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ORIGINAL ARTICLE

Parent–child conflict as an etiological moderator of childhood conduct problems: an example of a 'bioecological' gene–environment interaction

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Background. Prior research has suggested that, consistent with the diathesis–stress model of gene–environment interaction (G×E), parent–child conflict activates genetic influences on antisocial/externalizing behaviors during adolescence. It remains unclear, however, whether this model is also important during childhood, or whether the moderation of child conduct problems by negative/conflictive parenting is better characterized as a bioecological interaction, in which environmental influences are enhanced in the presence of environmental risk whereas genetic influences are expressed most strongly in their absence. The current study sought to distinguish between these possibilities, evaluating how the parent–child relationship moderates the etiology of childhood-onset conduct problems.

Method. We conducted a series of 'latent G by measured E' interaction analyses, in which a measured environmental variable was allowed to moderate both genetic and environmental influences on child conduct problems. Participants included 500 child twin pairs from the Michigan State University Twin Registry (MSUTR).

Results. Shared environmental influences on conduct problems were found to be several-fold larger in those with high levels of parent–child conflict as compared with those with low levels. Genetic influences, by contrast, were proportionally more influential at lower levels of conflict than at higher levels.

Conclusions. Our findings suggest that, although the diathesis–stress form of G×E appears to underlie the relationship between parenting and conduct problems during adolescence, this pattern of moderation does not extend to childhood. Instead, results were more consistent with the bioecological form of G×E which postulates that, in some cases, genetic influences may be most fully manifested in the absence of environmental risk.

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Introduction

A key framework for understanding the joint contribution of genetic and environmental influences on child outcomes is the gene–environment interaction (G×E), whereby exposure to a given environmental risk factor moderates the importance of genetic and/or environmental contributions to a given outcome (Plomin *et al.* 1977; Rutter *et al.* 2006). This etiological moderation can take many forms (Pennington *et al.* 2009; Burt, 2011). The most widely accepted of these represents a specific instantiation of the more general diathesis–stress model, in which genetic risk for a given behavior or outcome is expressed more fully in response to an environmental pathogen (Moffitt *et al.* 2006; Rutter *et al.* 2006; Gottlieb, 2007).

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Available work studying the influence of parenting on antisocial behavior in adolescence and emerging adulthood has provided strong empirical support for this model of G×E (Caspi *et al.* 2002; Foley *et al.* 2004; Spatz Widom & Brzustowicz, 2006; Feinberg *et al.* 2007; Button *et al.* 2008; Hicks *et al.* 2009; Beach *et al.* 2010; Li & Lee, 2010; Aslund *et al.* 2011). Feinberg *et al.* (2007), for example, found that genetic influences on adolescent behavior problems were potentiated in the face of parental negativity. Hicks *et al.* (2009) found nearly identical results in their analysis of more than 1300 pairs of 17-year-old twins. Such results strongly imply that genetic contributions to adolescent externalizing are accentuated in the presence of poor-quality parenting (i.e. the diathesis–stress form of G×E).

Critically, however, nearly all of these studies examined externalizing behavior in adolescence and emerging adulthood. The single largest exception examined antisocial behavior in 975 boys and found only trend-level evidence ($p=0.16$) of a diathesis–stress

G×E in childhood (Kim-Cohen *et al.* 2006). We thus know very little about G×E in childhood conduct problems, a surprising gap in the literature given that child-onset conduct problems are typically conceptualized as more severe and chronic than those beginning later in life (Moffitt, 1993, 2003). Although one could argue that studies conducted on adult antisocial behavior generalize to child conduct problems (as the former is often a consequence of the latter), this conclusion would be premature. Indeed, recent work has suggested that the etiological processes linking particular risk experiences to conduct problems may change over the course of development (Kendler *et al.* 2008). For example, although studies have uniformly indicated that deviant peer affiliation exacerbates genetic influences on conduct problems/externalizing behaviors during adolescence (Cleveland *et al.* 2005; Button *et al.* 2007; Harden *et al.* 2008; Beaver *et al.* 2009; Hicks *et al.* 2009), a study conducted during childhood was unable to replicate these results (Burt & Klump, 2013*b*). Instead, genetic influences on child conduct problems appeared to be proportionally more important in the absence of deviant peer affiliation, whereas shared environmental influences were more important in the presence of deviant peer affiliation.

Such findings are noteworthy, not only because they highlight etiological distinctions between child and adolescent conduct problems, but also because findings of shared environmental moderation are difficult to rectify with the diathesis–stress model of G×E. How might we understand the moderation of shared environmental influences? There is another, less frequently discussed, model of G×E that predicts shared environmental moderation in particular: the 'bioecological interaction' (Bronfenbrenner & Ceci, 1994; Pennington *et al.* 2009). The logic of this model is best illustrated through Lewontin's analogy of genetically variable seeds planted in either nutrient-rich or nutrient-deprived soil (Lewontin, 1995). Because all plants receive adequate nutrition in nutrient-rich soil, individual differences in plant height would be largely a consequence of genetic differences between plants. The environmental adversity conferred by the deprived soil, by contrast, should eventuate in a field populated largely by short plants, regardless of the plants' genetic predispositions for height. Put differently, it may be that some adverse experiences provide such a strong 'social push' for a given outcome that the importance of genetic factors in these environments is effectively diminished (Raine, 2002; Legrand *et al.* 2008). Only in the absence of these risks are genetically mediated individual differences fully manifested.

The diathesis–stress and bioecological models of G×E thus represent fundamentally different models of G×E. Under the diathesis–stress model, G×E would manifest as stronger genetic effects in the presence of environmental risk. In more specific terms, the diathesis–stress model would predict absolute (or unstandardized) increases in genetic influences with increasing environmental risk exposure. There are no clear predictions for environmental influences on the outcome. Under the bioecological model, by contrast, deleterious environments are thought to amplify (shared) environmental influences, whereas genetic influences are more important under normal environmental conditions. In this case, the model would specifically predict absolute increases in environmental influences with increasing environmental risk exposure. Genetic influences on the outcome would be expected to decrease. However, the latter effect may only be observable when examined relative to the environmental moderation (i.e. via standardized estimates): 'unlike in a diathesis–stress model, the environmental factor in a bioecological interaction does not necessarily act on the same biological substrate as the genetic risk factors. Instead, it may just allow those genetic risk factors to account for more of the variance in outcome, because environmental risk factors that affect that outcome have been minimized' (Pennington *et al.* 2009, p. 80).

Current study

Prior work has strongly suggested that the diathesis–stress model of G×E characterizes the etiological moderation of adolescent antisocial behavior by negative/conflictive parenting. It remains unclear, however, whether this model of G×E is also important during childhood, or whether the moderation of child conduct problems by negative/conflictive parenting is better characterized as a bioecological interaction. The current child twin study sought to distinguish between these possibilities, evaluating whether and how the parent–child relationship moderates the etiology of childhood-onset conduct problems.

Method

Participants

The Michigan State University Twin Registry (MSUTR) includes several independent twin projects (Burt & Klump, 2013*a*). The 500 twin pairs (50.2% monozygotic; MZ) included in the current study were assessed as part of the Twin Study of Behavioral and Emotional Development in Children within the MSUTR. Participating twins did not differ from non-participating twins in their average levels of conduct problems, emotional symptoms or hyperactivity (Cohen's $d = -0.05$, 0.01 and -0.08, respectively).

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Moreover, participating families endorsed ethnic group memberships at rates comparable with area inhabitants (e.g. Caucasian: 86.4% and 85.5%, African-American: 5.4% and 6.3% for the participating families and the local census, respectively). More detailed information regarding the design, recruitment procedures, representativeness and participation rates have been provided elsewhere (Burt & Klump, 2013*a*).

The twins were 47.0% female and ranged in age from 6 to 10 years, although a small handful (n=14 pairs) had turned 11 years old by the time the family participated [mean age for full sample=8.2 (S.D.=1.46) years]. Zygosity was established using physical similarity questionnaires administered to the twins' primary caregiver (Peeters *et al.* 1998). On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of 95% or better.

Primary analyses

Measures

Child conduct problems. Parents completed the Achenbach Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) separately for each twin, while the twins completed the Semistructured Clinical Interview for Children and Adolescents (SCICA; McConaughy& Achenbach, 2001), the corresponding interview for youth aged 6–18 years. Twins were interviewed in separate rooms by different interviewers. In the current study, we made use of the Diagnostic and Statistical Manual of Mental Disorders (DSM)-oriented Conduct Problems (CP) scale (Achenbach & Rescorla, 2001; McConaughy & Achenbach, 2001), which comprises 17 CBCL items and 19 SCICA items (with nearly identical item content) viewed as 'very consistent' with the DSM-IV diagnostic category of Conduct Disorder (e.g. stealing, fighting, setting fires, cruelty to animals, etc.). Further validation work (Achenbach *et al.* 2001) indicated that the DSM-oriented CP scale accurately captures conduct-disordered behavior and DSM-IV diagnoses. Internal consistency reliabilities for the CBCL scales were adequate (α=0.80 and 0.76 for mother- and father-informant reports, respectively). Roughly 10% of SCICA interviews were videotaped to obtain inter-rater reliability (the average intraclass correlation across raters was 0.88).

Maternal and paternal reports of twin CP were available for 996 and 862 twins, respectively. Child interviews were available for 996 twins. As expected based on prior meta-analyses of informant effects (Achenbach *et al.* 1987), the various informant reports of CP were moderately intercorrelated (maternal- and paternal-informant reports were correlated 0.43; child interviews were correlated 0.32 and 0.27 with maternal- and paternal-informant reports, respectively; all p<0.01). CP data were averaged across informants to create a CP composite. When only one informant report was available (n=3 twins), that report was used for analyses. The use of this combined informant approach is thought to allow for a more complete assessment of twin symptomatology than would the use of any one informant alone (Achenbach *et al.* 1987). Consistent with manual recommendations (Achenbach & Rescorla, 2001), analyses were conducted on the raw CP composite. Data were log-transformed prior to analysis to adjust for positive skew (skews before and after transformation were 2.28 and 0.54, respectively).

Parent–child conflict. The parent–child conflict scale on the Parental Environment Questionnaire (PEQ; Elkins *et al.* 1997) was administered to assess conflict in each parent–child relationship (12 items; e.g. 'My parent often criticizes me'; 'My parent and I often get into arguments'). Mothers and fathers individually rated their relationships with each of their participating children, while children individually rated their relationships with their mother and their father. Items were the same for parents and children, with alterations in wording appropriate for particular raters. Each item was rated on a four-point scale (1=definitely true; 2=somewhat true; 3=somewhat false; 4=definitely false). The PEQ was read to twins with reading levels under 5th grade (as assessed via a brief reading screen; Torgesen *et al.* 1999) to assure comprehension of the items. The conflict scale displayed good internal consistency reliability, with α's between 0.74 and 0.87 across all age groups (i.e. 6–8 and 9–11 years old) and individual informants (i.e. parents and children). Maternal and paternal reports of conflict were available for 990 and 857 twins, respectively. Child reports of their relationships with their mother and father were available for 985 and 960 twins, respectively. As with CP, the various informant reports were modestly to moderately correlated (r=0.16–0.32, all p<0.01). Consistent with prior work (Burt *et al.* 2003, 2005), the four informant reports were averaged together to create a composite of parent–child conflict.

Analyses

Twin studies leverage the difference in the proportion of genes shared between MZ twins (who share 100% of their segregating genes) and dizygotic (DZ) twins (who share roughly 50% of their segregating genes) to estimate additive genetic (A), shared environmental (i.e. environmental factors that make twins similar to each other; C) and non-shared environmental (i.e. factors that make twins different from each other, including measurement error; E) contributions to a

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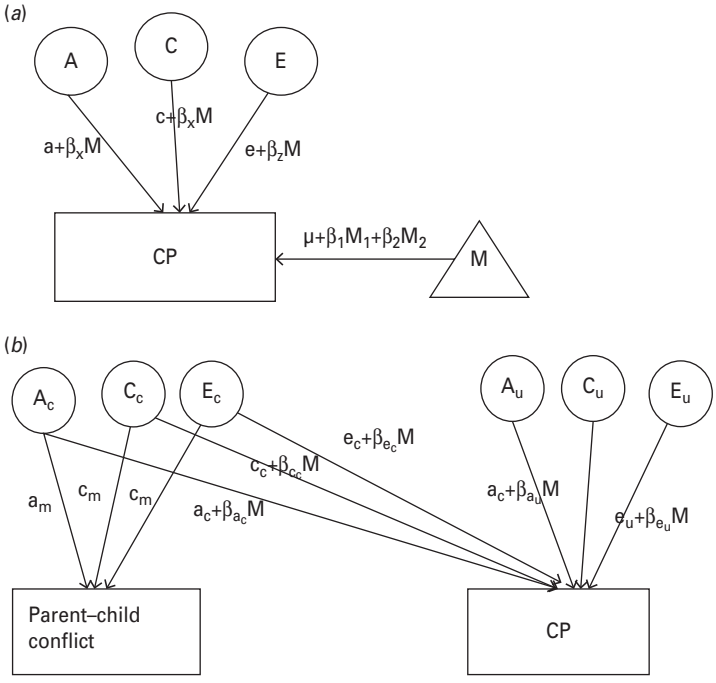

Fig. 1. (*a*) The extended univariate gene–environment interaction (G×E) model. (*b*) The bivariate G×E model. A, C and E represent genetic, shared environmental, and non-shared environmental influences, respectively. For ease of presentation, the co-twin variables and paths are omitted here, though they are estimated in the models. In model 1*a* (van der Sluis *et al*. 2012), the variance decomposition of Conduct Problems (CP) was modeled as a function of parent–child conflict (the moderator, M). To circumvent possible r_{GE} confounds, the parent–child conflict values of both twins were entered in a means model of CP. Linear moderation was then modeled on the residual CP variance (i.e. that which does not overlap with parent–child conflict), separately for each component of variance (i.e. β_XM, β_YM and β_ZM for a, c and e paths, respectively). The non-linear moderators are not shown. In model 1*b* (Purcell, 2002), the moderator is entered twice: once as a variable that is allowed to correlate with the outcome and once as the moderator. A_C and A_U, respectively, represent genetic influences on CP held in common with the moderator and those unique to CP. Interactions with the moderator (e.g. β_{ac}M and β_{au}M) are added to these common and unique genetic influences. Only the latter are thought to index 'true' G×E. The same interpretation holds for C and E effects.

given phenotype. More information on twin studies is provided elsewhere (Plomin *et al*. 2012).

For our primary analyses, we evaluated whether parent–child conflict moderated the etiology of CP using the 'extended univariate G×E' model (Purcell, 2002; van der Sluis *et al*. 2012). In this model (see Fig. 1*a*), the variance decomposition of CP was modeled as a function of parent–child conflict. To circumvent possible gene–environment correlational confounds (in which genetic effects overlap across the moderator and the outcome), the moderator values of both twins were entered in a means model of each twin's CP. Moderation was then modeled on the residual CP variance (i.e. that which does not overlap with parent–child conflict). The first and least restrictive of these models allows for linear and non-linear moderation. We then fitted a series of more restrictive moderator models, constraining the moderators to be zero and evaluating the reduction in model fit.

The extended univariate G×E model is quite flexible. Twins are not required to be concordant on the value of the moderator (although they can be), and the moderator can be either continuous or categorical, although it should include zero. The conflictive parenting variable was thus floored at zero and collapsed into nine groups (from an observed range of 0 to 24.5), so that each level of conflictive parenting contained roughly 100 twins. Although the interpretation of standardized or proportional ACE estimates may be useful in some cases, it is generally recommended that unstandardized or absolute ACE estimates be presented (Purcell, 2002). We thus standardized our log-transformed CP score to have a mean of 0 and a standard deviation of 1 to facilitate interpretation of the unstandardized values.

Mx, a structural-equation modeling program (Neale *et al*. 2003), was used to fit models to the transformed raw data using full-information maximum-likelihood techniques. When fitting models to raw data,

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variances, covariances and means are first freely estimated (minus twice the log-likelihood; −2lnL). Model fit for the more restrictive biometric G×E models was then evaluated using four information-theoretic indices that balance overall fit (via −2lnL) with model parsimony: the Akaike's Information Criterion (AIC; Akaike, 1987), the Bayesian Information Criterion (BIC; Raftery, 1995), the sample size-adjusted BIC (SABIC; Sclove, 1987) and the deviance information criterion (DIC; Spiegelhalter *et al*. 2002). The lowest or most negative AIC, BIC, SABIC and DIC among a series of nested models is considered best. Because fit indices do not always agree (they place different values on parsimony, among other things), we reasoned that the best-fitting model should yield lower or more negative values for at least three of the four fit indices (Hicks *et al*. 2009).

Confirmatory analyses

To evaluate the robustness of our primary G×E results, we conducted four sets of confirmatory analyses.

Analysis 1

We first sought to evaluate whether our primary results persisted to observer ratings of videotaped parent–child interactions. Interactions took place in laboratory space restructured to resemble a living room (i.e. couch, coffee table, area rug, pictures, etc.). Each parent–child dyad (i.e. mother–twin 1, mother–twin 2, father–twin 1, and father–twin 2) was asked to complete an 8-min task that was mildly to moderately frustrating (i.e. use an Etch-a Sketch to draw specific pictures, but parent and child could each use only use one dial, thereby requiring cooperation). Interaction data were coded using the Twin Parent–Child Interaction System (Deater-Deckard *et al.* 1997). Each observer received approximately 85 h of training and was required to pass observation examinations before coding videotapes. Observers attended bi-weekly coder meetings for ongoing training and to prevent 'rater drift'. Observer reliability was assessed by randomly assigning 10% of all tapes to be rated by a second observer, and then comparing the primary and secondary ratings using intraclass correlations. Following training, each video was watched three times: once to code the behavior of the parent, once to code the behavior of the child, and once to code dyadic behaviors between parent and child. To reduce the possibility of rater bias, each parent–child dyad was coded by a research assistant who was blind to all participant data. Further, different coders rated each of the four parent–child dyads within a family, eliminating the possibility of shared method variance.

In the current study, we focused on the parental negativity (or negative content) variable, which assesses parental use of physical control and criticism during the interaction (inter-rater intraclass correlations were 0.97). Observer ratings of maternal and paternal negativity were modestly associated with maternal- and paternal-informant reports of their own conflictive parenting (r=0.12 and 0.16, respectively). Although small, these associations are fully consistent with the modest observer rating/informant report correlations seen in other studies (Arsenault *et al*. 2003; Burt *et al*. 2011). A composite rating of maternal and paternal negativity was available for 941 twins.

Analysis 2

Van der Sluis *et al*. (2012) recommended that researchers confirm positive findings of etiological moderation using the bivariate G×E model (see Fig. 1*b*; Purcell, 2002), since the extended univariate G×E model is unable to distinguish between moderation of the covariance path and moderation of the residual path (only the latter of which represents 'true' G×E). Although useful for confirming G×E in this way, the bivariate G×E model otherwise suffers from a number of problems, including issues of identifiability (Rathouz *et al*. 2008). Given these problems, we restricted our core G×E analyses to the extended univariate model, and made use of the bivariate model only to confirm those results.

Analysis 3

We also sought to confirm that our results persisted to other operationalizations of child CP, as G×E modeling results can be influenced by the scaling and distribution of the outcome variable (Purcell, 2002). This re-analysis was conducted in two ways. First, rather than log-transforming our CP variable to adjust for positive skew, we instead rank-normalized and Blom-transformed CP separately by sex (skew after this transformation was 0.30). Our primary G×E analyses were then re-run using the Blom-transformed CP variable as our outcome. Second, we created a second measure of CP, in which the maximum CP score according to any one informant was used as an index of child CP. Analyses were then re-run using 'maximum CP' as our outcome variable.

Analysis 4

We also examined whether the G×E effect identified using the global measure of parent–child conflict persisted to maternal–child and paternal–child conflict, as well as individual informant reports of parent–child

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Table 1. *Intraclass twin correlations and univariate heritability estimates*[a]

	Intraclass correlations		Parameter estimates, %		
	MZ	DZ	A	C	E
CP (n=1000)	0.61	0.45	35.4	27.2	37.4
Conflict (n=999)	0.58	0.52	19.1	52.8	28.1
CP at lowest level of conflict (n=108)	0.50	0.30	–	–	–
CP at average level of conflict (n=126)	0.43	0.34	–	–	–
CP at highest level of conflict (n=73)	0.52	0.50	–	–	–

MZ, monozygotic; DZ, dizygotic; A, genetic parameter estimate; C, shared environmental parameter estimate; E, non-shared environmental parameter estimate; CP, Diagnostic and Statistical Manual of Mental Disorders-oriented Conduct Problems.

[a] All intraclass correlations were calculated using double-entered data, in keeping with standard practices. The top half of the table presents results for the full sample. Intraclass correlations in the bottom half of the table are presented separately by level of parent–child conflict (note that we are only presenting correlations for the lowest, highest and middle levels of conflict, not for all nine levels of conflict). A, C and E estimates at the various levels of conflict are examined in subsequent gene–environment interaction (G×E) models, and so are not presented here. All correlations and ACE estimates were significantly greater than zero at p<0.05.

conflict. To do so, we re-ran our G×E analyses separately for maternal and child reports of maternal–child conflict, and for paternal and child reports of paternal–child conflict.

Results

In keeping with the expected rate of child conduct problems in the general population (Moffitt, 1993, 2003), 9.6% of participants evidenced clinically significant levels of CP (as defined in the CBCL and SCICA manuals) according to both parental report and child interview. Boys evidenced significantly higher rates of raw CP than did girls [mean=1.98 (S.D.=2.11) for boys and mean=1.20 (S.D.=1.49) for girls; Cohen's d=0.43, p<0.05). Mean levels of parent–child conflict were also higher in boys as compared with girls (Cohen's d=0.23, p<0.05). Although parent–child conflict was not significantly associated with twin age (r=0.01, N.S.), CP demonstrated a small and negatively signed associated with age (r=–0.11, p<0.05). Sex and age were thus regressed out of CP prior to analyses (McGue & Bouchard, 1984). As expected, CP was positively associated with both conflictive parenting (r=0.41, p<0.001) and observer ratings of parental negativity (r=0.17, p<0.01).

Intraclass correlations and univariate estimates

Basic estimates of heritability are presented in Table 1. As seen there, both CP and parent–child conflict evidenced small-to-moderate genetic influences. Shared environmental influences, by contrast, were moderate to large in magnitude, in keeping with prior work (Burt, 2009*b*). We also present MZ and DZ intraclass correlations separately by level of conflict experienced. Although these results do not constitute a formal test of etiological moderation, the pattern of MZ–DZ differences observed across the cells is consistent with either decreasing genetic influences and/or increasing shared environmental influences on CP with increasing levels of parent–child conflict.

Primary analyses

Formal tests of moderation were conducted next. As seen in Table 2, the best-fitting model was the linear C moderation model. Estimated paths and moderators from the full and best-fitting linear models are presented in Table 3. Unstandardized or absolute genetic and environmental variance contributions to CP at each level of parent–child conflict are plotted in Fig. 2. A and E contributions to CP were significantly greater than zero and small to moderate in magnitude across all levels of conflict. C contributions, by contrast, were near zero at the lowest levels of conflict (the 'c' path was small and non-significant), but increased dramatically (and significantly) with increasing levels of conflict. Indeed, shared environmental influences on CP at high levels of parent–child conflict were many-fold larger than those at low levels of conflict[1]†.

Although the above findings indicate that parent–child conflict moderates only the shared environmental component of variance, there is one key consequence of the absolute increase in C for the genetic (and non-shared environmental) components of variance: namely, when A, C and E estimates are considered relative to one another, A appears to be proportionally more important to CP at low levels of conflict than at high levels of conflict (even as its absolute contribution remains unchanged). To empirically evaluate this possibility, we computed standardized estimates of A at the lowest and highest levels of conflict in the best-fitting model. We then computed the difference score between these standardized estimates of A, as

† The notes appear after the main text.

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Table 2. *Fit indices*

Model	–2lnL	df	AIC	BIC	SABIC	DIC
Parent–child conflict (composite of parent- and child- informant reports)						
(1a) Linear and non-linear ACE moderation	2504.46	983	538.46	−1801.27	−241.22	−897.95
(1b) Linear ACE moderation	2507.97	986	535.97	−1808.83	−244.02	−902.75
(1c) Linear A moderation only	2513.33	988	537.33	−1812.36	−244.38	−904.45
(1d) Linear C moderation only[a]	2511.56	988	535.56	−1813.25	−245.26	−905.34
(1e) Linear E moderation only	2515.19	988	539.19	−1811.43	−243.45	−903.52
(1f) No moderation	2526.15	989	548.15	−1809.06	−239.49	−900.23
Parental negativity (observer ratings of parent–child interactions)						
(2a) Linear ACE moderation	2453.13	928	597.13	−1628.30	−155.66	−775.53
(2b) Linear A moderation only	2456.05	930	596.05	−1632.99	−157.17	−778.38
(2c) Linear C moderation only[a]	2453.41	930	593.41	−1634.32	−158.50	−779.71
(2d) Linear E moderation only	2459.39	930	599.39	−1631.32	−155.50	−776.71
(2e) No moderation	2460.18	931	598.18	−1634.01	−156.60	−778.48

2lnL, Minus twice the log-likelihood; df, degrees of freedom; AIC, Akaike's Information Criterion; BIC, Bayesian Information Criterion; SABIC, sample size-adjusted Bayesian information criterion; DIC, deviance information criterion; A, genetic parameter estimate; C, shared environmental parameter estimate; E, non-shared environmental parameter estimate.

[a] Best-fitting model for a given set of analyses, as indicated by the lowest AIC, BIC, SABIC and DIC values for at least three of the four fit indices.

well as the 95% confidence interval (CI) of that difference score[2]. Difference score CIs that did not overlap with zero would indicate that the two estimates were significantly different from one another (Cumming & Finch, 2005; Knezevic, 2008), even though the absolute or unstandardized contribution of A remained constant. Results of these *post hoc* analyses are presented in Table 4. As seen there, A accounted for 53.5% of the variance in CP at low levels of conflict, but only 32.1% at high levels of conflict. Moreover, this difference was statistically significant, as indicated by difference score CIs that did not overlap with zero. When viewed alongside the absolute increases in C with parent–child conflict, such findings are fully consistent with the bioecological model of G×E.

Confirmatory analyses

(1) Do these findings persist to other operationalizations of parent–child conflict?

To confirm that our results were not simply a function of shared informant effects (both CP and conflict were measured via parent- and child-informant reports), we re-ran our primary analyses using observer ratings of parental negativity. Given the above results, analyses were restricted to the various linear and no-moderation models. Results are presented in Tables 2 and 3. As seen there, the C moderation model again provided the best fit to the data. The parameter estimates were also fully consistent with those for parent–child conflict. Moreover, the standardized estimates of A at low levels of parental negativity (63.3%) were again significantly larger (at $p<0.05$, results not shown) than those at high levels of parental negativity (43.4%). Such findings imply that our results are robust to the specific operationalization of parent–child conflict.

(2) Do our findings of moderation persist to the bivariate G×E model?

We sought to further confirm the above results using the bivariate G×E model (Purcell, 2002), as recommended by van der Sluis *et al.* (2012). For observer ratings of parental negativity, the unique C moderator was estimated at 0.20 ($p<0.05$) and the common C moderator was estimated at 0.00. For parent–child conflict, the unique C moderator was estimated at 0.08 ($p<0.05$) and the common C moderator was estimated at 0.05. Although this latter moderator was also statistically significant, the unique moderator results are generally very similar to those reported in Table 3, serving to bolster our conclusion that parent–child conflict/parental negativity moderates shared environmental influences on CP.

(3) Do our findings persist to other operationalizations of child CP?

We next re-ran our primary G×E analyses using different transformations and measures of child CP. The linear C moderation-only model provided the best fit to the Blom-transformed CP data by all four fit indices (results available on request). Moreover, the C moderator was estimated to be 0.09 ($p<0.05$), whereas the

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Table 3. *Unstandardized path and moderator parameter estimates for the full and best-fitting models*[a]

	Paths			Linear moderators		
	a	c	e	A_1	C_1	E_1
Parent–child conflict (composite of parent- and child-informant reports)						
Full Linear ACE moderation model	0.69 (0.21–0.82)*	−0.08 (–0.61 to 0.33)	0.48 (0.39–0.59)*	−0.02 (–0.09 to 0.09)	0.10 (0.03–0.15)*	0.03 (–0.01 to 0.05)
Best-fitting linear C moderation model	0.63 (0.45–0.71)*	−0.13 (–0.41 to 0.30)	0.57 (0.52–0.63)*	–	0.11 (0.04–0.15)*	–
Parental negativity (observer ratings of parent–child interactions)						
Full linear ACE moderation model	0.75 (0.07–0.86)*	−0.01 (–0.70 to 0.70)	0.57 (0.49–0.67)*	−0.02 (–0.14 to 0.24)	0.18 (–0.29 to 0.29)	−0.01 (–0.07 to 0.05)
Best-fitting linear C moderation model	0.75 (0.53–0.82)*	−0.13 (–0.39 to 0.47)	0.55 (0.50–0.63)*	–	0.19 (0.03–0.28)*	–

[a] A, C and E (upper and lower case), respectively, represent genetic, shared, and non-shared environmental parameters on child conduct problems (CP). 95% confidence intervals are presented in parentheses alongside their respective estimates. Because the lowest level of parent–child conflict/parental negativity was dummy coded as 0 in all models, the genetic and environmental contributions to CP at this level can be obtained by squaring the path estimates (i.e. a, c and e). At each subsequent level, linear moderators (i.e. A_1, C_1, E_1) were added to the paths using the following equation: unstandardized variance total = $[a + A_1(\text{parent–child conflict})]^2 + [c + C_1(\text{parent–child conflict})]^2 + [e + E_1(\text{parent–child conflict})]^2$.

* Estimate is significant ($p < 0.05$).

Fig. 2. Moderation of Diagnostic and Statistical Manual of Mental Disorders (DSM)-oriented Conduct Problems (CP) by parent–child conflict. (*a*) Full linear ACE moderation model, as reported in Tables 2 and 3. (*b*) Best-fitting linear C moderation-only model, as reported in Tables 2 and 3. A, C and E represent unstandardized genetic, shared, and non-shared environmental influences, respectively. These unstandardized estimates index absolute changes in genetic and environmental influences on CP with level of parent–child conflict. Of note, because variance is a second-order statistic, the linearity in the best-fitting linear model is at the level of effect rather than the level of variance component (Purcell, 2002); as a result, the variance components may appear to shift non-linearly even though the model itself is linear.

A and E moderators (when estimated) ranged from −0.02 to 0.01. We then re-ran the analyses using each participant's maximum CP score according to any one informant as an index of his or her level of CP. There was again evidence of significant environmental, but not genetic, moderation of CP by conflict. The genetic moderator was estimated at −0.01 (N.S.), whereas the shared environmental moderator was estimated at 0.12 (95% CI 0.07–0.17, $p < 0.05$). Such findings are fully consistent with those reported above, implying that our results are robust to the specific operationalization of CP.

Table 4. *Standardized genetic and environmental variances at the lowest and highest levels of parent–child conflict*[a]

	Parameter estimates, %		
	A	C	E
Lowest level of parent–child conflict (n=108)	53.5 (31.1–61.6)*†	2.4 (0–19.1)†	44.2 (31.6–60.4)*†
Highest level of parent–child conflict (n=73)	32.1 (18.0–44.7)*†	41.4 (24.1–54.9)*†	26.5 (19.3–37.4)*†
Difference score	21.4 (10.4–29.0)*	−39.0 (–54.2 to –17.9)*	17.6 (7.3–29.9)*

[a] A, C and E represent standardized genetic, shared, and non-shared environmental parameters on child conduct problems (CP), respectively. 95% confidence intervals are presented in parentheses alongside their respective estimates.

* Parameter estimate is significantly greater than zero (p<0.05).

† Standardized parameter estimates at the lowest and highest levels of parent–child conflict (i.e. those within a given column) differ from one another (p<0.05), as indicated by a difference score confidence interval that does not overlap with zero.

(4) Do these findings persist to maternal–child and paternal–child conflict, respectively?

As a final confirmation, we clarified whether these results persisted to the mother–child and the father–child relationship, when analysed separately. We thus re-ran our G×E analyses separately for maternal and child reports of maternal–child conflict, and for paternal and child reports of paternal–child conflict. In all four cases, the C moderation model provided the best fit to the data by at least three of the four fit indices (results available on request). Moreover, the C moderator values ranged from 0.06 to 0.12 (all p<0.05). Our findings thus appear to be robust to both mother–child and father–child relationships, as well as to specific informant reports of those relationships.

Discussion

The goal of the current study was to evaluate whether and how parent–child conflict moderated the etiology of child CP. Results offered strong support for the presence of shared environmental moderation of CP by parent–child conflict: C did not significantly contribute to CP in those with low levels of parent–child conflict, but increased dramatically and significantly with increasing levels of parent–child conflict. A and E contributions to CP, by contrast, were unchanged across all levels of parent–child conflict. Despite their absolute etiological stability, however, the relative proportion of variance accounted for by A and E changed significantly across high and low levels of parent–child conflict, such that A and E were proportionally more influential at low *versus* high levels of parent–child conflict. These results fully persisted to observer ratings of the parent–child relationship, as well as to other operationalizations of child CP, to individual informant reports of conflict, and to the mother–child and father–child relationships. Such findings thus serve to not only illuminate the origins of child CP, but also provide important empirical support for the bioecological model of G×E.

Despite the strength of these results, there are several limitations that should be considered. First, although our sample is only moderately sized by current twin study samples, previous power analyses (Purcell, 2002) suggest that it is more than adequate for the G×E models used here. Nevertheless, analyses incorporating sex would probably be unwieldy and underpowered in this sample. It thus remains unclear whether the G×E identified here varies across sex (although it is worth noting that CP heritability estimates in general do not vary significantly across sex; Burt, 2009*a*, *b*). We also did not directly examine the effects of age. As such, the current results should not be applied to other developmental periods. Next, although shared environmental influences on CP were moderated in these analyses, examinations of other environmental risk and protective factors in these data have revealed evidence of genetic moderation (Humbad *et al.* 2012). The current results are thus specific to the association between parent–child conflict and child CP, and do not imply that all G×E underlying childhood CP are bioecological in nature.

Conclusions

The current study found evidence that high levels of parent–child conflict exacerbate shared environmental influences on child CP, and, in doing so, effectively dampen the proportion of variance accounted for by genetic influences. Moreover, because the models we used control for gene–environment correlation

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confounds (i.e. r_{GE}; non-random exposure to particular environmental experiences), the exacerbation of shared environmental influences on CP by high levels of conflict is likely to represent 'true' G×E. Such findings have several interrelated implications. First, they clearly suggest that G×E are not restricted to the diathesis–stress model of moderation, but can take other forms as well. In this case, results were more consistent with a bioecological G×E, which postulates that, in some cases, adverse environments may provide such a strong 'social push' for negative outcomes that the importance of genetic factors on individual differences is accordingly diminished. What might this mean in concrete terms? Our results suggest that children experiencing conflictive parenting are, on average, prone to higher levels of CP, and that this increase is due largely to the common family environment. By contrast, to the extent that children experiencing low-risk parenting engage in CP, their behavior appears to be largely a function of genetic influences. Put differently, because such children are experiencing little by way of environmental risk for CP, the remaining individual differences are largely genetic in origin.

Second, our finding of shared environmental moderation stands in sharp contrast to those studies conducted on adolescent and emerging adult samples (e.g. Feinberg *et al.* 2007; Hicks *et al.* 2009), for which conflictive/negative parenting appears to directly exacerbate genetic influences on conduct problems. This same developmental pattern (bioecological moderation in childhood, diathesis–stress moderation in adolescence) has also been identified for delinquent peer affiliation (e.g. Harden *et al.* 2008; Hicks *et al.* 2009). Such findings collectively (if speculatively) imply that the moderating effects of particular environmental experiences may shift in meaningful ways over the course of development. Although this possible developmental shift is inconsistent with the common (if implicit) assumption that G×E are unaffected by human development (Burt, 2011), it does dovetail quite nicely with the broader literatures regarding heritability and r_{GE} (Scarr & McCartney, 1983). For example, prior work has convincingly demonstrated that the importance of genetic influences on CP change dramatically from childhood through adolescence (Lyons *et al.* 1995; Burt & Klump, 2009; Burt & Neiderhiser, 2009). It thus stands to reasons that the constituent pieces of these heritability estimates (which include most G×E and r_{GE}; Purcell, 2002) may also shift during this developmental period. Future work should examine more directly the possibility that, much like r_{GE}, G×E may be influenced in meaningful ways by the challenges and experiences characteristic of particular developmental periods.

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Declaration of Interest

None.

Notes

[1] To further ensure that our results were reflective of C moderation in particular, we ran a model that included one linear moderator parameter for A, C and E (i.e. a general etiological moderator), as well as a second linear moderator parameter just for C (i.e. a shared environmental-specific moderator). The general moderator value was near zero (0.016) and non-significant. The C-specific moderator, by contrast, was moderate in magnitude (0.127) and significantly larger than zero at $p<0.05$. When viewed in conjunction with our other results, such findings strongly imply that the etiological moderation of CP is specific to its shared environmental influences.

[2] The difference score method was favored here because the more standard constraint analyses effectively forced the C moderator to zero in order to constrain the standardized A estimates to be equal. Nevertheless, it is worth noting that our conclusion is identical when using constraint models in place of difference scores (i.e. the standardized A estimates at high and low levels of conflict, respectively, cannot be constrained to be equal without a significant decrement in fit).

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