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Differential Parent–Child Relationships and Adolescent Externalizing Symptoms: Cross-Lagged Analyses Within a Monozygotic Twin Differences Design

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Abstract

Research has indicated that differential parental treatment is linked to differences in externalizing symptomology (EXT) across siblings, even those siblings who are genetically identical. However, the direction of causation and longitudinal significance of this relationship remains unclear. Thus, in the present study, the authors examined 486 monozygotic twin pairs, assessed at ages 11, 14, and 17 years, within a cross-lagged twin differences design. Results revealed that differential parent–child conflict at age 11 years uniquely contributed to differential sibling EXT 3 years later but only in the most discordant twin pairs. In the full, unselected sample, this relationship was not significant. These results suggest that markedly different parent–child conflict has an environmentally mediated impact on child behavior through mid-adolescence, findings that yield insights into environmental influences on behavior.

Keywords

parent-child relationships; environmental influences; twin differences; externalizing disorders

In 1987, Plomin and Daniels published a seminal and widely cited article in which they noted that the largest environmental contributions to behavioral outcomes occur at the child-specific (i.e., nonshared environment) level rather than at the family-wide (i.e., shared environment) level. They consequently reasoned that, to the extent that parents influence children's outcomes, they likely do so at this child-specific level. To examine this provocative hypothesis, they charged researchers with the following tasks: (a) identify differential experiences among siblings in the same family; (b) relate these differential experiences to differences in sibling behavior; and (c) determine the causal nature of the relationship between differential experiences and differences in sibling behavior.

To date, researchers have established both that there are significant sibling differences in parental treatment within families (Feinberg & Hetherington, 2001; Reiss et al., 1995) and that these differences are predictive of child and adolescent outcomes (Anderson, Hetherington, Reiss, & Howe, 1994; Conger & Conger, 1994; Feinberg & Hetherington, 2001; Kowal, Krull, & Kramer, 2004). For example, parent–child conflict appears to contribute to adolescent adjustment largely at the child-specific level (Anderson et al., 1994), results that appear to

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persist over time (Conger & Conger, 1994). Specifically, researchers found that the adolescents treated in a more hostile fashion by their parents in early adolescence exhibited relatively more delinquent behavior during mid-adolescence, even when controlling for the association between hostility and delinquent behavior during early adolescence. Although these studies certainly establish a role for differential parental treatment in child outcomes, their interpretative significance is limited by two issues. First, the mechanisms through which differential parental treatment/parent–child relationships affect child outcomes have yet to be firmly resolved. Although they still remain unclear, researchers recently have offered one possibility, suggesting that social comparisons between the child's own treatment and that of his or her sibling may lead to attributions regarding who is the favored child (Baker & Daniels, 1990; Kowal et al., 2004; McHale, Crouter, McGuire, & Updegraff, 1995).

A second more critical interpretive concern, however, is that genetic influences on child/adolescent outcomes were not fully controlled. Accordingly, it remains possible that the child's predisposition towards misbehavior is eliciting differential parental treatment, at least in part. It thus remains unclear whether these effects represent true nonshared environmental influences. Recently, however, researchers have begun to evaluate the impact of differential parental treatment, making use of a *monozygotic (MZ) differences design* (Deater-Deckard et al., 2001; O'Connor, Hetherington, Reiss, & Plomin, 1995). This design capitalizes on the unique features of MZ (identical) twins, specifically that they share both 100% of their genes and their shared environment. As differences between MZ twins, accordingly, cannot be confounded by these factors, this design enables researchers to evaluate more explicitly whether differences in sibling treatment were *environmentally* linked to child-specific behavior problems.

Asbury, Dunn, Pike, and Plomin (2003) used this design in a large sample of 4-year-old MZ twin pairs ($n = 2,353$) to examine whether sibling differences in parental treatment were linked to child-specific behavior problems. Of importance, however, they also explicitly evaluated whether extreme sibling differences in parental treatment (and extreme differences in sibling behavior problems) potentiated this nonshared environmental relationship. They found that differential parental treatment accounted for 3% of the variance in differential sibling outcomes across all participants but accounted for 11% of the variance in highly discordant pairs. Furthermore, they found that differences in child conduct problems had a somewhat stronger association with differential parental treatment in more chaotic homes and with more depressed mothers. Such findings suggest that the impact of the nonshared environment is most pronounced when differences in sibling experiences are extreme or take place in higher risk environments.

Researchers also have begun to examine the predictive value of the measured nonshared environment over time using the MZ differences design. Caspi et al. (2004) examined associations between differences in observed maternal expressed emotion at age 5 years and differences in child antisocial behavior at age 7 years (controlling for prior antisocial behavior at age 5 years) in 565 MZ twin pairs. They found that, regardless of informant, differences in maternal expressed emotion predicted differences in child antisocial behavior, accounting for roughly 1%–2% of the variance 2 years later. As they are longitudinal, such findings offer the strongest evidence to date of measured nonshared environmental influences on child outcomes.

However, although the studies reviewed above make strides toward resolving the impact of the nonshared environment on behavioral outcomes, both the persistence of these influences over time and the direction of causation between them remain unclear. First, as noted by Caspi et al. (2004), they controlled for continuity in antisocial behavior over time but were not able to control for similar continuity in maternal expressed emotion (as it was not assessed at age 7 years). Accordingly, it remains possible that the association between maternal expressed

emotion and child antisocial behavior is, in part, a function of the stability of maternal expressed emotion. Thus, it is necessary for researchers to determine whether the longitudinally predictive aspects of the measured nonshared environment identified in Caspi et al. (2004) persist over and above the stabilities of both parenting and antisocial behavior.

Second, as noted by Asbury et al. (2003) and Caspi et al. (2004), studies of nonshared environmental effects typically interpret their findings within a parent-driven framework such that differential parental treatment *causes* sibling differences in child outcomes. However, these conclusions may be premature. In numerous studies (both phenotypic studies and those examining genetic and environmental influences), researchers have found evidence of bidirectional associations between parental treatment and child outcomes (Burt, McGue, Krueger, & Iacono, 2005a; Campbell, Pierce, Moore, Marakovitz, & Newby, 1996; Neiderhiser, Reiss, Hetherington, & Plomin, 1999). Reciprocity therefore remains a possibility for nonshared environmental effects, as well. For example, it may be that differential environmental experiences (e.g., obstetrical complications, peer influences) elicit differences among genetically identical children, which then elicit differential parental treatment. However, we know of no MZ twin difference study in which researchers have examined the respective influence of both child- and parent-driven pathways on nonshared environmental relationships. Thus, we cannot be sure whether these effects are indeed parent driven.

With the goal of further broadening our understanding of the measured nonshared environment, the current study examined sibling differences in parent–child conflict and in conduct disorder (CD) and oppositional defiant disorder (ODD) symptoms (collectively referred to as *externalizing symptoms*; EXT) at ages 11, 14, and 17 years within a sample of MZ twins. We used a cross-lagged model (Plewis, 1985), enabling us to examine the direction of causation between sibling differences in parent–child conflict and EXT over time, regardless of their preexisting relationships. Analyses were conducted on the full, unselected sample and on highly discordant twins (Asbury et al., 2003). In this way, we evaluated whether and how differential parent–child relationships and adolescent EXT affected one another over time at a solely environmental level and whether this association was a function of the severity of sibling differences.

Method

Participants

The sample was drawn from participants in the ongoing Minnesota Twin Family Study (MTFS). The MTFS is a population-based, longitudinal study of same-sex adolescent twins born in Minnesota and their parents. More than 90% of twin births between 1971 and 1985 were located through the use of public databases. Participating families were broadly representative of the Minnesota population at the time the twins were born; approximately 98% are Caucasian. Further information regarding the design, recruitment procedures, participation rates, and zygosity determination procedures of the MTFS can be obtained elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999).

Participants in the current research ranged in age from 10 years to 12 years, averaging age 11 years, at the time of their intake visit. Our sample consisted of 974 (508 male, 466 female) same-sex, reared-together MZ twins (487 pairs). Of these participants, 476 (94%) of the boys and 436 (94%) of the girls completed the first follow-up assessment approximately 3 years later. Roughly 3 years after that, 415 (82% of the original sample) male participants and 409 (88% of original sample) female participants completed the second follow-up assessment. Those who did not complete the follow-up assessments did not have more mental health problems or parent–child conflict than those who did complete follow-up (McGue, Elkins, Walden, & Iacono, 2005a).

Assessment of Mental Disorders

During their intake and follow-up visits, all participants and their parents were assessed in-person for *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., revised; *DSM-III-R*; American Psychiatric Association, 1987) mental disorders (*DSM-III-R* was current at the study's onset) by trained bachelor's- and master's-level interviewers. CD and ODD were assessed through use of the *Diagnostic Interview for Children and Adolescents-Revised* (DICA-R; Reich & Welner, 1988). The MTFS version of this instrument contained supplementary probes and questions, which we added after consultation with one of the authors of the *DICA-R* to ensure complete coverage of each symptom. Mothers reported on symptom presence separately for each twin; twins reported only on themselves. Family members were interviewed by separate interviewers. At intake, the reporting period was the twin's lifetime. At both follow-up assessments, only symptoms present during the last 3 years provided the basis of the symptom count.

Two *DSM-III-R* symptom count variables were used in the present study. These variables corresponded to the nine Criterion A symptoms of ODD and 12 of the 13 Criterion A symptoms of CD (the exception was Symptom 9, "has forced someone into sexual activity with him or her"). Following the interview, a clinical case conference was held in which the evidence for every symptom was discussed by at least two advanced clinical psychology doctoral students. Mother and child interviews were not discussed during the same clinical case conference. Only symptoms that were judged to be clinically significant in both severity and frequency were considered present. As actual diagnoses were not used, duration rules were excluded for both disorders.

After assigning clinically significant symptoms, we used computer algorithms to sum the number of assigned symptoms using a combined informant approach. Specifically, a symptom was considered present if it was endorsed by either the mother or the child. Symptoms endorsed by both mother and child were counted as only one symptom. The use of this combined informant approach allowed for a more complete assessment of symptomatology than would the use of either informant alone, as previous studies have indicated that each informant contributes a considerable amount of uniquely predictive information (Achenbach, McConaughy, & Howell, 1987; Burt, McGue, Krueger, & Iacono, 2005b). Of note, although mothers reported on ODD symptom presence at all three assessments, they reported on CD symptom presence only at ages 11 and 14 years. Twins reported on their own CD and ODD symptom presence at all three assessments. Therefore, CD symptoms at age 17 years were indexed only by twin reports, whereas those CD symptoms at ages 11 and 14 years were indexed by both informants. Sample sizes at each wave of assessment are presented in Table 1.

We then summed the CD and ODD symptom counts to create an overall measure of EXT. To create the signed EXT symptom difference scores used in the final analyses, we subtracted the symptom count of the second born (Twin B) from that of the firstborn (Twin A). Accordingly, at any given assessment, we included diagnostic data only if it was present on both members of the twin pair. At intake, data were available on all 486 pairs. At Follow-ups 1 and 2, data were available on 453 and 391 pairs of twins, respectively.

Assessment of the Family Environment

We administered the Parental Environment Questionnaire (PEQ) to tap perceptions of the parent-child relationship. Mothers and twins rated 50 items assessing aspects of their relationships on a 4-point scale (1 = *definitely true*, 4 = *definitely false*). The mothers rated their own relationships with each twin, and twins each rated their relationship with their mothers and fathers. Items were essentially the same for both mothers and twins, with alterations in wording appropriate for particular raters. This questionnaire was developed for

use by the MTFS (see Elkins, McGue, & Iacono, 1997) and has been factor-analyzed and shown to assess, among other things, parent–child conflict. The Parent–Child Conflict scale comprised 12 items (see Table 2 for a list of items on this scale). In a normative sample, such as the present one, this questionnaire likely assesses parental criticism. The internal consistencies for this scale at age 11 years in the current sample (and at age 17 years in the older cohort of the MTFS, which is not used in the present study) ranged between .81 and .88 for twin and parent informants. The consistency of the alphas across ages and across independent samples strongly suggests that conflict is a consistently reliable construct.

PEQs were mailed to families prior to their onsite assessments. Participants were asked to bring their completed PEQ with them to their in-person visit. If a completed PEQ was not obtained by the end of the assessment, participants were asked to complete it at home and return it by mail. One telephone prompt was made if a completed PEQ still was not received. If one item was missing, that item was prorated and added to the scale score. If two or more items were missing, the scale score was coded as missing.

In creating the conflict variable, we averaged all informant reports (Burt, Krueger, McGue, & Iacono, 2003). However, to ensure that twin reports were not weighted more heavily than were mother reports, we first averaged twin report of mother and twin report of father. This decision is bolstered by the very high intercorrelations between twin reports of mother and father (ranging from .73 to .89 across assessments). We allowed one of these reports to be missing. We then averaged the twin composite and the mother report of twin to capture more fully the relational aspect of parent–child conflict (correlations between mother and twin informant reports were .28, .28, and .23 at ages 11, 14, and 17 years, respectively). Previous work in this sample has indicated that both mother and twin informant reports of parent–child conflict independently contribute to teacher-reported grades and behavior problems, indicating that each informant provides unique and predictive information that is not provided by other informants (as reported in Burt et al., 2005a). To maximize the number of participants with conflict data, we allowed for missing twin or mother data. Sample sizes at each wave of assessment are presented in Table 1.

To create the signed conflict difference scores used in the final analyses, we again subtracted the conflict score of Twin B from that of Twin A. Accordingly, at any given assessment, we included conflict data only if they were present on both members of the twin pair. Thus, at intake, conflict data were available on 439 pairs. At Follow-Ups 1 and 2, data were available on 447 and 401 pairs of twins, respectively.

Statistical Analyses

MZ twins share 100% of their genetic material as well as 100% of those family-wide environmental forces that act to further increase their similarity. Accordingly, differences between these reared-together twins are due only to child-specific or unique environmental influences (and measurement error), making this sibling-differences approach a direct estimate of nonshared environmental forces (i.e., e^2).

Although we were missing only a relatively small amount of data, we used Full-Information Maximum-Likelihood (FIML) raw data techniques, which produce less biased and more efficient and consistent estimates than do other techniques, such as pairwise or listwise deletion, in the face of missing data (Little & Rubin, 1987). We used AMOS, a structural equation modeling program (Arbuckle, 2003), to fit cross-lagged models to the observed raw data (see Figure 1). For better approximation of normality, the symptom count variables were individually Blom-transformed and rank-normalized, separately by sex, prior to model-fitting, a procedure that was found to optimize model selection (van den Oord et al., 2000). This

procedure involved replacing raw symptom counts with their rank values. Ties were assigned the mean rank of the tied values.

The model requires all cross-age associations to function as partial regression coefficients. The cross-age but within-trait coefficients (i.e., b_{11} , b_{22} , b_{33} , b_{44}) index the stability of differences in conflict and EXT over time, controlling for the cross-lagged contributions of the other trait. The cross-lagged coefficients (i.e., b_{12} , b_{21} , b_{34} , b_{43}) allowed us to determine whether differences in conflict and EXT at ages 11 and 14 years independently affected one another at ages 14 and 17 years, controlling for both stability and any cross-lagged contributions of the other trait. Next, we were able to evaluate the relationship between differences in conflict and differences in EXT at each age via age-specific correlations (i.e., r_1 , r_2 , r_3). Because of the stability and cross-lagged coefficients, these correlations functioned as residuals at ages 14 and 17 years, thereby revealing the age-specific association between differences in conflict and EXT independent of their preexisting association. Any path or correlation greater than zero is indicative of nonshared environmental mediation. Modeling analyses were conducted on signed sibling differences, thereby allowing us to evaluate the direction of any significant effects.

In spite of its utility for examination of the mechanisms underlying child-specific environmental associations over time, this design has not, to our knowledge, been used previously to examine MZ twin differences in either unselected or highly discordant twin samples. In the current study, we attempted to remedy this situation, conducting these cross-lagged analyses on the full, unselected sample of MZ twin pairs ($n = 486$) and then repeating these analyses (separately) for those twins most discordant for EXT symptomology (top 25%) and those twins most discordant for parental conflict (top 25%). In choosing our cutoff point, we attempted to balance both the need for a discordant sample large enough to permit statistical testing and the need for a sufficiently discordant sample. Thus, we chose a difference approximating one standard deviation as the cutoff point (i.e., twins discordant by three or more EXT symptoms, $n = 124$ pairs, or by six or more points on the conflict scale, $n = 120$ pairs) but used a percentile threshold to ensure that the analyses of EXT and conflict were based on similarly sized samples. Roughly one third of the pairs ($n = 41$) overlapped across the two discordant samples, a finding that is not surprising given the robust phenotypic relationship between conflict and EXT (Burt et al., 2005a).

The moderating effects of gender also were examined in these analyses, as it is well-known that male adolescents display a higher frequency of externalizing behaviors than do female adolescents. Previous analyses have not revealed evidence of sex differences in estimates of genetic and environmental contributions to the variance in these traits (Burt et al., 2005a; Rhee & Waldman, 2002), and hence, we expected that we would find little evidence for gender differences within the current model.

Results

To index the severity of externalizing symptomatology, we computed mean combined symptom counts separately by gender and age (see Table 3). Across all ages, independent-sample t tests indicated that the mean EXT symptom counts and conflict scores differed significantly by gender ($p < .01$), with boys having higher symptom counts and more conflict than girls. Furthermore, paired-sample t tests indicated that in boys, EXT symptom counts increased from age 11 years to age 14 years and decreased from age 14 years to 17 years. In girls, there were no changes in EXT symptom prevalence with age. Conflict scores increased from age 11 years to age 14 years for girls but did not differ across assessment for boys. These statistics of central tendency and range collectively indicate that boys in this sample were more symptomatic and had more conflictive relationships with their parents than did girls. These

statistics also suggest that there is a great deal of growth in externalizing symptomatology in early adolescence that either stabilizes or dissipates by the end of adolescence.

Consistent with the overall growth in these behaviors from early adolescence to mid-adolescence, sibling difference scores in conflict increased from age 11 years and age 14 years for both boys and girls (see Table 3; please note that we present the absolute values of the difference scores to highlight the magnitude of sibling differences. However, to permit examination of the direction of effects, we conducted final analyses on signed sibling differences). Boys also exhibited an increase in sibling EXT difference scores from age 11 years to age 14 years. However, from age 14 years to age 17 years, this increase in sibling differences appeared to plateau, as there was no appreciable change in sibling difference scores for either EXT or conflict in girls or boys.

Correlations

Full, unselected sample—Correlations were computed among MZ sibling differences in conflict and in EXT symptoms from age 11 years to age 17 years (see Table 4). As mentioned, nonsignificant correlations are indicative only of a lack of nonshared environmental (E) mediation rather than a lack of association between those phenotypes in general (indeed, at the phenotypic level, conflict and EXT were correlated between .32 and .44, $ps < .001$, at ages 11, 14, and 17 years). In boys, there was evidence of E-mediated within-trait stability for EXT from age 11 to age 14 years and from age 14 to age 17 years. There was also evidence of E-mediated within-trait stability for conflict from age 14 years to age 17 years. The age-specific relationships between difference scores in EXT and conflict at ages 11 and 14 years were similarly suggestive of E mediation. Similar relationships were found for girls. When the male and female samples were combined, there was evidence of E mediation of the cross-trait, within-age association between EXT and conflict difference scores at all three ages. Further, there was some evidence of E-mediated within-trait stability for both phenotypes.

Most discordant pairs—The pattern of results was remarkably similar for twin pairs highly discordant for EXT and for those highly discordant for conflict (see Table 5). In both samples, the relationships between EXT difference scores at ages 11 and 14 years and at ages 14 and 17 years are suggestive of E mediation, as are those for conflict. The age-specific relationships between difference scores in EXT and conflict at ages 11 and 14 years were also significant (those at age 17 years were approaching significance, as well). Finally, the relationship between conflict at age 11 years and EXT at age 14 years was indicative of E mediation in both samples.

Multivariate Modeling

Because the variables were standardized to have a mean of zero and a standard deviation of 1 separately by gender, we first constrained the variances in each variable to be equal across gender. This procedure resulted in a chi-square change of less than 0.1 on six degrees of freedom. After this procedure, the cross-lagged model was fit, thus allowing for differences in parameter estimates across gender, $\chi^2(14, N = 486) = 10.05$, Akaike information criterion (AIC) = 90.05, and constraining the parameter estimates to be equal across gender, $\chi^2(25, N = 486) = 18.82$, $p = .240$, AIC = 78.80. The improved fit of the no-gender-differences model, as indicated by a smaller AIC value, suggests that although boys are more symptomatic than girls, the nonshared environmental relationship between difference scores in parenting and EXT does not vary across gender. Further, the no-gender-differences model provided an excellent overall fit to the data with a root-mean-square error of approximation (RMSEA) of .00.

Figure 2 presents the standardized path diagram for the no-gender-differences model for the full, unselected sample. The percentage of variance accounted for by a given path can be obtained by simply squaring the path coefficient. Sibling differences in EXT symptoms at ages

11 and 14 years (i.e., b11, b33) evidenced some stability over time, predicting 2.0% and 4.5% of the variance in sibling differences in EXT, respectively, 3 years later. Sibling differences in conflict evidenced significant stability only from age 14 years to age 17 years (i.e., b44), predicting 7.7% of the variance at age 17 years. However, in no case did sibling differences in EXT or conflict predict the other 3 years later. Specifically, the cross-lagged paths (i.e., b12, b21, b34, b43) uniformly accounted for less than 1.0% of the variance. In contrast, the significant age-specific correlations among the traits (i.e., r1, r2, r3) suggest that sibling differences in conflict and EXT are uniquely correlated at each age and account for 2.0%–3.8% of the other's variance. Moreover, as the age 14- and 17-year correlations are residuals and are only minimally smaller than their corresponding interclass correlations (as presented in Table 4), the results suggest that most of the association between sibling differences in conflict and EXT is age specific, at least within unselected samples.

When we examined the pairs most discordant for EXT and conflict, respectively (see Figure 3), we noted that the results were generally similar to, if somewhat larger in magnitude, than those of the unselected sample. Sibling differences in EXT symptoms again evidenced some stability across all assessments, predicting 3.3%–6.7% of the variance 3 years later. Sibling differences in conflict were stable only from age 14 years to age 17 years, predicting between 4.8% and 10.8% of the variance at age 17 years. In addition, significant correlations at each age again suggest that sibling differences in conflict and EXT continue to evidence unique, age-specific associations. Of importance, however, there was evidence of a cross-lagged association within the most discordant pairs, regardless of grouping. For pairs highly discordant on EXT and pairs highly discordant on conflict, differential parent–child conflict at age 11 years uniquely predicted differential EXT at age 14 years, accounting for 3.8%–6.7% of the variance. Moreover, the association was positive, such that the twin with more parent–child conflict engaged in higher levels of EXT behaviors 3 years later. However, the association appeared to be specific to the developmental period of early adolescence, as the association did not persist from age 14 years to age 17 years.

Discussion

The aim of the present study was to evaluate whether and how child-specific environmental processes affected the association between parent–child conflict and adolescent externalizing behavior over time. To do so, we examined MZ twin differences in conflict and EXT symptoms from age 11 years to age 17 years within a cross-lagged model. Final analyses revealed two major findings. In the unselected sample, the cross-lags were uniformly nonsignificant, and the residual correlations at ages 14 and 17 years were only minimally smaller than their corresponding interclass correlations. Such results collectively indicate that, at the population level, most of the child-specific association between differential parent–child conflict and differences in adolescent outcomes is age specific and does not persist over time. However, this conclusion does not hold for the most discordant twin pairs. For twins highly discordant in either EXT or conflict, we found that the twin with more parent–child conflict engaged in higher levels of EXT behaviors 3 years later, although this association was specific to the developmental period of early to mid-adolescence. Further, this association was not a function of a preexisting association between the traits, their stability over time, or a misidentified *child-driven effect* (in which the child's EXT behavior is eliciting, rather than being elicited by, parent–child conflict). Instead, these findings indicate that markedly different levels of parent–child conflict at age 11 years exert an environmentally mediated influence on adolescent misbehavior at age 14 years, even when controlling for the adolescents' genetically influenced characteristics.

Our results are notably consistent with those of other studies of the nonshared environment. In the full sample, we found that differential parent–child conflict cross-sectionally accounted for

between 2.0% and 3.8% of the total variance in EXT differences. Such findings are quite similar to cross-sectional results reported in other unselected samples (Deater-Deckard et al., 2001; Turkheimer & Waldron, 2000). Further, although these results are based on only MZ twins, the specific proportions of nonshared environmental variance in the unselected cross-lagged effects reported here are virtually identical to those found in Burt et al. (2005a). Of note, Burt et al. (2005a) did not independently examine highly discordant siblings, so we cannot be sure how these findings would compare. Finally, our highly discordant twin results are quite similar in magnitude to those reported in Asbury et al. (2003), offering confirmation that their findings are not solely a function of the cross-sectional nature of their design.

In contrast, our unselected sample findings do differ from those of Caspi et al. (2004), who found that differential maternal expressed emotion at age 5 years accounted for 1%–2% of the differences in antisocial behavior at age 7 years, controlling for antisocial behavior at age 5 years (across their full sample). The discrepancy between these results and those of the present study may be a function of the different age ranges of the two samples (early childhood vs. adolescence), as maternal influences may be stronger in early childhood than in adolescence. Alternately, it may be that their results, in part, reflect the stability of expressed emotion from age 5 years to age 7 years, which was not assessed in their sample.

The present study has several limitations. First, our measure of conflict was not based on direct observation but rather on mother and child self-reports. Although it remains unclear how these results would have changed with the use of observer-reported conflict, it is noteworthy that the percentages of nonshared environmental variance accounted for in behavioral outcomes are essentially the same when researchers use either observer-rated or self-reported parenting variables (Caspi et al., 2004; Deater-Deckard et al., 2001). Similarly, we were unable to examine differential parenting by mothers and fathers, as child self-reports did not sufficiently differentiate between parents. Future research should examine both observer-rated conflict and self-reports from both mothers and fathers.

Second, CD was assessed only in adolescents (i.e., no mother report) at age 17 years but was assessed through use of both mother and child reports at ages 11 and 14 years. This may have attenuated the magnitude of the EXT stability coefficient observed from age 14 years to age 17 years. However, reanalyzing the data without maternal report of CD at ages 11 and 14 years did not substantively alter the stability coefficients. Third, the origins of the extreme differences in EXT observed between MZ twins at age 11 years remain unexplored. Thus, future researchers should examine childhood precursors of highly discordant MZ twin pairs, with a focus on environmental experiences that are likely to differ between twins (i.e., obstetrical complications, peer influences, and parenting).

Next, although the notion that genes play at least a modest role in child development has gained acceptance in recent years (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000), the statistical techniques and assumptions (i.e., the Equal Environments Assumption; EEA) underlying traditional behavioral genetic methodology have been criticized on both ideological (Greenberg, 2005) and interpretive (Greenberg, 2005; Partridge, 2005) grounds. In particular, both Greenberg (2005) and Partridge (2005) questioned the interpretive significance of genetic proportions of variance. Although they were thoughtful in their critiques, we would argue that many of their concerns are either overstated in importance or do not accurately reflect the methodologies and techniques presently used by many behavioral geneticists (see McGue, Elkins, Walden, & Iacono, 2005b, for a detailed response to these criticisms). Moreover, a major limitation of the Greenberg (2005) and Partridge (2005) critiques is that they fail to provide a framework for exploring gene–environment interplay in adolescent development (cf. Rutter, Silberg, O'Connor, & Simonoff, 1999). In the current study, we used a novel behavioral genetic design (i.e., a longitudinal study of MZ twin differences) to investigate the role of the

parent–adolescent relationship in adolescent misbehavior, thereby highlighting the important role that behavioral genetics research has to play in developmental psychology.

Finally, although differences between genetically identical (and accordingly, like-gendered and like-aged) siblings offer researchers the opportunity to directly study nonshared environmental processes, the absence of these confounds also reduces the generalizability of these findings to other types of sibling relationships. Of importance, however, differential parent–child conflict and differential parental hostility have been linked to differential sibling outcomes, including delinquency, in studies that included other types of sibling pairs (i.e., full siblings, half siblings; Anderson et al., 1994; Conger & Conger, 1994; Feinberg & Hetherington, 2001; Kowal et al., 2004). Thus, the present results are thought to generalize to other types of sibling relationships and, in doing so, provide an important extension of prior research.

In spite of these limitations, the results of the current study yield several conclusions. First, our findings support recent suggestions (Rutter et al., 1999; Turkheimer & Waldron, 2000) that within low-risk, unselected populations, measured nonshared environmental factors may be less powerful influences on human behavior than was originally proposed (Plomin & Daniels, 1987). For the full sample of identical twin pairs, the associations between sibling differences in EXT and conflict at age 14 years and at age 17 years were not a function of their previous associations nor did they persist over time. Moreover, the vast majority of variance in both differential parent–child conflict and differences in sibling EXT was not explained in our analyses, results that collectively argue against potent identifiable nonshared environmental influences on adolescent outcomes at the population level.

However, rather than further weakening our confidence in the importance of nonshared environmental influences, our results are thought to augment their role. When we restricted our analyses to twin pairs discordant in either EXT or conflict by one standard deviation or more, we found that higher levels of parent–child conflict relative to one’s sibling predicted increases in child externalizing symptomatology 3 years later (although this effect did not persist through late adolescence). Such findings indicate that nonshared environmental influences may be attenuated in unselected samples.

Collectively, these results have two important implications. First, they highlight the salience of the parent–child relationship on early adolescent behavior, thereby suggesting that previous interpretations of nonshared environmental influences as largely parent driven are indeed accurate (Asbury et al., 2003). However, as this association was not replicated from age 14 years to age 17 years, these findings also may suggest a weakening in the salience of the parent–child relationship in regards to adolescent behaviors by late adolescence. This interpretation is consistent with the hypothesized decrease in environmental influences and the simultaneous increase in genetic influences across adolescence and into adulthood (Scarr & McCartney, 1983), a hypothesis that has been supported for antisocial behavior (Lyons et al., 1995) and for parent–child relationships (McGue et al., 2005a). Specifically, it appears that as individuals age and strive toward independence, their genetic predispositions are more fully manifested (i.e., increase in magnitude), whereas the impact of the environment (and, likely, the home environment in particular) on the individual’s behavior becomes less salient.

Second, the present findings expand researchers’ understanding of the impact of environmental influences on psychopathology in general. Behavioral geneticists have long maintained that their findings of moderate-to-large genetic influences likely apply only to the “average, expectable” environment, arguing that more extreme or unfavorable experiences are more likely to exert direct and sustainable environmental influences (Scarr & McCartney, 1983). However, little proof of this assertion actually has been offered. Our findings provide some

circumstantial support for this conjecture, such that only more extreme differences in the parent–child relationship acted as “environmental main effects” on adolescent outcomes. Future researchers should seek to clarify further the role of the nonshared environment on behavioral outcomes.

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Figure 1.

Cross-lagged model of sibling difference scores in externalizing symptoms (EXT) and parent-child conflict (CON) across ages 11, 14, and 17 years. Cross-age paths (i.e., partial regression coefficients) are indicated by a “b” followed by two numerals. Within-age correlations are indicated by an “r” followed by a single numeral. The residual variance in conflict and EXT difference scores at ages 14 and 17 years is represented by an “e” followed by a single numeral.

Figure 2.

No-gender-differences model for full, unselected sample. Externalizing symptoms (EXT) and parent-child conflict (CON) represent sibling difference scores in externalizing symptoms and conflict scores at ages 11, 14, and 17 years. For single-headed arrows (i.e., paths), the standardized regression coefficients are presented. For double-headed arrows, the correlation is presented. ** $p < .01$, two-tailed. * $p < .05$, two-tailed.

Figure 3.

Models for the twin pairs most discordant on externalizing symptom count (top 25%) and most discordant on family conflict (top 25%). Externalizing symptoms (EXT) and parent–child conflict (CON) represent sibling difference scores in externalizing symptoms and parent–child conflict scores at ages 11, 14, and 17 years. Standardized cross-lagged results for highly discordant twin pairs are presented (those highly discordant for EXT are first, followed in parentheses by those highly discordant for CON). For single-headed arrows (i.e., paths), the standardized regression coefficients are presented. For double-headed arrows, the correlation is presented. All significant paths/correlations were significant for both models (i.e., for those twins highly discordant on EXT and those highly discordant on CON). * $p < .05$, two-tailed.

Table 1

Monozygotic Twin Sample Sizes

Phenotype	Age of female participants (years)			Age of male participants (years)		
	11 (n = 466)	14 (n = 436)	17 (n = 409)	11 (n = 508)	14 (n = 476)	17 (n = 415)
Externalizing symptoms						
Child report	465	433	405	505	465	410
Mother report	466	425	382	506	470	405
Combined ^a	466	436	405	506	476	410
Parent-child conflict						
Child report	445	422	362	452	431	380
Mother report	402	410	356	428	423	376
Combined ^a	451	435	409	461	466	415

^a Reflects the input of multiple informants (when available).

Table 2

Items on the PEQ Parent–Child Conflict Scale

-
1	My parent often criticizes me.
2	Before I finish saying something, my parent often interrupts me.
3	My parent often irritates me.
4	Often there are misunderstandings between my parent and myself.
5	I treat others with more respect than I treat my parent.
6	My parent often hurts my feelings.
7	My parent does not trust me to make my own decisions.
8	My parent and I often get into arguments.
9	I often seem to anger or annoy my parent.
10	My parent often loses her/his temper with me.
11	My parent sometimes hits me in anger.
12	Once in a while I have been really scared of my parent.
-

Note. Items presented here compose the twin version of the questionnaire. For the parent version, items were essentially the same, with alterations in wording appropriate for parental informants (e.g., “I sometimes hit my child in anger”). Mothers and twins each rated these items on a 4-point scale (1 = *definitely true*; 4 = *definitely false*). The items were the same at both intake and follow-up assessments. PEQ = Parental Environment Questionnaire.

Table 3

Mean Level of Individual Externalizing Symptoms and Parent–Child Conflict Scores at Ages 11, 14, and 17, and Sibling Differences in Those Scores

Individual scores ^a						Absolute sibling difference scores ^b				
Measure	M	SD	Min	Max	n	M	SD	Min	Max	n
EXT_11	2.31	2.47	0	18	Male participants 506	1.18	1.52	0	12	253
EXT_14	3.64*	3.48	0	19		1.48*	1.76	0	10	237
EXT_17	2.74*	2.77	0	15		1.45	1.77	0	10	197
CON_11	21.27	4.91	12	40.25	Female participants 466	2.41	2.20	0	13.0	218
CON_14	21.19	5.12	12	36.75		2.95*	2.18	0	11.5	231
CON_17	21.02	5.22	12	43.0		3.01	2.64	0	13.0	201
EXT_11	1.49	1.81	0	10	Female participants 466	0.79	1.10	0	5	233
EXT_14	1.42	1.88	0	14		0.83	1.12	0	7	216
EXT_17	1.46	1.40	0	9		0.80	1.09	0	7	194
CON_11	18.76	4.13	12	33.5	Female participants 451	2.58	2.41	0	17.5	221
CON_14	20.34*	4.91	12	39.0		2.94*	2.44	0	20.0	216
CON_17	20.35	4.95	12	37.0		3.08	2.70	0	15.25	200

Note. Min = minimum; max = maximum; EXT_11, EXT_14, EXT_17 = individual externalizing symptoms, ages 11, 14, and 17 years, respectively; CON_11, CON_14, and CON_17 = parent–child conflict, ages 11, 14, and 17 years, respectively.

^aEXT symptom counts conceivably could range from 0 to 21, and parent–child conflict scores conceivably could range from 12 to 48.

^bSibling difference scores are presented in absolute value form to highlight the true magnitude of sibling differences. However, to examine the direction of any significant effects, we conducted final analyses on signed sibling differences.

* Indicates a within-trait change in sibling difference scores across age at $p < .05$.

Table 4
Correlations Among Sibling Differences in Externalizing Symptoms and in Parent–Child Conflict at Ages 11, 14, and 17 Years in Full Sample

Measure	1	2	3	4	5	6
Male participants						
1. EXT_11	—					
2. EXT_14	.13*	—				
3. EXT_17	.03	.21*	—			
4. CON_11	.19*	.05	-.04	—		
5. CON_14	-.01	.18*	.13	.10	—	
6. CON_17	-.13	.13	.13	.06	.26*	—
Female participants						
1. EXT_11	—					
2. EXT_14	.18*	—				
3. EXT_17	.14*	.24*	—			
4. CON_11	.14*	.13	-.02	—		
5. CON_14	.13*	.23*	.08	.09	—	
6. CON_17	-.04	-.02	.18*	-.04	.30*	—
Combined male and female participants						
1. EXT_11	—					
2. EXT_14	.15*	—				
3. EXT_17	.09	.23*	—			
4. CON_11	.16*	.09	-.03	—		
5. CON_14	.06	.21*	.11*	.09	—	
6. CON_17	-.08	.06	.16*	.01	.28*	—

Note. EXT_11, EXT_14, and EXT_17 = individual externalizing symptoms, ages 11, 14, and 17 years, respectively; CON_11, CON_14, CON_17 = parent–child conflict, ages 11, 14, and 17 years, respectively.

* Correlation is significantly greater than zero, $p < .05$.

Table 5

Correlations Among Sibling Differences in Externalizing Symptoms and in Parent–Child Conflict at Ages 11, 14, and 17 in Highly Discordant Pairs

Measure	1	2	3	4	5	6
Pairs ($n = 124$) highly discordant for EXT						
1. EXT_11	—					
2. EXT_14	.26*	—				
3. EXT_17	.15	.26*	—			
4. CON_11	.31*	.26*	-.01	—		
5. CON_14	.08	.28*	.12	.18	—	
6. CON_17	-.20	.07	.17	.01	.25*	—
Pairs ($n = 120$) highly discordant for CON						
1. EXT_11	—					
2. EXT_14	.30*	—				
3. EXT_17	.12	.20*	—			
4. CON_11	.25*	.30*	-.08	—		
5. CON_14	.15	.31*	.16	.20*	—	
6. CON_17	-.06	.13	.19	-.02	.33*	—

Note. EXT_11, EXT_14, and EXT_17 = individual externalizing symptoms, ages 11, 14, and 17 years, respectively; CON_11, CON_14, CON_17 = parent–child conflict, ages 11, 14, and 17 years, respectively.

* Correlation is significantly greater than zero, $p < .05$.