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Genetic and Environmental Influences on Parent-Child Conflict and Child Depression Through Late Adolescence

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Abstract

Objective—Few studies have investigated potential gender differences in the genetic and environmental influences on the prospective associations between parent-child conflict and later depression, a notable gap given substantial gender differences in rates of depression and suggestive evidence of differences in the etiology of depression among females and males. To fill this gap, we evaluated whether the prospective relationship between parent-child conflict and major depressive disorder (MDD) symptoms varied as a function of parent-child gender composition.

Method—A combined twin and adoption sample was used (53% female; 85% European ancestry), containing 1,627 adolescent sibling pairs (789 monozygotic twin pairs, 594 dizygotic/full-biological pairs, 244 genetically unrelated pairs) with assessments at two time points in adolescence (ages ~15 to ~18).

Results—Prospective associations between parent-child conflict and subsequent adolescent depression were explained predominately through common genetic influences for mother-daughter and mother-son pairs, but less so for father-daughter and father-son pairs.

Conclusion—Processes of gene-environment correlation involved in the prospective associations between parent-child conflict and later adolescent depression appear to be less relevant to father-child relationships in comparison to mother-child relationships. Notably, results did not show parent-child conflict was more relevant to the etiology of MDD for girls than boys; gender differences in depression do not appear to be due to differences in the associations between parent-child conflict and child depression.

Keywords

Behavior Genetics; Depression; Gender; Parent-Child Conflict; Parent-Child Relationships

Rates of depression increase rapidly during adolescence. Although point prevalence estimates of major depressive disorder (MDD) are quite low in childhood (1% to 3%; Costello, Erkanli, & Angold, 2006), estimates in adolescence are comparable to those for adults (5% to 7%; Costello et al., 2006; Kessler, Chiu, Demler, Merikangas, & Walters, 2005). A greater degree of depression in adolescence is associated with both concurrent and

prospective impairment in a range of important psychosocial domains, including family and peer relationships and academic functioning, as well as increased psychiatric comorbidity and suicidality (Angold, Costello, Farmer, Burns, & Erkanli, 1999; Hammen, Brennan, Keenan-Miller, & Herr, 2008; Jaffee et al., 2002; Lewinsohn, Rohde, Klein, & Seeley, 1999; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003; Pickles, Rowe, Simonoff, Foley, Rutter, & Silberg, 2001; Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013; Wilson, Hicks, Foster, McGue, & Iacono, 2015).

As depression in adolescence has such pervasive, negative implications, there has been considerable effort devoted to identifying risk factors for its development and course (see Burcusa & Iacono, 2007; Dobson & Dozois, 2008). One extensively investigated putative etiological factor has been the quality of the parent-child relationship. The parent-child relationship has long been seen as an important source of influence on child and adolescent health and adjustment (see Berg-Nielsen, Vikan, & Dahl, 2002; Steinberg & Morris, 2001). In particular, parent-child conflict and hostility have been implicated as playing a key role for depression in childhood and adolescence, with evidence of both concurrent and prospective links between parent-child conflict and adolescent depression (Burge & Hammen, 1991; Marmorstein & Iacono, 2004; Schwartz, Dudgeon, Sheeber, Yap, Simmons, & Allen, 2012; Sheeber, Hops, Alpert, Davis, & Andrews, 1997; Sheeber, Davis, Leve, Hops, & Tildesley, 2007).

Although prior research has demonstrated a consistent link between negative aspects of parenting and depression in childhood and adolescence (for a meta-analysis, see McLeod, Weisz, & Wood, 2007), that does not mean this link is necessarily causal. The relationship between parenting and child depression is likely bidirectional in nature (e.g., see Branje, Hale, Frijns, & Meeus, 2010; Pettit & Arsiwalla, 2008) and explained by a number of factors, including parental depression (Lovejoy, Graczyk, O'Hare, & Neuman, 2000; Wilson & Durbin, 2010). Following this, twin and family studies indicate that the heritability of adolescent depression is moderate to substantial in magnitude, with a meta-analysis of existing studies indicating that estimates of genetic influences account for 44% of the variance in MDD symptoms in samples of children and adolescents (Burt, 2009) and 37% in samples of adults (Sullivan, Neale, & Kendler, 2000). Studies decomposing the genetic and environmental influences common to the relationship between parenting and adolescent depression have shown this relationship is predominately explained by common genetic, rather than shared or nonshared environmental, influences (Neiderhiser, Reiss, Hetherington, & Plomin, 1999; Pike, McGuire, Hetherington, Reiss, & Plomin, 1996; Rice, Lewis, Harold, & Thapar, 2013).

Evidence of common genetic influences on the relationship between parenting and adolescent depression is often interpreted within a gene-environment correlation framework (Jaffee & Price, 2008; Scarr & McCartney, 1983). Parents with a genetically influenced depressive temperament may be more likely to engage in conflictual interactions, and thus transmit both a depressive genotype to the adolescent while also fostering a conflictual family environment (i.e., *passive* gene-environment correlation). Adolescents with a genetically influenced depressive temperament may frustrate parents and elicit conflictual responses (i.e., *evocative* gene-environment correlation). Depression-prone adolescents may

also seek out peer relationships and/or situations and environments that align with their genetically influenced depressive temperament (i.e., *active* gene-environment correlation), which may indirectly lead to increased conflict with their parents. Notably, both passive and non-passive (i.e., evocative and active) gene-environment explanations for the quality of parent-adolescent relationships have received support (Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007; Neiderhiser, Reiss, Pedersen, Lichtenstein, Spotts, & Hansson, et al., 2004).

Although prior research has demonstrated common genetic influences for the relationship between parenting and depression in childhood and adolescence (Neiderhiser et al., 1999; Pike et al., 1996; Rice et al., 2013), the extent to which this relationship is comparable across gender remains unclear. Gender differences in the rates of depression are well established, with females approximately twice as likely to experience depression as males (Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Hasin, Goodwin, Stinson, & Grant, 2005). Notably, gender differences emerge in adolescence, with comparable rates among girls and boys in childhood, but much more rapidly increasing rates among girls relative to boys in adolescence (Hankin, Abramson, Moffitt, Silva, McGee, & Angell, 1998). Thus, there may be important differences in the antecedents or overall etiology of depression for males and females.

Extensive research has sought to elucidate factors that account for the increased rates of depression among females relative to males. Three large-scale twin studies have shown heritability estimates tend to be larger in women than men (Bierut, Heath, Bucholz, Dinwiddie, Madden, Statham et al., 1999; Kendler, Gardner, Neale, & Prescott, 2001; Kendler, Gatz, Gardner, & Pedersen, 2006); however two recent meta-analyses find generally comparable heritability estimates of depression by gender in children and adolescents (Burt, 2009) and adults (Sullivan, Neale, & Kendler, 2000). It is important to note that even though research has shown consistent age-related changes in the heritability of depression, with large shared environmental influences in childhood (i.e., non-genetic factors that make family members similar to one another) that decrease over time, and small additive genetic influences in childhood (i.e., genetic factors that make family members similar to one another) that increase over time (Scourfield, Rice, Thapar, Harold, Martin, & McGuffin, 2003; Thapar & McGuffin, 1996), comparable estimates of additive genetic influence have been found in the adult and adolescent depression meta-analyses. Specifically, Sullivan et al.'s meta-analysis on adult depression showed 37% of the variance in MDD symptoms was due to additive genetic factors, 0% of the variance in MDD symptoms to shared environmental influence, and 63% to nonshared environmental influence (i.e., non-genetic factors that make family members different from one another). Burt's (2009) meta-analysis on children and adolescents showed 44% of the variance in MDD symptoms was due to additive genetic factors, 14% to shared environmental factors, and 42% to nonshared environmental influence.

One limitation of Sullivan et al.'s (2000) meta-analysis was that adoption studies were not included in the computation of overall heritability (as none were available at that time) and only one adoption study was included in Burt's (2009) meta-analysis of depression. Twin and adoption designs rely on key assumptions (e.g., equivalent environments in comparing

sibling groups such as monozygotic/MZ vs. dizygotic/DZ twins), and the inclusion of both twin and adoption samples offers complementary strengths to individual limitations. For example, twin studies may underestimate shared environmental influences due to the partial confounding of shared environmental and non-additive genetic effects (Matteson, McGue, & Iacono, 2013; Rutter, 2007). Adoption studies, on the other hand, provide a direct measure of shared environmental influences because adopted siblings share none of their segregating DNA (Buchanan, McGue, Keyes, & Iacono, 2009).

The primary way this study extends prior research is through a thorough investigation of genetic and environmental influences in the prospective relationship between parent-child conflict and subsequent MDD symptoms in late adolescence as a function of parent and child gender. Although prior research has shown the relationship between parenting and child depression is predominately accountable by genetic influences, there is some evidence that effects may vary for girls and boys, and among mother-child and father-child pairs.

For example, in a sample of 4–10 year-old children, Rice et al. (2013) found that mothers' positivity mediated the association between maternal depression and child depression only among genetically related mother-daughter (but not mother-son) pairs, while fathers' positivity mediated the association between paternal depression and child depression among both genetically related father-daughter and father-son pairs. This indicates that the gene-environment correlation effect (linking parent-child relationship quality and child depression) may be gender-specific for mothers but not fathers. Such etiological differences may help to explain the disparity in rates of MDD in adolescent girls versus boys, but replication and extension into later developmental periods when depression is more common are needed. Neiderhiser et al. (1999) found predominately genetic influences on associations between parental conflict-negativity and adolescent depression among both mother- and father-adolescent pairs; however, the effects of child gender were not examined. Lewis, Collishaw, Thapar, and Harold (2014) found genetic influences on associations between parental hostility and adolescent depression among both mother- and father-adolescent pairs, but also found some evidence of common nonshared environmental influence, but only among mother-daughter pairs. This may indicate that the environmental influence of maternal hostility may be more relevant to girls' than boys' development of depression, but again, replication and exploration of this finding via prospective design are needed..

The present study extends previous investigations of genetic and environmental influences on the association between parenting and child depression through: 1) the use of a combined twin and adoption sample, 2) prospective data assessment, and 3) the evaluation of parent and child gender differences. Specifically, we included multiple samples of twin and adopted/biological sibling pairs and their parents, prospectively assessed during the transition from mid- (~ age 15) to late (~ age 18) adolescence. The inclusion of a large number of female and male adolescents, as well as their genetically related and unrelated mothers and fathers, allowed us to examine the extent to which genetic and environmental influences on the association between parent-child conflict and subsequent depression in late adolescence differed by parent-child gender composition.

Method

Participants and Procedures

Participants were drawn from several ongoing longitudinal studies conducted at the Minnesota Center for Twin and Family Research (**MCTFR**), including the Minnesota Twin and Family Study (**MTFS**, Iacono, Carlson, Taylor, Elkins, & McGue, 1999), the Enrichment Study (**ES**, Keyes, Malone, Elkins, Legrand, McGue, & Iacono, 2009), and the Sibling Interaction and Behavior Study (**SIBS**, McGue, Keyes, Sharma, Elkins, Legrand, Johnson, et al., 2007). MTFS and ES consist of twin samples (MZ and DZ twin pairs) and SIBS consists of an adopted and biological sibling sample (adopted/genetically-unrelated and full biological sibling pairs). These studies and samples have been described at length elsewhere (Iacono et al., 1999; Keyes et al., 2009; McGue et al., 2007; also see Hicks, Schalet, Malone, Iacono, & McGue, 2011; Iacono, McGue, & Krueger, 2006) and are only briefly reviewed here. All studies include extensive assessment of twins/siblings and their parents, including information on the quality of the parent-child relationship and psychiatric diagnoses. All participating families were from the state of Minnesota and lived within a day's drive to the research lab. All studies were approved by the University of Minnesota Institutional Review Board. Parental consent was obtained for parents' own participation and for twins/siblings younger than 18 years. Twin/sibling assent was obtained prior to age 18, and consent was obtained after age 18.

MTFS is a longitudinal, cohort-sequential twin study of reared-together twins and their parents. Same-sex twin pairs were identified using publicly available birth records and twins residing within 1 day's drive of Minneapolis, living with at least one biological parent, and without physical or intellectual deficiencies that could interfere with assessment were recruited. Over 90% of eligible twins were located, and over 80% of eligible and located twins were enrolled. The MTFS includes two cohorts: the younger cohort was 11 years old at the first visit and the older cohort was 17 years old at the first visit. Follow-up assessments with both MTFS cohorts were conducted every 3–5 years through age 29 (with overlapping assessments at ages 17, 20, 24, and 29). Retention over assessment periods was 87%. Demographics for the MTFS sample are representative of Minnesota during the targeted birth years (95% Caucasian); see Iacono et al. (1999) for a detailed overview of MTFS.

ES was designed to supplement the MTFS by oversampling twins with childhood disruptive disorders. As for the MTFS, eligible same-sex twin pairs were identified using birth records, but were additionally randomly assigned to unscreened (24%) or screened (76%) groups. Recruitment of unscreened twins was identical to that for the MTFS. Recruitment of screened twins included assessment using parent reports of twin symptoms of conduct disorder, attention-deficit/hyperactivity disorder, or oppositional defiant disorder; twins with elevated symptoms were recruited¹. Over 80% of eligible twins were located, and over 80% of located eligible twins were enrolled. Just over one third of participating twins met criteria for a childhood disruptive disorder diagnosis. The ES sample was 11 years old at the first

¹We ran supplementary analyses that excluded the screened ES sample ($n = 242$ pairs with elevated childhood disruptive disorders) and found the same general pattern of results. To retain maximal power, we included the entire ES sample in the reported results below.

visit, and follow-up assessments were conducted at ages 14 and 17. Retention over assessment periods was 91%. Demographics for the ES sample are representative of Minnesota during the targeted birth years (91% Caucasian); see Keyes et al. (2009) for a detailed overview of ES.

SIBS is a longitudinal study of adopted and biologically related sibling pairs and their parents. Adoptive families were identified through three local adoption agencies, and non-adoptive families were identified using publically available birth records. Adoptive families with adopted (permanently placed into the adoptive home prior to age 2) and biologically unrelated sibling pairs and non-adoptive families with full biological sibling pairs within the targeted age range (between ages 11 and 21, sibling pairs no more than 5 years apart in age), residing within 1 day's drive of Minneapolis, and without physical or intellectual deficiencies that could interfere with assessment were recruited. Ninety percent of eligible adoptive families and 85% of eligible non-adoptive families were located, and over 60% of located adoptive and 57% of located non-adoptive families participated. Comparing parents' education and marital status to 2000 Census data showed that the study sample was generally representative of the region from which it was sampled (McGue et al., 2007); however, adoptive parents had slightly higher SES and less psychopathology than non-adoptive parents. The restriction in range of adoptive parent SES and psychopathology had no effect on the magnitude on the adopted sibling correlations for IQ, substance use, or delinquency, supporting the use of SIBS for behavior genetic studies using important psychological variables (see McGue et al., 2007). The SIBS sample was, on average, 15 years old at the first visit, and follow-up assessments were conducted at average ages of ~18 and ~22. Retention over assessment periods was 92%. Demographics for adopted offspring reflect high rates of international adoption (67% Asian, 13% other non-Caucasian); demographics for non-adoptive families and the parents in adoptive families are representative of Minnesota during the targeted birth years (95% Caucasian); see McGue et al. (2007) for a detailed overview of SIBS.

Total sample size and ages of assessment for the present study—In total, 1,627 sibling pairs (3,254 individuals) and their parents were included in the present study. This included 789 MZ twin pairs (395 brothers, 394 sisters), 594 DZ twin pairs or full biological sibling pairs (281 brothers, 313 sisters), and 244 adopted/genetically unrelated pairs (96 brothers, 148 sisters); only same-sex sibling pairs from SIBS were included in the present study for consistency with the inclusion of same-sex twin pairs from MTFs and ES. Given our interest in adolescence, and in order to maximize the available sample size for analyses while ensuring comparability in ages across the MTFs, ES, and SIBS samples, we used data from the follow-up one and follow-up two assessments for MTFs (11 year-old cohort only) and ES ($M_{\text{ages}} = 14.9$ ($SD = .55$) and 18.1 ($SD = .63$), respectively) and from the Intake and follow-up one assessments for SIBS ($M_{\text{ages}} = 15.0$ ($SD = 1.97$) and 18.5 ($SD = 2.2$), respectively), hereafter referred to as the age 15 and age 18 assessments. Altogether, approximately 85% of the adolescents were of European ancestry, 9% of Asian ancestry, and 6% of Native American, African, or Mixed ancestry.

Measures

Parent-child conflict—Adolescents and their parents reported on the quality of the parent-child relationship at ages 15 and 18 using the Parental Environment Questionnaire (PEQ) (Elkins, McGue, & Iacono, 1997). The conflict scale of the PEQ consists of 12 items rated on a scale ranging from 1 (*Very True*) to 4 (*Very False*); sample items are, “Often there are misunderstandings between [my parent/child] and myself” and “[My parent/child] and I often get into arguments.” Cronbach’s alphas for the conflict scale of the PEQ were .89 across reporters and assessments. Mother and child reports of conflict were correlated .42 at age 15 and .45 at age 18. Father and child reports of conflict were correlated .32 at age 15 and .40 at age 18 (all correlations significant at $p < .001$). Analyses used averaged (after standardizing) mother-child and father-child reports of conflict.

MDD symptoms—MDD symptoms were assessed in semi-structured interviews with adolescents and their mothers using the Diagnostic Interview for Children and Adolescents—Revised (DICA-R; Welner, Reich, Herjanic, Jung, & Amado, 1987) for adolescents aged 15 and younger, and the Structured Clinical Interview for *DSM-III-R* (SCID; Spitzer, Williams, Gibbon, & First, 1987) for those aged 16 and older. The age 15 MDD symptom count represents a lifetime symptom count through the age 15 assessment. The age 18 MDD symptom count represents symptoms since the age 15 assessment. MDD symptoms were considered present if endorsed by either adolescents or mothers. MDD symptoms were based on *DSM-III-R* criteria to maintain continuity with the diagnostic system in use at the time of MTFS intake. Full-level symptoms were counted as 1.0 and sub-threshold symptoms were counted as .5 in the weighted MDD symptom count (symptoms were included only if initial gateway MDD criteria were met). Diagnostic interviews were conducted by extensively trained interviewers with Bachelor’s or Master’s degrees in psychology or a related discipline. All diagnostic interviews were reviewed in case conferences with at least two advanced clinical psychology graduate students, and consensus was required prior to assigning each symptom. Interrater reliability for MDD diagnoses was assessed on a randomly selected subsample of 600 MTFS participants ($\kappa = .78$ and above).

Analysis Plan—Our primary aim was to evaluate the genetic and environmental influences on the prospective association between parent-child conflict and subsequent MDD symptoms and whether these associations varied as a function of parent-child gender composition (mother-daughter, mother-son, father-daughter, and father-son pairs). To address this aim, we first conducted phenotypic cross-lagged analyses of parent-child conflict and MDD symptoms assessed in mid- and late adolescence. Next, we conducted preliminary univariate analyses of genetic and environmental influences on parent-child conflict and MDD symptoms, then multivariate analyses of genetic and environmental influences on the prospective associations between parent-child conflict and MDD symptoms over time. Consistent with prior research (see McGue & Bouchard, 1984), all phenotypes were age- and sex-adjusted prior to univariate and multivariate genetic analyses².

²As outlined in McGue & Bouchard (1984), when main effects of age and sex exist, it is recommended that they are regressed out and analyses are conducted with the residualized scores in order to reduce the overestimation of the twin/sibling correlation. It is important

Phenotypic cross-lagged analyses evaluated the degree to which parent-child conflict and MDD symptoms assessed in mid-adolescence were related to subsequent parent-child conflict and MDD symptoms in late adolescence, after accounting for auto-regressive effects (i.e., stability of parent-child conflict and MDD symptoms over time) and correlated residual variance (i.e., the correlation between parent-child conflict and MDD symptoms at each time point). For an overview of cross-lagged models, see Kenny & Harackiewicz (1979).

Phenotypic cross-lagged analyses were conducted using Mplus 7.2 (Muthén & Muthén, 1998–2014); the nested nature of the family data was accounted for using the CLUSTER specification. We were particularly interested in whether there is support for the prospective influences of parent-child conflict on subsequent MDD symptoms, as well as whether evocative (or child-driven) effects are present (i.e., the effects of MDD symptoms on subsequent parent-child conflict). Results from these analyses were used to guide subsequent multivariate genetic analyses (discussed further below).

Next, we evaluated sibling correlations and univariate genetic and environmental influences on all study phenotypes using Mx (Neale, 2006). These analyses give an overall understanding of the *total* genetic and environmental influences on each study phenotype. Genetic and environmental influences are estimated using matrix algebra based on the degree of genetic relatedness among different sibling groups. Additive genetic influence (A) refers to the influence of all genetic variants added up across the entire genome. MZ twins share 100%, DZ and full biological siblings share 50%, and genetically unrelated siblings share 0% of their additive genetic variants. Shared environmental influence (C) refers to any non-genetic influence on sibling similarity. Shared environmental estimates are assumed to be equivalent across sibling pairs. Nonshared environmental influence (E) refers to any non-genetic influence on sibling difference, and also includes measurement error (for an overview of ACE modeling, see Rijdsdijk & Sham, 2002).

Our ultimate goal was to evaluate for gender differences in the genetic and environmental influences on the associations between parent-child conflict and depression over time. To do this, we tested a multivariate Cholesky decomposition using Mx (Neale, 2006). This type of analysis is a basic variance/covariance decomposition of ACE influences where the ordering of variables has meaning. The Cholesky model was specifically chosen as it is a way to evaluate common genetic and environmental influences on the covariance of phenotypes over time, as well as account for unique ACE influences above and beyond common ACE influences. Common genetic influences between parenting and child MDD symptom phenotypes can be interpreted as supporting (passive and/or non-passive) gene-environment correlation processes. Common shared or nonshared environmental influences can be interpreted as supporting environmental causation.

We specified the ordering of variables in the multivariate Cholesky decomposition based on the results of the phenotypic cross-lagged results. When there was clear evidence for directional effects of parent-child conflict on subsequent MDD symptoms (but not for directional effects of MDD symptoms on subsequent parent-child conflict), the phenotypes

to note that regressing out the main effect of gender on each variable does not account for whether gender differences are found in the covariances between variables. We outline our analytic plan for testing for moderation by gender later in the *Analysis Plan* section.

were ordered such that parent-child conflict at Time 1 was entered first, followed by MDD symptoms assessed at Time 1 (i.e., covering lifetime MDD symptoms through the age-15 assessment), followed by parent-child conflict at Time 2, then MDD symptoms assessed at Time 2 (i.e., covering MDD symptoms between ages 15 and 18) (Figure 1 illustrates this primary model). When there was clear evidence for directional effects of MDD symptoms on subsequent parent-child conflict (but not vice versa), parent-child conflict at Time 1 was entered first, followed by MDD symptoms at Time 1, parent-child conflict at Time 2, and MDD symptoms at Time 2 (eFigure 1 illustrates the secondary model).

The multivariate Cholesky decomposition model was also chosen because specific paths (e.g., a_{21} in Figure 1) can be squared and summed to determine the proportion of ACE variance accounted for. Thus, Cholesky decomposition results can be used to calculate the percentage of ACE variance explained in the dependent variable (e.g., MDD symptoms) by the independent variable (e.g., parent-child conflict) after accounting for the stability of variables and residual associations between variables over time. For example, to determine the proportion of genetic variance explained in MDD symptoms at age 18 by parent-child conflict at age 15, path a_{41} (Figure 1) is squared, summed, and divided by all squared and summed paths predicting MDD symptoms at age 18 (a_{41} , a_{42} , a_{43} , a_{44}). Ninety-five percent confidence intervals (CIs) for each path were obtained in order to determine the statistical significance. Notably, in Mx, missing data are handled using full information maximum likelihood (FIML), which has been shown to be superior to other ways of handling of missing data (Enders & Bandalos, 2001).

The phenotypic, univariate, and multivariate analyses all followed the same general strategy. We evaluated the relationship between parent-child conflict and MDD symptoms using averaged parent and child reports, analyzed separately for mother- and father-child pairs, by child gender. Significant differences between girls and boys were tested by constraining relevant parameters to be equivalent and using the chi-square difference test to test for significant differences between the free and constrained models.

Results

Analyses of gender differences in mean levels of the study measures and their intercorrelations produced expected effects; these can be found in Tables 1 and 2. Females tended to report more MDD symptoms than males, and these differences became larger in magnitude at age 18 (Cohen's $d = -.29$) relative to age 15 (Cohen's $d = -.17$). Similar levels of parent-child conflict were reported for both sons and daughters across time (Cohen's d ranged from $-.02$ to $.06$), with the exception of father's report of greater father-son than father-daughter conflict at age 18; in this sole case, fathers tended to report more conflict with sons than daughters (Cohen's $d = .18$). A similar pattern of intercorrelations was found by child gender as well. Mother- and father-child conflict were significantly correlated with MDD both within and across time (r 's range from $.12$ to $.27$).

Phenotypic Cross-Lagged Analyses

We conducted a series of cross-lagged analyses, separately for mother- and father-child pairs, in order to examine the phenotypic associations between parent-child conflict and

MDD symptoms over time. Figure 2 shows all results from the cross-lagged analyses. In these analyses, the associations between parent-child conflict at age 15 and subsequent MDD symptoms assessed at age 18 (via past three years report), and between MDD symptoms assessed at age 15 (via lifetime report) and subsequent parent-child conflict at age 18, represented as cross-paths, are of primary interest. Results were generally comparable across girls and boys for both mother- and father-child conflict (see Panels A-B for mother-child conflict and Panels C-D for father-child conflict by child gender). The cross-path of parent-child conflict at age 15 predicting subsequent MDD symptoms assessed at age 18 was significant for girls and boys for both mother- and father-child conflict (cross-paths ranged from .09 – .13, all p 's < .01). There were no significant differences in the cross-paths across girls and boys for either mother-child conflict ($\chi^2(1) = 1.01, p = .32$) or father-child conflict ($\chi^2(1) = .21, p = .65$; detailed chi-square difference statistics for all paths are shown in the Supplementary Materials, eTable 1). Notably, the cross-path of MDD symptoms assessed at age 15 (via lifetime report) predicting parent-child conflict at age 18 was not significantly different from zero across girls and boys for either mother- or father-child conflict. Thus, phenotypic results support the use of the primary model for subsequent multivariate genetic analyses. It is also notable that the total variance explained in MDD symptoms assessed at age 18 by the phenotypic model was generally twice the proportion for females than males; this was true across mother- and father-child conflict (see Panel A vs. B, and Panel C vs. D in Figure 2).

Univariate Analyses

We conducted univariate analyses examining the total genetic and environmental influences on parent-child conflict and MDD assessed at ages 15 (via lifetime report) and 18 (via past three years report). Intraclass correlations (ICCs) for sibling pairs differing in their level of genetic relatedness and univariate estimates of ACE influence on MDD symptoms and parent-child conflict by child gender are shown in Table 3. Notably, the proportion of additive genetic influence on MDD symptoms assessed at age 15 (via lifetime report) was significant and moderate in effect for females (21%) but non-significant for males (0%), and this difference was significantly different ($\chi^2(1) = 3.90, p = .05$). The shared environmental influence on MDD symptoms assessed at age 15 (via lifetime report) was significant for males (27%) but not females (12%) and this difference was marginally significant ($\chi^2(1) = 3.38, p = .07$). There was no significant difference in the nonshared environmental influence on MDD symptoms assessed at age 15 (via lifetime report) across females (67%) and males (73%). On the other hand, there was no significant difference in ACE influences on MDD symptoms assessed at age 18 (via past three years report) across females ($a^2 = 40\%$, $c^2 = 3\%$, $e^2 = 57\%$) and males ($a^2 = 32\%$, $c^2 = 3\%$, $e^2 = 65\%$). This suggests there are gender differences in the etiology of MDD symptoms in the lifetime measurement of MDD by age 15, but not in later adolescence.

We also evaluated for differences in the total genetic and environmental influence on parent-child conflict across mother-daughter, mother-son, father-daughter, and father-son pairs; detailed results are shown in rows 5 through 12 of Table 3. Results were generally comparable across gender, with two exceptions. There was greater shared environmental influence on mother-daughter conflict at age 15 ($a^2 = 41\%$, $c^2 = 34\%$, $e^2 = 25\%$) compared

to mother-son conflict ($a^2 = 55\%$, $c^2 = 17\%$, $e^2 = 28\%$; $\chi^2(1) = 4.80$, $p = .03$). There was greater nonshared environmental influence on father-daughter conflict at age 18 ($a^2 = 37\%$, $c^2 = 20\%$, $e^2 = 43\%$) compared to father-son conflict ($a^2 = 40\%$, $c^2 = 29\%$, $e^2 = 31\%$; $\chi^2(1) = 7.30$, $p = .007$). Altogether, however, genetic and environmental influences on parent-child conflict were more similar than different across child gender.

Multivariate Analyses

Our ultimate aim was to evaluate the genetic and environmental influences on the associations between parent-child conflict and depression over time, through late adolescence. Table 4 shows results for mother-child conflict and Table 5 shows results for father-child conflict. Each table shows ACE influences on each cross-path, stability path, and residual association. Specifically, and as shown in column 1, each row corresponds to a specific path from the Cholesky decomposition, labeled in terms of cross-path, stability paths, and residual association paths. To remind the viewer, the corresponding zero-order correlations and coefficients from the cross-lagged phenotypic models (associated with the pairing of phenotypes from each row) are provided in columns 2 and 3, respectively. Of particular interest is columns 4a, 4b, and 4c, which shown the proportion of ACE influences explained in the dependent variable (e.g., MDD at Time 2) by the independent variable (e.g., parent-child conflict at Time 1)³.

Mother-child conflict—In Table 4, row 1 corresponds to the cross-path of mother-child conflict on subsequent MDD symptoms for mother-daughter dyads and row 6 refers to the same cross-path for mother-son dyads. These are the rows of primary interest as they pertain to the ACE influences on the prospective associations between mother-child conflict and MDD symptoms across time, controlling for the stability of phenotypes and residual associations at each time point, across child gender. As shown in column 4a of row 1 and 6, respectively, mother-child conflict at age 15 explained a significant proportion of additive genetic variance in MDD symptoms assessed at age 18 (via past three years report) for both girls (16%) and boys (32%). These proportions were not significantly different across child gender (see row 11, $\chi^2(1) = .69$, $p = .41$). Neither shared nor nonshared environmental covariance for the relationship between mother-child conflict at age 15 and MDD symptoms assessed at age 18 (via past three years report) was significantly different from zero for either girls or boys (see columns 4b and 4c in rows 1, 6, and 11 for details). Altogether, results are consistent with the notion that the prospective associations between mother-child conflict on subsequent MDD symptoms is predominately influenced by common genetic factors for both girls and boys.

Other notable effects concern the within-time (or concurrent residual association) effects. As shown in column 4a, there was significant additive genetic influence on the residual association between mother-child conflict at age 15 and MDD symptoms assessed at age 15 (via lifetime report) for boys (see row 9, 76% additive genetic variance explained in MDD symptoms) but not girls (see row 4, 7% additive genetic variance explained), but these

³For reference, standardized coefficients from the full multivariate Cholesky results are also shown in the Supplementary Materials, eFigures 2–5, for mother-daughter pairs, mother-son pairs, father-daughter pairs, and father-son pairs, respectively.

estimates were not significantly different across child gender; $\chi^2(1) = 2.62, p = .11$. A similar pattern was found for the within-time associations at age 18 (see rows 5 and 10). As shown in column 4c, there was a small but significant nonshared environmental influence on the residual associations at both age 15 and age 18 between mother-child conflict and MDD symptoms for girls (see rows 4–5, 3–7% nonshared environmental variance explained) but not boys (see rows 9–10, 1–2% nonshared environmental variance explained), but these estimates were not significantly different across child gender (see rows 14–15 and Table 4 for details).

Father-child conflict—Table 5 shows results for father-child conflict and follows the same general format as Table 4. Rows 1 and 6 are the rows of primary interest as they represent the ACE influences on the prospective association between father-child conflict and MDD by child gender. As shown in columns 4a, 4b, and 4c, father-child conflict at age 15 did not significantly predict genetic or environmental variance in MDD symptoms assessed at age 18 (via past three years report), for either girls (row 1: A = 3%, C = 4%, E = 2%) or boys (row 6: A = 8%, C = 1%, E = 1%).

On the other hand, and comparable to those results for mother-child conflict, there was a slightly larger additive genetic influence on the with-time residual association between father-child conflict at age 15 and MDD symptoms assessed at age 15 (via lifetime report) for boys (see row 9, 65% additive genetic variance explained in MDD symptoms) relative to girls (see row 4, 2% additive genetic variance explained) but neither estimate was significantly different than zero. A similar pattern was found for the within-time association at age 18 (see rows 5 and 10). Like results for mother-child conflict, within-assessment results also showed small but significant nonshared environmental influence on the residual associations (at both age 15 and age 18) between father-child conflict and MDD symptoms for girls (see rows 4–5, 3–6% nonshared environmental variance explained) but not boys (see rows 9–10, 1–2% nonshared environmental variance explained).

Taken together, results of the multivariate analyses suggest that the prospective association between parent-child conflict and subsequent depression in late adolescence is largely due to additive genetic influences for mother-daughter and mother-son pairs. Results suggest that father-child conflict appears to be less relevant to explaining additive genetic variance in MDD symptoms than mother-child conflict for both boys and girls. In addition to these prospective results, there are some effects of nonshared environmental influence for the residual relationships between both mother- and father-child conflict and their sons and daughters at each time point, but the total proportion of nonshared environmental variance explained was quite small (i.e., less than 5% of variance explained).

Discussion

The purpose of this study was to extend prior research and evaluate gender differences in genetic and environmental influences on the association between parenting and child depression through late adolescence (Jacobson & Rowe, 1999; Lewis et al., 2014; Neiderhiser et al., 1999; Rice et al., 2013) by considering both mother-child and father-child pairs, examining the effects of child gender, and using prospective assessment. In total,

results build on prior research demonstrating substantial genetic influences on associations between parenting and depression in childhood and adolescence for both mothers and fathers (Neiderhiser et al., 1999; Pike et al., 1996), but suggest such findings may not be as relevant to father-son and father-daughter pairs as mother-daughter and mother-son pairs. Results do not support parent-child conflict as a major source explaining gender differences in MDD (i.e., why there are higher rates of MDD in girls than in boys), as they did not show parent-child conflict was more relevant to daughter's MDD symptoms compared to sons. On the other hand, results suggest processes of gene-environment correlation involved in the prospective associations between parent-child conflict and MDD symptoms in late adolescence may be less relevant to father-child than mother-child relationships.

These results are somewhat inconsistent with earlier reports demonstrating common genetic influences on associations between both mother and father hostility (assessed during a family interaction) and adolescent depression, for both boys and girls (Lewis et al., 2014). One potential reason for this difference in findings is the difference in measurement (observed hostility versus perceived conflict), although more study is needed to better understand such differences. For example, it could be that processes of gene-environment correlation are more important for observed parent hostility in relation to children's depression, compared to parent and child perceptions of conflict.

It is important to point out that while our study was powered to detect shared environmental influence through the inclusion of the adoption design (Buchanan et al., 2009; Rutter, 2007), we found little evidence that shared environmental influences contributed to the prospective relationship between parent-child conflict and depression in late adolescence. We did find that shared environmental influences on MDD symptoms assessed at age 15 (via lifetime report) were significant and moderate in effect for boys, and less so for girls (27% versus 12% of total variance explained). Results did not show that either mother- or father-child conflict explained a significant proportion of shared environmental variance in MDD symptoms assessed at age 15 (via lifetime report) for boys or girls. This suggests factors other than parent-child conflict contribute to shared environmental influence on depression in childhood and mid-adolescence, and such factors may be especially pertinent for boys.

Our phenotypic cross-lagged results did not show support for evocative, or "child-driven" effects; MDD symptoms assessed at age 15 (via lifetime report) did not significantly predict parent-child conflict at age 18 (after accounting for the stability of parent-child conflict and MDD symptoms over time and residual correlations between parent-child conflict and MDD symptoms assessed at each time point). Taken together with results demonstrating common genetic influence for the relationship between parent-child conflict and MDD symptoms for mother-daughter and mother-son pairs, it may be that evocative gene-environment correlation is less relevant to these associations than passive gene-environment correlation. On the other hand, MDD symptoms assessed at age 15 (via lifetime report) were less common relative to later adolescence (see Table 1), so evocative effects cannot be entirely ruled out. Additionally, only one aspect of the parent-child relationship was evaluated (parent-child conflict); it may be that other aspects of parent-child and family relationships (e.g., family stressors, parent emotional support) are more relevant to evocative processes which were not evaluated here.

Altogether, study findings suggest that parent-child conflict at age 15 relates to subsequent depression in late adolescence similarly in girls and boys, and that these prospective relationships are best explained by processes of gene-environment correlation, perhaps more so for mother-child than father-child relationships. Although the etiological associations between parent-child conflict and MDD symptoms were not different across girls and boys, other forms of gene-environment interplay may be relevant to understanding gender differences in depression that were not evaluated here, such as gene-environment interaction (Lau & Eley, 2008; Rice, Harold, Shelton, & Thapar, 2006).

Notably, we found that the heritability estimate for MDD symptoms assessed at age 15 (via lifetime report) was greater in girls than boys. It may be that this greater heritability for girls is more closely linked to hormonal changes occurring earlier in adolescence and that such hormonal changes more readily associate with internalizing disorders for girls and externalizing for boys around the timing of puberty, as has been suggested by Martel (2013). On the other hand, as our age 15 measure was a lifetime measurement of MDD symptoms, it could be that symptoms occurred earlier in childhood rather than solely in adolescence. In combination with earlier research suggesting the prevalence of depression is generally rare and somewhat comparable in boys and girls in childhood (Costello et al., 2006) but that during adolescence and after we see marked differences, such that girls and women are more likely to meet clinical depression thresholds than boys and men (Costello et al., 2006; Hankin et al., 1998; Kessler et al., 2005), we hypothesize the differences we found in the heritability of MDD symptoms from the age-15 assessment may be best explained by changes in early adolescence, specifically. Nonetheless, more research is needed better understand this gender difference.

Clinical implications of this study support the utility of assessing both mother- and father-child conflict in working with children and adolescents with depression. Both were predictive of subsequent adolescent MDD symptoms, for both girls and boys. Furthermore, results suggest that mother-child conflict may be more relevant to subsequent depression through processes of gene-environment correlation rather than direct causal influence.

Strengths of this study include the prospective study design, the large sample of genetically related and unrelated sibling pairs (MZ and DZ twins, adopted siblings, biological siblings) made possible through the pooled sampling design, the inclusion of diagnostic interview to assess MDD symptoms, the use of both parent and child reports of conflict, and the inclusion of mothers and fathers and girls and boys. There are also limitations to the present study. First, the lifetime measurement of MDD symptoms by age 15 vs. the current measurement of parent-child relationship conflict at age 15 may have biased our ability to detect equivalent cross-path effects in the phenotypic cross-lagged models. An important area of future research will be to use a concurrent measure of MDD symptoms (e.g., past year). A second limitation is that the present study focused exclusively on the relationship between parent-child conflict and MDD symptoms. Although the importance of the parent-child relationship is emphasized for child and adolescent health and adjustment (see Berg-Nielsen et al., 2002; Steinberg & Morris, 2001), the zero-order correlations and results from cross-lagged analyses indicated that parent-child conflict generally had only modest cross-sectional and prospective associations with MDD symptoms assessed in mid- and late

adolescence. Thus, other sources of social influence (e.g., peer and romantic relationships) may act as more potent risk factors, and/or it may be that child depression through late adolescence is accountable by the aggregate of several social-relationship factors that individually have only modest effects. Finally, while the sample was somewhat diverse due to the inclusion of internationally adopted children, the majority of the sample was of European ancestry and reared in two-parent families. Replication and extension is needed to confirm effects across demographic groups and in a variety of family types.

In conclusion, this study showed support for a gene-environment correlation hypothesis rather than a direct causal influence concerning the prospective relationship between parent-child conflict and subsequent child depression through late adolescence. This appears to be the case more so for mother-daughter and mother-son pairs than father-daughter and father-son pairs. In total, study results add to previous literature showing the etiology of depression appears to be different by gender early adolescence (Jacobson & Rowe, 1999; Rice, Harold, & Thapar, 2002) but this does not appear to be an effect of differences in the influence of parent-child conflict (Rice et al., 2013). Additional, well-powered studies are needed to extend these findings, for example, by evaluating concurrent versus longitudinal gene-environment interaction in these relationships.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Multivariate Cholesky Decomposition: Primary Model

This Figure demonstrates the primary model, in which the genetic and environmental influences on the association between parent-child conflict at Time 1 and child MDD at Time 2 are evaluated (controlling for the genetic and environmental influences on the stability of MDD and parent-child conflict over time, and the genetic and environmental influences on the relationship between MDD and parent-child conflict at each time point). MDD = Major Depressive Disorder symptom count. Variance of each phenotype is decomposed into additive genetic effects (A1, A2, A3, A4), shared environmental effects (C1, C2, C3, C4), and nonshared environmental effects (E1, E2, E3, E4). C paths and labels are shown in gray to ease the clarity of presentation. Paths, represented by lowercase letters followed by two numbers (e.g., a11, c11, e11).

Figure 2. Results of Phenotypic Cross-Lagged Analyses: Mother- and Father-Child Conflict by Child Gender

MDD = major depressive disorder symptom count. Time 1 = mean age ~15. Time 2 = mean age ~18. Showing standardized coefficients. All coefficients are significant at $p < .05$ unless denoted in gray italics with subscript *ns* (non-significant); non-significant paths are dashed for clarity of presentation. Panel A shows results for mother-daughter pairs. Panel B shows report for mother-son pairs. Panel C shows results for father-daughter pairs. Panel D shows report for father-son pairs. For both mother- and father-child conflict, there were no significant differences in the cross-paths by child gender.

Table 1

Descriptive Statistics for Raw Study Variables

Variable	<i>M (SD)</i>		Percentage With MDD Diagnosis ^a		
	Males	Females	Cohen's <i>d</i>	Males	Females
MDD at age 15	.68 (1.79)	1.02 (2.14)	-.17*	6.1%	9.4%
MDD at age 18	.77 (1.97)	1.47 (2.73)	-.29*	7.3%	14.7%
Mother-Child Conflict at 15 (child report)	21.92 (6.44)	21.69 (7.43)	.03		
Mother-Child Conflict at 15 (mother report)	20.45 (5.85)	20.57 (6.09)	-.02		
Father-Child Conflict at 15 (child report)	21.99 (6.58)	21.59 (7.19)	.06		
Father-Child Conflict at 15 (father report)	22.39 (6.25)	22.09 (6.32)	.05		
Mother-Child Conflict at 18 (child report)	21.98 (6.54)	22.02 (7.21)	-.00		
Mother-Child Conflict at 18 (mother report)	20.75 (5.83)	20.52 (5.85)	.04		
Father-Child Conflict at 18 (child report)	22.10 (6.59)	21.92 (7.12)	.03		
Father-Child Conflict at 18 (father report)	21.82 (6.34)	20.73 (5.87)	.18*		

Notes. *M* = mean, *SD* = standard deviation. MDD = major depression disorder symptom count; MDD symptoms ranged from 0 to 9. MDD at age 15 was assessed as lifetime report by age 15. MDD at age 18 was assessed as a report of MDD symptoms since age 15.

^aDefined as meeting *DSM-III-R* diagnostic criteria for MDD.

Parent-child conflict was measured as a sum of 12 items each answered on a scale of 1 to 4; scores ranged from 12 to 48 and a higher score indicated more conflict. Significant differences in means across groups is denoted by * $p < .05$. Cohen's *d* effect size can be interpreted as small if .2–.3, medium if around .5, and large if .8+ (Cohen, 1988).

Table 2

Zero-order Correlations Among Age- and Sex-Adjusted Study Variables

Variable	1	2	3	4	5	6
1. MDD at age 15	--	.27	.20	.35	.18	.16
2. Mother-Child Conflict at age 15	.18	--	.62	.22	.61	.39
3. Father-Child Conflict at age 15	.15	.61	--	.16	.40	.53
4. MDD at age 18	.24	.14	.12	--	.24	.22
5. Mother-Child Conflict at age 18	.14	.64	.43	.21	--	.58
6. Father-Child Conflict at age 18	.16	.40	.53	.15	.58	--

Notes. MDD = major depressive disorder symptom count. MDD at age 15 was assessed as lifetime report by age 15. MDD at age 18 was assessed as a report of MDD symptoms since age 15. Parent-child conflict is the average of the standardized parent and child reports of conflict. Correlations for females ($n = 1,710$) are shown above the diagonal, correlations for males ($n = 1,544$) are shown below the diagonal. All correlations were significant at $p < .05$.

Table 3
Intraclass Sibling Correlations and Univariate Estimates for Study Phenotypes by Child Gender

Variable	MZ pairs (<i>n</i> = 789)	DZ pairs (<i>n</i> = 466)	Full Bio pairs (<i>n</i> = 128)	Unrelated pairs (<i>n</i> = 244)	a^2	c^2	e^2
MDD by Child Gender:							
1. Age 15 MDD: Girls	.31 (.21, .40)	.26 (.14, .38)			.21 (.04, .37)		.67 (.59, .76)
2. Age 15 MDD: Boys	.27 (.18, .37)	.20 (.06, .33)	.36 (.12, .56)	.36 (.17, .52)		.27 (.15, .34)	.73 (.65, .80)
<i>Test of gender difference</i>							
3. Age 18 MDD: Girls	.37 (.27, .46)	.19 (.06, .32)			χ^2 (1) = 3.90		
4. Age 18 MDD: Boys	.31 (.21, .41)	.26 (.13, .39)			.40 (.23, .51)		.57 (.49, .67)
<i>Test of gender difference</i>							
					.32 (.14, .44)		.65 (.56, .75)
Mother-Child Conflict by Child Gender:							
5. Age 15 mother-daughter conflict	.72 (.66, .76)	.55 (.45, .63)	.41 (.19, .60)	.39 (.25, .52)	.41 (.30, .51)	.34 (.24, .44)	.25 (.21, .30)
6. Age 15 mother-son conflict	.68 (.62, .73)	.45 (.33, .55)	.34 (.10, .54)	.28 (.09, .46)	.55 (.41, .70)	.17 (.03, .29)	.28 (.24, .34)
<i>Test of gender difference</i>							
7. Age 18 mother-daughter conflict	.69 (.63, .74)	.28 (.15, .40)	.34 (.09, .54)	.22 (.06, .38)	.61 (.48, .74)	χ^2 (1) = 4.80	.30 (.25, .35)
8. Age 18 mother-son conflict	.70 (.64, .75)	.38 (.25, .50)	.33 (.08, .55)		.61 (.45, .74)		.30 (.26, .36)
<i>Test of gender difference</i>							
Father-Child Conflict by Child Gender:							
9. Age 15 father-daughter conflict	.56 (.49, .63)	.50 (.39, .59)	.42 (.20, .60)	.22 (.06, .37)	.30 (.15, .45)	.29 (.17, .41)	.41 (.35, .48)
10. Age 15 father-son conflict	.57 (.49, .64)	.28 (.14, .41)	.40 (.17, .59)	.25 (.05, .44)	.35 (.16, .55)	.20 (.02, .35)	.45 (.38, .52)
<i>Test of gender difference</i>							
11. Age 18 father-daughter conflict	.51 (.43, .59)	.44 (.32, .55)		.21 (.04, .37)	.37 (.21, .53)	.20 (.06, .32)	.43 (.37, .52)
12. Age 18 father-son conflict	.69 (.63, .75)	.36 (.22, .49)	.60 (.39, .75)	.22 (.01, .42)	.40 (.25, .57)	.29 (.12, .42)	.31 (.27, .37)
<i>Test of gender difference</i>							
							χ^2 (1) = 7.30

Notes. Standardized coefficients (95% confidence intervals) from univariate analyses. MZ = monozygotic. DZ = dizygotic. Bio = biological. a^2 = total additive genetic, c^2 = total shared environmental, e^2 = total nonshared environmental (plus measurement error). MDD = major depressive disorder symptom count. MDD at age 15 was assessed as lifetime report by age 15. MDD at age 18 was assessed as a report of MDD symptoms since age 15. Coefficients are significantly different from zero if the 95% confidence interval does not cross zero; non-significant coefficients are denoted in gray italics with subscript *ns* (non-significant) for clarity of presentation. *Significant differences* in ACE influences across child gender were tested by constraining the parameter to be equivalent and using the chi-square difference (χ^2) test to compare free and constrained models (1 degree of freedom change); χ^2 3.85 is needed to achieve statistical significance at $p < .05$ with 1 degree of freedom change.

Table 4

Results from Primary Models: Zero-Order Correlations, Cross-Lagged Coefficients, and Genetic and Environmental Contributions to the Associations between Mother-Child Conflict and Major Depression across Time Points by Child Gender

1. Path	2. Zero-order Correlation	3. Coefficients from the Phenotypic Cross-Lagged Model	4. Multivariate Decomposition Results: Genetic and Environmental Variance Proportion Explained in Dependent Variable by Predictor		
			4a. A Variance Explained	4b. C Variance Explained	4c. E Variance Explained
Mother-Daughter Dyads					
1. Cross-path ($T1$ conflict $\rightarrow$ $T2$ MDD)	.22 (.09, .21)	.13 (.07, .19)	.16 (.02, .44)		
2. Conflict stability ($T1$ conflict $\rightarrow$ $T2$ conflict)	.61 (.57, .65)	.61 (.56, .65)	.52 (.36, .69)	.91 (.42, 1.0)	.10 (.04, .16)
3. MDD stability ($T1$ MDD $\rightarrow$ $T2$ MDD)	.35 (.29, .42)	.32 (.25, .39)	.36 (.06, .93)		
4. Residual association 1 ($T1$ conflict $\rightarrow$ $T1$ MDD)	.27 (.22, .33)	.27 (.22, .33)			.07 (.03, .13)
5. Residual association 2 ($T2$ conflict $\rightarrow$ $T2$ MDD)	.24 (.18, .30)	.14 (.08, .20)			.03 (.01, .08)
Mother-Son Dyads					
6. Cross-path ($T1$ conflict $\rightarrow$ $T2$ MDD)	.15 (.09, .21)	.11 (.05, .17)	.32 (.07, .95)		
7. Conflict stability ($T1$ conflict $\rightarrow$ $T2$ conflict)	.66 (.62, .70)	.66 (.62, .69)	.70 (.53, .88)		.12 (.06, .19)
8. MDD stability ($T1$ MDD $\rightarrow$ $T2$ MDD)	.24 (.15, .33)	.22 (.13, .31)			
9. Residual association 1 ($T1$ conflict $\rightarrow$ $T1$ MDD)	.18 (.12, .24)	.19 (.13, .25)	.76 (.04, 1.0)		
10. Residual association 2 ($T2$ conflict $\rightarrow$ $T2$ MDD)	.21 (.14, .27)	.16 (.09, .22)			
Test of difference in paths across Mother-Daughter and Mother-Son pairs					
11. Cross-path	10.34			NA	NA
12. Conflict stability		7.18			
13. MDD stability	14.57	5.55		NA	NA
14. Residual association 1	10.40	9.66		NA	
15. Residual association 2	6.09		NA	NA	

Notes. Zero-order correlations (95% confidence intervals) are presented in Column 2, standardized coefficients from cross-lagged analyses (95% confidence intervals) are presented in Column 3, and proportion of genetic and environmental variance explained from multivariate genetic analyses (95% confidence intervals) is presented in Columns 4 to 6. A = additive genetic, C = shared environmental, E = nonshared environmental (plus measurement error). MDD = major depressive disorder symptom count. $T1$ = Time 1 (mean age ~15), $T2$ = Time 2 (mean age ~18). Coefficients are significantly different

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Table 5

Results from Primary Models: Zero-Order Correlations, Cross-Lagged Coefficients, and Genetic and Environmental Contributions to the Associations between Father-Child Conflict and Major Depression across Time Points by Child Gender

1. Path	2. Zero-order Correlation	3. Coefficients from the Phenotypic Cross-Lagged Model	4a. A Variance Explained	4b. C Variance Explained	4c. E Variance Explained
<i>Father-Daughter Dyads</i>					
1. Cross-path ($T1$ conflict $\rightarrow$ $T2$ MDD)	.16 (.10, .21)	.09 (.03, .14)			
2. Conflict stability ($T1$ conflict $\rightarrow$ $T2$ conflict)	.54 (.49, .58)	.53 (.48, .58)	.62 (.26, 1.0)	.33 (.06, .77)	.11 (.05, .18)
3. MDD stability ($T1$ MDD $\rightarrow$ $T2$ MDD)	.35 (.29, .42)	.33 (.26, .40)	.43 (.09, 1.0)		
4. Residual association 1 ($T1$ conflict $\rightarrow$ $T1$ MDD)	.20 (.15, .26)	.21 (.15, .26)			
5. Residual association 2 ($T2$ conflict $\rightarrow$ $T2$ MDD)	.22 (.16, .28)	.16 (.10, .21)			.06 (.02, .12)
<i>Father-Son Dyads</i>					
6. Cross-path ($T1$ conflict $\rightarrow$ $T2$ MDD)	.12 (.06, .19)	.09 (.03, .15)			
7. Conflict stability ($T1$ conflict $\rightarrow$ $T2$ conflict)	.55 (.50, .61)	.55 (.49, .60)	.87 (.49, 1.0)		.06 (.02, .12)
8. MDD stability ($T1$ MDD $\rightarrow$ $T2$ MDD)	.24 (.15, .33)	.23 (.14, .31)		.87 (.21, 1.0)	
9. Residual association 1 ($T1$ conflict $\rightarrow$ $T1$ MDD)	.16 (.10, .22)	.17 (.11, .22)			
10. Residual association 2 ($T2$ conflict $\rightarrow$ $T2$ MDD)	.15 (.08, .22)	.10 (.03, .16)			
<i>Test of difference in paths across Father-Daughter and Father-Son pairs</i>					
11. Cross-path			NA	NA	NA
12. Conflict stability					
13. MDD stability	14.57	6.91			NA
14. Residual association 1	3.99		NA	NA	NA
15. Residual association 2	7.55	4.58	NA	NA	4.41

Notes. Zero-order correlations (95% confidence intervals) are presented in Column 2, standardized coefficients from cross-lagged analyses (95% confidence intervals) are presented in Column 3, and proportion of genetic and environmental variance explained from multivariate genetic analyses (95% confidence intervals) is presented in Columns 4 to 6. A = additive genetic, C = shared environmental, E = nonshared environmental (plus measurement error). MDD = major depressive disorder symptom count. $T1$ = Time 1 (mean age ~15), $T2$ = Time 2 (mean age ~18). Coefficients are significantly different

from zero if the 95% confidence interval does not cross zero; non-significant coefficients are denoted in gray italics with subscript *ns* (non-significant) for clarity of presentation. Significant differences across child gender were tested by constraining the parameter to be equivalent and using the chi-square difference test to compare free and constrained models (1 degree of freedom change).

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