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Maternal Warmth and Directiveness Jointly Moderate the Etiology of Childhood Conduct Problems

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Abstract

Background—Prior studies exploring gene-environment interactions (GxE) in the development of youth conduct problems (CP) have focused almost exclusively on single risk experiences, despite research indicating that the presence of other risk factors and/or the absence of protective factors can accentuate the influence of a given risk factor on CP. The goal of the current study was to fill this gap in the literature, evaluating whether risky and protective aspects of parenting might combine to jointly moderate the etiology of CP.

Methods—The sample consisted of 500 child twin pairs from the Michigan State University Twin Registry (MSUTR). Child CP was assessed using multiple informant reports. Maternal warmth and directiveness were assessed via videotaped dyadic interactions between mothers and each of their twins.

Results—Biometric GxE analyses revealed that directiveness and warmth did appear to jointly moderate the etiology of CP. In particular, shared environmental influences were accentuated by colder, less directive or “less engaged” mothering, while genetic influences were strongest when the child was experiencing warmer, more directive or “more authoritative” mothering.

Conclusions—Such findings serve to highlight the synergistic effects of risky and protective experiences on child outcomes. They also provide additional empirical support for the bioecological form of GxE which postulates that, in some cases, genetic influences may be most strongly expressed in the presence of low risk environments.

Keywords

child conduct problems; maternal directiveness or control; maternal warmth; GxE

Gene-environment interaction (GxE) is thought to constitute a fundamental mechanism through which genes influence human behavior and mental health (Rutter, Moffitt, & Caspi, 2006). GxE may be defined as differential responsiveness to environmental risk as a function of genotype, such that the environment alters the impact of genetic influences (Plomin, DeFries, & Loehlin, 1977). Available latent GxE work has strongly supported the presence of GxE in the development of antisocial/externalizing behaviors (Burt & Klump, in press; Cleveland, 2003; Feinberg, Button, Neiderhiser, Hetherington, & Reiss, 2007; Hicks, South, DiRago, Iacono, & McGue, 2009; Lipscomb et al., 2012; Tuvblad, Grann, & Lichtenstein, 2006), as has extant molecular GxE work (Caspi et al., 2002; Kim-Cohen et

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al., 2006). Feinberg and colleagues, for example, examined whether parental negativity and warmth (as examined in separate analyses) moderated genetic and environmental influences on adolescent behavior problems, and found that genetic influences were potentiated in the face of negative parenting or low warmth.

Despite this robust support, both latent and molecular GxE studies to date have focused all but exclusively on individual or single risk/protective experiences, even though few would argue that any one risk factor captures the full array of environmental forces impinging on children (Forehand, Biggar, & Kotchick, 1998; Rothbaum & Weisz, 1994). An example of the potential advantage gained by the simultaneous examination of multiple risk factors can be seen in the work of Forehand and colleagues (1998). They found that a broad index of risk, in which multiple risk factors were considered simultaneously, explained far more variance in youth conduct problems than did the sum of its parts. Furthermore, it is not necessarily the case that high levels of risk always co-occur with low levels of protection. Rather, it is the relative balance of risk and protective processes that predicts whether an aggression-prone child ultimately experiences a deviant or a healthy outcome (Tolan, Guerra, & Kendall, 1995). A meta-analysis of the relationship between parental caregiving and child externalizing behavior, for example, found that composite indicators of risky and protective parenting (high and low levels, respectively) best predicted child externalizing outcomes, increasing the mean study effect size from .23 (when variables were examined separately) to .45 (Rothbaum & Weisz, 1994). We would thus expect risk and protective factors to influence youth conduct problems *synergistically*, such that the impact of high risk environments can be reduced by the simultaneous presence of protective experiences (or could be further accentuated by low levels of protective experiences).

Two variables that may serve to jointly moderate the etiology of child conduct problems are the orthogonal parenting dimensions of warmth and demandingness or directiveness (Baumrind, 1967; Maccoby & Martin, 1983). Low levels of warmth or affection have proven to be a robust predictor of youth conduct problems (Dodge, Pettit, & Bates, 1994; Hipwell et al., 2008; Stormshak, Bierman, McMahon, & Lengua, 2000). Directiveness, or the provision of control and expectations for behavior, has demonstrated less consistent links with conduct problems, however. Some studies have reported that conduct problems are predicted by high levels of parental directiveness (Campbell, Pierce, Moore, Marakovitz, & Newby, 1996), while others have suggested that low levels of directiveness are more predictive (Pettit, Laird, Dodge, Bates, & Criss, 2001). These inconsistencies across studies may partially reflect differences in the definitions and extent of directiveness found in various datasets (e.g., clear expectations for behavior versus over-controlling or harsh parenting). However, they may also reflect the known synergies between directiveness and warmth (illustrated in Figure 1, as described by Maccoby & Martin, 1983). Essentially, the presence or absence of parental warmth may alter the effects of parental directiveness on child outcome. Authoritative (i.e., high warmth, high directiveness) parenting, for example, has been described as a particularly beneficial parenting style (Fletcher, Darling, Steinberg, & Dornbusch, 1995), while authoritarian (i.e., low warmth, high directiveness) parenting and disengaged (i.e., low warmth, low directiveness) have been associated with psychological and behavioral problems in the children (Deater-Deckard, Ivy, & Petrill, 2006; Lamborn, Mounts, Steinberg, & Dornbusch, 1991; Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994).

Current Study

The current study sought to build on the above research, while also filling a critical gap in the extant GxE literature, by examining how maternal directiveness and warmth might jointly moderate the etiology of child conduct problems. Although these sort of synergistic

links between predictors are likely to capture a more nuanced and complete representation of the child's parenting environment, we know of no GxE study to *simultaneously* examine environmental risk and protective factors. This exploratory study should thus serve to inform our understanding of GxE processes.

Methods

Participants

The Michigan State University Twin Registry (MSUTR) includes several independent twin projects (Burt & Klump, 2013). The 500 twin families included in the current study (N=1,000 twins and their parent/s) were assessed as part of the Twin Study of Behavioral and Emotional Development in Children (TBED-C) within the MSUTR. Recruitment procedures are outlined in detail in Burt & Klump (2013). To be eligible for participation, neither twin could have a cognitive or physical condition (e.g., significant developmental delay) that would preclude completion of the roughly 4-hour assessment (as assessed during the initial phone screen). Participating twins were 47.0% female and ranged in age from 6 to 10 years, although a small handful (n=14 pairs) had turned 11 by the time they participated (mean age (SD) = 8.2 years (1.46)). Informed consent was obtained from parents for themselves and their twins. Informed assent was obtained from the twins.

Our final sample was broadly representative of the area population and of recruited families (as detailed in Burt & Klump, 2013). In brief, participating families endorsed ethnic group memberships at rates comparable to 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). Moreover, participating twins were not experiencing higher levels of conduct problems, emotional symptoms, or hyperactivity than were non-participating twins (Cohen's *d* effect sizes = -.076; ns).

Zygosity was established using physical similarity questionnaires administered to the twins' primary caregiver (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of 95% or better. Approximately half of the twin pairs (N=251) were monozygotic.

Measures

Child Conduct Problems—Mothers and fathers completed the Achenbach Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) separately for each twin, while the twins individually completed the Achenbach Semistructured Clinical Interview for Children and Adolescents (SCICA; McConaughy & Achenbach, 2001). The DSM-oriented Conduct Problems or CP scale comprises 17 CBCL items and 19 SCICA items 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, Dumenci, & Rescorla, 2001) indicated that the CP scale accurately captures conduct disordered behavior and diagnoses. Internal consistency reliabilities for the CBCL scales were adequate ($\alpha = .80$ and $.76$ for mother and father informant-reports, respectively). Roughly 10% of the SCICA interviews were videotaped to obtain inter-rater reliability (the intraclass correlation for CP across all raters was $.88$).

Consistent with the modest cross-informant correlations seen in other studies (Achenbach, McConaughy, & Howell, 1987), the various informant-reports of CP were correlated $.27$ to $.43$ (all $p < .01$). 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). There were no missing CP data following the creation of the composite. CP data were log-transformed prior to analysis to adjust for positive skew (skews before and after transformation were 2.28 and 0.54, respectively).

Parenting—Each mother-child dyad used an Etch-a Sketch to draw specific pictures, but each participant could only use one dial, thereby requiring their cooperation (Deater-Deckard, Pylas, & Petrill, 1997). Trained raters then coded interpersonal behaviors using a computer joystick apparatus (the Microsoft Sidewinder Force Feedback 2) and related software (Sadler, Ethier, Gunn, Duong, & Woody, 2009). The joystick-monitoring software program displays a Cartesian plane with the axes of interpersonal warmth and interpersonal control or directiveness (very similar to Figure 1). The software program records the x and y coordinates (ranging from -1000 to 1000) of the joystick's position twice per second. Warmth ranged from smiling or leaning towards her child, verbal praise, eye contact, and warm physical contact to looking away, unresponsiveness, and rude or sarcastic comments. Directiveness ranged from following the child's lead and asking for input to giving repeated instructions, and in more extreme cases, grabbing the etch-a-sketch from the child.

Coders were divided into teams of three to four raters, all of whom coded the interactions assigned to that team. Different teams coded the two dyads within a given family (e.g., mother and twin 1; mother and twin 2), thereby eliminating the possibility of "shared rating" effects. Ratings were averaged across team members and across all moments to create overall ratings of maternal warmth and directiveness (available for 929 and 928 twins, respectively). Inter-rater alphas were .89 for directiveness and .70 for warmth.

Analyses

Twin studies leverage the difference in the proportion of genes shared between monozygotic or MZ twins (who share 100% of their genes) and dizygotic or DZ twins (who share an average of 50% of their segregating genes) to estimate the relative contributions of additive genetic (A), shared environmental (i.e., factors that make twins similar to each other; C), and non-shared environmental effects r (i.e., factors that make twins different from each other, including measurement error; E) to the variance within observed behaviors or characteristics (phenotypes). More information on twin studies is provided elsewhere (Neale & Cardon, 1992).

We evaluated whether maternal warmth and directiveness might jointly moderate the etiology of CP using a two-moderator version (Purcell, 2002) of the 'extended univariate GxE' model (van der Sluis, Posthuma, & Dolan, 2012). A key advantage of the van der Sluis et al. (2012) model is that it allows us to circumvent confounding by non-random exposure to maternal warmth and directiveness (e.g., whereby the child elicits particular maternal behaviors). To accomplish this, the moderator values of both the twin and the co-twin (twins are not required to be concordant on the moderator) are entered in a means model of CP separately across twins and zygosity. The linear moderation model is then modeled on the residual CP variance (i.e., that which does not overlap with the moderators).

In the linear moderation model, linear moderators are added to the baseline genetic and environmental paths using the following equation: $Unstandardized\ Variance_{Total} = (a + a_{warmth}(moderator\ value) + a_{directiveness}(moderator\ value))^2 + (c + c_{warmth}(moderator\ value) + c_{directiveness}(moderator\ value))^2 + (e + e_{warmth}(moderator\ value) + e_{directiveness}(moderator\ value))^2$. This model thus allowed us to both examine the unique effects of each individual moderator, as well as to examine the joint effects of the moderators via the parenting style framework described by Maccoby & Martin (1983; as represented in Figure 1). To do this, we first dichotomized the warmth and directiveness variables using a median split (warmth

was dichotomized as cooler = 1, warmer = 0, N's were 465 and 464, respectively; directiveness was dichotomized as more directive = 1, less directive = 0, both N's were 464). We then examined these dichotomized indicators of directiveness and warmth as simultaneously, independent moderators. In this way, we were able to evaluate whether the etiology of CP varied across the four maternal styles described in previous literature. We could also fit a series of GxE 'constraint' sub-models, in which etiologic influences on CP were constrained to be equal across each pair of maternal styles. This approach thus allowed us to explicitly evaluate whether the magnitude of specific etiologic influences on CP varied across warmth and directiveness jointly, and if so, which specific pairs of styles varied from one another (an approach analogous to following up a significant one-way ANOVA with a series of independent t-tests).

Mx, a structural-equation modeling program (Neale, Boker, Xie, & Maes, 2003), was used to fit models to the transformed raw data using Full-Information Maximum-Likelihood. When fitting models to raw data, variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; $-2\ln L$). Model fit was then evaluated using the Akaike's Information Criterion (AIC; Akaike, 1987), the most commonly employed fit index within the field of behavioral genetics (Markon & Krueger, 2004). The AIC balances model fit with model parsimony. The lowest AIC among a series of nested models is considered best.

Results

Raw warmth ranged from -601.2 to 687.5 , with a mean of 213.4 ($SD = 193.6$). Raw directiveness ranged from -647.4 to 942.1 , with a mean of 307.4 ($SD = 284.8$). Directiveness and warmth were moderately associated ($r = -.27$, $p < .001$), indicating that cooler mothers were somewhat more directive. CP was modestly correlated with both directiveness and warmth ($r_s = .14$ and $-.17$, respectively; both $p < .001$). Moreover, mean levels of CP varied across the four maternal styles (i.e., cooler, more directive = 0.88 , cooler, less directive = 0.80 , warmer, more directive = 0.74 , warmer, less directive = 0.67 ; $F(3, 924) = 6.00$, $p < .001$). Subsequent t-test comparisons revealed that mean CP was significantly higher in the presence of cooler mothering than in the presence of warmer mothering regardless of the level of directiveness.

Mean levels of CP varied across sex (Cohen's $d = .43$, $p < .05$), such that boys demonstrated higher rates of CP than did girls. CP also demonstrated a small negative association with age ($r = -.11$, $p < .05$). Sex and age were thus regressed out of the log-transformed CP data prior to analyses (McGue & Bouchard, 1984). The CP data were then standardized to have a mean of zero and a standard deviation of one to facilitate interpretation of the unstandardized parameter estimates.

GxE Analyses

We next fitted the GxE model to the data ($-2\ln L = 2359.72$ on 879 df, $AIC = 601.72$). Parameter estimates are presented in Table 1. As seen there, genetic influences on CP were stronger in the presence of more directive mothering. Shared environmental influences on CP, by contrast, were stronger in the presence of cooler mothering.¹

¹To confirm that these results were robust to our specific analytic approach, we evaluated whether our full GxE model results persisted when warmth and directiveness were analyzed continuously. To do so, warmth and directiveness were each divided by $1,000$, so that they ranged from -1 to 1 . The pattern of GxE modeling results was quite similar to that reported in Table 1. For directiveness, the A and C moderators were moderate in magnitude ($.56$ and $-.33$, respectively), whereas the E moderator was near zero ($-.04$). For warmth, the C moderator remained moderate in magnitude ($.35$) whereas the A and E moderators remained small ($.04$ and $.13$). In short, our results appear to be fairly robust to our specific analytic approach.

To clarify the joint role of warmth and directiveness in the moderation of child CP, we next calculated genetic and environmental influences on child CP for each of the four maternal styles (as plotted in Figures 2 and 3). We then fitted a series of GxE ‘constraint’ models, constraining each pair of unstandardized parameter estimates to be equal to one another (constraining each pair of the 4 maternal styles yields 6 constraint models). The fit of each constraint model was then compared to the fit of the full GxE model (in which all estimates were free to vary). As seen in Table 2, AIC values increased relative to that of the full model when shared environmental influences on cooler, less directive mothering were constrained with any of the other maternal styles. Such findings indicate that shared environmental influences on CP are significantly more pronounced in the presence of cooler, less directive mothering as compared to any other maternal style. Conversely, shared environmental influences on CP were significantly smaller in the presence of warmer, more directive mothering as compared to any other maternal style.

Genetic influences on CP also appeared to vary across maternal styles, although less consistently so. As seen in Table 3, genetic influences on CP were larger in the presence of warmer, more directive mothering than the presence of cooler, less directive or warmer, less directive parenting. They did not differ from those found in the presence of cooler, more directive parenting, however.

Discussion

The current study sought to evaluate whether maternal warmth and directiveness jointly moderated the etiology of child CP. Results revealed that, when warmth and directiveness were examined individually, genetic influences on CP were accentuated by high levels of maternal directiveness or control, while shared environmental influences were accentuated by lower levels of maternal warmth. When warmth and directiveness were examined jointly, however, shared environmental influences were found to be potentiated in the presence of cooler, less directive (or “less engaged”) mothering, while genetic influences were potentiated in the presence of warmer, more directive (or “more authoritative”) mothering.

Such findings have several broad implications. First, our results indicate that the complex interplay between genes and environments does not universally involve the activation of genetic risk by environmental risk, but can take other forms as well. In particular, etiologic moderation can sometimes take the form of a biocological GxE (Bronfenbrenner & Ceci, 1994; Burt & Klump, in press; Lipscomb et al., 2012; Pennington et al., 2009) in which genetic influences are most strongly expressed in low risk environments, whereas less favorable environments serve to enhance the explanatory power of the environment. Put differently, because individuals are experiencing little by way of environmental risk in more advantageous conditions, the remaining individual differences are genetic in origin. Less advantageous environmental conditions, by contrast, would amplify environmental influences on the outcome.

Importantly, these findings of bioecological GxE are notably similar to those in the one other published study of GxE in externalizing during childhood (Lipscomb et al., 2012). In particular, Lipscomb et al. (2012) reported that genetic risk for negative emotionality predicted toddler negative emotionality only under conditions of low environmental adversity. Critically, however, these results in childhood stand in sharp contrast to those of studies conducted on adolescent samples (e.g., Feinberg et al., 2007; Hicks et al., 2009), for which low levels of parental warmth (or high levels of parental negativity) appear to exacerbate genetic influences on CP, results that are far more in keeping with the diathesis-stress model of GxE. As noted in prior research (Burt & Klump, in press), such findings may well imply that, akin to the well-known changes in the importance of particular forms of

gene-environment correlation with age (Scarr & McCartney, 1983), the manifestation of GxE may also shift in meaningful ways over the course of development (at least for particular risk factors). Future research should seek to clarify the role of development in GxE.

Lastly, our findings serve to illuminate the synergistic effects of risk and protective factors on the etiology of CP. Directiveness and warmth were both found to significantly moderate the etiology of child CP, and moreover, appeared to do so in concert. Such findings imply that future GxE research should simultaneously consider the role of multiple environmental experiences, including protective experiences.

Limitations

There are some limitations to the study that should be considered, however. First, although our use of observer-ratings of mothering along with a multi-informant indicator of child CP means that shared informant effects cannot account for our results, we were nevertheless missing father-reports of child CP from 65 families. To confirm that our conclusions were not affected by these missing data, we thus repeated our primary analyses on those 435 families with father-reported CP data. The A and C moderators for directiveness were again moderate in magnitude (.37 and $-.32$, respectively), whereas the E moderator was smaller ($-.10$). For warmth, the C moderator appeared more moderate in magnitude (.14) whereas the A and E moderators remained small ($-.08$ and $.05$). In short, our results appear more or less robust to the missing father data.

Second, given the possible role of age and development in GxE and CP, the current results should be considered specific to the developmental period of middle childhood, and should not be applied to other developmental periods (i.e., adolescence). Similarly, the results apply only to mothering, as fathering was not examined. Third, given our sample size ($n=500$ pairs, 47% female), analyses incorporating sex in a meaningful way would have been unwieldy and underpowered. Fortunately, prior meta-analyses (Burt, 2009) have indicated that heritability estimates for conduct problems do not vary significantly across sex. Next, because our sample was population-based, rates of clinically-significant CP were relatively low (8–10%). Consistent with this, mean levels of maternal warmth and directiveness were both positively signed, indicating that the typical mother in this sample was providing low risk mothering to her twins. Future studies should seek to extend these results to a high-risk sample.

Finally, the phenotypic associations between CP and warmth and directiveness were both small, and when examined jointly, were not necessarily in the direction we expected (e.g., both warmer, less directive and warmer, more directive mothering predicted lower levels of CP, even though prior research has suggested that only the latter is advantageous). It is worth noting, however, that presence of a small or even non-significant phenotypic association between the moderator and the outcome has no substantive bearing on the extent of etiologic moderation (as described by Purcell, 2002), with the lone exception of reducing concerns about possible gene-environment correlational confounds (Rathouz, Van Hulle, Rodgers, Waldman, & Lahey, 2008; van der Sluis et al., 2012).

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Key Points

- Maternal warmth and directiveness jointly moderate the etiology of child conduct problems
- Shared environmental influences on child conduct problems appear to be accentuated by cooler, less directive mothering while genetic influences were strongest when the child was experiencing warmer, more directive mothering
- Genetic influences may sometimes be most strongly expressed in the presence of normal or optimal environments

Figure 1.
Schematic of maternal style (as described in Maccoby & Martin, 1983)

Figure 2. Genetic and environmental influences on Conduct Problems (CP) by maternal warmth and directiveness, when modeled jointly

The results of the two moderator moderation model plotted in a radar graph. A, C, and E represent unstandardized genetic, shared, and non-shared environmental influences, respectively. The ACE estimates for each maternal style are indicated by the specific data points on their axis. Data points in the center of the graph would indicate estimates of 0, while data points on the edge of the graph would indicate estimates of 0.7. Changes in heritability estimates across the various maternal styles are indicated by connections between the data points. Note that, although directiveness and warmth were both dichotomized at their median, the small association between the two variables results in somewhat uneven sample sizes across the four maternal styles.

Figure 3. The Etiology of Conduct Problems (CP) by maternal style

Another representation of the results of the two-moderator moderation model. A, C, and E represent genetic, shared, and non-shared environmental influences, respectively, on child CP. Although directiveness and warmth were each dichotomized at their median, the small association between them resulted in somewhat uneven sample sizes across the four maternal styles.

Table 1

Unstandardized parameter estimates for the full linear moderation model

PATHS			LINEAR MODERATORS			
a	c	e	a _{warmth}	c _{warmth}	e _{warmth}	a _{directiveness}
Maternal warmth and maternal directiveness						
0.46 [*] (0.10, 0.72)	0.53 [*] (0.21, 0.72)	0.59 [*] (0.50, 0.68)	-0.12 (-0.38, 0.09)	0.22 [*] (0.01, 0.46)	0.06 (-0.07, 0.18)	0.33 [*] (0.05, 0.62)
						-0.24 [~] (-0.49, 0.04)
						-0.06 -0.18, 0.07)

Note. a, c, and e respectively represent genetic, shared, and non-shared environmental parameters on CP. Warmth was coded such that 0 = warmer and 1 = cooler. Directiveness was coded such that 0 = less directive and 1 = more directive. Because warmer, less directive was therefore dummy coded as 0, the genetic and environmental contributions to CP at this level can be obtained by simply squaring the path estimates (i.e., a, c, and e). At each subsequent level, linear moderators were added to the paths using the following equation: $Unstandardized\ Variance\ Total = (a + a_{warmth(moderator\ value)} + a_{directiveness(moderator\ value)})^2 + (c + c_{warmth(moderator\ value)} + c_{directiveness(moderator\ value)})^2 + (e + e_{warmth(moderator\ value)} + e_{directiveness(moderator\ value)})^2$.

^{*} indicates that estimate is significant at p<.05;

[~] indicates that the estimate is marginally-significant at p<.10. Estimates calculated this way are presented in Figures 2 and 3.

Table 2

Constraining shared environmental influences on Conduct Problems (CP) to be equal across the different maternal styles

Model	Model Fit		
	-2lnL	df	AIC
1) Full	2359.72	879	601.72
<u>Constraint Models</u>			
2) Cooler, Less Directive & Warmer, Less Directive	2363.59	880	603.59*
3) Cooler, Less Directive & Cooler, More Directive	2362.60	880	602.60*
4) Cooler, Less Directive & Warmer, More Directive	2365.94	880	605.94*
5) Warmer, More Directive & Warmer, Less Directive	2362.60	880	602.60*
6) Warmer, More Directive & Cooler, More Directive	2363.59	880	603.59*
7) Cooler, More Directive & Warmer, Less Directive	2359.73	880	599.73

Note. The Full model allows estimates of shared environmental influences to vary across the four maternal styles. These estimates are then constrained to be equal across each pair of maternal styles, and the resulting model fit is reported. An increase in the AIC value (highlighted with an asterisk) indicates that the estimates cannot be constrained without a significant reduction in fit, and thus appear to differ significantly from one another.

Table 3

Constraining genetic influences on Conduct Problems (CP) to be equal across the different maternal styles

Model	Model Fit		
	-2lnL	df	AIC
1) Full	2359.72	879	601.72
<u>Constraint Models</u>			
2) Cooler, Less Directive & Warmer, Less Directive	2360.93	880	600.93
3) Cooler, Less Directive & Cooler, More Directive	2365.19	880	605.19*
4) Cooler, Less Directive & Warmer, More Directive	2365.79	880	605.79*
5) Warmer, More Directive & Warmer, Less Directive	2365.19	880	605.19*
6) Warmer, More Directive & Cooler, More Directive	2360.93	880	600.93
7) Cooler, More Directive & Warmer, Less Directive	2360.68	880	600.68

Note. The Full model allows heritability estimates to vary across the four maternal styles. These estimates are then constrained to be equal across each pair of maternal styles, and the resulting model fit is reported. An increase in the AIC value (highlighted with an asterisk) indicates that the estimates cannot be constrained without a significant reduction in fit, and thus appear to differ significantly from one another.