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Parenting moderates the etiology of callous-unemotional traits in middle childhood

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Background: Callous-unemotional (CU) traits are associated with chronic and escalating trajectories of antisocial behavior. Extant etiologic studies suggest that heritability estimates for CU traits vary substantially, while also pointing to an environmental association between parenting and CU traits. **Methods:** We used twin modeling to estimate additive genetic (A), shared environmental (C), and nonshared environmental (E) influences on CU traits, measured with the Inventory of Callous-Unemotional Traits (ICU) and its subscales. Our sample included 600 twin pairs (age 6–11, 230 monozygotic) from neighborhoods with above-average levels of family poverty, a risk factor for antisocial behavior. We examined the extent to which correlations between parenting, measured via parent and child report on the Parental Environment Questionnaire, and CU traits reflected genetic versus environmental factors. Then, we tested whether parenting moderated the heritability of CU traits. **Results:** In the context of lower-income neighborhoods, CU traits were moderately to highly heritable (A = 54%) with similar moderate-to-high nonshared environmental influences (E = 46%). Bivariate models revealed that associations between CU traits and *warm* parenting were genetic (rA = .22) and environmental (rE = .19) in origin, whereas associations between CU traits and *harsh* parenting were largely genetic in origin (rA = .70). The heritability of CU traits decreased with increasing parental warmth and decreasing harshness. **Conclusions:** Callous-unemotional traits are both genetic and environmental in origin during middle childhood, but genetic influences are moderated by parenting quality. Parenting may be an important target for interventions, particularly among youth with greater genetic risk.
Keywords: Harshness; warmth; Genotype × Environment (G × E) interaction; twin model.

Introduction

Antisocial behavior, including aggression and rule breaking, is a major public health concern due to its high prevalence, the emotional and financial cost to victims, and broad cost to society (Foster & Jones, 2005; Nock, Kazdin, Hiripi, & Kessler, 2006; Odgers et al., 2007). At severe levels, youth antisocial behavior is diagnosed as conduct disorder and may co-occur with elevated levels of callous-unemotional (CU) traits ('with limited prosocial emotions' specifier in DSM-5 and ICD-11; American Psychiatric Association, 2013; World Health Organization, 2020). CU traits are defined by low empathy, remorselessness, and shallow affect, and are related to more chronic and escalating antisocial behavior. CU traits identify youth with a potentially different etiology to their antisocial behavior and thus different treatment needs (Frick, Ray, Thornton, & Kahn, 2014).

Heritability estimates for CU traits range widely from 25% to 80% (for reviews, see Moore, Blair, Hettema, & Roberson-Nay, 2019; Viding & McCrory, 2012). Much of the work in this area has focused on adolescence or early childhood (for reviews, see Moore et al., 2019; though see Twin Early Development Study work, e.g. Takahashi, Pease, Pingault, & Viding, 2021; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Frick, & Plomin, 2007) and/or has not used the now-standard measure of CU traits, the Inventory of Callous-Unemotional Traits (ICU; Essau, Sasagawa, & Frick, 2006; Kimonis et al., 2008). Thus, it is unclear whether heritability estimates may differ using this more extensive and reliable measure (Frick, 2021) and during middle childhood (i.e. ages 6–11), a time when antisocial behavior is relatively more stable, and less common, before escalating in adolescence (Moffitt, 2018). Moreover, as the ICU has become more commonly used, questions have arisen about the potentially divergent etiology of the unemotional subscale (Cardinale & Marsh, 2020; Henry, Pingault, Boivin, Rijsdijk, & Viding, 2016), prompting a need to examine the etiology of each subscale.

The role of parenting in the etiology of CU traits

In parallel, a growing literature is highlighting the role that parenting plays in the development of CU traits, with a particular interest in the role of parental warmth and harshness (Waller & Hyde, 2017). These dimensions of parenting are separable, though overlapping: Harshness includes critical parental behaviors, such as negative comments and threats, whereas warmth involves positive parental involvement, such as physical affection and encouragement (Power, 2013). Observational studies have shown that harsh parenting and parenting with little warmth are correlated with higher

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CU traits (Waller, Gardner, & Hyde, 2013), potentially because harshness interferes with empathy development and low warmth undermines building positive parent–child relationships, key to empathy development (Frick & Kemp, 2021). However, these associations could reflect gene–environment correlations (rGE) – that parenting practices and child CU traits covary due to shared genes (e.g. parent genes affect both child CU traits and parenting), rather than parenting being a truly causal environmental mechanism. This concern prompted the use of adoption (Hyde et al., 2016) and twin difference studies (Waller, Hyde, Klump, & Burt, 2018) to isolate environmental effects. These studies suggested that parenting–CU trait associations were due, at least partially, to environmental mechanisms (though see Viding, Fontaine, Oliver, & Plomin, 2009). Though several of these studies support the notion that parenting effects on CU traits are not only due to gene–environment correlation, little work has directly quantified the extent to which associations between parenting and CU traits are environmental versus genetic, a key consideration in designing effective parenting-focused interventions.

Genetically informed studies have also suggested the presence of Genotype × Environment (G × E) interactions between parenting and CU traits in which parenting may moderate the etiology of CU traits by increasing or decreasing the relative importance of genetic or environmental influences on CU trait development. For example, an adoption study of preschoolers found that warm parenting buffered genetic risk for the development of CU traits (i.e. genetic risk only predicted CU traits in children receiving less warm parenting; Hyde et al., 2016). Similarly, increased parental warmth was associated with decreased heritability of CU traits in a sample of twin pairs in middle childhood (Henry et al., 2018). Although such findings indicate that warm parenting may moderate the etiology of CU traits by buffering genetic risk, these studies have not examined whether harsh parenting may also impact heritability, an important gap given the separability of warm versus harsh parenting (Elkins, McGue, & Iacono, 1997; Pasalich, Witkiewitz, McMahon, & Pinderhughes, 2016). Moreover, understanding whether warm and/or harsh parenting moderates heritability of CU traits, especially when accounting for gene–environment correlation, is a crucial step toward designing effective interventions for children at (genetic) risk for CU traits (i.e. is increasing parental warmth uniquely important for the treatment of CU traits? Pasalich et al., 2016).

The current study

We assessed the heritability of CU traits in middle childhood (ages 6–11) using the ICU and its subscales in a sample of 600 twin pairs recruited via birth records. Because residence in low-income neighborhoods is a robust risk factor for antisocial behavior, this sample was oversampled for families living in lower-income neighborhoods, a novel approach that captures greater levels of risk and subsequent antisocial behavior than most other twin studies, which typically contain fewer families facing substantial adversity. Additionally, we examined whether overlapping genetics, environment, or both accounted for associations between parenting and CU traits. We then examined whether parenting moderated the etiology of CU traits, using a method that accounts for gene–environment correlation. Based on findings from adoption and twin difference designs, we predicted that the association between parenting and CU traits would be primarily explained by nonshared environmental influences. Additionally, consistent with previous seminal G × E interaction work (Henry et al., 2018), we predicted that greater warmth would be associated with lower heritability of CU traits and potentially that greater harshness would be associated with higher heritability.

Methods

Participants

The present study included data from families assessed as part of the Twin Study of Behavioral and Emotional Development in Children (TBED-C; for details, see Burt & Klump, 2019), a project within the Michigan State University Twin Registry (MSUTR). The 1,030 families participating in the TBED-C were identified through birth records and recruited into two cohorts – a population-based cohort that represented families living within 120 miles of Michigan State University, and an at-risk cohort recruited from the same area, but only including families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (the mean for the state of Michigan at the time; e.g. Burt, Klump, Gorman-Smith, & Neiderhiser, 2016). The present study only included those 600 families recruited to, or meeting criteria for, the second 'at-risk' cohort and who had CU data (this measure was added after the population-based study was nearly complete). This strategy yielded a sample representative of families living in neighborhoods with above-average levels of poverty, a unique sampling frame for a behavior genetics study, as few twin studies have been explicitly sampled for environmental risk. The twins were 6–11 years old (mean = 97 months, SD = 18 months; 50.5% male). The breakdown of twins' parent-reported ethnicity reflected the surrounding area (81% European American, 10% African American, 6% Other, 1% Native American, 1% Latino/Latina, and <1% Asian). Zygosity was established using physical similarity questionnaires (administered to the twins and/or their parents) that show accuracies of 95% or better (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). Discrepancies were resolved through review of zygosity items or by DNA markers. Four twin pairs were excluded for missing zygosity, age, or sex information, leaving a final N of 596 twin pairs (230 MZ, 366 DZ). Parents provided informed consent and children provided assent in compliance with the policies of the Institutional Review Board of Michigan State University.

Measures

Callous-unemotional traits. Callous-unemotional (CU) traits were assessed via mother report on the Inventory of Callous-Unemotional Traits (ICU; Essau et al., 2006; Kimonis et al., 2008). The ICU consists of 24 items and includes three subscales: (e.g. 'Does not care who gets hurt to get what s/he wants'), uncaring (e.g. 'Cares about how well s/he does at school or work'; reversed), and unemotional (e.g. 'Does not show his/her emotions to others') traits. The ICU has shown acceptable internal consistency for the total score and subscales across multiple studies (Cardinale & Marsh, 2020), and higher scores predict differential developmental trajectories for youth with antisocial behavior (Frick et al., 2014). Recent work in a nationally representative sample, including families across a wide range of incomes, demonstrated measurement invariance for the ICU across parent sex (Bansal, Babinski, Waxmonsky, & Waschbusch, 2020). Consistent with prior studies (Waller et al., 2015), we utilized a total 22-item sum score, excluding items 10 and 23 (α = .78). We also examined the callousness (α = .67; 11 items), uncaring (α = .82; 8 items), and unemotional (α = .83; 5 items) subscales.

Parenting. The Parental Environment Questionnaire (PEQ; Elkins et al., 1997) was administered to assess warmth (via the involvement scale) and harshness (via the conflict scale) in each parent–child dyad. The involvement subscale (12 items) assesses communication, closeness, and support in the parent–child relationship (e.g. 'I praise my child when he/she does something well'). The conflict subscale (12 items) assesses disagreement, tension, and anger in the parent–child relationship (e.g. 'I often criticize my child'). Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with each parent. Consistent with prior work (e.g. Burt, Krueger, McGue, & Iacono, 2003), we created a composite of all reporters for each subscale to assess the overall parenting environment for each child, averaging twin report on mother and father, and averaging that score with mother and father report. When any of these reporters were missing (N = 218 individuals were missing at least 1 informant report, primarily father), we calculated the composite from the available reports. Using this method, most twin pairs with ICU data had composite parenting data (N = 585 involvement, 586 conflict).

To evaluate the robustness of our GxE results, we repeated analyses using observer ratings of parenting (PARCHISY; Deater-Deckard, Pylas, & Petrill, 1997; details on the interaction task and coding system are presented in Appendix S1). The current study focused on two measures of the parent–child relationship: *dyadic reciprocity* measured warmth, and captured shared positive affect during the task, whereas *dyadic conflict* measured harshness, and captured shared negative affect during the task.

Analyses

Twin studies leverage the difference in the proportion of genes shared between monozygotic (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 nonshared environmental (i.e. factors that make twins different from each other, including measurement error; E) contributions to a given phenotype (see Plomin, DeFries, Knopik, & Neiderhiser, 2012). Before performing model-fitting analyses, we calculated intraclass twin correlations (ICCs) using the double-entry method, which removes variance due to twin ordering within each pair (Knopik, Neiderhiser, DeFries, & Plomin, 2017, p. 355).

We evaluated the etiology of CU traits using a univariate ACE model. We also tested an alternate, AE model, in which C is set to 0. We then fit bivariate ACE models to decompose the covariance between each parenting construct and CU traits. The bivariate ACE model parses the phenotypic covariance into that which is due to genetic, shared environmental, and nonshared environmental factors; these covariances can then be standardized on their respective variances to produce genetic and environmental correlations. As child age and sex correlate with CU traits (Essau et al., 2006), we regressed out age and sex effects from CU traits (i.e. used the residuals from a regression with age and sex predicting CU traits); additionally, it is generally recommended that unstandardized or absolute ACE estimates be presented (Purcell, 2002); thus, we used the standardized residual from this regression as our CU trait score (mean = 0, SD = 1) to facilitate interpretation of the unstandardized values.

We then evaluated whether parenting moderated the etiology of CU traits using the 'extended univariate GxE model' (Purcell, 2002; van der Sluis, Posthuma, & Dolan, 2012; Figure 1A), running separate analyses for parent–child involvement and conflict. In this model, the variance decomposition of CU traits was modeled as a function of each parenting variable. To eliminate gene–environment correlational confounds in this model, the moderator values of both twins were entered in a means model of each twin's CU traits (i.e. the overlap between each twin's CU traits and parenting score is residualized out of the model, thus eliminating potential gene–environment correlations between the outcome and the moderator). Moderation was then modeled on the residual CU trait variance (i.e. that which does not overlap with parenting). The first and least restrictive of these models allows for independent linear moderation of all three variance components (A, C, and E). We then fit a series of more restrictive moderator models, constraining the moderators to be zero and evaluating the reduction in model fit. As recommended by van der Sluis et al. (2012), we also ran a bivariate GxE model to confirm that the etiologic moderation we observed was indeed present on the variance that is unique to CU traits (Figure S1b). This more computationally intensive model calculates variance component estimates for both the variable of interest and the moderator variable, as well as the overlap between the two. We floored each parenting composite at 0 and then divided each value by the maximum value within our sample, providing a continuous moderator variable with minimum 0 and maximum 1.

We used Mplus version 8.5 (Muthén & Muthén, 2020) to fit all models using full-information maximum-likelihood techniques. We evaluated model fit for the G × E models using three indices that balance overall fit with model parsimony: the Akaike's information criterion (AIC; Akaike, 1987), the Bayesian information criterion (BIC; Raftery, 1995), and the sample size-adjusted BIC (ssBIC; Sclove, 1987); for these indices, more negative values indicate better fit. In line with previous G × E work (e.g. Burt & Klump, 2014; Hicks, South, Dirago, Iacono, & McGue, 2009), we considered the best-fitting model to be the one with lower or more negative values for the majority of these three fit indices. When available, we also considered root mean square of approximation (RMSEA; Yuan & Bentler, 2000) for which a lower value indicates better fit.

To explore the robustness of our results, we also ran three sets of supplemental analyses, evaluating whether G × E results persisted (1) across the various informant reports of parenting (i.e. parent vs. child report), (2) across the assessment method for parenting (i.e. parent/child reports vs. observational), and (3) across both parenting dimensions (warmth, harshness), when controlling for overlap with the other.

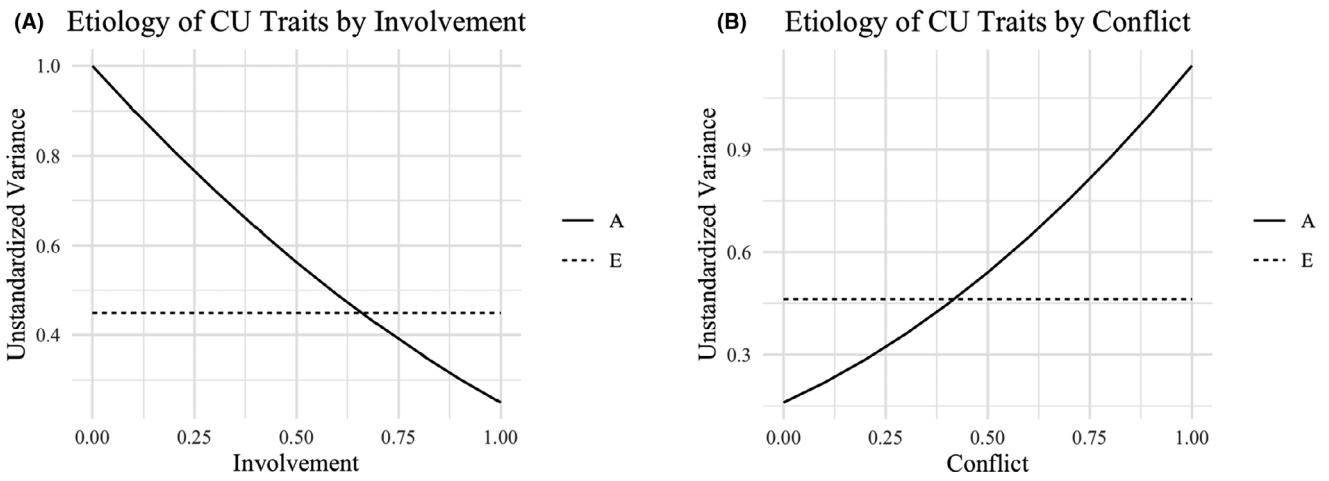

Figure 1 Parent–child involvement and conflict moderate the heritability of callous-unemotional (CU) traits. *Note*. This figure depicts unstandardized additive genetic (A) and nonshared environmental (E) contributions to CU traits as predicted by the best-fitting Genotype x Environment (G × E) interaction models at varying levels of the moderators: (A) parent–child involvement (N = 585 pairs, 227 monozygotic) and (B) and parent–child conflict (N = 586 pairs, 228 monozygotic). Heritability of CU traits decreases with increasing reported involvement ($p < .01$) and with decreasing reported conflict ($p < .001$)

Results

Descriptive statistics are reported in Tables S1–S3. Consistent with the enriched sampling design, ICU scores showed substantial variability for a community sample, with 12.4% of twins meeting a clinical cutoff score of >30 for parent report (Docherty, Boxer, Huesmann, O'Brien, & Bushman, 2017). Parent–child involvement and conflict were moderately negatively correlated ($r = -.36$) and correlated with total CU traits ($r = -.13$ and .31, respectively; Table S1). ICCs for CU traits were .47 for MZ twins and .20 for DZ twins (N = 596 pairs, 230 MZ), while ICCs for parent–child involvement were rMZ = .62 and rDZ = .55, and for parent-child conflict were rMZ = .69 and rDZ = .60 (see Table S2 for cross-trait cross-twin correlations).

Primary etiological models

We found no evidence for shared environmental (C) influences on CU traits (Table S4). The best-fitting univariate model was the AE model that estimated genetic influences (A) at 0.54 (95% CI [0.42, 0.64]) and nonshared environmental influences (E) at 0.46 (95% CI [0.36, 0.58]; Table S4), indicative of moderate-to-high genetic and moderate-to-high nonshared environmental influences (the confidence intervals of these estimates were highly overlapping). Similarly, for callous and uncaring subscales, there were moderate-to-high genetic and nonshared environmental influences. However, for the unemotional subscale genetic influences were small (.20) and nonshared environmental influences were larger (.80; Table S4).

We then examined the etiology of the associations between each parenting construct and CU traits. The best-fitting bivariate models estimated ACE components for parenting and AE components for CU traits. For parental involvement, the association with CU traits was due to both nonshared environment (52%) and overlapping genetic influences (48%). The association between conflict and CU traits was largely genetic in nature (92% shared A; see Table S4).

The best-fitting extended bivariate G × E model for both involvement and conflict was the genetic-moderation-only AE model (Table 1). The involvement model indicated decreasing heritability of CU traits with increasing parental involvement (A1 = −.50, $p < .01$; Figure 1A). The conflict model indicated increasing heritability of CU traits with increasing conflict (A1 = .67, $p < .001$; Figure 1B). To confirm that the etiologic moderation we observed was present for the variance unique to CU traits (rather than paths that overlapped across parenting and CU traits; van der Sluis et al., 2012), we also ran the bivariate GxE model (Purcell, 2002). Results indicated that the etiologic moderation in question was indeed specific to the unique paths (i.e. A1 = −.51 and .67, $p < .01$ and $p < .001$ for involvement and conflict respectively; Table S5).

Sensitivity analyses

To evaluate the robustness of the GxE results, we reran the best-fitting moderation models separately by reporter (Table S6). Across informants, the heritability of CU traits consistently decreased with increasing involvement and increased with increasing parent–child conflict. The overall pattern of results was strikingly consistent across informants; however, the genetic moderators were only statistically significant for twin reports of involvement and for mother and father reports of conflict.

We reran the best-fitting models using observed measures of parenting on the subset of families with

TABLE 1 Parenting Genotype × Environment interaction models

Model	A	A1	C	C1	E	E1	AIC	BIC	ssBIC
Involvement									
ACE, ACE moderation	.86***	−.28	.60	−.99*	.75***	−0.15	3,169.98	3,222.44	3,184.35
AE, AE moderation	.94***	−.39			.74***	−0.11	3,167.06	3,210.77	3,179.03
AE, A moderation	**1.00*** **	**−.50** **			**.67*** **		**3,165.44**	**3,204.79**	**3,176.21**
Conflict									
ACE, ACE moderation	.48***	.49	.00	.00	.61***	0.23	3,108.68	3,161.16	3,123.07
AE, AE moderation	.48***	.46			.61***	0.22	3,104.68	3,148.42	3,116.67
AE, A moderation	**.40*** **	**.67*** **			**.68*** **		**3,104.18**	**3,143.54**	**3,114.97**

This table depicts estimates and model fit statistics for the Genotype × Environment (G × E) interaction models. $N = 585$ pairs (227 monozygotic) for involvement, and 586 (228 monozygotic) for conflict. The first model listed for each moderator is the full ACE model with linear moderation (A1, C1, E1) allowed on A, C, and E terms. The best-fitting models are indicated in bold.
*$p < .05$, **$p < .01$, and ***$p < .001$.

available observational data ($N = 511$ pairs) using median splits of dyadic reciprocity and conflict due to skew in the conflict variable (skew = 6.8). The skew in the conflict variable was largely due to zero-inflation: There was no conflict observed during the task for 84% of twin–parent dyads. This was not the case for dyadic reciprocity (skew = −0.38). Consistent with parent/child reports of parenting, we found evidence of decreasing heritability of CU traits with observations of increasing parental warmth/reciprocity (A1 = −.15, $p = .02$; Table S6). However, there was no evidence of moderation by conflict (A1 = −.04, $p = .63$; Table S6), perhaps unsurprising given the limited conflict observed in the laboratory.

As a final clarification, we reran our best-fitting models using involvement and conflict variables that residualized out their overlapping variance (i.e. this modeled the variance unique to involvement or conflict). The conflict moderation on CU trait heritability remained significant (A1 = .62, $p < .01$; Table S7). However, removing the covariance with conflict substantially weakened the moderation by involvement (A1 = −.35, $p = .07$; Table S7).

Discussion

In a community sample of families living in neighborhoods with above-average levels of poverty, in univariate ACE models using the ICU we found evidence that CU traits were moderately to highly heritable (54%) in middle childhood, with similar moderate-to-high nonshared environmental contributions (46%), and little evidence for shared environmental influences. Associations between parenting warmth and CU traits were due to both genetic and environmental influences, whereas associations between parental harshness and CU traits were mostly due to shared genes. G × E analyses indicated that the heritability of CU traits was qualified by an interaction with parenting such that parenting higher in warmth and lower in harshness was associated with lower heritability, even when accounting for gene–environment correlation. These G × E results were consistent in strength and direction across informants and even when using observational measures of parental warmth. Such findings highlight the important heritable and environmental influences on the etiology of CU traits, the environmental role of parental warmth in the etiology of CU traits, and the role parenting can play in buffering or activating genetic risk for CU traits. These results also extend existing knowledge of the etiology of CU traits to school-aged children living in lower-income neighborhoods.

Our initial etiologic results replicate and extend those from normative population-based samples: Our heritability estimates for CU traits (54%) are consistent with estimates in young children and adolescents (e.g. range 25%–80%; Moore et al., 2019), as well as recent and early work in middle childhood (Takahashi et al., 2021; Viding et al., 2005). Moreover, our results emphasize that estimates of moderate-to-high heritability and nonshared environmental influences are present in middle childhood, when using the ICU to measure CU traits, and within families with greater exposure to disadvantaged neighborhoods, an important step given that heritability estimates are specific to time, developmental stage, and place (i.e. to each sample).

Additionally, our findings of consistent heritability estimates for the callous and uncaring subscales, but a different pattern of mostly nonshared environmental influences (80%) for the unemotional subscale, are consistent with similarly divergent heritability estimates of the ICU subscales found previously (Henry et al., 2016). The lower heritability and high estimate of E (which contains both nonshared environment and nonsystematic error), along with a meta-analysis of the ICU subscales suggesting low internal consistency and low external validity of the unemotional scale (Cardinale & Marsh, 2020), add to questions regarding the strength of this scale as it relates to interpersonal callousness, uncaring, and antisocial behavior (Cardinale & Marsh, 2020; though see Ray & Frick, 2020). At the least, the present data suggest the unemotional scale has a strikingly different etiology than the rest of the ICU.

We found that both parenting variables, but especially conflict, had genetic overlap with CU traits (92% of the shared variance with conflict, 48% with involvement). Thus, harsh parenting and CU traits may co-occur primarily because parents and children share genes (potentially consistent with Viding et al., 2009). This evidence of gene–environment correlation underscores the importance of genetically informed research when studying family influences on the development of CU traits. At the same time, for parental warmth there was also a substantial nonshared environmental contribution to CU traits. This finding is consistent with twin difference and adoption studies that identified environmental effects of warm parenting (Hyde et al., 2016; Waller et al., 2018). Moreover, the current results, which used another methodological approach (bivariate twin modeling) beyond twin difference and adoption designs, emphasizes that, though there is substantial gene–environment correlation between parental warmth and CU traits, parental warmth has a substantial *environmental* influence on the development of CU traits.

Beyond direct effects of parenting, we were particularly interested in whether parenting behaviors might buffer or activate risk for children with genetic vulnerability. Replicating and extending seminal work by Henry et al. (2018), we found that the heritability of CU traits decreased with increasing parental involvement and increased with increasing conflict. This pattern was largely consistent across informants and method. This finding adds to a growing body of work showing that parental warmth buffers genetic vulnerability to CU traits (e.g. Hyde et al., 2016). These results also provide new evidence that genetic influences on CU traits may emerge more strongly in a context of risk (i.e. harsh parenting). Notably, the models used here to test for moderation accounted for gene–environment correlation and suggested that the unique variance in harsh (versus warm) parenting was most important for this G × E. Taken together, our findings indicate that even when accounting for the substantial gene–environment correlation between parenting and CU traits, parenting still has an important role in buffering (warmth) or activating (harshness) genetic risk for CU traits. Thus, our findings suggest that parenting interventions for CU traits should focus on both increasing parental warmth and decreasing harshness. Warmth has a clear direct impact on CU traits and may also buffer genetic risk, highlighting its importance in treatment. In parallel, though parental harshness may not have a direct environmental impact on CU traits, harshness does appear to unmask genetic risk, making it an important treatment target for youth at higher genetic risk who may also have harsher parents (Dotterer, Burt, Klump, & Hyde, 2021).

It is important to note that the primary measure of the parent–child relationship used in this study was designed to assess specific *dyadic* aspects of warmth and harshness (McGue, Elkins, Walden, & Iacono, 2005) and was not comprehensive in measuring all developmentally appropriate positive or negative parenting behaviors. For example, the involvement scale used here to index warmth includes items tapping communication, closeness, and support, but does not cover all possible elements of warm parenting such as positive physical touch or positive reinforcement (e.g. Hyde et al., 2016). Thus, we may be underestimating the impact of parental warmth (versus harshness) in buffering genetic risk for CU traits. Additionally, all measures of parenting in this study contained dyadic elements. Youth CU traits may influence these dyadic processes in ways that make the study not just about parenting but about the parent–child relationship (which is likely influenced by CU traits; e.g. Pasalich, Dadds, Hawes, & Brennan, 2012). Thus, it may be safer to interpret our findings as being about warm and conflictive parent–child relationships and their impact on CU trait etiology, rather than about parenting as a unidirectional construct (see Trentacosta et al., 2019, for evidence that youth with CU traits also evoke harsher parenting).

The study had several strengths, including the examination of a unique twin sample living in lower-income neighborhoods and the use of multiple informants and observational methods. However, there are several limitations worth noting. First, this sample is only moderately sized by current twin study standards. However, power analyses (Burt, Clark, Pearson, Klump, & Neiderhiser, 2020; Purcell, 2002) suggest that it may be adequate, particularly since we focused on a reduced AE model. Second, though our enrichment strategy for families from lower-income neighborhoods was successful, and we used a strong sampling frame from birth records, we did not use sample weights. Thus, our unique sampling frame may impact heritability estimates if neighborhood income moderates the heritability of CU traits — an important next step in this research. Moreover, given the sampling frame, these results should generalize to families living in lower-income neighborhoods in the Midwestern United States; however, it is less clear whether they would generalize beyond that (e.g. outside of the Midwest or to families living in more advantaged neighborhoods). Third, age and sex information were regressed out of the CU traits variable to eliminate mean differences; however, given associations between age, sex, and CU traits, a next step would be to investigate whether parenting effects on CU traits differ across sex or age. Finally, the observed conflict variable had little variance, which may have undermined efforts to replicate these specific G × E findings.

Conclusions and clinical implications

Overall, the results suggest that variability in parenting is associated with CU traits at least in part via nonheritable, environmental pathways and that

parenting appears to alter the importance of genetic influences on CU traits. Warm parent–child relationships may buffer genetic risk, whereas harsh parent–child relationships may 'unmask' latent genetic risk. At the same time, substantial heritable influences on CU traits, combined with genetic overlap between parenting and CU traits, may mean that genetic risk for CU traits manifests in colder, more conflictive parent–child relationships. Moreover, this work highlights that youth with CU traits may have parents with higher CU traits (Dotterer et al., 2021), which may undermine motivation to seek treatment and engagement of families that do seek treatment (Viding & Pingault, 2016). Thus, parent-focused intervention efforts should take into account both child and parent characteristics when targeting parental warmth and harshness. Indeed, though previous studies using randomized clinical trial designs have found that parenting-focused interventions for youth with CU traits are effective (e.g. Hyde et al., 2013; Waller et al., 2013; White, Frick, Lawing, & Bauer, 2013), children with CU traits start higher on antisocial behavior than other children and may thus need 'extra effective' treatments to bring their antisocial behavior into normative ranges (Bansal et al., 2019). Therefore, modified versions of parenting interventions explicitly targeted to youth with CU traits are needed (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012; Kimonis et al., 2019).

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Supplemental methods.

Table S1. Means, standard deviations, and correlations with confidence intervals.
Table S2. Cross-trait, cross-twin correlations with confidence intervals.
Table S3. Internal consistency and zero-order correlations for parental conflict and involvement across informants.
Table S4. Model estimates and model fit statistics for univariate and bivariate models.
Table S5. Bivariate G × E models confirm that the GxE effects are present on variance unique to CU traits.
Table S6. G × E interaction models examining effects across different informants and methods.
Table S7. The unique G × E interaction effects of involvement versus conflict on CU traits.
Figure S1. Extended univariate and bivariate gene-environment interaction (G × E) models.

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Key points

- We examined the heritability of CU traits in a sample of children living in neighborhoods with above-average poverty.
- CU traits showed moderate-to-large heritability estimates (54%) with similar moderate-to-large nonshared environmental influences (46%) in middle childhood (ages 6–11).
- Both warm and harsh parenting had substantial genetic overlap with CU traits (48% and 92% of shared variance, respectively). Warm parenting had substantial nonshared environmental overlap with CU traits (52%).
- Accounting for gene–environment correlation, parental warmth and harshness each moderated the etiology of CU traits: CU traits were less heritable for children who received parenting that was warmer and less harsh.
- Parenting is likely to serve as an important target for effective interventions for CU traits, even for youth with genetic risk.

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