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Longitudinal Links Between Callous-Unemotional Behaviors and Parenting in Early Childhood: A Genetically-Informed Design

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Abstract

Objective—Most research on callous-unemotional behaviors (CU) and parenting does not focus on directions of effect, and work that does so has not been genetically-informed. The present study is the first to examine potential reciprocal effects between parenting and CU in a community sample of early childhood. Use of a twin sample also allows us to distinguish child-based genetic effects from environmentally-driven effects, which is necessary before translating this research to interventions.

Method—The present study used biometric cross-lagged models to investigate the relation between CU and parenting in twins at 2- and 3-years of age (MZ=145, DZ=169 twin pairs). CU was assessed using the parent-reported Child Behavior Checklist 1.5–5. Scores were residualized to control for conduct problems assessed on the Revised Rutter Parent Scale for Preschool Children. Parents' reports of negative and positive parenting were obtained using parent ratings of discipline and parent feelings from the Parent Feelings Questionnaire.

Results—CU and negative parenting were significantly correlated at both ages. Cross-lagged analyses revealed a unidirectional effect with CU at age 2 predicting negative parenting at age 3. These child-driven effects were primarily genetically mediated, though there were modest nonshared environmental contributions. CU and positive parenting were not consistently correlated, and further biometric analyses were not performed.

Conclusions—Children's genetically-influenced CU behaviors can have an impact on the parenting that they experience. Child-driven CU effects, though less examined in the literature, are important and should receive more attention in future work.

Lay Summary

In a community sample of twins at ages 2 and 3, children who were rated by their parent as displaying higher levels of callous-unemotional behaviors were reported to experience more negative parenting. This effect is child driven where genetically-influenced callous-unemotional behaviors of the child at age 2 predicted negative parenting at age 3, but not the other way around. This highlights the importance of children's genetically-influenced behaviors on the parenting they receive, and suggests that parenting interventions in early childhood should also target the child's behavior.

Conflict of Interest

The authors declare that they have no conflict of interest.

Keywords

Early childhood; callous-unemotional behaviors; parenting; genetic; longitudinal

Research over the last several decades has focused on callous-unemotional (CU) traits as an important diagnostic component of conduct problems. CU traits, characterized by low guilt, empathy, and affect [1], denote a more severe and stable trajectory of behavior problems [2,3], and are associated with socio-emotional maladjustment even in the absence of psychopathology [4]. These behaviors in middle childhood and beyond are viewed as "traits" as they are moderately to highly stable and enduring across time and situations [5]. A similar constellation of behaviors has been identified in early childhood (i.e., lack of guilt, empathy, and affect). Like CU traits in older children, these early emerging behaviors are related to other behavior problems such as ADHD and ODD [6,7] and demonstrate moderate stability across early childhood [8,9]. However, because at such an early age, this set of behaviors has not yet been demonstrated to be a long-lasting component of the child's personality or behavioral repertoire, they are viewed more cautiously as CU *behaviors.* Nonetheless, these early CU behaviors are developmentally significant and research has attempted to identify their developmental origins.

Whether conceptualized as a trait or early emerging behaviors, CU is genetically influenced, with heritability estimates ranging from .40-.70 in both community and high-risk samples [10], even as early as 2 years of age [8]. Yet, a growing number of studies have found that parenting is associated with CU. Specifically, both higher levels of harsh/negative parenting and lower levels of warm/positive parenting have been related to CU in childhood and adolescence [11]. This suggests that parenting may be a malleable protective factor that can be targeted in prevention efforts. However, prior to the development of parent-based interventions, a better understanding of the direction of effects between parenting and CU behavior is needed. The association between CU behaviors and parenting could arise via "parent-driven" effects that demonstrate an effect of parenting on child behavior, and/or "child-driven effects" whereby the child's behavior elicits specific parenting behaviors. For example, does parenting predict later CU behavior, such that harsher parenting leads to greater CU (i.e., parent-driven effects)? Or does CU lead to parental harshness and lower warmth as parents cope with their children's challenging behaviors (i.e., child-driven effects)? The current study explores both possibilities through testing cross-lagged models of CU and parenting, which incorporate bidirectional influences. A genetically-informed twin design allowed us to further distinguish between child-and parent-driven effects [12,13].

Parenting and CU behaviors

It has been proposed that negative parenting affects CU because it makes the internalization of parental messages about prosociality more difficult, and because it models aggression and other negative behaviors [14]. Positive parenting is thought to buffer against CU because it can promote positive socialization such as empathy and prosociality [15]. Consistent with this view, research on CU and parenting has largely focused on the extent to which negative and positive parenting affects children's CU. Longitudinal work finds support for these parent-

driven effects [16–19] but there are also inconsistencies or null associations [11,16]. Further clarification of the relation between parenting and CU is needed, particularly in very young children when behaviors related to CU are coming online and parenting may be especially relevant.

One missing piece from this puzzle is the impact children have on the parenting they receive (i.e., child-driven effects), yet few studies consider this possibility in their theoretical and statistical approaches. Children's CU could elicit more negative and less positive parenting. This possibility does not, however, negate parent-driven effects. Rather, both parent- and child-driven effects may operate at the same time, as described in models of coercive family processes [20]. Longitudinal studies that examine the reciprocal effects of child behavior and parenting over time are needed to test the relevance of this model for the development of CU.

To date, only a few studies have used cross-lagged models to examine the reciprocal effects between CU and positive parenting [12,13,21] and negative parenting [13,21]. These studies indicate that the timing and consistency of these effects may differ for positive and negative parenting. For example, reciprocal effects have been found between CU and observed parental warmth across 2 to 3 years of age [12]; and between CU and overall parent-reported positive parenting (e.g., parental involvement and positive reinforcement) in a 3-wave study of children ages 9 to 12, but only across the last two waves [13]. Another study, however, found only child-driven effects from age 3 to 6 years [21]. For negative parenting (e.g., inconsistent discipline, corporal punishment, and/or poor monitoring), only child-driven effects were found in early childhood [21]; and neither child- nor parent-driven effects were demonstrated in middle childhood [13]. Differences amongst these studies may reflect differences in how parenting was assessed, the length of time between time points or whether the sample included clinical vs. at-risk populations. Collectively, however, findings suggest that early childhood may be a crucial developmental period to capture both parent- and child-driven effects in the relation between CU and parenting.

Although a developmental picture of the reciprocal effects of parenting and child CU is emerging, previous studies are limited by their focus on phenotypic associations. For example, the finding that parenting predicts CU over time is often used to support a causal effect of parents on children. However, when samples include children and their biological parents, associations between parenting and children's CU could be a result of shared genes between parents and their children that both predispose children to higher CU and parents to more negative or less positive parenting (i.e., passive gene-environment correlation [22]). Alternatively, associations between parenting and CU could represent a "true" parenting effect, wherein the environment that parents provide influences their children's behaviors.

Genetically-informed designs, such as twin studies, can disentangle whether these links are explained by children's genetic makeup or are environmentally mediated. A previous twin study on a related construct, antisocial behavior problems, in early-to-middle childhood demonstrated reciprocal effects between antisocial behaviors and negative parenting that was both genetically and environmentally mediated [23]. The current study extends this work by focusing on CU and both negative and positive parenting during toddlerhood.

Current Study

This study is the first to investigate the reciprocal effects of parenting and CU in a community sample. Moreover, we examine reciprocal relations between CU and both negative and positive parenting using a longitudinal genetically-informed approach in toddlerhood—when CU is first emerging. Given that CU and parenting are both consistently related to conduct problems[24,25], we controlled for conduct problems in CU in order to examine unique effects between parenting and CU, rather than behavior problems more broadly. Biometric cross-lagged models examined the bidirectional effects from ages 2 to 3 years, as well as the underlying genetic and environmental sources of relations between CU and parenting within and across age. These analyses allow us to distinguish child-based genetic effects from environmentally driven effects, which is necessary before translating this research to interventions. We hypothesized that cross-age effects would be bidirectional, and be both genetically and environmentally mediated.

Methods

Participants

The Boston University Twin Project twin sample was recruited from the Massachusetts Registry of Vital Records. All procedures were approved by the university's Institutional Review Board, and primary caregivers provided informed consent. Three hundred and fourteen same-sex twin pairs (145 MZ and 169 DZ) participated at age 2 and of these, 304 pairs (141 MZ and 163 DZ) were reassessed at age 3. Ethnicity was generally representative of Massachusetts (85.4% Caucasian, 3.2% Black, 2% Asian, 7.3% Mixed, 2.2% Other). Socioeconomic status according to the Hollingshead Four Factor Index [26] ranged from low to upper middle class (range=20.5–66; M=50.9, SD=14.1). Zygosity was determined using DNA obtained through cheek swab samples. In cases where DNA was not available (n=3), zygosity was determined using parent responses on physical similarity questionnaires, which have been shown to be more than 95% accurate when compared to DNA markers [27].

Measures

CU.—Following Willoughby et al. [6], a 5-item screening measure from the *Child Behavior Checklist 1½–5* [28] was used to assess CU for each twin. Items include "doesn't seem to feel guilty after misbehaving", "punishment doesn't change behavior", "seems unresponsive to affection", "shows little affection toward people" and "shows too little fear of getting hurt", and do not overlap with ADHD and ODD scales. The primary caregiver rated items 3-point scale (0=not true, 1=sometimes true, and 2=always true). Possible scores range from 0 – 10, with our non-clinical sample ranging from 0 – 7. Means (see Table S1, available online) were consistent with other early childhood community samples [6,29]. This CBCL measure of CU has demonstrated good reliability and validity in 2-year-olds [8,30] and 3-year-olds [6,9]. For example, predictive validity is indicated by the finding that scores on the CBCL CU scale at age 2 predicted both antisocial and externalizing behaviors in middle childhood [30]. CU has early risk factors (e.g., fearless temperament) and socio-emotional and behavioral correlates in early childhood (e.g., lower moral regulation) that are distinct from ADHD and ODD [6,29]. Indeed, when comparing 1-, 2- and 3-factor models for the CU, ADHD, and ODD CBCL

items, a 3-factor model fits the data best [6,8,30]. Furthermore, though the CBCL CU, ADHD and ODD scales all load onto a common externalizing factor, each behavioral construct has genetic and environmental factors that are behavior-specific, providing biometric support for parents' ability to differentiate between the three behavioral domains in very young children [7]. Lastly, there is phenotypic factor invariance of the CBCL CU scale across ages 2 and 3 years [8], demonstrating that item loadings onto a latent CU factor could be equated across age and that CU is not structurally different at ages 2 and 3. In the current sample, reliability as indexed by Cronbach's alpha was .55 at age 2 and .61 at age 3. These estimates are consistent with previous research using the same measure in young children which report alphas ranging from .55 to .65 [6,9,29].

Conduct Problems.—The 8-item Conduct Problems from the *Revised Rutter Parent Scale for Preschool Children* [31] was used to control for conduct problems given their strong association with CU in childhood [24]. Parents rated their child on specific behaviors using a 3-point scale (0=not true, 1=sometimes true, and 2=certainly true). Cronbach's alphas for this scale were .91 and .98 at ages 2 and 3, respectively.

Parenting.—Parents rated their negative and positive feelings toward their children on the *Parent Feelings Questionnaire* [32]. This measure includes 24 items indexing parental negativity or positivity and 10 items assessing parental emotions toward each child. Scores were composited according to Deater-Deckard [33].

A 14-item parent-report measure based on a modification of widely-used semi-structured interview [32,33] and formatted for use with twins [34] provided information about discipline strategies used for each twin. Factor analysis indicated that that yelling, arguing, spanking, and asking someone else to deal with the child loaded on harsh discipline at both ages. At age 2, harsh discipline also included ignoring the child. Reasoning and praising loaded on positive discipline at both ages; however, at age 2 being firm and calm as well as joking also loaded on positive discipline, whereas at age 3 hugging/being affectionate loaded on positive discipline. All factor loadings were .4 or higher. The slightly different negative and positive discipline factors across age reflect developmental changes in the child, yet because subsequent analyses look at rank order differences results are not affected by different items at each age.

An overall measure of negative parenting at each age was formed by averaging standardized scores for negative feelings and emotions, and negative discipline at each age (age 2 α =.90; age 3 α =.93). Positive parenting was similarly formed by averaging standardized scores for parent positive feelings and emotions, and positive discipline (age 2 α =.74; age 3 α =.79).

Data Transformation

Because twin covariances can be inflated by variance due to sex [35], all CU and parenting scores were residualized for sex effects. To control for the possible influence of conduct problems on CU, CU scores were also residualized for conduct problems. Finally, CU at age 3 and negative and positive parenting at both ages were rank transformed to provide a more normal distribution.

Twin Design

The twin design decomposes phenotypic variance of a variable into additive genetic (A), shared (C) and nonshared (E) environmental variance components. *Heritability* is the proportion of phenotypic variance that can be attributed to genetic factors and describes the genetic effect size. Genetic influences are implied when cotwin similarity covaries with their degree of genetic relatedness. *Shared environmental variance* is familial resemblance that is not explained by genetic variance. Thus, it comprises environmental influences that are shared by family members and make them similar to each other (e.g., family demographics, one's rearing neighborhood, shared friends). *Nonshared environmental variance* is a residual variance that includes environmental influences unique to each individual. These unique environmental influences make members of the same family different from one another. Possible sources of nonshared environmental variance include differential parental treatment; relationships with friends, peers and teachers; and nonsystematic factors such as accidents, illness and measurement error [36]. When parenting is modeled as a child phenotype, genetic influences on parenting indicate the extent to which parenting is influenced by heritable aspects of the child.

Statistical Analysis Plan

Correlations between CU and negative and positive parenting were examined. When both significant within- and across-age correlations were found biometric cross-lagged models [37] were used to examine genetic and environmental sources of: a) variance at each age; b) covariance between CU and parenting within each age; and c) on the transmission of effects between and within variables across age (i.e., stability and cross-lagged effects, respectively). The model decomposes the variances and covariances between variables within and across age into their genetic and environmental components (i.e., A, C, E). A genetic correlation (r_g) between variables indicates the extent to which common genetic influences operate across variables, independent of the heritability of each phenotype. A similar logic applies to shared (r_c) and nonshared (r_e) environmental correlations. Models were fit to raw data using a maximum likelihood pedigree approach implemented in Mx structural equation modeling software [38]. This approach allows the inclusion of participants with incomplete data.

Results

Correlations

As seen in Table 1, CU and positive parenting were moderately stable, and negative parenting was highly stable. CU was modestly to moderately correlated with negative parenting, but demonstrated little or no association with positive parenting. Given this, biometric cross-lagged analyses were only applied to CU and negative parenting. Twin intraclass and cross correlations are presented in supplementary materials (see Table S2, available online).

Model-fitting

Model-fit statistics are presented in Table S.3 in supplementary materials. Models were reduced by dropping all nonsignificant paths in the full model. It was possible to drop C on CU at both ages, consequently, there was no shared environmental covariance between CU and negative parenting. The residual C on negative parenting at age 3 could also be dropped, as well as r_e at age 2, and all residual correlations *(*r_g, r_c, and r_e*)* at age 3, and the cross-lagged path from negative parenting to CU. The reduced model did not fit the data significantly worse, and is presented for parsimony of results.

Genetic and Environmental Influences on Variances and Covariances at Age 2

At age 2, genetic factors explained 62% (CI: 52–69%) of the variance in CU and 39% (CI: 25–54%) for negative parenting. Shared environmental influences explained 49% (CI: 34–62%) of the variance in negative parenting and no significant variation in CU. Nonshared environmental influences accounted for the remaining 36% (31–48%) of the variance in CU and 12% (CI: 9–16%) in negative parenting. There was moderate genetic covariance (r_g=.33) between CU and negative parenting, and it was these shared genetic factors that fully explained the phenotypic relation between CU and negative parenting at age 2.

Genetic and Environmental Contributions to Total Variances at Age 3

At age 3, genetic factors accounted for approximately 50% (CI: 38–60%) of the total variance in CU and 57% (CI: 49–65%) in negative parenting at age 3. Although these estimates of heritability vary somewhat from those at age 2, confidence intervals overlap across age and consequently, provide little evidence of differential heritability. The remaining variance in CU was explained by the nonshared environment (50%; CI: 40–62%), which also did not differ in magnitude from age 2. For negative parenting, the shared environment explained 20% (CI: 13–28%) of the variance, a significant decrease from age 2 as indicated by non-overlapping confidence intervals for the age 2 and age 3 estimates. In contrast, the nonshared environmental variance in negative parenting increased at age 3 (23%; CI: 19–28%).

Transmission Across Age

Tables 2 and 3 decompose these total genetic, shared environmental and nonshared environmental variances at age 3 into effects that are transmitted from age 2 (stability, cross-lagged, and common effects) and novel (residual) effects at age 3. The total variance is the sum of the component variances. Because effects are standardized, total variance for each phenotype at age 3 sum to 1.

There was substantial stability in negative parenting across age and moderate stability in CU. Submodels that dropped stability paths resulted in a significant decrement in fit (see Table S3, available online). For both variables, these stability effects account for most of the variance that is transmitted across age. For CU 12% of the variance at age 3 was a result of stability variance at age 2, whereas 40% of the variance in age 3 negative parenting was due to stability effects. As seen in Table 2, the majority of stability effects for CU were genetically influenced. Stability effects for negative parenting were significantly influenced

by all three etiologic sources, though genes and shared environments made the largest contributions.

Contrary to our hypothesis, the cross-age association between CU and negative parenting was unidirectional from age 2 CU to age 3 parenting. It was possible to drop the cross-lagged path from negative parenting at age 2 to CU at age 3 without worsening the fit of the model (see Table S3, available online). Because our model controls for stability and covariance at age 2, cross-lagged effects typically tend to be modest [39]. CU at age 2 explained approximately 1% of the variance in age 3 negative parenting, and this was mediated via genetic and modest nonshared environmental effects.

Given that only genetic factors linked CU and negative parenting at age 2, the common effects that are transmitted to negative parenting at age 3 are entirely genetic (Table 2). However, these common effects from age 2 explained only 1.5% of the variation in negative parenting at age 3.

Novel Effects and Covariance at Age 3

Variance at age 3 that was not transmitted from age 2 represents residual effects that are novel to age 3 and signify developmental change. That is, they are new effects that are independent of effects at age 2. As seen in Table, there was substantial change. For negative parenting 57% of the variance at age 3 was due to new age-specific effects, and for CU approximately 80% of the variation was novel to age 3. Developmental change in both CU and negative parenting was influenced by genetic and nonshared environmental factors. The *novel* effects on CU and parenting at age 3 do not correlate, as indicated by non-significant genetic and environmental residual correlations (Figure 1). However, because there are common genetic effects transmitted from age 2, there is a modest genetic correlation between CU and negative parenting at age 3 (r_{g2}=.10; CI=.05-.15).

Discussion

The overall goal of this study was to examine genetic and environmental bases for associations between CU and parenting during early childhood. There was no consistent association between CU and positive parenting, providing little evidence in support of either child- or parent-driven effects linking these two variables in an early childhood community sample. In contrast, CU and negative parenting were associated within and across age and cross-lagged models revealed only child-driven effects. CU behaviors at age 2 predicted negative parenting at age 3, however the reciprocal effect of parenting influencing later CU was not significant. In further support of child-driven effects on parenting, this longitudinal association was primarily explained by genetic factors. In addition, child-based genetic factors accounted for variance in negative parenting at both ages, and concurrent associations between CU and parenting at age 2 were explained by child-based genetic factors. This suggests that children's genetically-influenced tendencies affect the parenting that they receive and partially account for changes in parenting over time. This has also been found between antisocial behaviors and parent-child conflict across early adolescence [37]. Thus, despite the current focus in the CU literature on parent-driven effects, cross-lagged results demonstrate that child-driven effects are also important.

Contrary to research examining links between negative parenting and the related construct of child antisocial behaviors that finds bidirectional influences in middle childhood [23,37], in our young sample, negative parenting did not predict CU after controlling for conduct problems. Interestingly, however, when we did *not* control for conduct problems, there was a bidirectional relation that mirrored past work on negative parenting and related problem behaviors (see Figure S1, available online). The fact that we find parent-driven effects only with our unadjusted CU measure that included variance shared with conduct problems suggests that negative parenting impacts later conduct problems, but not CU specifically. That is, conduct problems are likely responsible for the parent-driven effects with the unadjusted measure, whereas child-driven effects appear to be particularly relevant to CU. Having a young child with high CU may be especially difficult for parents, and increase negativity. This, in turn, may also affect parent-child attachment, which can continue to have an impact on later development and the parent-child relationship.

Phenotypic cross-lagged models using clinical samples of older children demonstrated either no effects between CU and negative parenting or only parent-driven effects [13,21], and found that positive parenting was the most relevant [12,13,21]. This is contrary to our findings; however, these studies used clinical or at-risk samples whereas we utilized a community sample. Taken in the context of our results, this suggests that the type of parenting most relevant to CU behaviors may differ depending on the level of severity in related behavior problems. Specifically, cross-lagged studies of behavior problems in community samples, such as ours and others [23,37], indicate that it may be most beneficial to focus on negative parenting practices before serious behavior problems have emerged (i.e., prevention work). However, bidirectional research with clinical samples of children with more severe problems suggests [12,13,21] that it may be profitable to focus on improving positive parenting practices (i.e., in interventions). More bidirectional work on CU and parenting in both at-risk and community samples is needed to further clarify the relative importance of different aspects of parenting across developmental stages and symptom severity.

Although only child-driven effects were found for negative parenting and CU, and positive parenting showed little to no association with CU, this does not imply that parenting is unimportant. Controlling our measure of CU for variance shared with CP helps us to understand what is CU-specific in relation to parenting, but CU often co-occurs with other difficult behaviors such as CP, which demonstrate parent-driven effects. Additionally, this work suggests that early CU behaviors can drive later negative parenting, which could in turn affect CU behaviors later in development. Longitudinal research examining multiple time points from toddlerhood to middle childhood are needed to address this possibility. Lastly, as previously mentioned, the type of parenting may vary across behavior problem severity or developmental stage.

Despite strengths of this study, including the use of longitudinal, genetically-informed data on both positive and negative parenting, limitations should be mentioned. First, this study relied on parent-reports of both parenting and CU thereby raising the issue of shared rater variance. Given that shared rater variance would be expressed as shared environmental influence in biometric models [40], and because we found no shared environmental covariance between parenting and CU, rater variance is not contributing to covariation between the

phenotypes. Additionally, the cross-lagged effects in our models are not subject to potential shared rater bias or 'halo effects' as they are independent of covariance between the measures at the first time point. Furthermore, the etiologic pattern of negative parenting in our sample is similar to work implementing observer ratings [23]. Nonetheless, future studies should include observer ratings of parenting when possible. Second, the CBCL measure of CU has low internal consistency, however, this is typical in the literature on CU in young children (for a more extensive discussion of this issue see [6,8,29]). Third, while child-driven effects explain a significant amount of the variance at age 3, these effects are small and only part of the puzzle in explaining CU behaviors. Replication with other methods and genetically-informed designs is encouraged. Finally, there is some debate about the construct of "CU" at age 2. Indeed, it is not as clearly established as at age 3, demonstrating lower reliability and validity [41]. Nonetheless, we find that the factor structures and heritabilities at ages 2 and 3 are similar [7,8]; and that there is moderate stability across these ages. Moreover, this stability is genetically mediated indicating that, to some extent, the same genetic effects operate on the behaviors at ages 2 and 3 [8]. We suggest that, at the very least, these very early CU-like behaviors at age 2 are developmentally significant as they are predictive of both CU and negative parenting at age 3.

Our results demonstrate the importance of child-driven effects in the relation between CU and negative parenting in young children. These children driven effects have both genetic and environmental underpinnings, and highlight the necessity of targeting child behavior in early CU interventions in an effort to decrease later negative parenting. This may help prevent a downward spiral from occurring into middle childhood that exacerbates CU behaviors.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Facebook Text

In a community sample of toddler twins, genetically-influenced callous-unemotional behaviors (low guilt, empathy and affect) predicted negative parenting one year later, but not the other way around. The unidirectional relation between callous-unemotional behaviors at age 2 and negative parenting at age 3 emphasizes the need for more research examining child-driven effects on the parent-child relationship, and reminds us that parenting does not occur in a vacuum.

Twitter Text

Children's genetically-influenced behaviors can impact the type of parenting they receive. In the Boston University Twin Project toddlers who showed low guilt, empathy and affect at age 2 elicited more negative parenting at age 3. Parenting did not influence these behaviors.

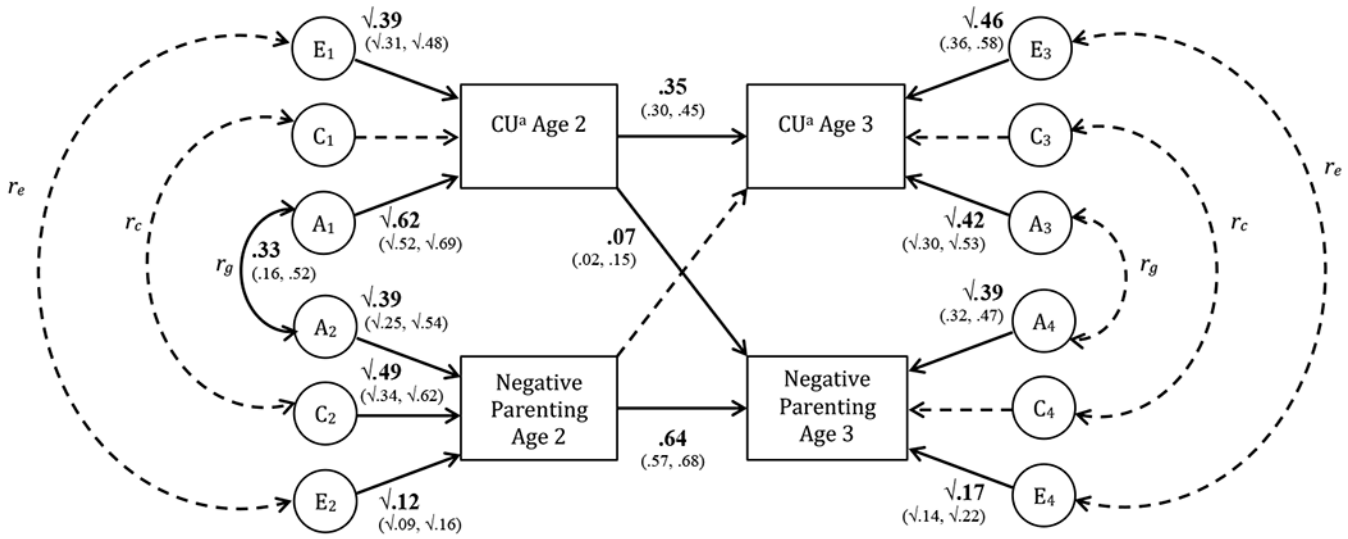

Figure 1: Biometric cross-lagged reduced model for Callous-Unemotional Behavior and negative parenting, shown for one twin.

Dashed paths indicate nonsignificant paths that could be dropped from the model. The model constrains all cross-age associations to function as phenotypic partial regression coefficients. The paths leading from CU at age 2 to CU at age 3 index stability, controlling for any prior association with parenting. The cross-lagged paths reflect the effects of CU at age 2 on parenting at age 3 and visa versa, above and beyond their preexisting relation at age 2 and stability effects. The variances of CU and parenting at age 2 are decomposed into their genetic (A), shared environmental (C), and nonshared environmental (E) components. The genetic and environmental effects at age 3 are residual effects that are independent of age 2, and reflect change. Paths between the latent A, C, and E factors and each phenotype are standardized partial regression coefficients, and indicate the relative contributions of A, C, and E factors to each phenotype. The square of these paths represents the genetic and environmental variance associated with each phenotype. Confidence intervals are presented in parentheses. r_g, r_c, and r_e denote the genetic and environmental correlations. A=Genetic influences; C=Shared environmental influences; E=Nonshared environmental influences; CU=Callous-Unemotional Behaviors[a]Residualized for sex and conduct problems.

Table 1.

Phenotypic Correlations (95% CI) Between Callous-Unemotional Behavior[a] and Parenting Within and Across Age

	Age 2			Age 3		
	CU	**NEG**	**POS**	**CU**	**NEG**	**POS**
CU Age 2	1					
NEG Age 2	**.17** (.09, .24)	1				
POS Age 2	**−.15** (−.23, −.07)	**−.37** (−.44, −.30)	1			
CU Age 3	**.35** (.27, .41)	**.11** (.03, .19)	−.04 (−.12, .04)	1		
NEG Age 3	**.20** (.12, .27)	**.68** (.63, .72)	**−.35** (−.42, −.28)	**.11** (.03, .19)	1	
POS Age 3	**−.09** (−.16, −.005)	**−.32** (−.39, −.24)	**.57** (.51, .62)	−.001 (−.08, .08)	**−.41** (−.47, −.34)	1

Note: CU=Callous-Unemotional Behaviors; NEG=Negative parenting; POS=positive parenting.

[a]Residualized for sex and conduct problems.

Bolded correlations are significant at $p < .05$.

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Table 2.

Genetic and Environmental Influences on Callous-Unemotional Behaviors[a] and Negative Parenting at Age 3 – Reduced Model

	Total variances	A	C	E
Age 3 CU				
Total variances due to:	1.000	.492	.000	.497
1. CU at age 2 (Stability effects)	.122	.075 (15%)	--	.047 (9%)
2. NEG at age 2 (Cross-lagged effects)	.000	--	--	--
3. Common effects at age 2	.000	--	--	--
4. Residual effects at age 3	.878	.422 (86%)	--	.456 (92%)
Age 3 NEG				
Total variances due to:	1.000	.571	.203	.226
1. NEG at age 2 (Stability effects)	.413	.159 (28%)	.203 (100%)	.051 (22%)
2. CU at age 2 (Cross-lagged effects)	.005	.003 (.5%)	--	.002 (1%)
3. Common effects at age 2	.015	.015 (2.5%)	--	--
4. Residual effects at age 3	.567	.394 (70%)	--	.173 (77%)

Note: The overall genetic and environmental variances for CU and NEG at age 3 can be decomposed into: (1) *stability effects*: influences specific to phenotype 1 at age 2 that are transmitted to age 3; (2) *cross-lagged effects*: influences specific to phenotype 2 at age 2 that are transmitted to phenotype 1 at age 3; (3) *common effects from age 2*: the effects common to both phenotypes at age 2 that are transmitted to phenotype 1 at age 3; and (4) *residual effects*: unique effects on phenotype 1 at age 3. The age 3 variances for phenotype 2 are similarly decomposed. Percentages of A (genetic), C (shared environmental), E (nonshared environmental) influences due to the four sources are provided in parenthesis. Deviations in percentages are due to rounding. A=Genetic influences; C=Shared environmental influences; E=Nonshared environmental influences; CU=Callous-Unemotional Behaviors; NEG=Negative Parenting

[a]Residualized for sex and conduct problems.