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THE ASSOCIATION BETWEEN PARENT-CHILD CONFLICT AND ADOLESCENT CONDUCT PROBLEMS OVER TIME: RESULTS FROM A LONGITUDINAL ADOPTION STUDY

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By

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ABSTRACT

THE ASSOCIATION BETWEEN PARENT-CHILD CONFLICT AND ADOLESCENT CONDUCT PROBLEMS OVER TIME: RESULTS FROM A LONGITUDINAL ADOPTION STUDY

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A handful of prior adoption studies have confirmed that the cross-sectional relationship between child conduct problems and parent/child conflict is at least partially shared environmental in origin. However, as the direction of causation between parenting and delinquency remains unclear, this relationship could be better explained by the adolescent's propensity to elicit conflictive parenting, a phenomenon referred to as an evocative gene-environment correlation. The current study thus examined the prospective relationship between conduct problems and parent-child conflict in a sample of adoptive families. Participants included 672 adolescents in 405 adoptive families assessed at two time points roughly 4 years apart. Results indicated that parent-child conflict predicts the development of conduct problems, whereas conduct problems do not predict increases in parent-child conflict. Such findings effectively rule out evocative gene-environment correlations as an explanation of prior shared environmental effects. Moreover, because the adolescents in this study do not share genes with their adoptive parents, the association between conduct problems and parent-child conflict is indicative of shared environmental mediation in particular. Implications of the findings are discussed.

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Introduction

From the financial burdens caused by destruction of property, to the much heavier human toll of assaults and child abuse, the callous disregard for personal and property rights that characterizes those with Antisocial Personality Disorder carries a high social cost. The adult criminal justice system represents society's most significant attempt to prevent and control these dangerous behaviors. However, it may be that more of society's resources should be spent on the treatment of those with child and adolescent conduct problems, since as many as 75% of those who meet DSM-IV criteria for conduct disorder will go on to meet criteria for Antisocial Personality Disorder (Gelhorn et. al., 2007). Consistent with the latter, the current study aims to better understand the developmental etiology of adolescent conduct problems, with the hope that such findings could ultimately inform interventions targeting this costly disorder.

There are several approaches to exploring the etiology of conduct problems, including a focus on genetic transmission, a focus on environmental transmission (i.e., via exposure to deleterious environmental experiences), and a synthesis of the two. Indeed, it is now well established that there are significant genetic influences on conduct problems. For example, Burt's (2009) meta-analysis of 100 twin and adoption studies of youth antisocial behavior found that 58% of the variance in child and adolescent conduct problems was attributable to genetic factors. Clearly then, genes play an important role in the development of these behaviors. That said, the proportion of variance accounted for by genetic influences is well short of 100%, indicating that environmental influences must also be important.

Behavioral genetic research distinguishes between two types of environmental influence: shared and non-shared. The non-shared environment is that part of the environment that differentiates members of a sibling pair, making them less similar to each other. The shared environment, by contrast, is that part of the environment that is common to both members of a sibling pair, and which acts to make them similar to each other. Shared environmental effects do not differ by proportion of segregating genes shared, and if acting alone, would make all sibling correlations similar in magnitude.

The shared environment had historically been set aside by the field of behavioral genetics, which has instead focused more attention on non-shared environmental influences. This focus on the non-shared environment was not arbitrary; indeed, because non-shared influences are consistently and significantly larger in magnitude than are shared environmental influences (particularly in adulthood), they thus seemed to be the more promising type of environmental influence. More recent research, however, has suggested that shared environmental influences may make important contributions to most forms of psychopathology during childhood and adolescence (Burt, 2009). In these cases, shared environmental influences are typically moderate in magnitude, persist over time (at least up through late adolescence), and perhaps most importantly, have proven to be identifiable sources of environmental influence. For example, research with twins has indicated that parenting is associated with adolescent delinquency partially via shared environmental mechanisms (Burt, McGue et al., 2007; Burt et al., 2003; McGue et al., 1996; Pike et al., 1996). Burt et al. (2003) examined a sample of more than 700 11 year-old twin pairs and their mothers and found that the shared environmental influences contributing to parent-child conflict accounted for roughly 12% of the total variance (or

23% of the shared environmental variance) in child externalizing disorders. Subsequent longitudinal research with these families further suggested that this association persists over time, such that parent-child conflict at age 11 influences the development of externalizing pathology via shared environmental mechanisms three years later (Burt et al., 2005). This relationship has also been supported in a longitudinal twin study of younger children (Larsson, et. al., 2008).

Parent-child conflict may thus be one source of shared environmental influence on child conduct disorder. Importantly, however, twin research has significant limitations when attempting to identify shared environmental influences. In particular, biological parents who are high on antisocial behaviors themselves would be expected to provide their children with both genes of risk for conduct disorder, as well as an increased likelihood of exposure to aversive life circumstances related to the parent's antisocial tendencies (Jaffee et. al., 2006). For example, to the extent that conflictual relationships are a function of the parent's own tendency towards antisocial behavior, the association between conflictual parenting and child conduct problems could be a reflection of common genes (a phenomenon referred to as a passive gene-environment correlation; passive rGE). Accordingly, in biological families (such as those used in child-based twin designs), passive rGE can mimic shared environmental influences (Neiderhiser et al., 2004) when the origins are in fact a function of common parent-child genes.

There is good reason to think passive rGE may impact the parent-child relationship. Indeed, there are several studies confirming some genetic influences on parenting. In a study of the parents of 2,334 Finnish twin pairs, genetic influences

accounted for a small to moderate proportion of the variance (17-30%) in parenting styles across four different measures of parental behavior (Harlaar et al., 2008). Studies examining the parenting practices of parents who are themselves twins or adoptees have also found modest genetic effects on several aspects of parenting (Perusse et al., 1994; Losoya et al., 2007). Neiderhiser et al. (2004) found evidence for passive rGE influence on mother's positivity and monitoring in a study involving a combination of a adolescent-based and parent-based design (Neiderhiser et al., 2004). McGue et al. (2005) similarly found heritability estimates ranging from .09 to .45 for parent-adolescent conflict. In sum, there is now reasonably strong evidence that parenting may be influenced, at least in part, by passive rGE.

Passive rGE thus represents a key confound in prior twin study findings of shared environmental mediation. As a result, more research is needed to confirm the presence of shared environmental influences on the association between parent-child conflict and child conduct problems. Adoption studies are ideal in this regard, since evidence of shared environmental influences in adoption studies cannot be attributed to passive rGE effects. Adopted children do not share any segregating genes with their adoptive parents, and thus, shared genes are not working to create similarities between these children and their parents. Instead, any similarities between them are necessarily shared environmental in origin (indeed, correlations between adoptive siblings are generally considered to be "direct" estimates of shared environmental effects). Fortunately, there are a small handful of adoption studies examining the association between parenting-child conflict and child externalizing. Burt et al. (2007) found evidence that parent-child conflict was as associated with adolescent delinquency in adoptive children as it was in

biological children (*r*s were roughly .2-.3), results that are indicative of shared environmental mediation of this association, over and above any passive rGE confounds. Burt et al. (2007) thus represents a critically important constructive replication of prior twin research.

That said, prior work has been unable to rule out other sorts of rGE. In particular, the child's genotype may influence the type of environmental events that he or she experiences, a phenomenon referred to as an evocative rGE. O'Conner, Deater-Deckard, Fulker, Rutter, and Plomin (1998), for example, demonstrated that adopted children at genetic risk for externalizing (based on information about their biological parents) were more likely to receive negative parenting from their adoptive parents. Although the presence of antisocial and related behaviors in biological parents is a rather simplistic and imperfect proxy for genetic risk in the adoptees, such results nevertheless suggest that the child's tendency toward externalizing and subsequent behavior may evoke negative parental responses from their caregivers. In this case, what appears to be a true shared environmental influence may again reflect genetic influences (specifically the child's genetic influences; the parent's genetic influences would be captured by passive rGE, as described previously).

One compelling method for disentangling evocative rGE from the shared environment involves the cross-lagged regression of data from longitudinal adoption samples. This approach would allow us to clarify the direction of the shared environmental association between parent-child conflict and child conduct problems over time regardless of their pre-existing relationships. Unfortunately, because all available adoption research on this topic (to our knowledge) is cross-sectional in nature, the

direction of causation between parenting and delinquency remains unclear. We thus cannot be certain whether parenting acts as a true shared environmental influence, or whether this relationship is better explained by the child's propensity to elicit conflictive parenting. A cross-lagged, longitudinal study of adoptive families is therefore needed to examine and rule-out evocative rGE (as well as reconfirm the absence of passive rGE), and in this way, more strongly infer shared environmental causality over time.

The current study sought to do just this, examining the association between parent-child conflict and adolescent conduct problems in a longitudinal sample of adoptive sibling pairs. A cross lagged model was fitted to the data in which the cross-age associations act as partial regression coefficients, indexing the unique longitudinal association between conflict and adolescent conduct problems over and above their respective stabilities and any preexisting associations. As a result, this design enables us to meaningfully examine (and potentially rule-out) the simultaneous influences of passive and evocative rGE on the association between parent-child conflict and adolescent conduct problems. If poor parenting is simply an index of antisocial tendencies in the parents (i.e., is a function of passive rGE), then the association between parent-child conflict and child conduct problems should not persist to a sample of adoptive youth (who do not share segregating genes with their adoptive parents). Evocative rGE can simultaneously be examined via the cross-age regression coefficients. A significant association between adolescent conduct problems at time 1 and parent-child conflict at time 2 would be an indication of an evocative rGE process in which the child's behavior is eliciting conflictual parent-child interactions. By contrast, should parent-child conflict at time 1 meaningfully influence adolescent conduct problems at time 2, and do so in our

sample of adoptive youth in particular, it would provide strong evidence of a shared environmental influence of parenting on adolescent conduct problems over time. Indeed, should the current study constructively replicate prior findings of shared environmental influences on the relationship between parent-child conflict and child conduct problems, it would provide the strongest, most unambiguous evidence of shared environmental mediation of this association available to date.

Methods

PARTICIPANTS

Participants were 672 adoptive adolescents in 405 families who participated in the Sibling Interaction and Behavior Study (SIBS), a population-based, longitudinal study of adoptive and biological adolescent siblings and their parents in the state of Minnesota. Adoptive families living in the Twin Cities greater metropolitan area were contacted based on records for the three largest adoption agencies in Minnesota (averaging between 600 and 700 placements a year). Families were selected to have 1) an adopted adolescent placed as an infant and first assessed between the ages of 11 and 19 years, and 2) a second non-biologically related adolescent sibling falling within the same approximate age range. The mean age of placement for the adopted adolescents was 4.8 months (SD=4.7 months). Additional requirements for eligibility included living within driving distance of the Minneapolis-based laboratory, siblings no more than 5 years apart in age, and the absence of cognitive or physical handicaps that would interfere with completion of the daylong intake assessment. Among eligible families, 63% of families participated. There were no significant differences between participating and non-participating adoptive families in terms of parental education, occupational status, and marital dissolution (McGue et al. 2007).

Sample retention at follow-up was excellent, with 94% of adolescents participating. At intake, participants ranged in age from 10 to 18 years (average 14.1 years). At follow-up, participants ranged in age from 14 to 24 years (average 18.2 years). The average age difference between siblings was 2 years (SD = 1 year). A little over half of the sample was female (55%). The adoptive parents were broadly representative of the

ethnic composition of the Minnesota population at the time they were born; approximately 95% were Caucasian. By contrast, due to predominantly international adoptions in Minnesota, the adopted adolescents were 67% Asian-American and 21% Caucasian. The remaining adopted adolescents consisted of African-American (2%), East Indian (2%), Hispanic/Latino (3%), South or Central American Indian (1%), mixed race (4%), and other (0.1%) ethnicities. More information on participant recruitment and participation rates is available in McGue et al. (2007).

MEASURES

Assessment of Conduct Problems

Our analyses centered on a composite of the following two measures: the Delinquent Behavior Index (DBI) and a self-reported DSM-IV "symptom count" variable corresponding to the sum of endorsed or partially-endorsed criterion A or C symptoms of Antisocial Personality Disorder. We will first describe each measure, followed by a description of the composite. The DBI (Burt & Donnellan, 2008; Burt, McGue et al., 2007; Farrington & West, 1971; Gibson, 1967) is an inventory of minor (e.g., skipping school) and more serious (e.g., using a weapon in a fight) delinquent behaviors, 21-items of which were administered in this sample (available on 664 adolescents at time 1, but only 429 at time 2, as the measure was not administered to those aged 19 or older at time 2). Participants were asked whether they had engaged in each behavior "as an adolescent" (0=no; 1=yes). Items were summed such that higher scores reflect endorsement of more delinquent behaviors. If fewer than two items were missing, items were prorated and added to the scale score. If more than two items were missing, the

score was coded as missing. The scale demonstrated good internal consistency reliability with alphas of .87 and .89 at intake and follow-up, respectively.

For the DSM-IV assessment, we assessed the two symptom dimensions (i.e., criterion A and C) that comprise DSM-IV Antisocial Personality Disorder. At time 1, we specifically examined the sum of endorsed or partially-endorsed criterion C symptoms (i.e., symptoms of Conduct Disorder or CD; available on 668 participants). At time 2, we examined the sum of endorsed or partially-endorsed criterion A symptoms (i.e., the adult-specific antisocial behavior (AAB) symptoms of Antisocial Personality Disorder; available on 529 participants, as those participants younger than 16 at time 2 were not administered the AAB interview).[1] The reporting period for time 1 CD was infancy through age 15 (the age cut-off specified in the DSM-IV), while the reporting period for time 2 AAB was since age 15. The reporting periods for these measures thus did not overlap across intake and follow-up.

All participants were assessed in-person by trained bachelor and masters-level interviewers for DSM-IV mental disorders. Siblings were interviewed by separate interviewers. CD was assessed using the Diagnostic Interview for Children and

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[1] The focus on CD at intake but AAB at follow-up (see Burt, McGue, & Iacono, in press) allowed us to accommodate some of the developmental change in antisocial behavior across adolescence, while maintaining a focus on clinically-meaningful levels of these behaviors. In childhood, for example, antisocial behaviors include lying, stealing, destroying property, setting fires, and being physically cruel. By early adolescence, these same youth are also likely to be truant from school, break curfew, and run away from home, behaviors that are quite rare prior to adolescence (Gelhorn, et. al., 2007). While some of these behaviors continue to characterize antisocial individuals in adulthood (e.g., stealing, lying, and physical cruelty), others are either no longer applicable (e.g., truancy, breaking curfew) or are rarely expressed (e.g., fire setting; Gelhorn, et al. 2007). The DSM-IV captured (some of) this developmental change in its diagnostic criteria for Antisocial Personality Disorder (i.e., CD is assessed until age 15, while AAB is assessed after age 15, but both are required for a diagnosis).

Adolescents-Revised (DICA-R) (Reich, 2000; Welner, Reich, Herjanic, Jung, & Amado, 1987). Of the 13 possible symptoms of CD, only symptom 9 ("has forced someone into sexual activity with him or her") was not assessed. AAB was assessed via the Structured Clinical Interview for personality disorders (SCID-II) (Spitzer, Williams, Gibbon, & First, 1987). Supplementary probes and questions were added to both interviews to ensure complete coverage of each symptom.

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. As actual diagnoses were not used in the current study, duration rules were excluded. Symptoms judged to be definitely present (i.e., they were clinically significant in both severity and frequency) were counted as one full symptom. Symptoms judged to be probably present (i.e., they were clinically significant in either severity or frequency, but not both) were counted as half of a symptom. The reliability of the consensus process was good, with a kappa of 0.79 for diagnoses of CD and .82 for "diagnoses" of AAB.

Conduct Problems Composites. In order to capture both clinically-significant (e.g., physical assault, setting fires) and less clinically-significant, but still problematic (e.g., driving a car without a license, carrying a weapon in a public place, etc.) conduct problems, we created a composite of the Delinquent Behavior Index (DBI) and (one of the two) self-reported DSM-IV symptom count variables. More specifically, we averaged the time 1 DBI and time 1 CD data for our time 1 conduct problems composite, and the time 2 DBI and time 2 AAB data for our time 2 conduct problems composite. The composite was available on 672 adolescents at time 1 and 632 adolescents at time 2.

The composite was created by standardizing and averaging the DBI and CD/AAB variables so as to ensure that, despite their different scoring metrics, scales were weighted equally. As noted above, this use of this composite is advantageous in that it allowed us to more completely assess the full range of delinquent acts, thereby increasing both statistical power and (arguably) the applicability of our results. Moreover, the use of a composite allows us to accommodate the fact that, at time 2, the DBI was not administered to adolescents over the age of 19, while AAB was not assessed in adolescents younger than age 15 (per the age cut-offs in the DSM-IV). Therefore, for those adolescents over the age of 19 years (n = 196) or under the age of 15 years (n = 44) at time 2, conduct problems were measured solely through the AAB or the DBI, respectively.

Assessment of the Parent-child Relationship

The Parental Environment Questionnaire (PEQ; Elkins, McGue, & Iacono, 1997) was administered to tap perceptions of the parent-child relationship. The current study focused on the 12-item parent-child conflict scale (e.g., "I often criticize my child"; "My child and I often get into arguments"). In this scale, mothers and fathers individually rated their relationships with each of their participating adolescents. Each informant rated items on a 4-point scale (1=definitely true; 2 = somewhat true, 3 = somewhat false; 4 = definitely false). As our goal was to examine overall levels of parent-child conflict, we thus made use of a parent informant composite (i.e., an average of mother and father reports of parent-child conflict). To accommodate any missing data, we allowed for up to one missing informant report. Items were scored such that high scores corresponded to

high levels of parent-child conflict. The conflict scale displayed good internal consistency reliability, with alphas ranging between .90 and .94 at intake and follow-up.

PEQ's were mailed to families prior to their assessment. Participants were asked to bring their completed PEQ to their in-person visit. If a completed PEQ was not obtained by the end of their in-person visit, participants were asked to complete it at home and return it by mail. One telephone prompt was made if the PEQ was still not received. Parental PEQ reports were available for 660 participants at time 1 and 625 participants at time 2.

ANALYSES

Analysis of these data was based on the fact that adoptive youth and their adoptive parents do not share any segregating genetic material. Shared environmental influences are thus implied by correlations significantly greater than zero. Using this framework, we examined the origins of the association between the parent-child relationship and adolescent conduct problems via a series of interrelated analyses. We first computed correlations between adolescent self-reported conduct problems and parent-child relationships across the entire sample, and separately for older and younger siblings (as done in McGue et al., 1996). Significant correlations for adopted youth are suggestive of shared environmental influences on this association (or at least an absence of passive rGE). In order to better approximate normality, all variables were log-transformed prior to analysis.

For our final analyses, we examined a cross-lagged regression model (see Figure 1), distinguishing between older and younger siblings both to allow for possible differences across siblings and to accommodate the non-independence of the data within

families, as analyses were conducted at the level of the family (the sample size for these analyses was the number of families, n = 405). This model requires all cross-age associations to function as partial regression coefficients. The cross-age but within-trait coefficients (i.e., b_{11}, b_{33} for the younger sibling and b_{22}, b_{44} for the older sibling) index the stability of conflict and conduct problems over time. The cross-lagged coefficients (i.e., b_{13}, b_{31} for the younger sibling and b_{24}, b_{42} for the older sibling) allowed us to determine whether conflict and conduct problems at time 1 *independently* impacted each other at time 2, controlling for the stability of both traits. Next, we were able to evaluate the cross-sectional relationship between parent-child conflict and adolescent conduct problems via assessment-specific correlations both within and across siblings. Because of the stability and cross-lagged coefficients, these correlations functioned as residuals at time 2, thereby revealing the association between conflict and conduct problems at time 2 in particular.

We fitted this model allowing cross-age associations to vary across older and younger siblings (i.e., unconstrained), as well as constraining associations to be equal across siblings (i.e., constrained). The latter model imposed four constraints (i.e., $b_{11} = b_{33}$; $b_{22} = b_{44}$; $b_{31} = b_{42}$; $b_{13} = b_{24}$). In this way, we were able to examine whether, for example, the stability of conduct problems varies across older and younger siblings. A non-significant change in the chi-square index of fit across the two models would indicate that the estimates do not differ across older and younger siblings. To accommodate missing data, we made use of Full-Information Maximum-Likelihood raw data techniques (FIML), which produce less biased and more efficient and consistent estimates than other techniques, such as pairwise or listwise deletion, in the face of

missing data (Little & Rubin, 1987). AMOS, a structural-equation modeling program (Arbuckle, 2003), was used to fit cross-lagged model to the observed raw data.

Results

Mean levels of DBI, CD symptom counts, AAB symptom counts, and parent-child conflict were calculated separately for males and females (see Table 1). Across both time points, independent-sample *t* tests indicated that mean DBI scores and CD/AAB symptom counts differed significantly by sex ($p < .01$), with boys consistently reporting higher levels of conduct problems. Parent-child conflict did not differ across sex at either time point. Paired-sample *t*-tests revealed little evidence for change in mean levels of parent-child conflict over time ($p > .70$). DBI scores, by contrast, increased significantly across time for males and females ($p < .01$). We did not evaluate changes for CD/AAB symptoms counts over time, as the number of possible symptoms naturally differs across the CD (time 1) and AAB (time 2) assessments.

Correlations

We computed correlations between log-transformed adolescent conduct problems and parent-child conflict both on the full sample, as well as separately for older and younger siblings (see Table 2). There was evidence of moderate to strong stability across time in both conduct problems and parent-child conflict (correlations range between .574 and .696, $p < .01$). This stability persisted when separately examining older and younger siblings. Parent-child conflict and conduct problems were moderately correlated with each other at both time points (i.e. within time, cross trait correlations range between .301 and .413, $p < .01$). These associations also persisted to older and younger siblings when

examined separately. The cross-time cross-trait correlations were similarly moderate in magnitude, such that conflict at time 1 was correlated .391 with conduct problems at time 2, and conduct problems at time 1 was correlated .207 with conflict at time 2 (both $p < .01$). Again, this relationship persisted across older and younger siblings (correlations range between .195 and .350, $p < .01$). As significant correlations between conflict and conduct problems are indicative of meaningful shared environmental influences on this association (or at least an absence of passive rGE), such findings highlight probable shared environmental influences on this association. [2]

Cross-lagged Analyses

For our primary analyses, we wanted to determine how conduct problems and parent-child conflict related to each other over time across the full sample (n = 405 families), as well as whether these estimates varied across older and younger siblings. We thus fitted the cross-lagged model allowing for differences in parameter estimates across older and younger siblings, (X^2 = 15.07 on 8 df, p = 0.06, AIC = 87.07), and constraining the parameter estimates to be equal across siblings, (X^2 = 16.52 on 12 df, p = 0.17, AIC = 80.52; ΔX^2 = 1.45 on 4 df, p = 0.84). The lower AIC and non-significant change in chi-square suggests that the constrained model provided a better fit to the data.

[2] As another way of examining these effects, we also computed sibling intraclass correlations for conduct problems and parent-child conflict at times 1 and 2. Conduct problems were moderately correlated between unrelated siblings at times 1 and 2 (r = .24 at both time points, $p < .01$), although this association only reached statistical significance at time 1. Parent-child conflict was also moderately correlated between unrelated siblings at both time points (r's range between .18 and .33, $p < .05$). These associations are consistent with shared environmental influences, and in this way, buttress the above suggestions of shared environmental effects.

We thus conclude that estimates can be constrained to be equal across older and younger siblings. The constrained model had a root-mean-square error of approximation (RMSEA) of 0.031, indicating an excellent overall fit to the data.

Path estimates from the better-fitting constrained model can be seen in Figure 2 and Table 3. The percentage of variance that is uniquely accounted for by a given path can be obtained by simply squaring its path coefficient. Conduct problems and parent-child conflict evidenced significant stability over time (i.e., 28-50%). Importantly, conduct problems at time 1 were no longer a significant predictor of parent-child conflict over time (i.e., b13, b24), accounting for .0001% (i.e., $.01^2$) of the variance in conflict at time 2. Such results suggest that the zero-order correlation between these variables (as seen in Table 2) may be a function of their robust cross-sectional associations and stability over time. By contrast, parent-child conflict at time 1 continued to significantly predict conduct problems 4 years later, accounting for 3.2% (i.e., $.18^2$) of the variance in conduct problems at time 2 (i.e., b31, b42). In short, once we account for stability over time and cross-sectional associations, parent-child conflict at time 1 appears to predict conduct problems at time 2, but not vice-versa. This suggests that a parent-driven, or a parent-child relationship-driven, effect better explains the association between parent-child conflict and conduct problems.

Follow-up Analyses

In order to ensure that our results persisted across sex, we re-fitted the model only for opposite-sex sibling pairs and families with only one adopted child (N = 270 families in total). Parameter estimates were allowed to differ across sex, (X^2 = 8.88 on 8 df, p =

0.35, AIC =80.88), and were constrained to be equal across sex (X^2 = 10.01 on 12 df, p = 0.62, AIC = 74.01; ΔX^2 = 1.13 on 4 df, p = 0.89). Both models provided a good fit to the data; however, estimates could be constrained to be equal without a significant decrement in fit. We can thus infer that estimates were equivalent across sex. Results of the better fitting model are presented in Table 3. As seen there, parent-child conflict at time 1 again predicted conduct problems at time 2, but not vice-versa. Such results suggest that the results of the full model are applicable to both boys and girls.

Additionally, because of the age range of the sample, many adolescents were no longer living with their parents at time 2 (n = 305 families). As this change in living arrangements could potentially alter the relationship between parent-child conflict and conduct problems, we re-fitted the model a third time, so as to ensure that results were applicable to both adolescents living at home as well as those no longer living at home. We allowed parameter estimates to differ across residential status (X^2 = 12.90 on 8 df, p = 0.12, AIC = 84.90) and then constrained them to be equal across residential status (X^2 = 14.27 on 12 df, p = 0.28, AIC = 78.27; ΔX^2 = 1.37 on 4 df, p = 0.85). As before, the constrained model provided a better fit to the data. Results are presented in Table 3, and again highlight the prediction of conduct problems at time 2 by conflict at time 1. We can thus infer that the impact of conflict on the development of conduct problems over time is not restricted to children still residing with their family of origin.

For our final set of follow-up analyses, we elected to statistically control for sex and age effects via regression techniques (McGue & Bouchard, 1984) in order to further

confirm that our results were not dependent on age or sex effects. We thus regressed age and sex out of conduct problems and parent-child conflict at both time points, and then fitted the cross-lagged model on the full sample. As with our primary analyses, we allowed for differences in parameter estimates across older and younger siblings, (X^2 = 12.27 on 8 df, $p = 0.14$, AIC = 84.3) and constrained them to be equal across siblings, (X^2 = 13.79 on 12 df, $p = 0.32$, AIC = 77.8; $\Delta X^2 = 1.52$ on 4 df, $p = 0.82$). Once again, the constrained model was the better fitting model. Parameter estimates (presented in Table 3) confirm prior results, and thereby provide additional evidence that our results are not a function of age or sex effects.

Discussion

The goal of the present study was to examine the relationship between adolescent conduct problems and parent-child conflict in a sample of adoptive families using a longitudinal cross-lagged model. In this way, we hoped to determine whether shared environmental influences of parenting on conduct problems persisted over time, or whether the relationship between conduct problems and parent-child conflict was better explained by rGE processes. Analyses revealed several important findings. First, parent-child conflict and adolescent conduct problems were associated both cross-sectionally and longitudinally in these data. As our sample consisted only of adoptive families (in which parents and their children do not share segregating genetic material), such findings provide additional confirmation that passive rGE do not explain the association between conduct problems and parent-child conflict. Second, once we controlled for cross-sectional associations and stability over time within our cross-lagged regression model, the association between conduct problems at time 1 and parent-child conflict at time 2 was no longer significant. Such results suggest that the presence of adolescent conduct problems does not contribute to the development or intensification of parent-child conflict over time, and thus argue against evocative rGE as an explanation for their association. Critically, however, the inverse relationship proved to be uniformly significant, such that parent-child conflict at time 1 significantly predicted the development of conduct problems at time 2. These results did not vary by adolescent sex or residence status (i.e., whether or not the adolescents reside with their family of origin), or when statistically controlling for both age and sex in the analyses. Such findings indicate that, rather than functioning as a passive or evocative rGE, the parent-child

relationship influences the development of conduct problems via shared environmental mechanisms.

These findings are generally consistent with those of extant twin studies, which suggest that parenting influences conduct problems partially through shared environmental mechanisms (Burt et al., 2003; McGue et al., 1996; Pike et al., 1996). Our results are also consistent with prior adoption studies implicating a shared environmental relationship between parent-child conflict and conduct problems (e.g., Burt, McGue et al., 2007). Critically, these adoption studies have uniformly been cross-sectional in nature and were thus unable to determine the direction of the association between conduct problems and conflict. The current study advances this prior work, and suggests that it is parent-child conflict that predicts the development of conduct problems, and not vice versa.

There are several limitations to the current study. First, the magnitude of the cross-lagged effect between conflict at time 1 and conduct problems at time 2 is relatively small, accounting for 3.2% of the variance. Importantly, however, phenotypes that account for 3% of the variance several years later are neither trivial nor unusual (i.e., several methodologically-rigorous longitudinal studies have reported similarly small percentages of variance explained; Campbell et al., 1996; Loukas et al., 2001; Reiss et al., 2000; Wasserman et al., 1996). There is also evidence to suggest that, in much the way that multiple genes are likely to impact a given mental disorder, environmental risks may involve a combination of several psychosocial risk factors, and thus individually account for only a small proportion of the variance (Rutter, Silberg, O'Connor, & Simonoff, 1999). This point is particularly salient in the case of parent-child conflict, as prior twin

work (Burt et al., 2003) has indicated that there is a largely direct relationship between parent-child conflict and adolescent externalizing disorders (i.e., their associations were not mediated by parental involvement with the child, parent regard for child, child regard for parent, parental structure, family cohesion and adaptability, marital discord, divorce of parents, or parental income). Accordingly, other psychosocial variables would likely account for additional variance over and above the effects of parent-child conflict. Given this, the finding that parent-child conflict accounts for 3.2% of the variance in conduct problems *several years later*, and does so via shared environmental mechanisms, is an important step towards identifying psychosocial variables that have a lasting influence on antisocial behavior.

Second, our measure of conflict was based on mother and father self-reports of their relationships with each of their children rather than on direct observation. Although it is unclear how these results may have changed with the use of observer-reported conflict, future research could include a measure of observer-rated conflict. That being said, we would argue that the use of different informants for conduct problems and parent-child conflict represents a strength of our study, as results cannot be a function of shared informant effects. Third, the results of this study are applicable only during adolescence and emerging adulthood, and not in other developmental periods. In particular, extant research suggests that the heritability of conduct problems varies by age and by age-of-onset (Burt & Neiderhiser, 2009), and that child versus adolescent onset groups have different etiological origins and trajectories (Moffitt, 2003). Thus, the results of the current study are unlikely to apply to earlier or later developmental stages. That being said, adolescence and emerging adulthood are critical stages in the

development of conduct problems, as conduct problems are more frequent during these periods than at any other point in the lifespan.

Finally, our measure of conduct problems differed somewhat from time 1 to time 2 (i.e., we made use of CD symptom counts at time 1 and AAB symptom counts at time 2). Although challenging from a measurement standpoint, the changes in age and developmental stage over the course of the study (i.e. participants were aged 10-18 at time 1 of the study and 14-24 at time 2) necessitated this shift in measures. For example, adolescents under the age of 15 are unlikely to engage in adult symptoms of ASPD (e.g., failure to honor financial obligations), while young adults are less likely to be engaging in symptoms of CD. Of note, however, the DBI has been validated on both adolescents and emerging adults and was identical across the two assessments. In order to ensure that our results were not a function of the CD/AAB distinction, we therefore re-ran the analyses using only the DBI as our measure of conduct problems. Cross-lagged results were quite similar to those reported herein (DBI at time 1 → conflict at time 2 = -.001, p = .86, conflict at time 1 → DBI at time 2 = .34 or 14% of the variance, p = .04). Such findings suggest that our use of the CD/AAB measure did not unduly affect our results, while still serving to increase the clinical utility of the analyses.

In spite of these limitations, the results of the current study have several important implications. Because adopted adolescents do not share genes with their adoptive parents, our findings of a shared environmentally mediated relationship between conduct problems and parent-child conflict cannot be explained by the effects of passive rGE, a possible confound in twin research on the shared environment. Similarly, the association does not appear to be a function of adolescents' genetic propensity for externalizing

behavior (i.e., evocative rGE) since conduct problems do not appear to significantly predict parent-child conflict over time. Thus, the best explanation for the relationship between parent-child conflict and conduct problems is that conflict itself plays a formative and shared environmentally-mediated role in the development of conduct problems over time. These findings provide the most unequivocal evidence in favor of shared-environmental mediation of this association available to date. They also lend further support to recent suggestions that, prior to adulthood, the shared environment plays an important role in the development of psychological and behavioral outcomes (Burt, 2009). The importance of this conclusion is augmented by the definitional properties of the shared environment. As discussed in Burt (2009), estimates of the shared environment are very unlikely to include G x E interactions, and thus the effects of the parent-child relationship on conduct problems are likely to represent a "main effect" of the environment. In this way, our findings lend further support to recent suggestions that, prior to adulthood, the shared environment plays an important role in the development of psychological and behavioral outcomes (Burt, 2009).

The implications of these "direct" effects of the parent-child relationship on adolescent conduct problems may be far-reaching. They not only suggest that interventions targeting the parent-child relationship during adolescence could be particularly effective in reducing or preventing the development of conduct problems (which is known), but more importantly, the current results suggest that this should be true regardless of the adolescent's genetic risk profile. Also of interest, the residence analyses indicate that family therapy may be an effective intervention strategy even when an adolescent or emerging adult no longer resides with his or her family of origin.

That said, the proximal shared environmental mechanisms through which parent-child conflict contributes to adolescent conduct problems cannot be determined herein. One possibility is that, in the presence of high levels of parent-child conflict, adolescents rely more heavily on their peers for emotional support. This increased affiliation with peers in general could lead to greater involvement in deviant behaviors. Consistent with this possibility, low levels of parental support has been linked with adolescent delinquency (Juang & Silberiesen, 1996), as have high levels of control (Farrington, 1989). Another possibility is that conflictual parenting styles alienate adolescents from their parents and "inspire" increased rebellion from them. In any case, future research should seek to more fully explicate the ways in which the parent-child relationship contributes to the etiology of conduct problems over and above the contribution of genetic influences in order to further capitalize upon this promising intervention opportunity.

APPENDIX

Table 1

Mean Level of Conduct Problems and Parent-Child Conflict at Time 1 and Time 2

Variable	Mean	SD	Min	Max	N
Males					
DBI Time 1	3.83**	4.09	0	20	302
CD symptom count Time 1	1.27**	1.66	0	9	303
Parent-Child Conflict- Time 1	22.68	5.48	12	38	299
DBI Time 2	5.16**	5.02	0	21	203
AAB symptom count- Time 2	1.38**	1.53	0	7	233
Parent-Child Conflict- Time 2	22.97	5.49	12	41	284
Females					
DBI Time 1	2.33**	2.90	0	17	362
CD symptom count Time 1	0.54**	0.84	0	5	368
Parent-Child Conflict- Time 1	22.20	5.83	12	42	361
DBI Time 2	3.21**	3.31	0	15	226
AAB symptom count- Time 2	0.84**	1.22	0	6	296
Parent-Child Conflict- Time 2	22.83	6.12	12	42	341

Note. Min = minimum; max = maximum; SD = standard deviation; DBI= Delinquent Behavior Inventory; CD = Conduct Disorder symptom count; AAB = Adult Antisocial Behavior symptom count

[a] DBI scores could conceivably range from 0 to 36, CD symptom counts from 0 to 12, AAB symptom counts from 0 to 7, and parent-child conflict from 0 to 48.

**Means are significantly different between sexes, $p < .01$.

Table 2

Phenotypic Correlations between Conduct Problems and Parent-Child Conflict at Time 1 and Time 2 in the Full Sample, and Separately by Older and Younger Siblings

Measure	1	2	3	4
Full Sample				
1. cp_1	-			
2. cp_2	.574***	-		
3. con_1	.301**	.318**	-	
4. con_2	.207**	.413**	.696**	-
Older Siblings				
1. cp_1	-			
2. cp_2	.583**	-		
3. con_1	.310**	.285**	-	
4. con_2	.195**	.401**	.690**	-
Younger Siblings				
1. cp_1	-			
2. cp_2	.562**	-		
3. con_1	.231**	.350**	-	
4. con_2	.215**	.432**	.711**	-

Note. cp_1 and cp_2 = conduct problems at time 1 and time 2, respectively; con_1 and con_2 = parent-child conflict at time 1 and time 2, respectively.

**Correlation is significantly greater than zero, $p < .01$.

Table 3

Path Estimates from the Best-Fitting Models for Each Set of Analyses

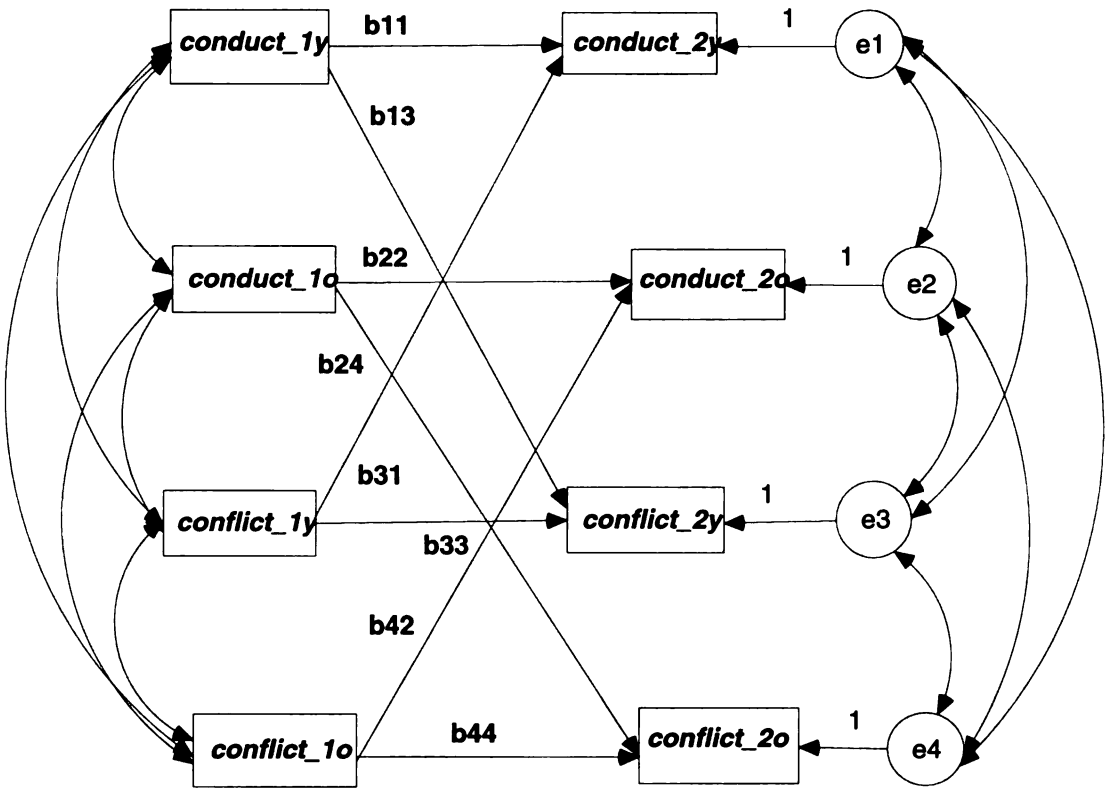
Model	Pathway	Standard-ized Estimate	*p*
Full Model (n = 405 families)	Stability of Conduct Problems over Time (b11, b22)	0.53	<.001
	T1 Conduct Problems → T2 Parent-Child Conflict (b13, b24)	0.01	0.726
	Stability of Parent-Child Conflict over Time (b33, b44)	0.71	<.001
	T1 Parent-Child Conflict → T2 Conduct Problems (b31, b42)	0.18	<.001
Sex Differences Model (n = 270 families)	Stability of Conduct Problems over Time (b11, b22)	0.47	<.001
	T1 Conduct Problems → T2 Parent-Child Conflict (b13, b24)	0.03	0.556
	Stability of Parent-Child Conflict over Time (b33, b44)	0.69	<.001
	T1 Parent-Child Conflict → T2 Conduct Problems (b31, b42)	0.24	<.001
Residence Differences Model (n = 305 families)	Stability of Conduct Problems over Time (b11, b22)	0.48	<.001
	T1 Conduct Problems → T2 Parent-Child Conflict (b13, b24)	-0.01	0.863
	Stability of Parent-Child Conflict over Time (b33, b44)	0.69	<.001
	T1 Parent-Child Conflict → T2 Conduct Problems (b31, b 42)	0.16	<.01

Table 3 (cont'd).

Model	Pathway	Standard-ized Estimate	*p*
Full Model	Stability of Conduct Problems over Time (b11, b22)	0.50	<.001
Age and Sex Regressed Out	T1 Conduct Problems → T2 Parent-Child Conflict (b13, b24)	0.02	0.645
	Stability of Parent-Child Conflict over Time (b33, b44)	0.69	<.001
(n = 405 families)	T1 Parent-Child Conflict → T2 Conduct Problems (b31, b42)	0.12	<.001

Figure 1.

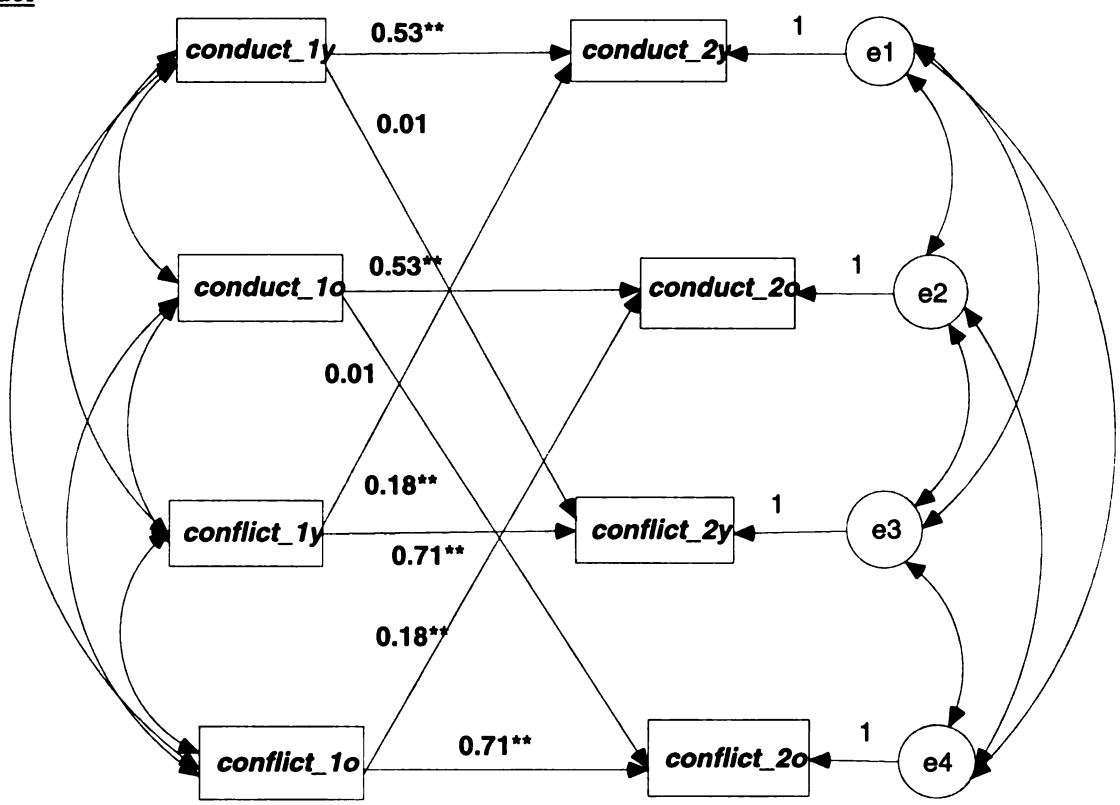
Cross-lagged model of the Association between Adolescent Conduct Problems (conduct) and Parent-child Conflict (conflict) Over Time.



Note. Time 1 variables are indicated on the left, and time 2 variables are indicated on the right. "y" and "o" indicate younger and older siblings, respectively. Cross-age paths (i.e., partial regression coefficients) are indicated by a "b" followed by 2 numerals. Within-age correlations are indicated by double-headed arrows on the far right and far left of the model. The residual variance in conduct problems and parent-child conflict at time 2 are represented by an "e" followed by a single numeral.

Figure 2

Cross-lagged model of the Association between Adolescent Conduct Problems (conduct) and Parent-child conflict (conflict) Over Time with Path Coefficients from the Best-Fitting (Constrained)
Model



Note. conduct_1y and conduct_2y = conduct problems at time 1 and time 2, respectively, in the younger siblings; conduct_1o and conduct_20 = conduct problems at time 1 and time 2, respectively, in the older siblings; conflict_1y and conflict_2y = parent-child conflict at time 1 and time 2, respectively, in the younger siblings; conflict_1o and conflict_2o = parent-child conflict at time 1 and time 2, respectively, in the older siblings.

** Path estimate is significantly greater than zero, $p < .01$

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