partners (fathers): Dutch or other-European (70); Cape Verdian (2); Dutch Antilles (3); Moroccan (4); Surinamese (6); Turkish (6); others (2)	Europe	Non-high risk	51	37.20	Sx	Ep, Bp	Sx (CBCL)	1	not stated	0.71
	Cents et al. (2011)[66]	687	Families: Dutch (100)	Europe	Non-high risk	51.40	36.30	Dx	Ep, Bp	Sx (CBCL)	1	yes	0.88
Cimino et al. (2013, 2015)	Cimino et al. (2015)[67]	80	Fathers: Caucasian (100)	Europe	Mix	-	27.60; 61.20	Sx	Ep, Bp	Sx (CBCL)	3	yes	0.56
	Cimino et al. (2013)[68]	64	Fathers: Caucasian (100)	Europe	Mix	50	28.80; 63.60; 91.20	Sx	Ep, Bp	Sx (CBCL)	3	yes	0.56
CEDAR	Clark et al. (1997)[69]	344	not reported	North America	High-risk	-	-	Dx	Anx	Dx (K-SADS)	5	yes	0.78
	Clark et al. (2004)[70]	344	Families (high-risk): African American (30); European American (68); other (2). Families (low-risk): African American (19); European American (78); other (3)	North America	High-risk	-	-	Dx	Anx	Dx (K-SADS)	5	yes	0.78
Coric et al. (2014)	Coric et al. (2014)[71]	109	not reported	Europe	Non-high risk	49.12	132	Sx	Anx	Sx (CDAS)	0	yes	0.29
Crego et al. (2013)	Crego et al. (2013)[72]	88	Offspring: the European Union (1.5); Latin America (9.0);	Europe	Non-high risk	55.20	148.80	Sx	Anx	Sx (MDAS)	0	not stated	0.71

			Spaniards (85.2); other countries (2.7)										
Dollberg et al. (2021)	Dollberg et al. (2021)[73]	77	not reported	Middle East	Non-high risk	55.13	47.56	Sx	Ep, Bp	Sx (CBCL)	1; 2	not stated	0.57
Donnelly et al. (2011)	Donnelly et al. (2011)[74]	169	Fathers: African American (1.4); Asian American (1.4); Caucasian (51.8); Hispanic (7.2)	North America	Non-high risk	60.71	219.60	Sx	Anx, Dep, General	Sx (MAS, BDI-II, RSES)	0	majority yes	0.43
Dubois-Comtois et al. (2019)	Dubois-Comtois et al. (2019)[75]	81	Families: Caucasian (majority)	Canada	High-risk	52	51	Sx	Bp	Sx (CBCL)	3	not stated	0.57
Dyba et al. (2019)	Dyba et al. (2019)[76]	14	Parents: German (95.6)	Europe	High-risk	-	75.12	Sx	Bp	Sx (SDQ)	3	majority yes	0.44
Dyba et al. (2022)	Dyba et al. (2022)[77] [g]	15	Families: German (88.5); other (11.5)	Europe	High-risk	-	-	Sx	Bp	Sx (SDQ)	3	majority yes	0.44
Fernandez-Mendoza et al. (2014)	Fernandez-Mendoza et al. (2014)[78]	135	not reported	Europe	Non-high risk	73.33	242.40	Sx	Ep, Bp, Anx, Dep	Sx (FIRST, ISI, POMS, PSS)	0	yes	0.57
Finsaas and Klein (2022)	Finsaas and Klein (2022)[79]	510	Offspring: Asian (2.7); Black (8.2); White (88.7); Native American (0.0); other (0.0). Hispanic (12.4)	North America	Non-high risk	43.50	111.60; 152.40	Sx	Anx, Dep	Dx (K-SADS)	4	not stated	1
TOPP	Fjermestad et al. (2017)[80]	337	Families: Norwegian (majority)	Europe	Non-high risk	56.10	174	Sx	Anx	Sx (CPNI)	0; 1; 2	not stated	0.57
Fliek et al. (2017)	Fliek et al. (2017)[81]	117	Offspring: Dutch (100)	Europe	Non-high risk	48.84	114.24	Sx	Anx	Sx (SCARED)	0	majority yes	0.57
Fliek et al. (2015)	Fliek et al. (2015)[82]	97	not reported	Europe	Non-high risk	41.90	51.24	Sx	Anx	Sx (PAS)	2	majority yes	0.57
VTSABD	Foley et al. (2001)[83]	850	Parents: Caucasian (majority)	North America	Mix	-	-	Dx	Anx	Dx (CAPA-C)	4	yes	1
Forresi et al. (2020, 2021)	Forresi et al. (2020)[84]	193	Offspring: Italian (94.7)	Europe	Non-high risk	49	132	Sx	General, Bp, Ep	Sx (SDQ)	0	not stated	0.33
	Forresi et al. (2021)[85]	193	not reported	Europe	Non-high risk	47.40	135.12	Sx	Ep	Sx (PTSD-RI)	0	not stated	0.56
Freedman-Doan (1994)	Freedman-Doan (1994)[86]	97	Offspring: White (majority)	North America	Non-high risk	46.10	108; 120	Sx	Anx	Sx (*ad hoc* questions)	0	not stated	0.75
Gamliel et al. (2018)	Gamliel et al. (2018)[87]	60	Families: Israeli (100)	Middle East	Non-high risk	46.67	47.52	Sx	Ep, Bp	Sx (CBCL)	1; 2	yes	0.57
Hajal et al. (2020)	Hajal et al. (2020)[88]	104	not reported	North America	High-risk	52.90	51.56	Sx	General, Bp, Anx	Sx (SCAS, ECBI)	3	not stated	0.71
Harold et al. (2012)	Harold et al. (2012)[89]	436, 170	not reported	Europe	Non-high risk	52.90	80.64	Sx	Bp	Sx (SDQ, DSM)	1	*n* = 436 yes, *n* = 170 no	0.71

Hastings et al. (2021)	Hastings et al. (2021)[90]	220	Families: White/European American (71)	North America	Non-high risk	49.55	164.04; 328.92	Sx	Anx, Dep	Sx (STAIC-trait, DACL)	0	majority yes	0.63
Hughes and Gullone (2010a)	Hughes and Gullone (2010a)[91]	148	Fathers: Australian (74)	Australia	Non-high risk	56	188.40	Sx	Ep	Sx (RADS+RCMAS, CBCL)	0; 1; 2	majority yes	0.57
Hughes and Gullone (2010b)	Hughes and Gullone (2010b)[92]	124	Fathers: Australian (73)	Australia	Non-high risk	59.89	194.40	Sx	Ep, Anx, Dep	Sx (RADS, RCMAS, CBCL)	0; 1; 2	majority yes	0.75
OCC	Hulgaard et al. (2021)[93]	621	not reported	Europe	Non-high risk	47.40	98.40	Sx	Anx	Sx (CIAS)	0	not stated	0.75
Johnson (2012)	Johnson (2012)[94]	552	Offspring: African American (2); Hispanic (6); White (88); other (4)	North America	Non-high risk	52	142.80	Sx	Anx, Dep	Sx (YSR)	0	majority yes	0.75
Kelley et al. (2017)	Kelley et al. (2017)[95]	97	Fathers: African-American (22.68); American Indian or Alaskan Native (4.12); Asian (0); Caucasian (58.76); Hispanic or Latino (7.22); multicultural/other (7.22)	North America	High-risk	48.96	131.64	Sx	Ep	Sx (CBCL)	1; 2	not stated	0.71
Kins et al. (2013)	Kins et al. (2013)[96]	119	Families: Belgian (100)	Europe	Non-high risk	44	300	Sx	Anx	Sx (PASAS)	0	yes	0.57
Kujawa et al. (2014, 2015)	Kujawa et al. (2015)[97]	144	Offspring: Hispanic (13.2). African American (4.9); Asian (2.1); Caucasian (92.4); Native American (0.7)	North America	Non-high risk	43.10	110.04	Dx	Anx, Dep	Sx (SCARED, CDI)	3	yes	1
	Kujawa et al. (2014)[98]	407	Offspring: Hispanic (11.1). African American (7.6); Asian (2.7); Caucasian (89.7)	North America	Non-high risk	45	110.16	Dx	Anx, Dep	Dx (K-SADS)	5	yes	0.86
Lara et al. (2012)	Lara et al. (2012)[99]	183	not reported	Europe	Non-high risk	48.60	112.56	Sx	Anx	Sx (CFSS-DS)	0	not stated	0.57
Lee et al. (2021)	Lee et al. (2021)[100]	126	Parents: Black (10.9); Hispanic (10.4); White	North America	Non-high risk	-	-	Sx	Anx	Sx (CBCL)	3	not stated	0.57

			(70.9); other (7.9)										
Leve et al. (2009)	Leve et al. (2009)[101 i]	95	not reported	North America	Non-high risk	43.20	18	Sx	Bp	Sx (CBCL)	1	yes	0.71
Liu et al. (2021)	Liu et al. (2021)[102]	477	Families: Chinese (100)	Asia	Non-high risk	40.88	100.56	Sx	Anx	Sx (SCAS-P)	3	not stated	0.71
NDIT	Low et at. (2012)[103]	454	Offspring: Canadian (95.8)	Canada	Non-high risk	53	244.80	Sx	Anx	Sx, Dx (*ad hoc* question, CIDI)	0	majority yes	0.75
Ma et al. (2016)	Ma et al. (2016)[104 h]	531	not reported	Asia	Non-high risk	49.72	-	Sx	Anx	Sx (SCAS-P)	2	not stated	0.57
Mackinaw-Koons (2001)	Mackinaw-Koons (2001)[105]	97	Offspring: African-American (2); Asian (2); biracial (3); Hispanic (2); Caucasian (90)	North America	Non-high risk	67.94	176.40	Sx	Anx	Sx (RCMAS)	0; 1; 2	majority yes	0.57
Mann and Sanders (1994)	Mann and Sanders (1994)[106]	40	not reported	North America	Non-high risk	0	116.40	Sx	Ep	Sx (CPAS)	0	yes	0.71
Mazaheri et al. (2011)	Mazaheri et al. (2011)[107]	220	not reported	Middle East	Non-high risk	50	-	Sx	General, Ep	Sx (SEQ-C)	0	not stated	0.29
MUSP	McClure et al. (2001)[108]	522	Families: Aboriginal (2.1); Asian (4.3); Australian Caucasian (91.4); Maori/Islander (2.2)	Australia	High-risk	49.26	180	Dx	Anx	Dx (K-SADS)	5	majority yes	1
VETR	McCutcheon et al. (2013)[109]	488	Offspring: Caucasian (87); Non-Caucasian (13)	North America	Non-high risk	48.50	272.40	Dx	Anx	Dx (SSAGA)	4	yes	1
	Xian et al. (2010)[110]	942	Fathers: White (93.5)	North America	High-risk	50.10	256.80	Dx	Bp	Sx (FTND)	0	majority yes	0.86
Medeiros et al. (2016)	Medeiros et al. (2016)[111]	243	not reported	Europe	Non-high risk	57.20	147.24	Sx	General	Sx (KIDSCREEN-10)	0	not stated	0.71
Milgram and Toubiana (1999)	Milgram and Toubiana (1999)[112 g]	354	not reported	Middle East	Non-high risk	52.82	-	Sx	Anx	Sx (AAS)	0	not stated	0.57
Mohammadi et al. (2020)	Mohammadi et al. (2020)[113]	29541	Offspring: Iranian (100)	Middle East	Non-high risk	51.10	141.60	Dx	Anx	Dx (S-SADS)	4	yes	1
Nikolic et al. (2016)	Nikolic et al. (2016)[114]	*n* = 44 high-risk, *n* = 61 non-high risk	Parents: Caucasian (93)	Europe	High-risk and Non-high risk	50.91	53.26	Sx	Anx	Sx (PAS)	3	not stated	1
Olak et al. (2013)	Olak et al. (2013)[115]	344	not reported	Europe	Non-high risk	45.35	111.60	Sx	Anx	Sx (CFSS-DS)	0	not stated	0.57
FORBOW project	Pavlova et al. (2022)[116]	299	Offspring: White (88.4); other (African,	Canada	Mix	50.20	128.40	Dx	Anx	Dx (K-SADS, SCID-5)	4	yes	1

			Chinese, East Indian, Inuit, Mi'kmaq, Métis, multiracial, 11.6)										
Peleg et al. (2015)	Peleg et al. (2015)[117]	88	Families: Israeli Jewish (100)	Middle East	Non-high risk	46.60	158.64	Sx	Anx	Sx (SAT)	0	yes	0.57
Pemble (2006)	Pemble (2006)[118]	50	Families: African American (12); Asian (2); Caucasian (84); Native American (2)	North America	Non-high risk	56	84.24	Sx	Ep	Sx (CBCL)	1	yes	0.86
Pickersgill et al. (1999)	Pickersgill et al. (1999)[119]	27	not reported	Europe	Non-high risk	100	-	Sx	Anx	Sx (FSS)	0	not stated	0.43
Proyer and Neukom (2013)	Proyer and Neukom (2013)[120]	160	not reported	Europe	Non-high risk	49.73	97.56	Sx	Anx	Sx (PhoPhiKat-30c)	0	not stated	0.71
Raouna et al. (2021)	Raouna et al. (2021)[121]	21	Fathers: British (90.48); other (9.52)	Europe	High-risk	57.10	10.81	Sx	General	Sx (ASQ:SE-2)	3	not stated	0.50
Reitman and Asseff (2010)	Reitman and Asseff (2010)[122]	122	Fathers: African American (7); Hispanic and Asian (<7); White (86)	North America	Non-high risk	54.50	228	Sx	Anx	Sx (STAIC-trait)	0	not stated	0.57
Renk et al. (2007)	Renk et al. (2007)[123]	272	Families: African American (12.9); Asian American (0.4); Caucasian (74.6); Latino/Latina (10.7); Native American (1.1); other (0.4)	North America	Non-high risk	47.79	162.24	Sx	Ep, Bp	Sx (YSR)	0	yes	0.86
Riskind et al. (2017)	Riskind et al. (2017)[124]	286	Offspring: Caucasian (100)	Europe	Non-high risk	43.20	259.20	Sx	Anx	Sx (PSWQ, BAI)	0	yes	0.57
CONFIA-20	Romero et al. (2020)[125]	102	Parents: from Galicia, NW Spain (94.2); other Spanish regions (5.8)	Europe	Non-high risk	50.40	87.48	Sx	Ep, Bp	Sx (SDQ)	3	not stated	0.71
Schick et al. (2013)	Schick et al. (2013)[126]	51	Families: Kosovar (100)	Europe	High-risk	66.67	171.60	Sx	Ep, Anx, Dep	Sx (DIKJ, SCAS, UCLA)	0	not stated	0.71
Schreier et al. (2010)	Schreier et al. (2010)[127]	336	not reported	Europe	Non-high risk	51	150.96	Sx	Ep, Bp, Anx	Sx (SASC-R, SDQ)	0	not stated	0.71
Sfeir et al. (2021)	Sfeir et al. (2021)[128]	401	Parents: Lebanese (100)	Middle East	Non-high risk	57.10	96.24	Sx	Bp	Sx (PSQ)	3	not stated	0.43
Sica et al. (2013)	Sica et al. (2013)[129]	288	Offspring: Caucasian (100)	Europe	Non-high risk	43.20	259.20	Sx	Ep, Anx	Sx (BAI, OBQ, OCI)	0	yes	0.71

Skoranski and Lunkenheimer (2021)	Skoranski and Lunkenheimer (2021)[130]	82	Fathers: Asian (1); Black (2); Latinx (53); White (29); multiethnic (8); Native American (2); other or unknown (4)	North America	Mix	47	48	Sx	Ep, Bp	Sx (CBCL)	1	not stated	0.63
Tam et al. (2017)	Tam et al. (2017)[131]	310	Families: Chinese (100)	Asia	Non-high risk	53.30	120.72	Sx	Dep	Sx (HADS)	0	not stated	0.71
CBS	Tambs (1991)[132]	814	Families: Norwegian (100)	Europe	Non-high risk	-	-	Sx	Anx	Sx (SCL-90)	0	not stated	0.71
Tazouti et al. (2018)	Tazouti et al. (2018)[133]	167	not reported	Europe	Non-high risk	52.69	124.80	Sx	Anx	Sx (*ad hoc* measure)	0	not stated	0.71
Trumello et al. (2021)	Trumello et al. (2021)[134]	102	Families: Italian (100)	Europe	Non-high risk	41	102	Sx	Ep, Bp	Sx (SDQ)	2	not stated	0.71
Wang and Zhou (2015)	Wang and Zhou (2015)[135]	119	not reported	Asia	Non-high risk	-	-	Sx	Bp	Sx (RCBC)	1; 2	not stated	0.71
Welch (1996)	Welch (1996)[136]	98	Families: Caucasian (majority)	North America	Mix	71.51	182.40	Dx	Ep, Bp	Sx (CBCL+TRF)	5	yes	0.75
CPAS	Westrupp et al. (2021)[137]	92	Parents: Aboriginal or Torres Strait Islander (2); Australian (98)	Australia	Non-high risk	48.60	103.92	Sx	Anx, Dep	Sx (BSC, SMFQ)	3	not stated	0.71
Woodhouse et al. (2010)	Woodhouse et al. (2010)[138]	189	Families: Asian (10); Black/African American (14); Hispanic (3); White/Caucasian (73)	North America	Non-high risk	62.43	198	Sx	Dep	Sx (CDI)	0	yes	0.86
Xing et al. (2017)	Xing et al. (2017)[139]	328	Families: Chinese (100)	Asia	Non-high risk	50.60	49.32	Sx	Ep	Sx (CBQ-VSF)	1	not stated	0.71
Youn et al. (2018)	Youn et al. (2018)[140]	158	not reported	Asia	Non-high risk	46.80	183.84	Sx	Bp	Sx (SAS)	0; 3	not stated	0.29
Zhang et al. (2022)	Zhang et al. (2022)[141]	1514	Families: Chinese (100)	Asia	Non-high risk	48.40	169.20	Sx	Anx, Dep	Sx (GAD-7, PHQ-9)	0	not stated	0.71
Zhao et al. (2022)	Zhao et al. (2022)[142]	595	Families: Chinese (100)	Asia	Non-high risk	45.80	54	Sx	Bp	Sx (CSHQ)	3	not stated	0.71

Note: 3D pregnancy cohort = Design, Develop, Discover pregnancy cohort; AAS = Academic Anxiety Scale; ADIS-C = Anxiety Disorder Interview Schedule for child psychopathology; ALSPAC = Avon Longitudinal Study of Parents and Children study; ASQ:SE-2 = Ages and Stages Questionnaire: Social-Emotional, Second Edition; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory—Second Edition; BSC = Brief Spence Children's Anxiety Scale; CAPA-C = Child and Adolescent Psychiatric Assessment-Children's Version; CBCL = Child Behavior Checklist; CBQ-VSF = Child Behavior Questionnaire - Very Short Form; CBS = Central Bureau of Statistics of Norway; CDAS = Corah Dental Anxiety Questionnaire; CDI = Children's Depression Inventory; CEDAR = Center for Education and Drug Abuse Research; CESD = Center for Epidemiological Studies Depression Scale; CFSS-DS = Children's Fear Survey Schedule-Dental Subscale; CIAS = Childhood Illness Attitude scales; CIDI = Composite International Diagnostic Interview; CIS-

R = Clinical Interview Schedule Revised; CONFIA-20 = Confinement Effects on Families and Children study; CPAS = Children's Perceptual Alteration Scale; CPAS = COVID-19 Pandemic Adjustment Survey; CPNI = Coolidge Personality and Neuropsychological Inventory for Children; CSHQ = Children's Sleep Habits Questionnaire; DACL = Depression Adjective Checklist; DIKJ = Depressionsinventar fur Kinder und Jugendliche; ECBI = Eyberg Child Behavior Inventory; EGDS = Early Growth and Development Study; FIRST = Ford Insomnia Response to Stress Test; FORBOW project = Families Overcoming Risks and Building Opportunities for Well-being project; FSS = Fear Survey Schedule; FTND = Fagerström Test for Nicotine Dependence; GAD-7 = Generalized Anxiety Disorder-7; HADS = Hospital Anxiety and Depression Scale; ISI = Insomnia Severity Index; K-SADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children; LPS = Lack of Protection Scale; MAS = Manifest Anxiety Scale; MDAS = Modified Dental Anxiety Scale; MUSP = Mater-University Study of Pregnancy; NAS = Need for Achievement Scale; NDIT = Nicotine Dependence in Teens Study; OBQ = Obsessive Beliefs Questionnaire; OCC = Danish Odense Child Cohort; OCI = Obsessive Compulsive Inventory; PAS = Preschool Anxiety Scale; PASAS = Parents of Adolescents Separation Anxiety Scale; PHQ-9 = Patient Health Questionnaire-9; POMS = Depression and Anxiety scales of the Profile of Mood States; PSQ = Pediatric Sleep Questionnaire; PSS = Perceived Stress Scale; PSWQ = Penn State Worry Questionnaire; PTSD-RI = Post-traumatic Stress Disorder Reaction Index; RADS = Reynolds Adolescent Depression Scale; RCBC = Rutter's Child Behaviour Checklist; RCMAS = Revised Children's Manifest Anxiety Scale; RSES = Rosenberg Self-Esteem Scale; SAS = Smartphone Addiction Scale; SASC-R = Social Anxiety Scale for Children-Revised; SAT = Separation Anxiety Test; SCAI = Spence Child Anxiety Inventory; SCARED = Screen for Child Anxiety Related Emotional Disorders; SCAS = Spence Child Anxiety Scale; SCAS-P = Spence Children's Anxiety Scale for Parents; SCID-5 = Structured Clinical Interview for DSM-5; SCL-90 = Symptom Checklist-90; SDQ = Strengths and Difficulties Questionnaire; SECCYD = Study of Early Child Care and Youth Development; SEQ-C = Self-Efficacy Questionnaire for Children; SMFQ = Short Mood and Feelings Questionnaire; SPAI-C = Social Phobia and Anxiety Inventory for Children; SSAGA = Semi-Structured Assessment for the Genetics of Alcoholism; STAIC = State-Trait Anxiety Inventory for Children; TAS = Test Anxiety Scale; TOPP = Tracking Opportunities and Problems in Childhood and Adolescence study; TRF = Teacher's Report Form; UCLA = UCLA Posttraumatic Diagnostic Scale; VETR = Vietnam Era Twin Registry; VTSABD = Virginia Twin Study of Adolescent Behavioral Development surveys; YSR = Youth Self-Report.

[a] We tried to identify information on the Race/Ethnicity of the paternal sample, or, if unavailable, their nationality, as reported in the original research; if unavailable in the primary study, we reported information on parents, mothers, or offspring. For cohort studies, when the sociodemographic characteristics were not reported in the primary study, we tried to retrieve data from the general cohort description. The term Caucasian is reported because used in the primary research, but we recognise that it is not correct.

[b] The risk level of the paternal sample was coded as high risk for clinical or at-risk samples (eg, samples drawn from war or earthquake zones), non-high risk for community samples, mix for samples drawn from both community and at-risk populations.

[c] The method of assessment was coded as symptom-based (sx) and diagnosis-based (dx).

[d] The offspring outcomes assessed were coded as General (i.e., general negative outcomes), emotional problems (Ep), behavioral problems (Bp), anxiety outcomes (Anx), and depression outcomes (Dep). Notably, anxiety and depression outcomes were included in the meta-analysis examining emotional outcomes.

[e] The raters of offspring outcomes were coded as: self-report (0), mother (1), father (2), both parents/unknown parent (3), expert (4), other (5).

[f] The quality assessment was based on the Newcastle-Ottawa Scale (NOS) for case-control and cohort studies. The item 'Demonstration that outcome of interest was not present at start of study' in the cohort studies checklist was considered irrelevant for our purposes and excluded from the assessment. Quality scores between 0.00-1.00 were generated from total scores in the NOS. Studies were generally highly rated in terms of comparability, adequacy of the assessment time of the outcomes (for cohort studies), method of ascertainment for cases and controls (for case-control studies). However, only in a minority of studies the assessment of exposure and of the outcomes was made via secure records or structured interviews, and in many studies there was no information regarding the representativeness of the sample.

[g] Paternal anxiety in this study refers to a retrospective self-report.

[h] Quality rating based on a translation to English.

[i] This study focused on the association between birth fathers' anxiety and adopted children's outcomes, hence 100% of the offspring sample had not been environmentally exposed to paternal anxiety.

Table 2: Summary of the Meta-Analyses and Sensitivity Analyses' Results

Offspring outcomes	Effect sizes (k)	Studies (k)	Samples (k)		Pooled *r*	95% CI		*p*	I^2 %
All outcomes	322	98	83		0.16	0.13	0.19	< .0001	89.92
				Lowest [a]	0.15	0.12	0.18	.000	50.49
				Highest [a]	0.16	0.13	0.19	.000	55.68
Behavioral outcomes	79	30	27		0.19	0.13	0.24	< .0001	90.37
				Lowest [a]	0.17	0.13	0.20	.000	0.001
				Highest [a]	0.19	0.13	0.27	.000	26.53
Emotional outcomes	235	84	71		0.15	0.12	0.18	< .0001	89.9
				Lowest [a]	0.14	0.11	0.17	0.000	32.69
				Highest [a]	0.16	0.12	0.19	0.000	51.52
Anxiety outcomes	124	58	52		0.13	0.11	0.16	< .0001	87.46
				Lowest [a]	0.13	0.10	0.15	0.000	0.002
				Highest [a]	0.14	0.11	0.17	0.000	0.05
Depression outcomes	21	14	13		0.13	0.03	0.23	0.01	88.55
				Lowest [a]	0.08	0.00	0.16	0.05	0
				Highest [a]	0.14	0.03	0.24	0.01	27.33

Note: [a] The values refer to the lowest and highest effect sizes obtained in the leave-one-out sensitivity analyses. The sensitivity analyses were conducted for a meta-analysis of aggregated samples, as it is not possible to run leave-one-out analyses in three-level meta-analytic models.

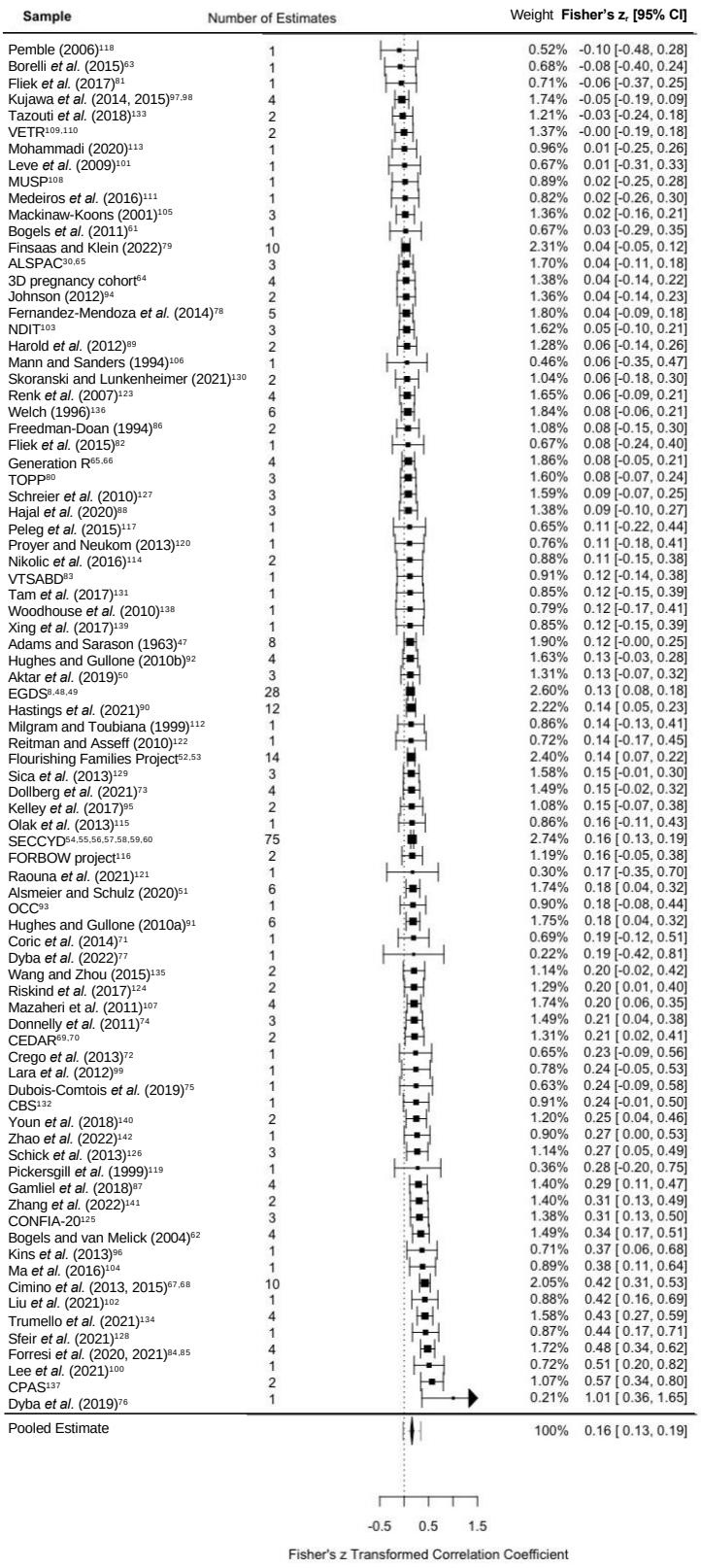
Table 3: Summary of the Results of the Moderator Analyses

	All emotional and behavioral outcomes								Behavioral outcomes								Emotional outcomes								Anxiety outcomes								Depression outcomes							
Categorical moderators																																									
	k	***df***	***r***		**95% CI**		***F***	***p***	**k**	***df***	***r***		**95% CI**		***F***	***p***	**k**	***df***	***r***		**95% CI**		***F***	***p***	**k**	***df***	***r***		**95% CI**		***F***	***p***	**k**	***df***	***r***		**95% CI**		***F***	***p***
					lb	**ub**							**lb**	**ub**							**lb**	**ub**							**lb**	**ub**							**lb**	**ub**		
Paternal anxiety assessment							3.18	0.08							-	-							3.37	0.07							1.92	0.17							-	-
Diagnostic interview	21	320	0.09	*	0.01	0.17			5								16	233	0.08		-0.01	0.16			10	122	0.08		-0.01	0.16			2							
No diagnostic interview	301	320	0.17		-0.01	0.16			74								219	233	0.16		-0.01	0.18			114	122	0.14		-0.03	0.16			19							
Offspring assessment							**4.57**	**0.03**							-	-							**4.90**	**0.03**							**7.19**	**0.01**							-	-
Diagnostic interview	24	320	0.09	*	0.01	0.16			0								24	233	0.08	*	0.00	0.15			21	122	0.06	*	0.01	0.12			3							
No diagnostic interview	298	320	0.17	*	0.01	0.16			79								211	233	0.16	*	0.01	0.16			103	122	0.15	**	0.02	0.15			18							
Offspring outcome rater							**23.58**	**< .0001**							**10.76**	**< .0001**							**24.50**	**< .0001**							**10.38**	**< .0001**							-	-
Self-report	111	316	0.15	***	0.11	0.18			9 [a]								96	221	0.14	***	0.11	0.18			64	114	0.13	***	0.10	0.17			16							
Mother	69	316	0.11		-0.08	0.01			26	63	0.12	***	0.06	0.17			43	221	0.10		-0.10	0.00			15	114	0.01	**	-0.20	-0.04			0							
Father	59	316	0.27	***	0.08	0.18			21	63	0.26	***	0.08	0.21			38	221	0.29	***	0.10	0.20			14	114	0.25	**	0.03	0.20			0							
Parents	55	316	0.20	*	0.00	0.11			19	63	0.23	*	0.02	0.20			34	221	0.20		0.00	0.12			13	114	0.23	*	0.02	0.18			2							
Expert	15	316	0.07		-0.18	0.03			0 [a]								15	221	0.06		-0.19	0.02			13	114	0.06		-0.16	0.01			2							
Other	13	316	0.13		-0.10	0.07			4 [a]								9 [a]								5 [a]								1							
Biological relatedness							0.60	0.62							-	-							0.33	0.80							0.08	0.97							-	-
Yes	63	318	0.14	***	0.09	0.19			21	75							42	231	0.15	***	0.09	0.21			20	120	0.15	***	0.08	0.21			4							
No	29	318	0.13		-0.19	0.16			1	75							28	231	0.13		-0.24	0.19			16	120	0.14		-0.14	0.14			0							
Majority yes	44	318	0.14		-0.08	0.08			3	75							40	231	0.13		-0.11	0.07			21	120	0.12		-0.12	0.08			9							
Not stated	186	318	0.17		-0.03	0.10			54	75							125	231	0.16		-0.06	0.08			67	120	0.13		-0.10	0.07			8							
Study location							**4.35**	**0.005**							**4.32**	**0.02**							2.62	0.05							0.69	0.41							-	-
Australia	13	318	0.21	***	0.09	0.32			0 [a]								13	231	0.21	***	0.09	0.33			3 [a]								2							
Asia and Middle East	27	318	0.23		-0.12	0.16			10	76	0.27	***	0.16	0.36			14	231	0.20		-0.16	0.14			6 [a]								2							
Canada and North America	200	318	0.10		-0.24	0.02			46	76	0.10	*	-0.30	-0.04			152	231	0.10		-0.24	0.02			82	113	0.12	***	0.08	0.15			15							
Europe	82	318	0.18		-0.16	0.10			23	76	0.22		-0.18	0.08			56	231	0.18		-0.16	0.10			33	113	0.14		-0.04	0.09			2							
Risk population							0.17	0.85							-	-							0.65	0.53							-	-							-	-
High-risk population	17	319	0.18	***	0.08	0.27			5								10	232	0.15	*	0.03	0.27			6								1							
Non-high risk	284	319	0.16		-0.12	0.08			65								213	232	0.15		-0.13	0.12			115								20							
Mix	21	319	0.18		-0.15	0.16			9								12	232	0.22		-0.11	0.23			3								0							
Association type							0.35	0.55							0.54	0.47							0.18	0.67							0.40	0.53							-	-
Cross-sectional	216	320	0.16	***	0.13	0.19			46	77	0.19	***	0.14	0.25			162	233	0.15	***	0.12	0.19			100	122	0.14	***	0.11	0.17			15							
Prospective	106	320	0.15		-0.04	0.02			33	77	0.17		-0.10	0.05			73	233	0.14		-0.05	0.03			24	122	0.12		-0.09	0.04			6							
Continuous moderators																																									

Intercept		305	0.11	***	0.06	0.17				74	0.17	**	0.07	0.26				224	0.11	**	0.04	0.17				114	0.08		0.00	0.15				19	0.11		-0.14	0.34		
Offspring age at assessment	307	305	0.00		0.00	0.00	2.57	0.11	76	74	0.00		0.00	0.00	0.24	0.63	226	224	0.00		0.00	0.00	1.45	0.23	116	114	0.00		0.00	0.00	1.82	0.18	21	19	0.00		0.00	0.00	0.09	0.77
Intercept		279	0.17	***	0.12	0.22				68	0.15		-0.02	0.31				201	0.17	***	0.11	0.22				103	0.16	***	0.09	0.22				19	0.01		-0.13	0.16		
Offspring sex	281	279	0.00		0.00	0.00	0.83	0.36	70	68	0.00		0.00	0.00	0.53	0.47	203	201	0.00		0.00	0.00	0.88	0.35	105	103	0.00		0.00	0.00	1.05	0.31	21	19	0.00	*	0.00	0.00	**7.12**	**0.02**
Intercept		320	-0.98		-1.00	1.00				77	-1.00	*	-1.00	-0.96				233	-0.86		-1.00	1.00				122	0.47		-1.00	1.00				19	-1.00		-1.00	1.00		
Year of publication	322	320	0.00		0.00	0.00	0.73	0.40	79	77	0.01	*	0.00	0.02	**5.09**	**0.03**	235	233	0.00		0.00	0.00	0.21	0.64	124	122	0.00		0.00	0.00	0.02	0.88	21	19	0.00		-0.02	0.03	0.18	0.67
Intercept		320	0.16	***	0.13	0.19				77	0.19	***	0.13	0.24				233	0.15	***	0.12	0.18				122	0.14	***	0.11	0.17				19	0.14	*	0.04	0.24		
Time lag	322	320	0.00		-0.01	0.01	0.04	0.84	79	77	0		-0.01	0.01	0	1.00	235	233	0.00		-0.01	0.01	0.01	0.93	124	122	0.00		-0.01	0.01	0.15	0.70	21	19	-0.02		-0.08	0.04	0.38	0.55

Note: Significant F-tests, and their p values, are reported in bold.
[a] Subgroup not included in the moderator analyses.
*p < *.05;* **p < *.01;* ***p < *.001*.

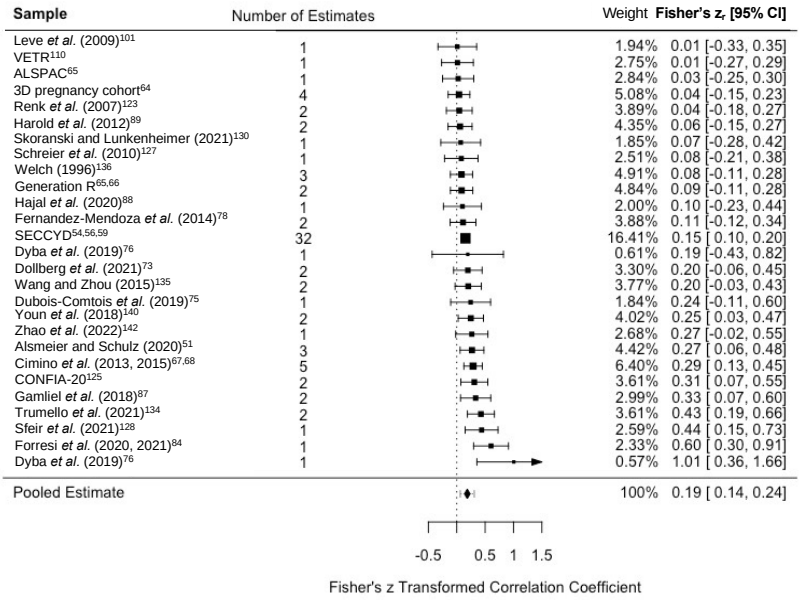
Figure 2: Forest Plot for Meta-Analysis of the Association Between Paternal Anxiety and All Offspring Emotional and Behavioral Outcomes



Note: The forest plot represents a meta-analysis of aggregated samples for readers' ease and it is an approximate representation of the three-level meta-analysis reported in the text. 3D pregnancy cohort = Design, Develop,

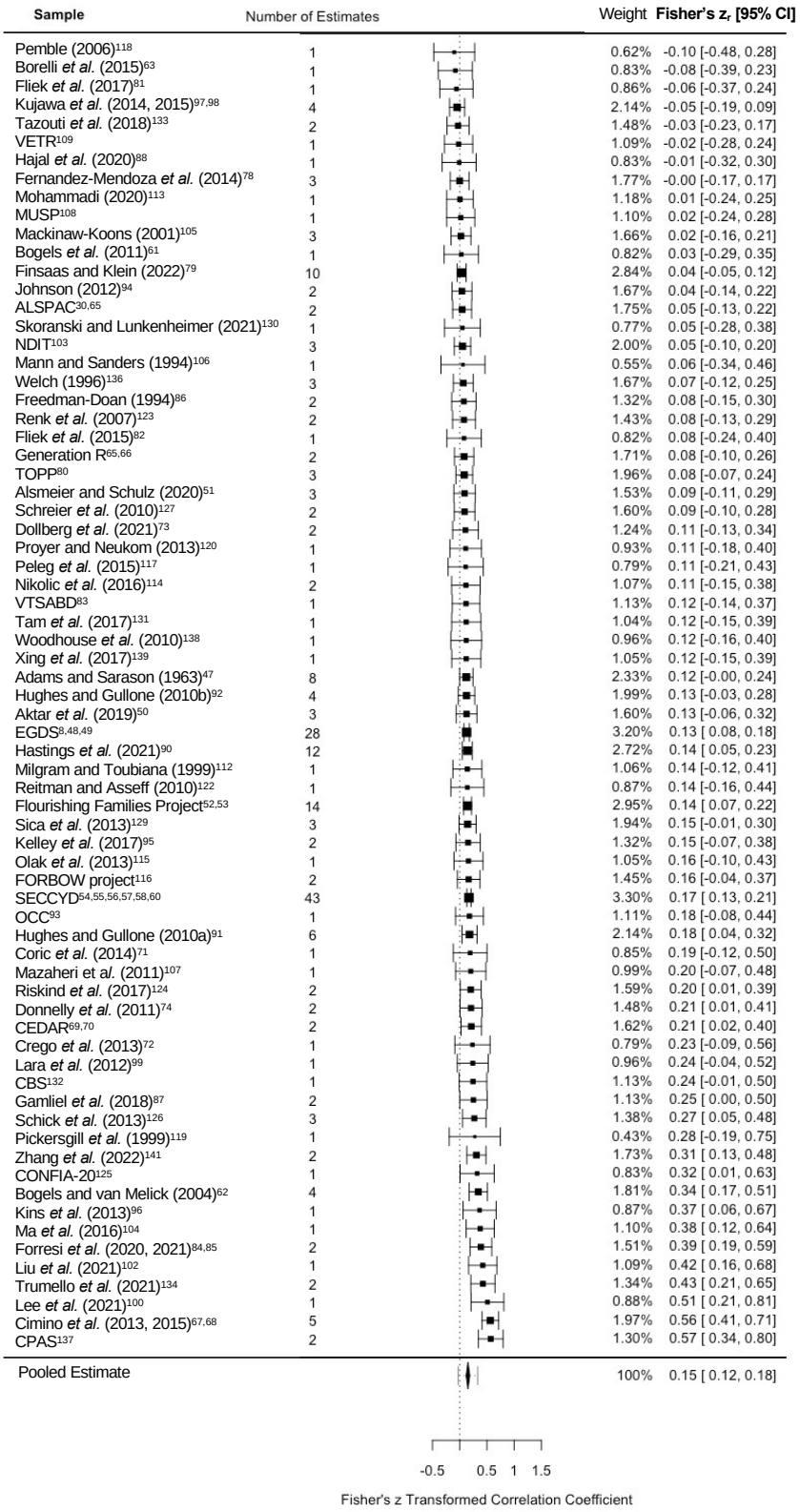
Discover pregnancy cohort; ALSPAC = Avon Longitudinal Study of Parents and Children study; CBS = Central Bureau of Statistics of Norway; CEDAR = Center for Education and Drug Abuse Research; CONFIA-20 = Confinement Effects on Families and Children study; CPAS = COVID-19 Pandemic Adjustment Survey; EGDS = Early Growth and Development Study; FORBOW project = Families Overcoming Risks and Building Opportunities for Well-being project; MUSP = Mater-University Study of Pregnancy; NDIT = Nicotine Dependence in Teens Study; OCC = Danish Odense Child Cohort; SECCYD = Study of Early Child Care and Youth Development; TOPP = Tracking Opportunities and Problems in Childhood and Adolescence study; VETR = Vietnam Era Twin Registry; VTSABD = Virginia Twin Study of Adolescent Behavioral Development surveys.

Figure 3: Forest Plot for Meta-Analysis of the Association between Paternal Anxiety and Offspring Behavioral Outcomes



Note: The forest plot represents a meta-analysis of aggregated samples for readers' ease and it is an approximate representation of the three-level meta-analysis reported in the text. 3D pregnancy cohort = Design, Develop, Discover pregnancy cohort; ALSPAC = Avon Longitudinal Study of Parents and Children study; CONFIA-20 = Confinement Effects on Families and Children study; SECCYD = Study of Early Child Care and Youth Development; VETR = Vietnam Era Twin Registry.

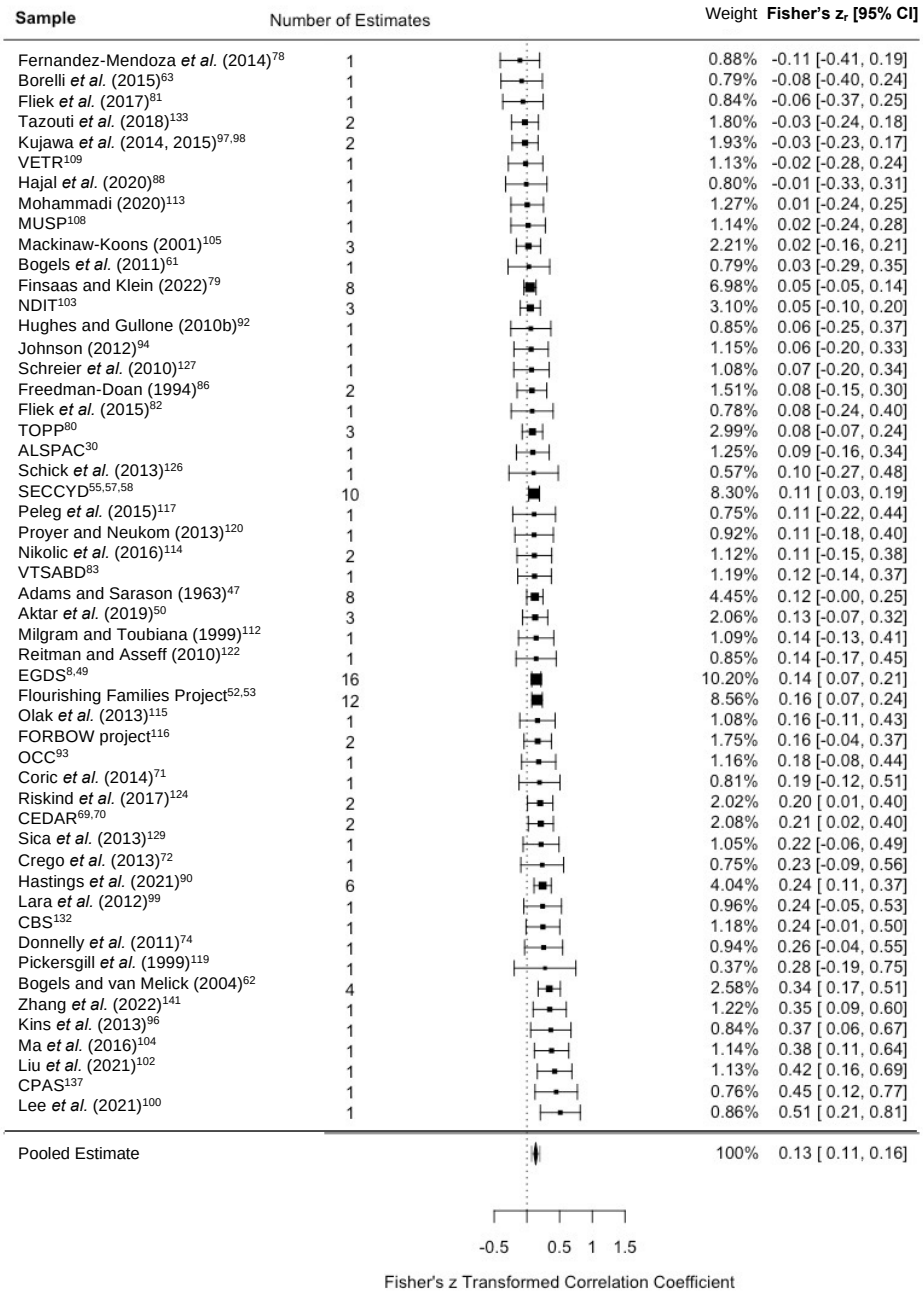
Figure 4: Forest Plot for Meta-Analysis of the Association between Paternal Anxiety and Offspring Emotional Outcomes



Note: The forest plot represents a meta-analysis of aggregated samples for readers' ease and it is an approximate representation of the three-level meta-analysis reported in the text. ALSPAC = Avon Longitudinal Study of

Parents and Children study; CBS = Central Bureau of Statistics of Norway; CEDAR = Center for Education and Drug Abuse Research; CONFIA-20 = Confinement Effects on Families and Children study; CPAS = COVID-19 Pandemic Adjustment Survey; EGDS = Early Growth and Development Study; FORBOW project = Families Overcoming Risks and Building Opportunities for Well-being project; MUSP = Mater-University Study of Pregnancy; NDIT = Nicotine Dependence in Teens Study; OCC = Danish Odense Child Cohort; SECCYD = Study of Early Child Care and Youth Development; TOPP = Tracking Opportunities and Problems in Childhood and Adolescence study; VETR = Vietnam Era Twin Registry; VTSABD = Virginia Twin Study of Adolescent Behavioral Development surveys.

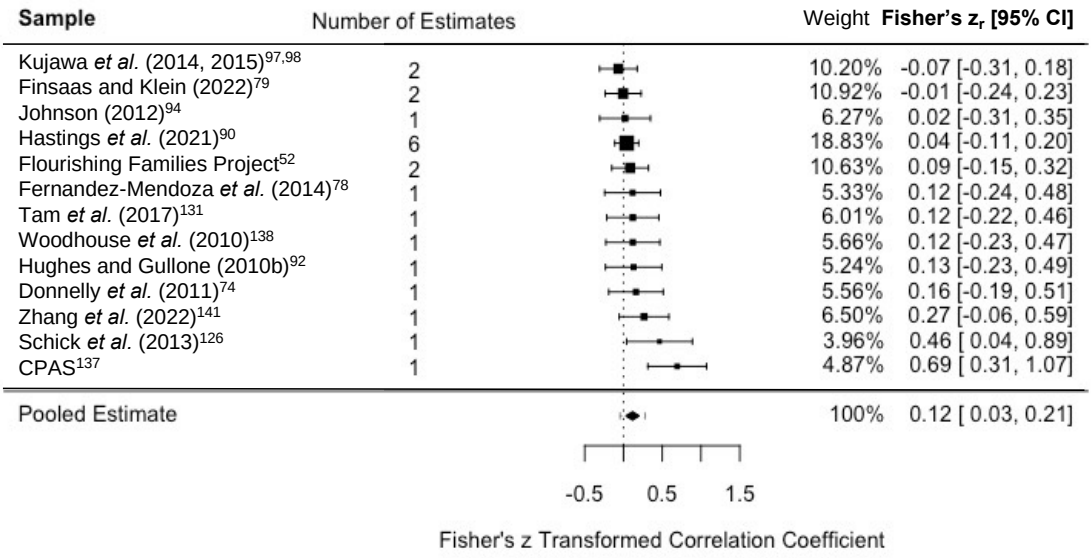
Figure 5: Forest Plot for Meta-Analysis of the Association between Paternal Anxiety and Offspring Anxiety Outcomes



Note: The forest plot represents a meta-analysis of aggregated samples for readers' ease and it is an approximate representation of the three-level meta-analysis reported in the text. ALSPAC = Avon Longitudinal Study of Parents and Children study; CBS = Central Bureau of Statistics of Norway; CEDAR = Center for Education and

Drug Abuse Research; CPAS = COVID-19 Pandemic Adjustment Survey; EGDS = Early Growth and Development Study; FORBOW project = Families Overcoming Risks and Building Opportunities for Well-being project; MUSP = Mater-University Study of Pregnancy; NDIT = Nicotine Dependence in Teens Study; OCC = Danish Odense Child Cohort; SECCYD = Study of Early Child Care and Youth Development; TOPP = Tracking Opportunities and Problems in Childhood and Adolescence study; VETR = Vietnam Era Twin Registry; VTSABD = Virginia Twin Study of Adolescent Behavioral Development surveys.

Figure 6: Forest Plot for Meta-Analysis of the Association between Paternal Anxiety and Offspring Depression Outcomes



Note: The forest plot represents a meta-analysis of aggregated samples for readers' ease and it is an approximate representation of the three-level meta-analysis reported in the text. CPAS = COVID-19 Pandemic Adjustment Survey.