

Special Issue Article

Illuminating the origins of the intergenerational transmission of psychopathology with a novel genetically informed design

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Abstract

Although it is well known that parental depression is transmitted within families across generations, the etiology of this transmission remains unclear. Our goal was to develop a novel study design capable of explicitly examining the etiologic sources of intergenerational transmission. We specifically leveraged naturally occurring variations in genetic relatedness between parents and their adolescent children in the 720 families participating in the Nonshared Environment in Adolescent Development (NEAD) study, 58.5% of which included a rearing stepparent (nearly always a stepfather). Results pointed squarely to the environmental transmission of psychopathology between fathers and children. Paternal depression was associated with adolescent depression and adolescent behavior problems (i.e., antisocial behavior, headstrong behavior, and attention problems) regardless of whether or not fathers and their children were genetically related. Moreover, these associations persisted to a subset of “blended” families in which the father was biologically related to one participating child but not to the other, and appeared to be mediated via father–child conflict. Such findings are not only fully consistent with the environmental transmission of psychopathology across generations, but also add to extant evidence that parent–child conflict is a robust and at least partially environmental predictor of adolescent psychopathology.

Keywords: adolescent behavior problems; adolescent depression; environment; intergenerational transmission; parental depression

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It is now abundantly clear that psychopathology is transmitted within families across generations. Children of depressed mothers, for example, are more likely to develop depression themselves and to develop other forms of psychopathology such as ADHD, anxiety, antisocial behavior (Fisher, 2017; McAdams et al., 2015; Natsuaki et al., 2014; Pemberton et al., 2010). Genetically informed studies have generally concluded that genetic influences account for a substantial, and usually the largest, portion of this familial resemblance (Polderman et al., 2015; Sullivan et al., 2000) – so much so that this conclusion is now considered a “law” (Turkheimer, 2000).

For all the insights gained, however, it is nevertheless the case that the vast majority of genetically informed studies (including classical twin studies and most molecular genetic studies) do not actually assess intergenerational transmission *per se*. Instead, genetic influences in these studies are measured in only a single generation with the implicit assumption that, by measuring genes in any one generation, we are *de facto* assessing genetic transmission across generations. This assumption is a reasonable one in many ways – DNA is necessarily inherited from one’s parents and contributes to outcomes in both parents and their children. That said, this assumption is more problematic than it might seem, for at least two reasons. First, each parent–offspring dyad share

only 50% of their nuclear DNA. The remaining 50% of the parent’s genes have not (by definition) been transmitted to the child. Although this reduction in genetic similarity across generations is already factored into studies of intergenerational transmission, the effects of genetic influences on our lives are so far-reaching that even environmental effects can still have genetic underpinnings. Alleles not transmitted from parents to their child, for example, may still indirectly contribute to intergenerational transmission by shaping the parent’s behaviors and choices as well as the parenting they provide (Koellinger & Harden, 2018; Kong et al., 2018). And although genetically influenced, the mechanism of intergenerational transmission in those cases would necessarily be environmental, since these alleles were not transmitted to the child in question (Koellinger & Harden, 2018; Kong et al., 2018).

A second key caveat is that only additive genetic influences on psychopathology are transmitted across generations. Additive genetic influences are defined as the effect of individual genes summed over loci and act to increase familial resemblance (including between parents and their biological children) relative to the proportion of genes shared. Additive genetic influences are distinct from nonadditive genetic influences, which are defined as the effect of interactions between alleles at a given locus (or at multiple loci). Because the latter involve interactions between alleles, and because each parent provides only one of the two alleles, nonadditive genetic effects do not contribute to parent–child resemblance. This additive-only nature of intergenerational genetic transmission is rarely viewed as problematic, since additive genetic

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influences are pronounced for most forms of psychopathology (Burt, 2009).

That said, we should be cautious when assuming that all genetic effects for a given phenotype are additive, since classical twin studies are prone to underestimating nonadditivity under some scenarios. Prior simulation studies have found that, when a given phenotype is simultaneously influenced by nonadditive genetic and shared environmental influences (or influences that increase familial relatedness regardless of the proportion of genes shared), it typically serves to inflate additive genetic influences at the expense of nonadditive genetic and shared environmental influences (Keller & Medland, 2008; Keller et al., 2010). As an example, Keller & Medland (2008) simulated data in which additive genetic, nonadditive genetic, and shared environmental parameters were equal to .40, .15, and .15, respectively. Using the classical twin model, the standard ACE model (which estimates additive genetic, shared environmental, and nonshared environmental influences, respectively) fitted the simulated data better than the ADE model (which estimates additive genetic, nonadditive genetic, and nonshared environmental influences, respectively). In other words, nonadditive genetic effects were estimated to be zero. Moreover, additive genetic effects were estimated at .60, and shared environmental influences were estimated at .02 (Keller & Medland, 2008). The simultaneous presence of nonadditive genetic and shared environmental influences thus led to an overestimate of additive genetic influences and an underestimate of nonadditive genetic and shared environmental influences within the classical twin design.

In short, given all of the above issues, we would argue that, assessments of additive genetic influences in the offspring should be considered at most an indirect index of the intergenerational transmission of genetically influenced outcomes. Fortunately, other designs are thought to more directly illuminate the origins of intergenerational transmission in particular. The Nuclear Twin Family Model (NTFM), for example, directly incorporates the twins' biological parents into the model. Doing so allows affords researchers two key gains. First, they can disambiguate intergenerational environmental transmission from generation-specific environmental influences. The former specifically captures environmental influences that increase the similarity of family members across generations regardless of the proportion of genes shared (e.g., SES, family religion), whereas the latter captures those environmental influences that create similarity between twin siblings but not between parents and their children (e.g., parenting, schooling, cohort effects). Second, although the NTFM still infers genetic influences only at the level of the twin offspring, it also leverages the degree of parent-child similarity to ascertain the presence of nonadditive genetic influences with more certainty than is possible in traditional twin studies (e.g., nonadditive effects would yield high levels of similarity between monozygotic (MZ) twins but very little between dizygotic (DZ) twins or between parents and their biological children). These innovations should facilitate our understanding of intergenerational transmission.

Adoption designs can also be quite useful for understanding intergenerational transmission. In many such studies, only the adoptive parent(s) and children are assessed. In a "full" parent-offspring adoption design, however, both the adoptive and the biological parents of an adopted child are recruited and assessed (in addition to the child themselves), allowing researchers to more fully disambiguate genetic transmission from environmental transmission (Leve et al., 2019). The similarity of adopted children

to adoptive parents on a given outcome, for example, permits inferences regarding environmental transmission across generations. By contrast, the similarity of adopted children to their biological parents, with whom they have not been reared, implicates genetic transmission, with important caveats for prenatal effects.

Finally, Children-of-Twin (CoT) designs are also able to quantify and illuminate the intergenerational transmission of psychopathology (D'Onofrio et al., 2003; McAdams et al., 2015). This design is predicated on a naturally occurring shake-up in typical patterns of familial relatedness amongst the children of MZ twins. Namely, the children of a given MZ twin are as genetically related to that parent's co-twin (i.e., their aunt or uncle) as they are to their parent (e.g., they share 50% of their genes with both their mother and her genetically identical sister). This stands in contrast to the children of DZ or fraternal twins, who share an average of 25% of their genes with their parent's co-twin (just as all children of full sibling pairs do). Following this logic even farther, children who are cousins via MZ twin parents are in fact genetic half-siblings. Pairing this design with a traditional twin study, an innovation referred to as an Extended CoT design (Narusyte et al., 2008), allows for especially strong inferences regarding genetic and environmental transmission across generations.

What we know so far

These innovative designs have each been used to identify sources of the intergenerational transmission of psychopathology, with mixed results. The NTFM, for example, has provided clear evidence of both additive and nonadditive genetic influences on youth ADHD, anxiety, and physical aggression (Burt & Klump, 2012; Burt et al., 2012; Ding et al., 2021), with estimates of additive genetic variance ranging from 9% to 50% of the total variance and estimates of nonadditive genetic variance ranging from 13% to as high as 41% of the total variance. Such results suggest some intergenerational genetic transmission of psychopathology (via the additive genetic component), as well as generation-specific genetic effects (via the nonadditive genetic component; since nonadditive genetic effects do not contribute to similarity between parents and children because each parent provides only one of the two interacting alleles). What's more, these same studies have collectively found little-to-no evidence of family-wide cultural environmental influences, arguing against prominent effects of environmental transmission across generations. Instead, results have more strongly supported generation-specific environmental influences that increase sibling similarity with each other but not with their parents.

In rather striking contrast, extant adoption studies have almost exclusively supported the environmental transmission of psychopathology across generations (Natsuaki et al., 2014). Depression in adoptive parents (and particularly in adoptive mothers) has been consistently correlated with internalizing and externalizing psychopathology in their adopted children (see review by Natsuaki et al., 2014), with correlations in the .10-.20 range across multiple samples, investigative teams, and participant developmental stages (early childhood, middle childhood, and adolescence). What's more, full adoption studies (i.e., those including a biological parent as well) have only inconsistently found evidence of significant associations between the depression of biological mothers and the psychopathology of their reared-apart children (Eley et al., 1998; McAdams et al., 2015). In short, adoption studies have generally pointed to environmental transmission of depression and related conditions across generations.

The results of extant CoT studies have generally supported those of adoption studies, with some exceptions (McAdams *et al.*, 2014). McAdams *et al.* (2015), for example, examined parental depression and youth psychopathology in 876 CoT families in Sweden. They found that parent depression was moderately correlated with their child's internalizing psychopathology, and that this association was significantly larger than its corresponding MZ avuncular correlation (r was .26 for children and their parents versus .07 for children and their parent's MZ twin). What's more, the correlation between cousins in MZ families was only slightly, and not significantly, larger than that in DZ families (i.e., .16 and .10, respectively). Very similar results were reported by Silberg *et al.* (2010) in their study of the intergenerational transmission of depression in a large sample of CoT families in Virginia (Silberg *et al.*, 2010). Singh *et al.* (2011) also reported results consistent with the environmental transmission of depression across generations from a large sample of CoT families in Australia.

The CoT results were a bit murkier for adolescent externalizing psychopathology. McAdams *et al.* (2015) found that associations with parental depression were significantly larger than the MZ avuncular correlation (the parent-child correlation was .18 while the MZ avuncular correlation was .09), while correlations between cousins in MZ families were only slightly, and not significantly, larger than those in DZ families (.22 and .14, respectively). Such findings again point squarely to environmental transmission. Silberg *et al.*, also found evidence of at least partial environmental transmission. Singh *et al.*, by contrast, found no evidence of environmental transmission. In other words, CoT studies of adolescents have reported evidence both for and against environmental transmission for externalizing psychopathology.

Regardless of the somewhat murkier results for youth externalizing, the general distinction by methodology remains: NTFM studies have typically found little-to-no evidence of environmental transmission of psychopathology across generations, whereas adoption and CoT studies have found much stronger evidence of environmental transmission across generations. How do we understand these differences? We suspect that the core explanation may reflect the fact that, in the NTFM, all parents share 50% of their genes and the family environment with their twin children. There is thus no variability in their genetic or environmental relatedness across the sample, and accordingly no opportunity to leverage that variability to inform our causal inferences regarding intergenerational transmission (even as the NTFM is very useful for other sorts of etiologic questions; e.g., Burt *et al.*, 2016). This same limitation does not apply to either adoption or CoT designs, both of which cleverly disambiguate genetic and environmental relatedness across parents and children to explicitly inform our understanding of intergenerational transmission. To be sure, these designs also have limitations: the CoT design, for example, relies on the MZ avuncular relationship as a key element of its causal inferences, but does not leverage variability in the child's genetic relatedness with their rearing parent. Adoption studies overcome this additional limitation. However, adoptive parents are highly selected and thus not representative of the general population (McGue *et al.*, 2007) (although there is a great deal of variability in adoptive families, including in symptoms of depression and anxiety; Leve *et al.*, 2019). As has been noted before (Stoolmiller, 1999), this selection may undermine the strength of causal inferences in adoption studies. In sum, there are clear methodologic reasons for the different findings observed across NTFM studies on the one hand and CoT and adoption studies on the other hand.

Yet another key limitation in prior work is that simply identifying intergenerational transmission as 'environmental' is in many ways incomplete. How is it that psychopathology is transmitted via the environment – what are the psychological underpinnings of that transmission across generations? Prior phenotypic work indicates that depression damages one's significant relationships, leading to reductions in warmth and increases in conflict (Hammen & Shih, 2014; Lovejoy *et al.*, 2000). What's more, there is a parallel literature indicating that parent-child conflict and harshness robustly predicts current and future child psychopathology (Gard *et al.*, 2017; Goulter *et al.*, 2020), and does so at least in part via environmental mechanisms (Burt *et al.*, 2021). This work raises the question as to whether depression is transmitted across parents and children by increasing conflict in the parent-child relationship. Should that be the case, it would not only provide an actionable target for possible interventions in the future, but would also significantly bolster our confidence in findings of environmental transmission.

A final concern with prior literature is it tends to focus more on the intergenerational transmission of maternal psychopathology relative to paternal psychopathology (as discussed in Natsuaki *et al.*, 2014). This is likely due to a confluence of circumstances, including more limited interest in the father-child relationship (relative to the mother-child relationship) across much of the literature, and the higher proportion of mothers participating in research studies (e.g., see Burt, 2009; McAdams *et al.*, 2015). In the case of adoption studies, this is further compounded by the fact that, when a non-rearing biological parent participates in a "full" parent-offspring adoption design, that participating parent is far more likely to be the adopted child's biological mother (Leve *et al.*, 2019). Regardless, the relative absence of fathers from the research literature is problematic, as fathers and the father-child relationship are also critical relationships in a child's life (Cabrera *et al.*, 2018). Indeed, studies examining father-child relationship quality (e.g., Neiderhiser, Reiss, Lichtenstein, *et al.*, 2007; Pemberton *et al.*, 2010) suggest that warm and supportive father-child relationships are related to fewer problem behaviors, more positive peer relationships, and academic success. What's more, fathers are less likely to reside with their children for a whole host of reasons (Livingston & Parker, 2011), allowing researchers to more easily disambiguate genetic and environmental relatedness between fathers and their children.

The current study

To address the above issues, we developed a novel genetically informed design that incorporates the core interpretative feature of adoption studies (e.g., rearing by a nonbiologically related parent) but does so using a more representative sample that also includes biologically related parents as a comparison. We specifically leveraged naturally-occurring variations in genetic relatedness between parents and their adolescent children in the 720 families participating in the Nonshared Environment in Adolescent Development (NEAD) study, 58.5% of which included a rearing stepparent. Note that, because the vast majority of residential step-parents in NEAD were fathers, we focused here on father-child transmission, although we did preliminarily evaluate mother-child transmission as well for completeness sake. We specifically reasoned that, should genes common to parent and child underlie the transmission of depression from parents to their children, this association would be observed only when examining parents and their biological children, and would not persist to

parents and their stepchildren (as they are not genetically related). By contrast, should the transmission of depression across generations be environmental in origin, we would expect to observe this association in parents and their stepchildren to a similar degree as seen between parents and their biological children. What's more, we would expect this association to be mediated at least in part by parent-child conflict, which has emerged as a particularly robust risk factor for youth psychopathology (Patterson et al., 2017). Given this framework, and building on the results of extant CoT and adoption studies, we hypothesized that depression would be transmitted from fathers to their children solely via environmental mechanisms. We further expected at least some evidence of environmental transmission for youth externalizing psychopathology. Finally, we expected that associations between paternal and child psychopathology would be mediated by father-child conflict even in stepfather-child pairings.

Method

Participants

The data for this study were collected as part of the Time 1 assessment in the NEAD project. Families were sampled by means of random-digit dialing and commercial market panels with family information to identify certain family types (e.g., twin, step, and non-divorced families). Eligibility required sibling pairs to be no more than 4 years apart in age, between the ages of 10 and 18 (although one 9 year-old was included in the 10 year-old group), and reside in the household at least half-time. Stepfamilies were additionally required to have been in existence for a minimum of 5 years. Of note, families were not recruited to be representative of their prevalence in the general population. Rather, family types were sampled with the express goal of facilitating examinations of genetic and environmental influences (e.g., twin pairs were over-sampled to power the estimation of genetic influences), consistent with the original goals of NEAD. The final sample consisted of 720 families with same-sex adolescent sibling pairs (51.6% boys) from 47 states in the United States.

The non-divorced or "intact" families included 598 adolescents nested in 299 families (93 MZ twin pairs, 99 DZ twin pairs, 12 twin pairs of uncertain zygosity, and 95 full sibling pairs). All adolescents in these families were biologically related to both their maternal and paternal heads of household (i.e., their biological mother and father). The stepfamilies included 842 adolescents nested in 421 families (182 full sibling pairs, 109 half-sibling pairs, and 130 stepsibling pairs). In 181 of the 182 full sibling pairs, both adolescents were biologically unrelated to the paternal head of household (i.e., their stepfather) but were biologically related to the maternal head of household (i.e., their mother). In the one outlying full sibling pair, the adolescents were biologically unrelated to the maternal head of household but were biologically related to the paternal head of household. In 108 of the 109 half-sibling pairs, both adolescents were biologically related to the maternal head of household, but only one of the two was biologically related to the paternal head of household. In the one outlying half-sibling pair, the adolescents were both biologically related to the paternal head of household, while only one was biologically related to the maternal head of household. Among the 130 stepsibling pairs, one adolescent was biologically related to the maternal head of household but not the paternal head of household, while their stepsibling was biologically related to the paternal head of household but not the maternal head of household. In total then, the NEAD sample contained 840 adolescents (in 539 families) being reared by their

biological father, and 600 adolescents (in 421 families) being reared by their stepfather. Similarly, the sample contained 1307 adolescents (in 588 families) being reared by their biological mother, and 133 adolescents (in 132 families) being reared by their stepmother.

The mean age of the older child was 14.5 years (± 2.2), and that of the younger child was 12.9 years (± 2.2). The families reported a wide range of family incomes (median family income was \$25,000-35,000 range, and families ranged from lower working-class to upper middle-class on the Hollingshead Four Factor Indicator of socioeconomic status) and education (mean years of education = 13.6 for mothers, 14.0 for fathers). Nearly all participants (i.e., 94% of mothers, 93% of fathers, and 93% of adolescents) identified as White or European American. Adolescents gave informed assent, while parents gave informed consent for themselves and their children. Additional details on the sample, measures, and zygosity procedures are available in prior publications (Neiderhiser, Reiss, & Hetherington, 2007; Reiss et al., 2000).

Measures

Parental depression symptoms

Mothers and fathers each completed the Center for Epidemiological Studies-Depression (CES-D; Radloff, 1977) scale. The CES-D is a 20-item inventory that asks participants to rate how often they have experienced symptoms associated with depression (e.g., depressed mood, restless sleep) over the past week. Items are rated using a 4-point scale, ranging from 0 (rarely or none of time) to 3 (most or all of the time). We focused here on the total score ($\alpha = .59-.69$ for fathers and mothers).

Child psychopathology symptoms

Informant-reports of child psychopathology for each adolescent were collected from adolescents, mothers, and fathers using the 32-item Behavior Problems Index (BPI) (Zill, 1985). The BPI was adapted from the Child Behavior Checklist (Achenbach & Edelbrock, 1983) and has been extensively used on projects like the National Longitudinal Survey of Youth 1979 (<https://www.nlsinfo.org/content/cohorts/nlsy79-children/topical-guide/assessments/behavior-problems-index-bpi>). Prior work (Singh & Ghandour, 2012) has shown that BPI scales strongly correlate with parent-reported diagnoses of depression, anxiety, oppositional defiant and conduct disorder, and ADHD and with child health status and school absence (r s ranged from .54 to .92). The BPI includes scales assessing depressive symptoms (six items; $\alpha = .72-.77$; e.g., sudden changes in mood; felt sad or depressed), antisocial behavior (six items; $\alpha = .72-.78$; e.g., got into trouble at school, bully/mean to others), headstrongness (five items; $\alpha = .66-.75$; e.g., argued too much, disobedient at home), and attention problems (five items; $\alpha = .69-.74$; e.g., difficulty concentrating and paying attention, impulsive, restless). Participants were asked to rate on a 3-point scale the extent to which a series of statements generally described their behavior (or that of their child) over the last 3 months. Mother, father, and adolescent informant-reports of adolescent psychopathology were correlated .31-.58 for antisocial behavior, .16-.44 for adolescent depression, .22-.46 for headstrongness, and .15-.45 for attention problems. These correlations are largely in line with expected patterns of small-to-moderate cross-informant correlations for youth psychopathology (Achenbach et al., 1987) and are thought to reflect the fact that different informants are exposed to different slices of the child's behavior and thus develop different opinions/attributions

regarding the same child (De Los Reyes & Kazdin, 2005). To capture this multi-informant perspective of the child's psychopathology and to maintain the approach taken in prior NEAD studies (Reiss *et al.*, 2000), all informant-reports were averaged prior to analysis.

Adolescent depression was also assessed using the Child Depression Inventory (CDI), a 27-item questionnaire assessing symptoms of depression in children aged 8–17 years (including disturbances in mood, hedonic capacity, and vegetative functions). Each item was rated on a 3-point scale (0–2) and assessed symptomatology over the preceding 2-week period ($\alpha = .73-.76$). Mother, father, and adolescent informant-reports of the CDI were moderately correlated with one another (r s ranged from .28 to .53). As above, all informant-reports within a given scale were averaged prior to analysis.

Parent-child conflict

We examined parent-child conflict as a potential mediator of the association between paternal depression and child psychopathology. The Parent-Child Relationship scale (Hetherington & Clingempeel, 1992) is a 46-item questionnaire that assesses how each adolescent-parent dyad relate to one another. We focused here on the parent-adolescent conflict scale (eight items; α ranges from .70 to .76 for father and adolescent informant-reports; e.g., how much do you yell at this person?; how much does this person nag you?; how much does this person criticize you?). Parent reports on each child and child reports on each parent were averaged in the current report.

Statistical Analyses

We examined the association between paternal depression symptoms and child psychopathology symptoms by regressing each child outcome on paternal depression in a series of multiple regression and structural equation models. All models were estimated in Mplus version 8.5 (Muthén & Muthén, 2021) using full information maximum likelihood estimation. Confidence intervals were derived via family-clustered, nonparametric, percentile bootstrapping with 10,000 draws. The clustered, nonparametric bootstrap performs well under a variety of data conditions, including the presence of nonindependence and non-normality (Chernick, 2011; Falk, 2018; Fritz & MacKinnon, 2007). Analyses were also facilitated by the Mplus Automation Package (Hallquist & Wiley, 2018) in R. To ensure that observed associations did not reflect downstream effects of assortative mating, since fathers and mothers were more similar to each other on in their depression than would be expected by chance (spousal r was .19 in non-divorced families and .14 in stepfamilies, both $p < .001$), we controlled for maternal depression in all analyses. As child age and sex are also known to influence rates of psychopathology (Rescorla *et al.*, 2007), we controlled for age and sex in all analyses as well.

Analyses began with a series of multiple regression models conducted separately for children reared by their biological father and children reared at least in part by their stepfather. Potential differences in father-child similarity based on genetic relatedness were then examined in a series of multiple-group multiple-regression models. The relevant path estimates (between paternal depression symptoms and child outcome) were constrained to equality across biological and stepchildren, and the fit of this model was compared to a model without these constraints (i.e., the fully unconstrained multigroup model). The change in log likelihood of

the two models was chi-square distributed on 1 degree of freedom, and could thus be used to indicate the extent to which paths differ across biological and nonbiological father-child relationships.

We then estimated the extent to which parent-child conflict mediated the association between paternal depression symptoms and child psychopathology symptoms via a series of structural equation mediation models (Figure 1). In these models, paths were specified from paternal depression symptoms to child psychopathology symptoms (PD→CP in Figure 1) and the intervening variable of parent-child conflict (PD→CON), respectively. Another path was specified from parent-child conflict to child psychopathology symptoms (CON→CP). In these models, both parent-child conflict and child psychopathology symptoms were regressed on the covariates (i.e., child age, child sex, and maternal depression). The path from paternal depression symptoms to child psychopathology symptoms in these models (PD→CP) captures the residual "direct effect", or the association between paternal depression symptoms and child psychopathology symptoms after controlling for parent-child conflict and the covariates. The indirect effect is the product of the paths from paternal depression symptoms to parent-child conflict, and from parent-child conflict to child psychopathology symptoms (i.e., PD→CON*CON→CP). This estimate reflects the extent to which the association between paternal depression symptoms and child psychopathology symptoms was mediated by conflict in the father-child relationship. As above, potential differences with genetic relatedness were examined by constraining the direct and indirect effect path estimates to equality across biological and stepfather pairings, and then comparing the fit of the more constrained model to a model without these constraints. The change in log likelihood of the two models in this case is chi-square distributed on 3 degrees of freedom.

Notably, all of the analyses described above were conducted twice for each outcome. We first ran the above models on the full sample, thereby maximizing our power to detect significant differences in father-child similarity as a function of their genetic relatedness. We then repeated all analyses in only the 238 blended families, or those in which one child was genetically related to the male head of household while their sibling was not (i.e., the half-sibling and stepsibling families). Comparisons of father-child similarity across biological and stepchildren in blended families yield especially strong causal inferences, as we are necessarily conditioning on the same male head of household. In doing so, we are able to control for a host of potential moderators and unmeasured confounds.

Results

Descriptives and zero-order correlations

Paternal depression was reasonably common in these data, with 15.1% of fathers considered at risk for clinical depression using the standard CES-D cut-point for the presence of clinically significant symptomatology (mean = 9.19 (7.63) with a range of 0 to 51). A paired samples t-test revealed that, as expected, fathers' mean level of depressive symptomatology was significantly lower than that for mothers (mean = 10.47 (8.17) with a range of 0–44 and 18.7% at or above the cut point of 16). Mean levels of paternal depression did not vary across non-divorced and stepfamilies (standardized Cohen's d effect size = .04, ns), although mean levels of maternal depression were slightly higher in stepfamilies than in non-divorced families ($d = .23$, $p < .01$). Mean levels of child psychopathology symptoms also varied across non-divorced and stepfamilies, with Cohen's d effect sizes ranging from .31 to .50

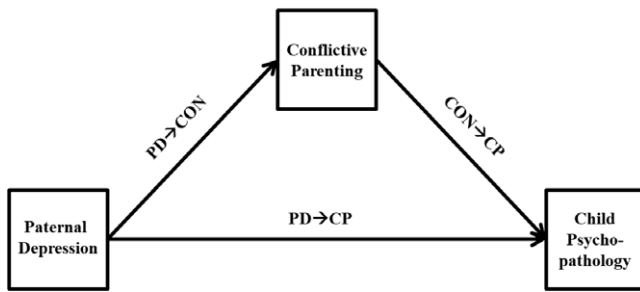


Figure 1. Conceptual path diagram of the simple indirect effects models. PD = paternal depression; CON = paternal conflict with child; CP = child psychopathology. All models included age, sex, and maternal depression as covariates of both parenting and child psychopathology (omitted from the figure for clarity of presentation).

across the various forms of psychopathology (all $p < .01$). Critically, however, when we compared children in stepfamilies who were being reared by their biological father to those being reared by their stepfather, we observed only a few differences (d s were $-.21, -.02, .03, .21$, and $-.09$ for adolescent depression symptoms on the CDI, adolescent depression symptoms on the BPI, antisocial behavior, headstrong, and attention problems, respectively; all nonsignificant except adolescent depression on the CDI and headstrong behavior, such that stepchildren had lower levels of CDI depression and higher levels of headstrong symptoms than those being reared by their biological father). Such results suggest that, although child psychopathology symptoms were clearly more common in stepfamilies than in non-divorced families, these differences were largely unrelated to whether or not the adolescents were biologically related to their rearing father.

Zero-order correlations between paternal depression symptoms, conflict in the father-child relationship, and child psychopathology symptoms are presented in Table 1, separately for biological and stepfather pairings. As seen there, paternal depression symptoms evidenced consistently small and positively signed associations with all forms of child psychopathology symptoms, and did so regardless of whether or not the male head of household shared genes with the child in question. Although only preliminary, such findings offer key support for an environmentally mediated link between paternal depression symptoms and common forms of child psychopathology symptoms. Similar results emerged for father-child conflict, which evidenced small-to-moderately sized associations with all forms of child psychopathology symptoms, and did so regardless of whether or not fathers shared genes with their child.

Direct associations between paternal depression and youth psychopathology

Table 2 presents the results of the multiple regression models estimating the association between paternal depression symptoms and child psychopathology symptoms adjusting for covariates. Paternal depression symptoms were associated with all measures of child psychopathology symptoms when evaluated across the full sample, an association that held regardless of whether or not fathers and their children were genetically related. Indeed, we were able to constrain the coefficients across biological and stepfather-child pairings in all cases without a significant decrement in fit (the change in χ^2 on 1 df was 0.84 or less). Associations were similar in magnitude (if less consistently statistically significant) when we restricted our analyses to those 238 “blended” families in which

Table 1. Zero-order correlations

Child psychopathology symptoms	Zero-order correlations	
	Paternal depression symptoms	Paternal conflict with child
<i>Adolescents being reared by their biological father</i>		
Depression, CDI	.18**/.16*	.28**/.25**
Depression, BPI	.20**/.21**	.27**/.27**
Antisocial behavior	.14**/.17**	.27**/.30**
Headstrong	.12**/.14*	.37**/.39**
Attention problems	.15**/.20**	.28**/.25**
<i>Adolescents being reared by their stepfather</i>		
Depression, CDI	.16**/.14*	.31**/.32**
Depression, BPI	.17**/.13*	.31**/.35**
Antisocial behavior	.11**/.14*	.27**/.36**
Headstrong	.16**/.18**	.34**/.34**
Attention problems	.18**/.14*	.26**/.23**

Note. Correlations in the full sample are presented before the slash, while correlations in the blended family subset are presented after the slash.
 *Indicate that the correlation is significantly larger than zero at $p < .05$.
 **Indicate that the correlation is significantly larger than zero at $p < .01$.

the father was biologically related to one participating child but not to the other. As above, we were able to constrain the coefficients across biological and stepfather-child pairings without a significant decrement in fit in all cases. Such findings collectively point to environmental transmission of psychopathology between fathers and their children.

To evaluate whether these effects were specific to paternal depression symptoms, we conducted a set of post hoc analyses evaluating the extent to which the overall pattern of intergenerational transmission of psychopathology persisted to the mother-child relationship as well, entering paternal depression, child age, and child sex as covariates. Of note, we had less power to detect associations between stepmothers and their children, as there were only 133 stepmother-child pairings in the data (versus 600 stepfather-child pairings). Results are presented in Table S1. Although we were able to constrain coefficients across biological and stepmother-child pairings for all phenotypes save attention problems, maternal depression symptoms significantly predicted youth psychopathology symptoms only when mothers were biologically related to their child.

Mediation by parent-child conflict

We next examined whether conflict in the father-child relationship might mediate links between paternal depression symptoms and child psychopathology symptoms. As shown in Table 3, we observed consistent evidence of mediation such that associations between paternal depression symptoms and the various forms of child psychopathology were all mediated in part via father-child conflict. Moreover, paths could be fully constrained to equality across biologically related and unrelated father-child pairs in all cases. As above, this pattern of results (i.e., significant indirect effects in stepfather-child pairings that were equivalent to those in biological father-child pairings) held for both the full sample and the subset of blended families. As a final point, we note that the direct path from paternal depression symptoms to child psychopathology symptoms was no longer statistically larger than

Table 2. Associations between paternal depression and youth psychopathology

Youth psychopathology symptoms	Standardized path coefficients		Constraining path coefficient to equality	
	Fathers and their biological children	Fathers and their stepchildren	$\Delta\chi^2$ (on 1df)	<i>p</i> -value
<i>All families (N = 720)</i>				
Depression, CDI	.15 (.08, .23)*	.14 (.05, .23)*	0.84	0.358
Depression, BPI	.17 (.10, .25)*	.15 (.07, .23)*	0.46	0.500
Antisocial behavior	.12 (.05, .20)*	.10 (.01, .20)*	0.09	0.764
Headstrong	.09 (.01, .18)*	.14 (.05, .23)*	0.44	0.507
Attention problems	.12 (.04, .20)*	.16 (.04, .26)*	0.10	0.752
<i>Blended families (N = 238)</i>				
Depression, CDI	.14 (.02, .25)*	.12 (−.01, .24)	0.12	0.727
Depression, BPI	.20 (.08, .32)*	.12 (.01, .24)*	0.74	0.390
Antisocial behavior	.15 (.02, .29)*	.11 (−.03, .25)	0.07	0.791
Headstrong	.09 (−.03, .22)	.16 (.04, .28)*	0.53	0.467
Attention problems	.18 (.04, .31)*	.12 (−.01, .26)	0.66	0.417

Note. Standardized path coefficients indexing the association between paternal depression and child psychopathology are presented. All models included age, sex, and maternal depression as covariates. 95% confidence intervals were derived via family-clustered percentile bootstrapping with 10,000 random draws. To evaluate the role of father-child genetic relatedness in the association between paternal depression and child psychopathology, the path coefficients were constrained to be equal across biologically related and unrelated father-child pairs.

* Indicates that the path coefficient was statistically larger than zero.

zero in stepfather-child pairings, although it was intermittently significant in biological father-child pairings.

Contextualizing the above results within traditional twin family analyses

Given that the above findings point to largely environmental transmission of paternal psychopathology across generations, one remaining question concerns the way in which these results might line up with traditional, single-generation approaches to understanding etiology. Presumably, we would see evidence of this environmental transmission at the child level as well. To evaluate this possibility, we conducted a second set of post hoc analyses, computing standard univariate heritability estimates and sibling intraclass correlations for each phenotype. Results are presented in Table 4. As seen there, all five phenotypes evidenced prominent genetic influences, but only one evidenced significant shared environmental influences (shared environmental influences on youth antisocial behavior were estimated to be 13%). What's more, the shared environmental estimate was exactly .00 for two of the five phenotypes (i.e., headstrongness and attention problems).

Discussion

The current study sought to evaluate the etiologic origins of the intergenerational transmission of psychopathology within families. To do so, we developed a novel genetically informed design that incorporates the core interpretive element of adoption studies (e.g., rearing by a nonbiologically related parent) but does so using a sample that also includes biologically related rearing parents as a comparison. Results pointed squarely to the environmental transmission of psychopathology between fathers and children. Paternal depression was consistently associated with child psychopathology, an association that held regardless of whether or not fathers and their children were genetically related. Moreover, the associations between paternal depression symptoms and the various

child psychopathology symptoms were all significantly mediated via father-child conflict, and persisted across the full sample and the 238 "blended" families in which the father was biologically related to one participating child but not to the other. The latter comparisons are especially revealing, since they necessarily measure biological and stepchild similarity with the same father, thereby controlling for many measured and unmeasured confounds.

Post hoc analyses sought to contextualize the above findings within traditional, single-generation approaches to understanding etiology. Because parental psychopathology is shared across siblings and associated with youth psychopathology via environmental mechanisms, we hypothesized that we would observe significant shared environmental influences on the various forms of psychopathology under study. Instead, two of five phenotypes estimated shared environmental influences to be exactly 0%, and only one of the five (antisocial behavior) estimated them to be significantly larger than zero. Such findings suggest that, although the transmission of paternal psychopathology between generations was clearly environmental in origin, these effects did not manifest as shared environmental influences as conceptualized within traditional twin study approaches to understanding etiology. Put another way, we observed environmental transmission of depression across generations within families regardless of the presence or absence of shared environmental influences on the outcome.

Such findings constructively replicate and extend those from other intergenerational designs. CoT studies, for example, have consistently pointed to the environmental transmission of depression across generations and (somewhat less consistently) to environmental mediation of the association between parental depression and child externalizing/conduct problems (McAdams et al., 2015; Silberg et al., 2010; Singh & Ghandour, 2012). Similarly, depression in adoptive parents (and particularly in adoptive mothers) has been consistently correlated with internalizing and externalizing psychopathology in their children, with

Table 3. Parental-child conflict (CON) as a mediator of the association between paternal depression symptoms (DEP) and child psychopathology symptoms (CP)

Child psychopathology symptoms	Fathers and their biological children				Fathers and their stepchildren				Constraining all paths to equality	
	DEP→CON	DEP→CP	CON→CP	Indirect effect estimate	DEP→CON	DEP→CP	CON→CP	Indirect effect estimate	$\Delta\chi^2$ (on 3 df)	<i>p</i> -value
<i>All families (N = 720)</i>										
Depression CDI	.15 (.07, .24)*	.12 (.04, .19)*	.25 (.18, .31)*	.04 (.02, .06)*	.24 (.14, .34)*	0.07 (−.02, .16)	.28 (.20, .36)*	.07 (.04, .11)*	5.10	0.164
Depression BPI	.15 (.07, .24)*	.13 (.06, .21)*	.25 (.18, .31)*	.04 (.02, .06)*	.24 (.14, .34)*	0.09 (.00, .17)	.27 (.19, .35)*	.07 (.03, .10)*	3.94	0.268
Antisocial behavior	.15 (.07, .23)*	.09 (.01, .17)*	.23 (.16, .30)*	.04 (.02, .06)*	.24 (.13, .34)*	0.04 (−.06, .14)	.26 (.18, .34)*	.06 (.03, .10)*	3.86	0.277
Headstrong	.15 (.07, .24)*	0.04 (−.04, .12)	.35 (.28, .42)*	.05 (.02, .09)*	.24 (.14, .34)*	0.06 (−.03, .15)	.32 (.24, .40)*	.08 (.04, .12)*	4.61	0.203
Attention problems	.15 (.07, .24)*	.08 (.01, .16)*	.25 (.18, .31)*	.04 (.02, .06)*	.24 (.14, .34)*	0.10 (−.01, .21)	.23 (.14, .31)*	.05 (.03, .09)*	3.89	0.274
<i>Blended families (N = 238)</i>										
Depression CDI	.18 (.04, .31)*	0.10 (−.02, .22)	.21 (.07, .34)*	.04 (.01, .08)*	.22 (.09, .35)*	0.05 (−.08, .19)	.29 (.17, .41)*	.06 (.02, .12)*	1.11	0.775
Depression BPI	.18 (.04, .31)*	.16 (.03, .28)*	.22 (.10, .35)*	.04 (.01, .08)*	.22 (.09, .35)*	0.05 (−.07, .17)	.33 (.21, .45)*	.07 (.03, .13)*	2.57	0.463
Antisocial behavior	.18 (.04, .31)*	0.11 (−.03, .24)	.26 (.15, .37)*	.05 (.01, .09)*	.22 (.09, .35)*	0.03 (−.10, .17)	.34 (.22, .45)*	.08 (.03, .13)*	1.77	0.622
Headstrong	.18 (.04, .31)*	0.03 (−.09, .16)	.34 (.22, .46)*	.06 (.01, .11)*	.22 (.09, .35)*	0.09 (−.03, .21)	.31 (.17, .45)*	.07 (.02, .12)*	1.92	0.589
Attention problems	.18 (.04, .31)*	.14 (.01, .28)*	.21 (.08, .33)*	.04 (.01, .08)*	.22 (.09, .35)*	0.08 (−.06, .22)	.20 (.07, .36)*	.05 (.01, .09)*	2.09	0.554

Note. Standardized path coefficients from unconstrained models are presented above. All models included age, sex, and maternal depression as covariates. 95% confidence intervals were derived via family-clustered percentile bootstrapping with 10,000 random draws. The Indirect effect estimates index the product of the paths from paternal depression to parenting and from parenting to child psychopathology. When testing equality constraints, the three paths were constrained to equality across biological and step children.

* Indicates that the path coefficient was statistically larger than zero.

Table 4. Sibling intraclass correlations, separately by sibling type, and univariate heritability estimates

Child psychopathology symptoms	Sibling intraclass correlations						Univariate heritability estimates		
	MZ	DZ	FS-ND	FS-D	HS	SS	A	C	E
Depression, CDI	0.53*	0.11	0.20*	0.17*	0.18*	.06	.51 (.29, .62)*	.01 (.00, .14)	.48 (.38, .60)*
Depression, BPI	0.64*	0.33*	0.15*	0.27*	0.24*	0.19*	.65 (.46, .78)*	.05 (.00, .16)	.30 (.22, .42)*
Antisocial behavior	0.76*	0.48*	0.37*	0.37*	0.30*	0.16*	.66 (.52, .79)*	.13 (.02, .23)*	.21 (.16, .28)*
Headstrong	0.62*	0.22*	0.34*	0.18*	0.21*	.04	.65 (.47, .73)*	.00 (.00, .10)	.35 (.27, .46)*
Attention problems	0.57*	0.25*	0.17*	0.29*	0.15*	.01	.66 (.49, .74)*	.00 (.00, .10)	.34 (.26, .44)*

Note. The left side of the table presents the sibling intraclass correlations for each form of child psychopathology. MZ, DZ, FS-ND, FS-D, HS, and SS represent monozygotic twins, dizygotic twins, full siblings from non-divorced or intact families, full siblings from divorced families, half-siblings, and stepsiblings, respectively. The right side of the table presents the univariate heritability estimates, and their 95% confidence intervals, for each form of child psychopathology. A, C, and E represent additive genetic, shared environmental, non-shared environmental influences, respectively. Confidence intervals that do not overlap with zero are considered statistically significant at $p < .05$ (indicated by bold font).

* Indicates that the correlation is larger than zero at $p < .05$.

correlations in the .10–.20 range, again pointing to environmental transmission. By contrast, NTFM results have not generally supported environmental intergenerational transmission of psychopathology, with minimal evidence of environmentally mediated similarity across parents and children. Although we cannot be sure what accounts for this discrepancy, we note that the approach taken in our paper is conceptually similar to an adoption design, in that we are comparing parent–child similarity across parent–child pairings with and without a genetic relationship. Somewhat similarly, CoT designs leverage differences in environmental relatedness between genetic “parents” and children (since the MZ avuncular relationship can be understood as a non-rearing biological parent–child relationship), although they do not include variability in the genetic relatedness of parents and their children. The NTFM model differs from both the CoT and the adoption design in this regard in that all parents are rearing parents that share 50% of their genes with their children. As such, there is no ability to specifically disambiguate genetic and environmental transmission across generations. Instead, genetic and environmental influences are inferred only at the level of the twins, and thus they cannot be considered “intergenerational” *per se*. Put another way, our results align with those from designs explicitly able to disambiguate genetic and environmental transmission from parents to children, but not with the one putatively intergenerational design that leverages only child (i.e., twin) differences in genetic relatedness.

The current study does have a few important limitations that should be considered. First, all offspring in this sample were adolescents between 11 and 18 years of age. This is a key point, since both the prevalence and etiology of most forms of psychopathology change with age and development (Burt, 2009; Rescorla et al., 2007). We thus cannot know whether the environmental transmission of paternal psychopathology across generations observed here during adolescence would also be observed during childhood, or if there are particular “sensitive periods” for exposure to parental depression. We also cannot know whether and how the current results might persist over time into emerging adulthood. This point is particularly salient given that the cross-sectional nature of these data does not permit any conclusions regarding the directionality of the reported associations (paternal depression predicting adolescent psychopathology or vice versa). This caveat is important given that at least one longitudinal adoption study suggested that adoptive parent depression may also be a consequence of youth psychopathology (McAdams et al., 2015), raising key

questions as to the (bi)directionality of the intergenerational transmission of psychopathology. What’s more, parental depression may have longstanding influences on child mental health that emerge only later in development, associations that we are not able to evaluate herein. Longitudinal extensions of either the current analyses or a CoT study would be necessary to resolve among these possibilities.

A related limitation stems from the current study’s inclusion criteria for recruitment. To be eligible for participation in NEAD, stepchildren were required to have resided with their genetically unrelated parent for a minimum of 5 years, but were not required to have resided with them since infancy. This represents a key distinction with adoption studies, nearly all of which require that the adopted child was placed with their adoptive family during infancy. The similarity of the current results with those of adoption studies is even more telling in this light.

Another limitation was that the NEAD sample was predominantly White, limiting the generalizability of our findings to families of color. Future genetically informed studies should make explicit efforts to collect more racially and ethnically diverse samples, as it remains possible that results differ for families with marginalized identities. Next, the current study specifically focused on parental depression given the longstanding interest in parental depression as a predictor of child psychopathology (Gelfand & Teti, 1990; Goodman et al., 2011). As such, our results are specific to the transmission of parental depression and do not extend to other forms of adult psychopathology. Future studies should expand this framework to understand the intergenerational transmission of other forms of psychopathology.

Finally, it was not clear whether the pattern of results observed for the transmission of paternal psychopathology persisted to the transmission of maternal psychopathology. On the one hand, we were able to constrain path coefficients across biological and stepmother–child pairings for all phenotypes save attention problems, results which are similarly suggestive of environmental transmission for adolescent depression, antisocial behavior, and headstrongness. On the other hand, maternal depression was significantly associated with youth psychopathology only when mothers were biologically related to their child. As the number of stepmother–stepchild pairings was small ($N = 133$), it is not entirely clear how to rectify these two sets of results, nor is it clear whether they might persist to larger sample. Future studies should rerun these analyses using a larger sample of stepmother–child pairings.

Despite these limitations, the current study has a number of key implications. First and foremost, despite the clear and robust presence of genetic influences on adolescent psychopathology in these data, the transmission of psychopathology between fathers and their adolescent children appeared to be environmental in origin. Paternal depression was positively associated with adolescent psychopathology regardless of whether fathers and their children were genetically related. This marked equivalence of father–stepchild similarity with father–biological child similarity was observed even when examining blended families in which the father was genetically related to one child and not the other. What's more, these associations were significantly mediated via father–child conflict, highlighting a specific aspect of the environment that appears to drive at least some of this association. Such findings are not only fully consistent with the environmental transmission of psychopathology across generations, but also add to extant evidence that father–child conflict is a robust and at least partially environmental correlate of adolescent psychopathology (Burt et al., 2021).

Second, we observed this environmental transmission of depression across generations even in the absence of shared environmental influences on the outcome. Indeed, only one of the five phenotypes we examined revealed any evidence of shared environmental influences. Such findings indicate that, although the transmission of psychopathology between fathers and their children appeared to be environmental in origin, these effects did not manifest as shared environmental influences within traditional approaches to understanding etiology. Such results imply that, somewhat surprisingly, the origins of intergenerational transmission may not sync up with single-generation ACE estimates of etiology in an obvious way.

How do we understand this seemingly foundational incongruence within the same dataset? One possibility relates to the aforementioned issues with directionality. Namely, should children's psychopathology symptoms be inducing symptoms of depression in fathers rather than vice versa, we would expect to observe environmental transmission across generations that was independent of the etiology of child psychopathology itself. Alternately, these results could suggest that, rather than functioning as shared environmental influences as typically conceptualized, parent psychopathology exerts its environmentally mediated effects on child psychopathology in a child-specific way, loading on the non-shared environmental component of variance (Turkheimer & Waldron, 2000). In other words, although parental depression is objectively shared across both siblings in a family, it may effectively operate as a non-shared environmental influence at a functional level. Yet another possibility relates to the developmental timing of genetic expression. Namely, it may well be the case that the genes relevant for depression in mid-life differ from those related to psychopathology during adolescence. A final possibility relates to the fact that, as has been documented many times, the inclusion of additional non-twin family members in biometric studies often yields different, and arguably more precise, estimates of etiology (Keller et al., 2010). Future studies should explore these possibilities and seek to clarify whether and how the results from classical twin studies inform our understanding of intergenerational transmission.

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Conflicts of interest. None.

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