Maternal and paternal influences on the developmental trajectory of childhood anxiety symptoms: a genetically sensitive investigation

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Abstract

One theory suggests that anxious fathers may pose a greater environmental influence on childhood anxiety than anxious mothers. This study uses the Early Growth and Development Study (EGDS) to test rearing parent anxiety influences from mothers and fathers on child anxiety symptoms between 18 months and 4.5, while considering inherited influences. The EGDS is a longitudinal, multisite study of adopted children recruited through US adoption agencies, and their adoptive and birth parents. Bayesian latent growth models of the trajectory of child anxiety symptoms over 3 years predicted from inherited (birth parent anxiety) and adoptive parent anxiety influences were compared for maternal and paternal measures. Parameter estimates and their HPD intervals provided evidence that the slope for anxiety symptoms between 18 and 54 months is trivially affected by both rearing parent anxiety and inherited influences from both mothers and fathers. Similarly, rearing parental anxiety and inherited influence from both mothers and fathers had only a very small effect on the intercept for growth (anxiety symptoms at 18 months old). The evidence for differences between mothers and fathers for any of these parameters was, at best, weak. Contrary to theoretical predictions, anxiety in the rearing father is unlikely to have a more important role in fostering child anxiety symptoms than that in the rearing mother.

General Scientific Summary

One theory suggests that anxious fathers may pose a greater environmental influence on childhood anxiety than anxious mothers. This study found that this theory is unlikely to be true.

Keywords: Parenting, Children, Anxiety symptoms, Adoption design

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Anxiety is the most common childhood psychological disorder (Cartwright-Hatton, McNicol, & Doubleday, 2006) affecting around 3% of children at any time (Ford, Goodman, & Meltzer, 2003) and with a lifetime prevalence of 15-20% in community samples (Beesdo, Knappe, & Pine, 2009). Anxiety is associated with serious impairments in children's academic and social functioning (Pine, 1997) and is a major risk factor for subsequent psychological problems (Kim-Cohen, et al., 2003; Kovacs, Gatsonis, Paulauskas, & Richards, 1989; Kushner, Sher, & Beitman, 1990). Childhood anxiety is also on the increase in the general population: levels of anxiety in American children increased to the extent that children in the 1980s scored higher than psychiatric patients in the 1950s (Twenge, 2000). It is no surprise that government initiatives in the UK and US emphasize early intervention and promotion of childhood emotional health (e.g., Department for Children Schools and Families, 2010; U.S. Public Health Service, 2000).

Developmental models of anxiety symptoms (i.e., attention, interpretation) suggest that much of the 'development' is likely to occur in the pre-school years. For example, Field and Lester (2010) distinguish integral bias, moderation, and acquisition trajectories for anxiety-related cognition. 'Integral bias' implies a flat trajectory in which the mechanisms underpinning anxiety-related symptoms (presumably inherited) are unaffected over time by the environment. 'Acquisition' implies a low propensity for anxiety-related symptoms that increase over time (predominantly over the first 7 years of life) as a result of (amongst other things) environmental influences. 'Moderation' also

implies environmental influences but with an initially high propensity for anxiety-related symptoms that lessens (is ‘moderated’) as a function of (amongst other things) environmental influences. In all three models, inherited influences could affect both the initial state of anxiety-related symptoms and how they change over time. Field and Lester conclude that the integral bias model is unlikely because at least some aspects of anxious symptomology show developmental change.

Field and Lester’s review suggests a moderation model is most likely for certain aspects of anxiety symptoms, which implies that the pre-school years are key to understanding how anxiety symptoms develop. Despite this conclusion, most research on the trajectory of anxiety symptoms has focused on school aged children (for a review, see Muris & Field, 2011). The models that Field and Lester present also highlight the importance of inherited and environmental influences on anxiety symptoms at this early age. The primary source of environmental influence on childhood anxiety symptoms during this time comes from parents and siblings (for a review, see Silverman & Field, 2011) because children are more physically and psychologically dependent on parents at this age (Shaw & Shelleby, 2014). Twin studies consistently demonstrate a role for shared environmental influences on child anxiety symptoms in pre-school children (Eley, et al., 2000; Eley, et al., 2003) and 8-17 year olds (e.g., Ogliari, et al., 2010; Spatola, Rende, & Battaglia, 2010; Zavos, Rijdsdijk, & Eley, 2012). These studies also show that the non-shared environment is important in shaping anxiety symptoms. However, because most of the extant literature has been conducted with biological parents raising their own offspring, it is unclear the degree to which parental behaviors escalate the inherited risk to child anxiety symptoms.

The ‘children-of-twins’ design enables genetic transmission to be separated from environmental transmission and in a recent example by Eley et al. (2015), data from 385 monozygotic and 486 dizygotic same-sex twin parents and their offspring (aged 11 to 22) were analyzed. The study showed that environmental mechanisms account for parent-child similarity but the data were equally consistent with children’s anxiety affecting parental symptoms as the reverse. One interpretation would be that parent anxiety is conducive to creating a rearing environment that fosters anxiety in the offspring, although the specifics of that environment were not explored.

Bögels and Perotti (2011) have argued that because of their typically different roles in child upbringing, mothers and fathers create rearing environments that differentially influence child anxiety symptoms. They argue (see also Möller, Majdandzic, de Vente, & Bögels, 2013) that because males and females have different reproductive strategies, males invest more in mating whereas females invest more in parenting. Humans are relatively unusual in that high paternal investment in offspring is common and, they argue, that the explanation for this lies in the complex social structures that humans have evolved. Survival depends upon the social competitiveness of the individual and Bögels and Perotti argue that it is the father who primarily develops these skills, through physical play, risk taking, overcoming obstacles, and engaging in social competition. Mothers, they believe, are more likely than fathers to foster skills in emotional bonding, empathy, and caring for others.

Bögels and Perotti also note that fathers may take more risks with their children than mothers (and therefore foster skills in overcoming challenging situations) because of the lower paternal than maternal certainty in the biological relatedness of the child, and

because of the relative ease with which fathers can reproduce. With respect to the development of child anxiety symptoms, the theory suggests that because fathers are typically more involved in the modelling of taking risk and facing challenge, they have a greater part to play in the development of child anxiety. The logic is that anxiety will inhibit fathers from encouraging their children to take risks, face challenges, or engage in social competition. Children of anxious fathers will, therefore, have underdeveloped skills to deal with threatening or novel situations, which in turn create anxiety in the child.

If the family environment is so critical to the development of anxiety symptoms in children, then Field and Lester's conclusion that the preschool years are where much of the development in anxious symptoms takes place might be explained by parents spending the most time with their children at this age. Consequently, the differential effects of mothers and fathers predicted by Bögels and Perotti's should be strongest during the pre-school years.

Bögels and Perotti's theory offers an alternative to the conventional wisdom that, because of their increased contact with their children, mothers might have a *greater* influence on child anxiety symptoms; for example, mothers spend, on average, 49.8 hours per week with their child compared to 31.4 hours for fathers (Raley, Bianchi, & Wang, 2012). Also, some of Bögels and Perotti's arguments to support a greater role for fathers in promoting anxiety symptoms plausibly support the opposite theory: for example, if mothers foster emotional skills more than fathers, then mothers will have a greater role in helping children to regulate their emotional reactions in challenging situations, and may be more likely (than fathers) to model emotional responses, such as anxiety, to novel

situations. A large comparison of maternal and paternal influences on pre-school anxiety symptoms (that considers inherited and parental anxiety influences) is needed to dissociate these opposing ideas.

Unfortunately, no such study exists: in fact, nearly all the research into parental influences on child anxiety symptoms has been conducted on mothers. The data that exist to compare maternal and paternal influences on child anxiety symptoms is, at best inconclusive with respect to Bögels and Perotti's theory (for reviews, see Bögels & Phares, 2008; Möller, et al., 2013)ⁱ.

On the one hand, several studies suggest little evidence that fathers have a more important role in child anxiety symptoms than mothers. For example, (1) autonomy granting from *both* mothers and fathers has been shown to predict some form of anxiety in 6 to 18 year old children (Lindhout, et al., 2006); (2) in social referencing experiments, reactions to potentially threatening situations in mothers and fathers did not significantly differentially affect children's (aged 8 to 13) anxious responses to those situations (Möller, Majdandzic, Vriends, & Bögels, 2014); (3) in other social referencing experiments, although mothers displayed significantly more intense encouraging facial reactions to their infant (aged 10-15 months) crossing a visual cliff than fathers, the infant's behavior was not differentially affected by whether the mother or father was present during the task (Möller, Majdandzic, & Bögels, 2014).

On the other hand, there *is* evidence that paternal, but not maternal, overinvolvement significantly predicts infant (10-15 months) anxiety (Möller, Majdandzic, & Bögels, 2015). Also, a 6-month longitudinal study of children aged 2 and 4 showed that lower levels of paternal, but not maternal, autonomy granting was

significantly associated with greater child anxiety symptoms; and fathers' challenging parental behavior predicted less subsequent social anxiety in their oldest child, whereas maternal challenging behavior was associated with more subsequent social anxiety (Majdandzic, Möller, de Vente, Bögels, & van den Boom, 2014).

To our knowledge, there are only three longitudinal studies that have looked at the relationships between both maternal and paternal anxiety symptoms/behaviors and pre-school (study age ranges 2 to 5) child anxiety symptoms (Edwards, Rapee, & Kennedy, 2010; Majdandzic, et al., 2014; McShane & Hastings, 2009). These studies showed that child anxiety symptoms were significantly predicted by specific rearing behaviors of both mothers *and* fathers. These studies used only relatively short follow ups (12 months at most), and none of them could dissociate the parental anxiety influence from the inherited influence from parents.

In summary, existing theories suggest that the preschool years might be an important developmental period for anxiety symptoms, and that mothers and fathers might have differential roles in how those symptoms develop. Specifically, one theory suggests that parental influences on child anxiety symptoms are stronger for anxious fathers than anxious mothers. The first step in testing whether paternal anxiety poses a stronger influence on child anxiety symptoms is to establish whether there are differential longitudinal associations between child anxiety symptoms and maternal and paternal anxiety symptoms. The existing research on pre-school aged children used only relatively short follow-ups (6 to 12 months) and could not dissociate the parental anxiety from the inherited influence on child anxiety symptoms.

The current study addresses these shortcomings by using data from the Early Growth and Development Study (EGDS) which uses a parent-offspring adoption design (Leve, et al., 2013). The EGDS tracks the developmental trajectory of child anxiety symptoms in the preschool years (~18 to ~54 months). Whereas children of twin designs directly estimate passive gene-environment correlations, the adoption design eliminates it because the adoptive parents provide the rearing environment but are genetically unrelated to the child. Therefore, estimates of the parental anxiety influence created by parent anxiety are not confounded by heritable characteristics of anxiety transmitted from rearing parent to child. Furthermore, the EGDS contains data from both mothers and fathers (adoptive and biological), which makes it particularly well suited to test Bögels and Perotti's theory that paternal anxiety is a greater influence on children's own anxiety symptoms than maternal anxiety.

Method

Participants

The data come from the Early Growth and Development Study (EGDS), a longitudinal, multisite study that follows 561 sets of adopted children (57.2% male), recruited through US adoption agencies, and their adoptive and birth parents. The EGDS is described in detail in Leve, et al. (2013). The EGDS sample has the following characteristics: (1) infants were placed domestically with an unrelated adoptive family; (2) placement occurred within 3 months of birth; (3) infants had no known major medical conditions (e.g., extreme prematurity or extensive medical surgeries); and (4) adoptive and birth parents could read or understand English to at least eighth-grade standard.

The focus of the current study was on the relative role of mothers and fathers on the child's anxiety symptoms across wave 1 (~4 months after birth for birth parents and 9 months for adoptive parents), 2 (18 months), 3 (27 months) and 4 (54 months). The comparison of mothers and fathers meant that children with same sex adoptive parents and single-parent adoptive families were excluded. The total remaining sample contained 502 linked adoption units (89% of the sample); this section described the EGDS sample, and for ease of readability we refer to the first parent who participated as "adoptive mother" and the second as "adoptive father," which captures over 90% of the parent genders accurately.

The ethnic composition within the EGDS sample is as follows (see Leve, et al., 2013): 70.1% of birth mothers were Caucasian (13.3% African American, 2.5% American Indian/Alaska Native, 1.8% Asian American, 0.2% Native Hawaiian/Pacific, 6.7% Hispanic or Latino, 4.9% more than 1 race, and 0.5% unknown or not reported); 69.9% of birth fathers were Caucasian (11.5% African American, 0.5% American Indian/Alaska Native, 9.6% Hispanic or Latino, 4.8% more than 1 race, and 3.3% unknown or not reported); 91.8% of the first adoptive parent (96.6% female) were Caucasian (3.9% African American, 0.2% American Indian/Alaska Native, 0.9% Asian American, 2% Hispanic or Latino, 0.9% more than 1 race, and 0.4% unknown or not reported); 90.4% of the second adoptive parent (96.6% male) were Caucasian (4.9% African American, 0.5% Asian American, 0.5% Native Hawaiian/Pacific Islander, 1.6% Hispanic or Latino, 1.1% more than 1 race, and 0.9% unknown or not reported).

With respect to educational background, 68.7% of birth mothers had completed a high school degree or lower. The same was true of 77.7% of birth fathers. In contrast,

78.4% of the primary adoptive parents and 73.3% of the secondary adoptive parents had achieved a 4-year college or university degree or graduate program qualification.

When comparing eligible families who enrolled in the study to those who declined, no significant differences were reported in education level, income, or age (see Leve, et al., 2013, for details). All infant placements occurred between 0 and 91 days from birth ($M = 6.2$ days, $SD = 12.45$, $Mdn = 2$ days).

Measures

Inherited influences on child anxiety symptoms (birth parent anxiety):

Inherited influences on child anxiety symptoms were assessed using a composite measure based on several widely used measures of adult anxiety and depressionⁱⁱ: the Beck Anxiety Inventory, BAI (Beck & Steer, 1993a), the Beck Depression Inventory, BDI (Beck & Steer, 1993b) and the Composite International Diagnostic Interview, CIDI (Andrews & Peters, 1998; Kessler & Üstün, 2004). For the CIDI, diagnosis based on DSM for both anxiety and depression correlated almost perfectly with the corresponding diagnoses based on ICD, robust correlation coefficients were .97 for birth mothers and .99 for birth fathers. Therefore, only diagnoses based on DSM were included.

Table 1 shows descriptive statistics and internal consistency for these measures for both birth mothers and fathers: the BAI and BDI were measured at around 4 and 18 months after birth, and the number of anxiety and depressive disorder diagnoses were scored with the CIDI at 18 months. BAI scores can be thought of as describing minimal (0–7), mild (8–15), moderate (16–25), and severe (26–63) anxiety. Table 1 shows that scores ranged into the severe anxiety category for both birth mothers and fathers and, on average, were higher than the population average of 6.6 ($SD = 8.1$) reported by Gillis,

Haaga and Ford (1995). The levels of anxiety in birth parents are perhaps not surprising given that (1) they are more likely to have long standing problems with psychopathology because of difficult life circumstances, and (2) childbirth and placing a child for adoption are likely to be stressful and anxiety evoking.

To see whether the 6 indicator variables could be reduced to a meaningful composite in birth mothers, parallel analysis was conducted using the *nFactors* package in R (Raiche, 2010) which yielded 1 factor using Kaiser's criterion, parallel analysis, optimal coordinates and the acceleration factor. Subsequent factor analysis yielded loadings on the factor between .46 and .81. Factor scores (regression method) were saved as a composite indicator of inherited influence from the birth mother. For birth fathers, parallel analysis similarly yielded a 1 factor solution, and factor analysis revealed loadings between .48 and .83. Factor scores were again saved as a composite indicator of inherited influence from the birth father.

In summary, inherited influences on anxiety symptoms from birth mothers and birth fathers were represented by factor scores generated from a factor analysis of BAI and BDI at both 3 and 18 months, and the number of lifetime DSM diagnoses of anxiety and depression (measured at ~18 months) from the CIDIⁱⁱⁱ. These scores were generated using the regression method and, therefore, are centred around ~0 with a standard deviation of ~1 (see, for example, Field, Miles, & Field, 2012).

Parental anxiety influences on child anxiety symptoms (adoptive parent anxiety): The influence on child anxiety of parental anxiety was assessed using a similar composite measure to that just described but using adoptive parents' responses to the BAI and BDI. Anxiety and depressive symptoms were relatively low in adoptive parents.

Table 1 shows that, in the extremes, scores ranged into the moderate to severe anxiety categories in both adoptive mothers and fathers but were, on average, lower than the aforementioned US norms (Gillis, et al., 1995). Although these measures were available at waves 1 to 3, the composite was based on waves 1 and 2 to make it comparable to the composite for inherited influence; doing so minimised the number of missing values for this measure^{iv}.

The composite was constructed in the same way as for inherited influence (described above): to see whether the 4 indicator variables could be reduced to a meaningful composite in adoptive mothers, parallel analysis was conducted which yielded 1 factor in both adoptive mothers and fathers. Subsequent factor analysis yielded loadings on the factor between .65 and .78 (adoptive mothers) and .59 and .84 (adoptive fathers). Factor scores (regression method) were saved from these models as the composite indicator of environmental influence from the adoptive mother and adoptive father respectively.

In summary, parental anxiety influences from adoptive mothers and fathers were represented by factor scores generated from a factor analysis of BAI and BDI at both 9 and 18 months. Like inherited influence, these scores are centred around ~0 with a standard deviation of ~1.

Child anxiety symptoms: The outcome variable was anxiety symptoms in the child as measured by the Child Behavior Checklist, which is a well-standardized, parent-report inventory of behavioral and emotional problem with excellent internal consistency, test-retest reliability, and validity (Achenbach & Rescorla, 2000). Items reflect behavioral and emotional scenarios to which parents respond on a three-point scale (0 = not true, 1 =

sometimes true, 2 = very true or often true). The 10 items reflecting DSM-Anxiety were totalled separately for adoptive mothers and adoptive fathers to create two scores (out of 20) representing child anxiety symptoms (one based on maternal report and the other on paternal report). The DSM-anxiety scale has a test-retest reliability of .85 over 8 days, and .60 over 12 months.

Table 2 shows that DSM anxiety scores ranged from 0 to 15 with means ranging from 1.68 to 2.81 (*SDs* from 1.59 to 2.55). These values are comparable with the US norms published in Achenbach (2013). The current sample, therefore, represents a good range of anxiety with a distribution of scores that overlaps with clinically diagnosed anxious children (although not representing the extremes of a clinical sample).

Results

All analyses were conducted using R 3.3.0 (R Core Team, 2016). Bayesian hypothesis tests of correlation were performed using the *BayesMed* package (Nuijten, Wetzels, Matzke, Dolan, & Wagenmakers, 2015) and Bayesian latent growth models were performed using the *blavaan* package (Merkle & Rosseel, 2016).

Missing values

Growth models were fitted to the full sample including missing values (see Tables 1 and 2 for the number of cases available for each variable in the model). Bayesian growth model parameters are estimated using an iterative process (Markov chain Monte Carlo, MCMC) and missing values are likewise estimated as unknown parameters (Gelman, et al., 2013). The process has two stages: an ‘imputation’ step draws missing values at iteration $t + 1$ from the probability distribution for the missing part of the data given the observed part and the posterior at iteration t ; next, the ‘posterior’ step draws

posterior values at iteration $t + 1$ from the probability distribution for posterior values given the observed data and the just-estimated values for the missing part at the same iteration (Merkle, 2011). This data augmentation approach has parallels to multiple imputation with the ‘imputation model’ being the growth model and imputations are being drawn at every iteration of the MCMC procedure; however, it benefits from using the same model for sampling missing values as is used to analyze the data (Merkle, 2011).

Descriptive statistics

The Bayesian correlations (and 95% credible intervals) between adoptive mother’s and adoptive father’s ratings of child anxiety symptoms were $r = .42$ [.34, .51], $BF_{01} = 2.44 \times 10^{17}$, posterior $p = 1$ at 18 months, $r = .51$ [.43, .60], $BF_{01} = 3.08 \times 10^{26}$, posterior $p = 1$ at 27 months, and $r = .52$ [.43, .62], $BF_{01} = 5.92 \times 10^{21}$, posterior $p = 1$ at 54 months. As such, there was overwhelming evidence for a strong relationship between adoptive mother and father ratings of child anxiety. Therefore, the average of the adoptive parent’s scores on the CBCL DSM Anxiety scale was used as the main outcome variable, which is consistent with similar work using the EGDS sample (e.g., Brooker, et al., 2014; Brooker, et al., 2015)^v.

The composite variables representing inherited and parental anxiety influences were trivially related when based on maternal data, $r = -.03$ [-.13, .07], $BF_{01} = 0.05$, posterior $p = .05$ and paternal data, $r = .10$ [-.08, .28], $BF_{01} = 0.14$, posterior $p = .12$. In both cases the Bayes factors and posterior ps strongly favor the null (i.e., no relationship) and the credible intervals suggest that assuming a relationship exists it is likely to be

small. The fact that these relationships were, at best, small suggests that evocative gene-environment mechanisms can be largely ruled out of the main model.

Environmental influence scores for adoptive mothers and fathers (which remember indicate overall internalizing symptoms) were not strongly correlated, $r = .12$ [.03, .22], $BF_{01} = 0.90$, posterior $p = .47$. The Bayes factor and posterior p suggest equal evidence for the null and alternative that a correlation exists, and estimates of the correlation suggest that if it does exist it is small. For inherited influence the correlation between birth mothers and fathers scores was also quite weak $r = .22$ [.06, .40], $BF_{01} = 2.38$, posterior $p = .70$. The Bayes factor and posterior p slightly favor the alternative hypothesis but the evidence is weak. Overall, this suggests negligible assortative mating.

Prior distributions for the model parameters

Spatola, et al. (2010) estimate the proportion of variance in CBCL anxiety accounted for by additive inherited influences (A) as being 0.57 with 95% CI limits of 0 and 0.74 from their ordinal ML model. For shared environmental influence (C), the mean was 0 with CI limits of 0 and 0.18. For inherited influences, these values convert to a change in CBCL DSM anxiety from a unit change in inherited influence between about 0 and 1.63. Our prior on inherited influence should encapsulate these values. The corresponding range of values for parental anxiety influence are 0 to 0.81.

The prior for inherited influence was, therefore, set as normal with mean of 1 and SD of 0.6. This places the bulk of our belief in inherited influence having an impact of between 0 and 2 points on the CBCL DSM anxiety scale. Bearing in mind that population data suggest the CBCL DSM anxiety scale has an SD of 2 (Kristensen, Henriksen, &

Bilenberg, 2010) our prior reflects a belief of 0 to 1 *SD* shift in scores due to inherited influence.

The prior for the influence of rearing parent anxiety was set as normal with mean of 0.5 and *SD* of 0.4. This prior places the bulk of our belief in environmental influence having an impact of between 0 and 1 point on the CBCL DSM anxiety scale (i.e., anywhere between a 0 and 0.5 *SD* shift).

In both cases the priors entertain bigger shifts than those stated, and a shift in the opposite direction (i.e., anxiety *reducing*) but only with low probability. Using the default uninformative priors from *blavaan*, rather than our subjective priors, did not change the conclusions of the analysis. For more detail see the supplementary material at <https://osf.io/zgcg2/>.

Bayesian latent growth models of maternal and paternal influences on child anxiety symptoms

A Bayesian latent growth model was fitted separately using maternal and paternal variables. The key model parameters that test the main hypothesis that paternal anxiety exerts greater influence on child anxiety symptoms than maternal anxiety, were explicitly compared between the maternal and paternal models.

Figure 1 shows the latent growth model fitted to both maternal and paternal data: child anxiety symptoms (CBCL DSM Anxiety) at 18, 27 and 54 months are endogenous observed variables predicted from latent variables representing the intercept and slope for growth (of anxiety symptoms) over time. The loadings for the paths from the slope latent variable to the three anxiety outcomes were constrained to be 0, 9 and 36 so that the intercept represents anxiety at 18 months old (rather than constraining the values to be

18, 27, 54, which would have resulted in an intercept representing anxiety symptoms at 0 months, which makes less practical sense). The indices of inherited influence and rearing parent anxiety are included as exogenous observed variables that predict the intercept and slope of growth in anxiety symptoms.

These models were fitted once for maternal variables and separately for paternal variables. In both models the outcomes were the mean of the child anxiety symptom ratings from adoptive mothers and fathers at each time points (18, 27, and 54 months after birth). In the maternal model the inherited influence and rearing parent anxiety variables were derived from birth mothers and adoptive mothers respectively whereas in the paternal model these variables derived from the birth father and adoptive father.

There are four key parameters in the model that tell us about the influence of inherited and rearing parent anxiety influence on the growth curve for child anxiety symptoms. In Figure 1, b_{IRi} , is the parameter for the effect of inherited influence on the intercept of the growth curve (i.e., child anxiety symptoms at 18 months), b_{IRs} , is the parameter for the effect of inherited influence on the slope of the growth curve (i.e., change in child anxiety symptoms over time), and b_{ERi} and b_{ERs} are the parameters for the effects of rearing parent anxiety on the intercept and slope of the growth curve respectively.

As well as looking at estimates of these key parameters we quantified the plausibility of their values being the same in the maternal and paternal model by inspecting the 95% credible intervals (in this case the Highest Posterior Density, HPD). The credible interval represents the limits within which values are more credible than values outside of the limits. In the case of a 95% credible interval, the bounds contain

95% of the posterior distribution, that is, we can believe with 95% probability that the population parameter value lies within the limits (assuming the effect exists). Unlike confidence intervals, credible intervals can be interpreted as direct probabilistic evidence for the likely population value of a model parameter^{vi}.

Table 3 shows the key parameter estimates for the maternal and paternal models. For the growth curve for anxiety symptoms, the slope parameters were 0.029 (maternal) and 0.031 (paternal), and the intercepts 1.71 (maternal) and 1.64 (paternal)^{vii}. The intercepts indicate the levels of anxiety at baseline (18 months from birth) whereas the slopes show that a unit shift in time (1 month) would lead to a 0.029/0.031 shift on the CBCL DSM anxiety subscale. This equates to about a 1-point (~half a standard deviation) increase in CBCL DSM anxiety over the three years encompassed by the model. The credible intervals for the slope suggest a 95% probability that the parameter value lies between 0.02 and 0.4 (maternal) and 0 and 0.06 (paternal), which equates to the change in anxiety from 18 to 54 months being anywhere between 0 and an increase of 2.16 points on the CBCL DSM anxiety at the extremes. In short, there were no dramatic increases.

One key aim of the study is to determine the extent to which these intercepts and slopes are influenced by inherited influence or rearing parent anxiety. There is strong evidence that the slope for anxiety symptoms is trivially affected by both rearing parent anxiety and inherited influences from both mothers and fathers. The parameter estimates are all very close to zero and have credible intervals similarly suggesting potential parameter values reflecting a trivial influence.

The effects are somewhat bigger for the intercept (that is, overall anxiety at 18 months). Figure 2 plots the expected values of the intercept (i.e. CBCL DSM anxiety at 18 months after birth) as a function of low (1 *SD* below the mean), average and high (1 *SD* above the mean) scores in inherited influence and rearing parent anxiety. The parameter estimates for the maternal model imply that inherited influence (gradients of the lines) changes baseline anxiety symptoms by ~ 0.36 points as it moves from 1 *SD* below to 1 *SD* above mean inherited influence. In the paternal model inherited influence affects baseline anxiety by only ~ 0.19 points. Rearing parent anxiety influence (distance between the lines) changes baseline anxiety symptoms by ~ 0.17 points as it moves from 1 *SD* below to 1 *SD* above mean rearing parent anxiety. The equivalent value in the paternal model is ~ 0.39 . These are all very small shifts on what is a 20-point scale (which, based on population values, has an *SD* ~ 2). The credible intervals similarly suggest that the effects of inherited influence and rearing parent anxiety on child anxiety symptoms at 18 months are unlikely to be anything other than very small.

The main aim of the study was to evaluate whether rearing parent anxiety or inherited influences are likely to differ in mothers and fathers. Figure 3 plots the credible intervals from Table 3 for the key parameters. In particular, the parameters for rearing parent anxiety are central to test the main hypothesis that fathers have a more dominant role in child anxiety symptoms because of rearing behaviors. The intervals for fathers are wider than for mothers (because they derive from less data). It is clear, however, that the maternal and paternal intervals overlap substantially. Remember that these intervals contain the population value (assuming an effect exists) with 95% probability, therefore we can gauge the plausibility of maternal and paternal parameters being the same by the

degree of overlap of their credible intervals. For the change in anxiety symptoms over time (slope), there is ~50% overlap of the intervals for both inherited influence and rearing parent anxiety suggesting – at best – as much evidence as not that the effect is the same for mothers and fathers. For anxiety at 18 months (intercept) the intervals have an overlap > 50% which provides greater evidence for the possibility that maternal and paternal effects are the same (or very similar) than for the possibility that they differ.

Discussion

To our knowledge, this is the first genetically sensitive study of the role of paternal and maternal anxiety influence on the developmental trajectory of child anxiety symptoms during the pre-school years. The key findings are that (1) the change in child anxiety symptoms over time was trivially affected by both inherited influence and rearing parent anxiety; (2) the effect of inherited influence and rearing parent anxiety on child anxiety symptoms at 18 months was, at best, very weak; and (3) there was, at best, as much evidence for as against the idea that maternal and paternal rearing influences on child anxiety symptoms differ.

There were two key theoretical questions that this research aimed to address. The primary one was that maternal and paternal rearing influences on child anxiety symptoms would differ. A secondary question was to look at the contribution of inherited and rearing parent anxiety influences on anxiety symptoms in the preschool years. On the first aim, the current study found little evidence for the Bögels and Perotti (2011) theory that anxiety in the rearing father has a stronger influence on child anxiety symptoms than anxiety in the rearing mother (after considering inherited influence). The parameter estimates for rearing parent anxiety from mothers and fathers overlapped considerably:

for both overall anxiety symptoms at 18 months and the change in anxiety over time, the credible intervals for mothers were consumed within the intervals for fathers. For overall anxiety symptoms at 18 months the maternal credible interval overlapped about 2/3rd of the paternal interval, and for the change over time the overlap was around 45-50%. This leaves little scope for maternal and paternal influences being substantively different (especially when you consider the range of plausible values encapsulated by the intervals and how they would translate to unit changes on the CBCL DSM anxiety).

As discussed earlier, contrary to the Bögels and Perotti (2011) model, there may be good reasons to assume that maternal anxiety rearing influences will be greater than paternal, but the current results lead to equally ambivalent beliefs in this idea: in the age range tested there is very little evidence for differences in anxiety influences in rearing mothers and fathers. Even if maternal and paternal effects fell at the opposite extremes of their plausible values (based on the credible intervals) this would equate to a difference of about 1 point on the CBCL DSM anxiety (about half a standard deviation based on scale norms) in anxiety at 18 months. Remember this value is an extreme though, and it is equally plausible that the difference is 0 and all values between.

One explanation is, of course, that theories proposing differences in parental anxiety influences on child anxiety are wrong. There are many studies in which rearing characteristics other than parental anxiety in *both* fathers and mothers predict child anxiety symptoms, for example: overprotection (Edwards, et al., 2010), parental control (van der Bruggen, Bögels, & van Zeilst, 2010) and negative control (Karreman, de Haas, van Tuijl, van Aken, & Dekovic, 2010). There are other explanations too, for example: (1) differential parental anxiety influences might take effect at later ages (a point to which

we will return) and so our sample missed the time window during which differential parental effects take place; (2) it is not general anxiety in the parent that matters but specific rearing practices conducive to fostering child anxiety symptoms. Put another way, our measure of parental anxiety influence was very specific and narrow, and may have been too blunt to capture differences between mothers and fathers.

The idea that the specifics of childrearing matter has some credibility given that the studies reviewed earlier that supporting the Bögels and Perotti (2011) model have tended to look at specific aspects of rearing practices (e.g., overprotection) whereas the current study looked at anxiety overall. It is noteworthy that across the reviewed literature there is little consistency in *which* aspects of maternal and paternal rearing behaviour differentially influence anxiety symptoms in the child. For example, parental over control as a predictor of child anxiety symptoms has been shown to be more important in mothers than fathers (Hudson, Comer, & Kendall, 2008), more important in fathers than mothers (van der Bruggen, Stams, Bögels, & Paulussen-Hoogeboom, 2010), and equally as important in mothers and fathers (Karreman, et al., 2010). One obvious next step is to repeat the current study looking at specific, theoretically-driven, aspects of parenting.

With respect to the secondary aim of the paper, we found little evidence for inherited influence or rearing parent anxiety on child anxiety symptoms in the preschool years. The finding that inherited influence had a trivial effect on the developmental trajectory of a child anxiety symptoms and a small effect on overall symptom levels at 18 months is consistent with recent children of twins work showing that an environment pathway, but not a genetic one, significantly predicted child anxiety symptoms (Eley, et al., 2015). Eley et al. concluded that although the lack of evidence for a genetic pathway

is surprising in light of the twin studies showing sizable heritability estimates, it is consistent with studies of childhood depression (e.g., Silberg, Maes, & Eaves, 2010). In addition, other work on the EGDS in similar age groups have found that (1) inherited influence does not significantly predict inhibition and social anxiety in 18 to 27 month olds (Natsuaki, et al., 2013); and (2) bidirectional effects of negative affect in 9 to 27 month old children on adoptive parent anxiety that seemed to be environmental and not due to birth parent negative affect (Brooker, et al., 2015). The current study further supports this position.

As Eley et al. (2015) suggest, evidence pointing to the limited inherited influence on familial transmission of anxiety supports developmental theories that assume that parents transmit anxiety through pathways such as modelling and verbal information (Creswell, Murray, Stacey, & Cooper, 2011; Field & Lester, 2010; Field & Purkis, 2011). However, the current study failed to provide strong evidence for rearing parent anxiety on either the trajectory or overall levels of child anxiety symptoms: like inherited influence, parental anxiety influences had virtually no effect on the developmental trajectory (i.e., the change over time) of anxiety symptoms, but had some (albeit weak) effect on overall levels at 18 months. At face value, this pattern of results contradicts Eley et al.'s data as well as the aforementioned theories that parents can transmit anxiety environmentally and the empirical work to support it (for a review, see Creswell, et al., 2011). However, the current study was based on a pre-school sample whereas Eley et al.'s sample was aged 11-22. One obvious explanation is that anxiety transmission through pathways such as modelling and verbal information becomes important after the preschool years as the

child develops greater linguistic and cognitive skills that help them to understand the implicational meaning of threat information.

However, twin studies have shown close to zero effects of the shared genetic environment with the non-shared environment having a much greater contribution to anxiety (Spatola, et al., 2010). Adoptions studies, such as the current one, measure shared environment, so the measures of rearing parental anxiety in the current study would align to the shared environment estimates in a twin study. In this sense, the current results are not surprising: it may not be the proximal family environment that matters so much as the broader environment (which was not measured in the current analysis).

In general, there could be any number of reasons why the current models yielded very small estimates for the inherited influence and rearing parent anxiety in child anxiety symptoms. Because our analysis focussed on estimation and not highly-problematic null hypothesis significance testing there is no issue of power here: instead we have quantified the likely size of rearing parent anxiety and inherited influence through point estimates and credible intervals. Some potential explanations for the very small rearing parent anxiety and inherited influences on the change in anxiety are: (1) the developmental trajectory of the child may already be relatively fixed by 18 months (the first time point at which child anxiety symptoms were measured in this study); (2) the developmental trajectory may not change in the time period captured by the current study; (3) the current sample was a community, not clinical, sample, so perhaps there was insufficient variability in anxiety over time to enabled us to detect moderators of that change; (4) because the current sample was a community sample, there may have been insufficient variability in birth parent to show up the effect on child anxiety symptoms; or

(5) related to the previous point, because they have undergone intense screening adoptive parents may have higher levels of mental health (and, therefore, lower variability) than the general population which would again mitigate detecting an effect on child anxiety symptoms (McGue, et al., 2007).

The first two possibilities are interesting in that they fit with developmental ‘moderation’ models, which propose that qualia of anxiety symptomology exist very early in life but are moderated as a function of later cognitive development, the socio-emotional landscape of the child and inherited influence (Field & Lester, 2010). For example, selective attentional to threat has been shown in infants as young as 5 months old (for a review, see LoBue & Rakison, 2013). In addition, attention control during toddlerhood has been shown to be a protective factor in genetically susceptible children who are raised in low-influence environments (Brooker, et al., 2014). Some moderation theories, such as Kindt’s inhibition model (Kindt & van den Hout, 2001) suggest that the moderation (or inhibition) of selective attention to threat may occur as late as 10 years old. Although support for this model is equivocal (for a review, see Nightingale, Field, & Kindt, 2010) it does raise the possibility that a three year longitudinal study during pre-school is too short to capture the critical points of change of the growth curve (and, therefore, the inherited and parental anxiety influence upon it). This would also explain the differences to Eley, et al. (2015), whose sample of offspring were considerably older (11 to 22) than ours.

The third possibility is plausible given the range of scores on the CBCL DSM Anxiety for children in the current sample. It is noteworthy, for example, that reliability statistics were quite low for the child anxiety symptoms measure at the first two waves,

which could mean that there was more measurement error in early waves than later waves, which mitigated the sensitivity of the models to detect moderators of the change in anxiety over time. Also, child anxiety symptoms scores did not meet the upper extremes of the scale even at older ages when you might expect some children to have developed extremes of anxiety.

The fourth explanation seems implausible for inherited risk because birth parents reported levels of anxiety and depression that ranged to clinical levels. However, explanation 5 has credibility because the BAI and BDI scores of adoptive parents were quite low (as you might expect in a group selected to be adoptive parents), so it is plausible that the parental anxiety risk composite had low variance and range which attenuated the parameter estimates (i.e., effect size estimates and their credible intervals). However, in general, adoption samples have been shown not to show range restriction in measures of mental health

There are other methodological limitations of study, including the sole reliance on questionnaires and parent report for assessing child and parent anxiety symptoms. Also, measures of parental anxiety influence (adoptive parent anxiety) and child anxiety symptoms came from the same informants which may have attenuated the potential relationships between them. Finally, the results may not be generalizable beyond adoption samples.

The take home message is that in the pre-school years, when controlling for inherited influences, child anxiety symptoms are relatively unaffected by parental anxiety symptoms. There is also little evidence for differential effects from mothers and fathers. These findings could suggest that by 18 months, anxiety symptoms are already in place

and unchangeable (except perhaps by non-family environment) until 4.5 years. This *could* mean that parental influence on anxiety takes place before 18 months or after 4.5 years, or that it has simply been overestimated in past theories and research.

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Table 1

Descriptive statistics for measures used to generate composite scores of inherited influences (birth parents) and parental anxiety influences (adoptive parents)

		4-9 months (Wave A)		18 months (Wave B)			
Measure		BAI	BDI	BAI	BDI	CIDI-Anx	CIDI-Dep
Birth Mother	<i>n</i>	496	491	444	445	457	457
	<i>Range</i>	0-52	0-55	0-41	0-43	0-4	0-3
	<i>Mdn</i>	8	8.42	6.5	8.42	1	0
	<i>M</i>	9.93	11.1	8.96	10.7	-	-
	α	.91	.92	.92	.91	-	-
Birth Father	<i>n</i>	169	165	147	147	164	
	<i>Range</i>	0-38	0-53	0-38	0-36	0-4	0-3
	<i>Mdn</i>	6	5	4	5	0	0
	<i>M</i>	8.19	7.40	6.84	7.52	-	-
	α	.88	.89	.89	.87	-	-
Adoptive Mother	<i>n</i>	495	495	458	457	-	-
	<i>Range</i>	0-21	0-17	0-28	0-25	-	-
	<i>Mdn</i>	3	2	3	3	-	-
	<i>M</i>	3.81	3.62	3.39	3.81	-	-
	α	.75	.71	.78	.79	-	-
Adoptive Father	<i>n</i>	477	477	446	442	-	-
	<i>Range</i>	0-19	0-27	0-23	0-22	-	-
	<i>Mdn</i>	2	2	1	2		
	<i>M</i>	3.00	2.95	2.32	2.86		
	α	.73	.75	.80	.81	-	-

CIDI = Number of anxiety (anx) and depressive (dep) disorders diagnosed (lifetime)

using the Composite International Diagnostic Interview; BAI = Beck anxiety inventory;

BDI = Beck depression inventory

Table 2

Descriptive statistics for adoptive mother and father ratings of child anxiety symptoms
(Child Behavior Checklist DSM-Anxiety)

Months from birth		18	27	54
Adoptive Mother	<i>n</i>	457	442	371
	<i>Range</i>	0-9	0-11	0-15
	<i>Mdn</i>	1	2	2
	<i>M</i>	1.74	2.05	2.81
	<i>α</i>	.45	.46	.72
Adoptive Father	<i>n</i>	442	424	342
	<i>Range</i>	0-14	0-10	0-12
	<i>Mdn</i>	1	2	2
	<i>M</i>	1.68	1.96	2.67
	<i>α</i>	.55	.55	.75

Table 3

Key model parameters for latent growth models of child anxiety symptoms (parameters excluded are the variances for the intercept, slope, and observed child anxiety symptoms at 18, 27 and 54 months)

Effect	<i>b</i> [95% HPD]	Posterior SD	<i>Prior</i>
Maternal model (<i>N</i> = 232)			
Intercept	1.713 [1.54, 1.90]	0.090	dnorm(1.6, 0.277)
Slope	0.029 [0.02, 0.04]	0.006	dnorm(1.6, 0.277)
Intercept~~Slope	-0.039 [-0.06, 0.02]	0.012	dbeta(1, 1)
Intercept~Inherited influence	0.179 [-0.03, 0.37]	0.103	dnorm(1, 2.78)
Intercept ~Env influence	0.086 [-0.11, 0.29]	0.102	dnorm(0.5, 6.25)
Slope~Inherited influence	0.000 [-0.01, 0.01]	0.007	dnorm(1, 2.78)
Slope~Env influence	0.006 [-0.01, 0.02]	0.007	dnorm(0.5, 6.25)
Paternal model (<i>N</i> = 83)			
Intercept	1.643 [1.36, 1.93]	0.146	dnorm(1.6, 0.277)
Slope	0.031 [0.00, 0.06]	0.014	dnorm(1.6, 0.277)
Intercept~~Slope	-0.020 [-0.10, 0.06]	0.040	dbeta(1, 1)
Intercept~Inherited influence	0.097 [-0.26, 0.45]	0.181	dnorm(1, 2.78)
Intercept ~Env influence	0.193 [-0.11, 0.49]	0.153	dnorm(0.5, 6.25)
Slope~Inherited influence	-0.007 [-0.04, 0.03]	0.019	dnorm(1, 2.78)
Slope~Env influence	0.011 [-0.02, 0.04]	0.016	dnorm(0.5, 6.25)

In keeping with the syntax for lavaan, ~ = predicted from, ~~ covariance, in keeping with conventions in JAGS (the engine behind the Bayesian estimation process) the second value in parentheses for the priors represents the *precision* (not the standard deviation)

Figure 1: Latent growth model of CBCL DSM-Anxiety fitted to the data.

Figure 2: Expected values of CBCL DSM Anxiety at 18 months after birth as a function of maternal and paternal inherited and parental anxiety influences.

Figure 3: Bayesian parameter estimates and 95% HPDs for parental anxiety and inherited influence on the slope and intercept of the growth curve for child anxiety symptoms.

Footnotes

ⁱ Although Bögels and Perotti's theory is specific to anxiety, work on other internalizing disorders is also inconsistent with respect to differential influence from mothers and fathers. For example, adoption studies on adolescent depression have shown that: (1) maternal but not paternal depression is a significant predictor of adolescent depression (Tully, Iacono, & McGue, 2008); (2) *both* maternal and paternal depression significantly predict adolescent depression (Marmorstein, Iacono, & McGue, 2012); and (3) paternal (but not maternal) substance use and nicotine dependence significantly predicts adolescent depression (Marmorstein, et al., 2012).

ⁱⁱ Because of the high comorbidity between anxiety and depression we decided to use both in constructing an index of inherited risk. A sensitivity analysis was conducted to compare factor scores derived from anxiety measures to those derived from both anxiety and depression measures. The factor scores based only on anxiety measures almost perfectly correlated (using a robust correlation) with the scores based on both anxiety and depression measures, $r = .93$ (biological mothers) and $.94$ (biological fathers). As such, the decision to include anxiety and depression measures will have a negligible effect on the subsequent analyses, but using more indicators should yield more stable scores.

ⁱⁱⁱ A sensitivity analysis was done in which the composite scores were created using ICD diagnostic variables instead of DSM to see whether the resulting composite would differ had we used ICD diagnoses. The robust correlation between the composite scores based on DSM and those based on ICD were 1 for both birth mothers and fathers indicating that the decision to use DSM made no difference at all to the index of inherited risk.

^{iv} A sensitivity analysis was performed to see what effect including Wave 3 BAI/BDI scores would have on the resulting index of environmental risk. This analysis was achieved by saving factor scores based on waves 1 and 2 only, or waves 1 to 3. The robust correlations between factor scores based on waves 1 to 3 and those based on only waves 1 and 2 were $r = .97$ (adoptive mothers) and $r = .96$ (adoptive fathers). For those for whom data were available at waves 1, 2 and 3 the composite scores carry almost exactly the same information regardless of whether wave 3 is included, suggesting that it is reasonable to base the composite on waves 1 and 2 to minimize missing values that would result from trying to include wave 3.

^v Using a composite of adoptive mother and father ratings of anxiety is also desirable to avoid comparisons of maternal and paternal influence being contaminated by systematic differences in parent's perceptions of their child's anxiety symptoms. For example, if fathers are more reluctant to report child anxiety than mothers then this introduces a confound in the outcome variable when comparing paternal and maternal influences.

^{vi} Frequentist confidence intervals contain the population parameter value in 95% of samples, so the probability that the interval in a given sample contains the population value is either 0 or 1 depending on whether the sample happens to be one of the 95% that contains it or one of the 5% that does not. Because you have no way of knowing which of

the two probabilities applies to your particular interval, they cannot provide evidence of the plausible similarity between two parameters.

^{vii} The CBCL variables defining the growth curve are the same in the maternal and paternal models so the estimates for the intercept and slope for growth differ in the two models only because of the differing quantities of missing values in the maternal and paternal datasets.