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## The Inventory of Callous-Unemotional Traits (ICU) in Children: Reliability and Heritability

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### Abstract

Callous-unemotional (CU) traits comprise the core symptoms of psychopathy, yet no study has estimated the heritability of CU traits in a community sample of children using an instrument designed solely to assess CU traits. The current study uses data from 339 twin pairs aged 9-14 to examine the reliability and heritability of the parent-report Inventory of Callous-Unemotional Traits (ICU) at two assessments approximately 3 weeks apart. Time-specific measurement error was taken into account to obtain a more accurate estimate of the heritability reflecting the latent liability to CU traits. Test-retest reliability was .84 and heritability at visit 1 was 39%. The heritability of the latent liability to CU traits was 47%. This latent liability contributed 79% of the variance in ICU score at visit 1 and visit 2. This is the first study to account for measurement error while examining the heritability of CU traits, furthering our understanding of psychopathy in children.

### Keywords

Callous-Unemotional Traits; Heritability; Reliability; Measurement Model; Twins

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## Introduction

Conduct disorder (CD) is a psychiatric disorder of childhood and adolescence that reflects socially debilitating psychopathology (American Psychiatric Association 2013). CD has been associated with a variety of negative health outcomes including poorer physical health (Bardone et al. 1998; Odgers et al. 2007), premature mortality (Laub and Vaillant 2000), comorbid psychiatric conditions (Kim-Cohen et al. 2003), and increased risk for legal problems (Simonoff et al. 2004). It is estimated that 12% of males and 7% of females will meet criteria for CD at some point in their lifetime (Nock et al. 2006).

There appears to be substantial heterogeneity in the developmental trajectories, corollaries, and treatment outcomes associated with CD (Frick 2012). This observed heterogeneity has contributed to a substantial literature on subtypes and features of CD that may delineate those individuals who are at the highest risk for future psychopathic behavior. Some of the most prominent subtyping efforts involve grouping individuals based on age of onset (Moffitt et al 2008), whether they display aggressive behaviors (Tackett et al 2005), socialized vs. undersocialized constructs (American Psychological Association, 1980), and callous-unemotional (CU) traits (Frick et al. 2014).

Psychopathic traits describe interpersonal (e.g., egocentric, manipulative) and affective characteristics (e.g., shallow affect, lack of guilt) rather than focusing on antisocial *behavior* (Frick et al. 2014). An extension of the psychopathic construct to children was first conceptualized in the DSM-III as the “undersocialized” CD subtype (American Psychological Association, 1980). Although the undersocialized subtype suffered from many conceptual problems (e.g., not corresponding to the adult psychopathy construct; incorrect interpretations of “socialized”) the more recent CU traits construct, which focuses on the affective characteristics of psychopathy, has emerged as a useful marker for designating youth with the most severe conduct problems (Frick et al. 2014). CU traits have been said to comprise the “core” symptoms of psychopathy (Frick and Morris 2004; Fowles and Dindo 2009).

The latest edition of the APA's Diagnostic and Statistical Manual (DSM-5) includes a specifier for CD diagnosis: limited prosocial emotions (American Psychiatric Association 2013). This specifier reflects a subtype of CD in which individuals display a variety of callous-unemotional (CU) traits. Within the DSM-5 CU traits describe characteristics such as shallow affect and lack of empathy and remorse (American Psychiatric Association 2013) and are associated with severe forms of CD and antisocial personality disorder (ASPD) (Robins 1978; Simonoff et al. 2004; Gelhorn et al. 2007; American Psychiatric Association 2013). Among individuals who meet criteria for CD, approximately 10–32% will also meet criteria for the more limited prosocial emotions (CU) specifier (Kahn et al. 2012). Amongst individuals with CD, those who meet criteria for the CU specifier are at risk for more severe developmental trajectories, such as ASPD (McMahon et al. 2010) and violent crime in adulthood (Kruh et al. 2005; Vitacco and Vincent 2006).

CU traits, as measured by a variety of instruments, have been shown to be under moderate to substantial genetic control, with genetic factors accounting for 40–78% of the phenotypic

variance (for a review see Viding and McCrory 2012). Although the heritability of CU traits has been extensively studied, less research has focused on examining the heritability of CU traits in children (e.g., Bezdjian et al. 2011; Humayun et al. 2014). One such study used items from the Antisocial Process Screening Device (APSD) (Frick and Hare 2001) and Strengths & Difficulties Questionnaire (SDQ) (Goodman 1997) to assess the heritability of CU traits in 7-year-old twins with elevated CU traits (i.e., > 1.3 SD above the mean). This study found a heritability of approximately 75% (Humayun et al. 2014). Similar studies using selected samples have also found childhood heritabilities for high levels of CU traits in the range of 80% (e.g., Viding et al. 2005; Viding et al. 2008), although these results are not generalizable to a community sample of children due to their reliance on the upper end of the CU trait distribution.

To date, only one study has examined the heritability of CU traits in a community sample of children, albeit indirectly. In this study, Bezdjian and colleagues (2011) assessed psychopathic traits in twins aged 9–10 using the Child Psychopathy Scale (CSP) (Lynam 1997). They found a two-factor solution with one factor representing a “callous/disinhibited” dimension. The heritability of this factor was estimated at 64% in boys and 49% in girls (Bezdjian et al. 2011), although the validity of this factor in assessing CU traits has not been established.

The Inventory of Callous-Unemotional Traits (ICU) (Frick 2004; Kimonis et al. 2008) was developed to directly assess CU traits via parent-, teacher-, and/or youth self-report. The ICU was developed from items within the APSD (Frick and Hare 2001) and expanded to provide a more complete assessment of CU traits in children (Fanti et al. 2009). Although the specific factor structure of the ICU has been variously reported, the majority of studies find three subscales of the ICU (i.e., ‘uncaring,’ ‘unemotional,’ and ‘callousness’) (e.g., Fanti et al. 2009; Roose et al. 2010; Ezpeleta et al. 2013; Pechorro et al. 2016). The youth self-report version of the ICU has demonstrated good test-retest reliability (Feilhauer et al. 2012), however the reliability of the teacher- and parent-report versions have yet to be tested.

Only two studies have examined the heritability of CU traits using the ICU (Mann et al. 2015; Henry et al. 2016). The first used data from 255 twin pairs between the ages of 13–21 and found the heritability of the self-report ICU to be approximately 40% (Mann et al. 2015). The second used data from 5,092 16-year-old twin pairs, and examined the heritability of separate factors within the parent-report ICU. This study revealed a general factor, a callous-uncaring factor, and an unemotional factor that were all under genetic influence, with heritabilities of 58%, 70%, and 79%, respectively (Henry et al. 2016).

Reviewing quantitative genetic studies of CU traits in children reveals two primary concerns: 1) the frequent use of selected samples, and 2) the use of instruments not specifically designed to assess CU traits. In regard to the first concern, researchers often use selected samples due to the relative rarity of psychopathic traits in children. Historically, psychopathological phenotypes have been considered in terms of categorical constructs (American Psychological Association 2013) and reliance on the upper end of the phenotypic distribution concurs with this view of psychopathology. However, many researchers now advocate for taking a dimensional approach to the study of psychopathological constructs (e.g., Widiger and Gore 2014). Specifically in genetic analyses, where a normal underlying

distribution is a primary statistical assumption, selected samples will produce biased estimates (e.g., Neale et al. 1989). It is therefore important that traits known to index 'extreme' phenotypes (e.g., psychopathy) be studied in individuals with lower levels of such traits (i.e., community samples), particularly when the research questions are genetic in nature. Regarding the second concern, instruments designed specifically to assess CU traits are fairly recent developments, and no studies have used such instruments to examine the heritability of CU traits in a community sample of children, leaving a critical research gap.

### Study Aims

The current study uses a genetically informed community sample of 339 twin pairs (N = 678) between the ages of 9-14 who were assessed at two separate visits (23 days apart, on average). The first aim of the current study was to investigate the test-retest reliability of the parent-report ICU, a measure designed to assess CU traits in children. The second aim was to obtain a stable and reliable estimate of heritability by including data from two assessments of parent-report ICU in a single model. Given that the reliability of the parent-report ICU has not been demonstrated, it is possible that current heritability estimates for this measure may not be reliable. Therefore, the current study is novel and innovative in that it is the first to combine the assessment of reliability and heritability into a single analytic model within a community sample of twins.

### Methods

Data came from the Virginia Commonwealth University Juvenile Anxiety Study (VCU-JAS) (Carney et al. 2016), an ongoing twin study of anxiety and related phenotypes in Caucasian children aged 9-14. The inclusion of only Caucasian twins ensured a homogeneous sample for the genetic aims of the larger study. All twin pairs were recruited through the Mid-Atlantic Twin Registry (MATR) (Lilley and Silberg 2013). Twins and parents completed a variety of self-report measures via REDCap (Research Electronic Data Capture) (Harris et al. 2009). In addition, each twin completed several laboratory tasks. Families were invited to return to the lab 2-4 weeks later to complete a reliability assessment. Only the parent-report about child measures from visit 1 and visit 2 are used in the current study. The Virginia Commonwealth University Institutional Review Board approved the study, and all participants provided informed consent (parents) and assent (children) before participating. Twins and parents were monetarily compensated for their participation in the study.

### Participants

The data included herein are from families in which a parent completed the Inventory of Callous-Unemotional Traits (ICU) about both twins at visit 1, visit 2, or both, and also completed the zygosity questionnaire. The analytic sample size for the current study was N = 678 (N = 61 monozygotic female [MZF] twin pairs; N = 56 monozygotic male [MZM] twin pairs; N = 63 dizygotic female [DZF] twin pairs; N = 57 dizygotic male [DZM] pairs; N = 102 dizygotic opposite-sex [DZOS] twin pairs). Two hundred forty individuals (N = 25 MZF pairs; N = 17 MZM pairs; N = 20 DZF pairs; N = 19 DZM pairs; N = 39 DZOS pairs) were re-assessed approximately 2-4 weeks later for the purpose of examining test-retest

reliability. The visit 1 and visit 2 samples did not differ significantly on age ( $p = .63$ ), sex ( $p = .63$ ), or zygosity ( $p = 1$ ).

## Measures

**Zygosity**—Zygosity was determined from standard questions about physical similarity between twins (Nichols and Bilbro 1966; Peeters et al. 1998). Concordance rates between the algorithm assigned zygosity and zygosity from placental/DNA testing reported by parents was kappa = 1.0 ( $N = 42$  twin pairs).

**Inventory of Callous-Unemotional Traits (ICU; Parent Report)**—For each twin, a parent completed the ICU (Frick 2004; Kimonis et al. 2008), a 24-item measure assessing traits relating to callousness, carelessness, and emotionless. Parents ranked each item on a 4-point Likert scale, from 0 (not at all true) to 3 (definitely true). A sum score was created for the entire ICU measure, with possible scores ranging from 0–72. In addition, sum scores were created for each of the three subscales most frequently reported in the literature (Fanti et al. 2009; Roose et al. 2010; Ezpeleta et al. 2013; Pechorro et al. 2016). Supplementary Table 1 shows the ICU item wording and indicates items used to construct the ICU sum score and traditional subscales. The self-report ICU has demonstrated good test-retest reliability, internal consistency and convergent validity (Kimonis et al. 2008; Feilhauer et al. 2012). All ICU items have been shown to load onto a general ICU factor under substantial genetic control, and therefore the ICU sum score is a valid construct for measuring the underlying genetic structure of CU traits (Henry et al. 2016).

## Statistical Analysis

Confirmatory factor analyses (CFA) were conducted to determine if the traditional uncaring, unemotional, and callous subscales provided a good fit to the current data. After examining the distribution of ICU scores (see Figure 1), test-retest reliability was estimated using Pearson's product-moment correlation. Correlation was computed for ICU sum scores as well as ICU subscale sum scores from visit 1 and visit 2.

Standard biometrical structural equation modeling (SEM) (Neale and Cardon 1992) was used to decompose the observed variation in callous-unemotional traits in terms of additive genetic ( $a^2$ ), shared environmental ( $c^2$ ) and unique, or non-shared, environmental ( $e^2$ ) risks. Additive gene action ( $a^2$ ) reflects the additive or average effect of individual alleles at genetic loci influencing a trait or behavior and contributes twice as much to the MZ twin correlation as the DZ twin correlation (because MZ twins share 100% of their genes, whereas DZ twins share, on average, 50% of their genes). Common environmental effects ( $c^2$ ) describe influences that make family members more alike compared to random pairs of individuals and contribute to the MZ and DZ twin correlations equally. Unique environmental effects ( $e^2$ ) capture aspects of the environment that are unique to each individual plus error and are therefore uncorrelated between twins in a family.

Biometrical SEM was used to decompose the variance in the ICU sum score at visit 1 into A, C and E factors (saturated model). Individual parameters were then constrained to zero (e.g., CE model, AE model and E model) to determine the best fitting, most parsimonious

model via comparison of  $-2 \log$ -likelihood ( $-2LL$ ) fit statistics. This procedure was then repeated for the visit 2 data.

The common factor measurement model, fully described elsewhere (Kendler et al. 1993; Kendler et al. 1999; Ystrom et al. 2011), uses data from two separate assessments and assumes that each individual possesses a latent liability to the phenotype of interest. The separate assessments are considered fallible indicators of the latent liability. Figure 2 displays the parameters of interest in the measurement model. The  $\lambda_1$  and  $\lambda_2$  paths represent the strength of the relationship between the true liability and the measured phenotype at two time points. The remaining variance in the observed phenotypes can be attributed to time-specific error, as indicated by the  $\delta_1$  and  $\delta_2$  paths. The latent liability to the phenotype of interest can then be further decomposed into A, C, and E factors, as described previously.

We used visit 1 and visit 2 ICU indicators to fit a saturated ACE measurement model. We then constrained individual parameters (e.g.,  $a^2$  &  $c^2$ ) to zero to determine the best fitting, most parsimonious model. Further constraints were imposed on the best-fitting model where  $\delta_1$  was constrained to equal  $\delta_2$  and  $\lambda_1$  was constrained to equal  $\lambda_2$  seeking a more parsimonious model.

All analyses were performed in the R statistical computing environment (R Core Team 2014). The Lavaan package (Rosseel 2012) was employed to examine CFA models, and twin models (ACE and measurement models) were estimated using the OpenMx 2.0 package (Boker et al. 2014). Full information maximum likelihood (FIML) estimation was employed to ensure that twins with data from only one visit would meaningfully contribute to the full measurement model.

## Results

### Sample Characteristics

For the analytic sample, the mean age at visit 1 was 11.24 ( $SD = 1.44$ ; range = 9–14.5) and was 51.6% female. At visit 2 the mean age was 11.32 ( $SD = 1.44$ ; range = 9–14) and was 53.8% female. The mean ICU score in the current sample (including both visit 1 and visit 2) was 17.46 ( $SD = 7.74$ ; range = 0–45). The mean ICU scores at visit 1 and visit 2 were 17.62 ( $SD = 7.63$ ; range = 1–41) and 16.97 ( $SD = 8.05$ ; range = 0–45), respectively. Figure 1 displays the distribution of ICU sum scores in the current sample (including both visit 1 and visit 2).<sup>1</sup>

Full CFA results for visit 1 can be found in Supplementary Table II. The fit of the traditional model was adequate (CFI = .923; RMSEA = .102), and the high correlation ( $r = .941$ ) between the uncaring and callous subscales was notable, especially in light of recent analyses showing similar results in adolescents (Henry et al. 2016). We additionally examined a bifactor model, which altered the traditional model in two ways: 1) combining the highly correlated callous and uncaring subscales into a single factor, and 2) adding a

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<sup>1</sup>Identical sets of reliability and SEM analyses were conducted with observations more than 3 standard deviations from the mean ( $N = 2$ ) removed, and the results were nearly identical to those reported here.



general factor on which all items were instructed to load. This model displayed improved fit (CFI = .989; RMSEA = .041), although several items failed to load significantly on the callous/uncaring factor. We thus examined a more restricted bifactor model that removed items from the sub-factors (unemotional and callous/uncaring) that did not reach factor loadings of at least .3 in the full bifactor model. As expected, the fit of this model (CFI = .986; RMSEA = .044) did not decrease significantly from the full bifactor model and was thus chosen as the best-fitting, most interpretable model. In all models, item #10 (“Does not let feelings control him/her”) did not load well on the callous, callous/uncaring, or general factors.

Table 1 displays the cross-twin cross-time ICU sum score correlations by zygosity. Additionally, table 2 presents the visit 1 and visit 2 twin correlations divided by individual zygosity groups. At visit 1, for which our sample size was largest, these correlations indicate that ICU score is substantially influenced by both unique environmental (as indicated by a MZ correlation of .39, substantially less than 1) and additive genetic factors (as indicated by a MZ correlation higher than the DZ correlation, .16). Additionally, the correlation for DZOS twin pairs (.05) is substantially lower than that of DZM (.22) or DZF (.29) twin pairs, indicating a potential qualitative sex-effect (Neale and Cardon 1992).

We examined potential covariates for inclusion in SEM models by conducting a multiple regression using both age and sex as predictors of total ICU score. Results revealed that female sex was significantly associated with lower ICU score ( $\beta = -1.60$ ;  $p = .005$ ), but age was not significantly associated ( $\beta = .25$ ;  $p = .21$ ). Given these results, and because our small sample size precluded formally testing for qualitative or quantitative sex effects, we chose to control for sex in all subsequent SEM analyses. By explicitly modeling the effect of sex on the mean in subsequent SEM analyses, we are able to parse out the effects of this covariate by decomposing only the residual variance that is not due to sex (Neale and Cardon 1992).

## Reliability

Two hundred nine children had ICU data at both visit 1 and visit 2. The mean time between visits was 23 days (range = 12–50 days). Test-retest reliability between parent-report ICU total at visit 1 and visit 2 was  $r = .84$  (95% CIs: .80–.88). Test-retest reliability estimates for the traditional uncaring, unemotional, and callousness subscales were  $r = .81$  (95% CIs: .76–.85),  $r = .75$  (95% CIs: .68–.80), and  $r = .67$  (95% CIs: .58–.74), respectively. Reliability estimates  $< .60$  are considered insufficient, .60–.69 marginal, .70–.79 acceptable, .80–.89 good, and  $> .90$  excellent (Barker et al. 1994). Accordingly, our results demonstrate good test-retest reliability for the ICU total, whereas the uncaring, unemotional, and callousness subscales reliabilities were good, acceptable, and marginal, respectively.

## Univariate Heritability

**Visit 1**—Biometrical SEM, described above, was used to estimate a full ACE model (model I). Individual parameters in the model were constrained to zero to test their significance, and  $-2LL$  values were compared. Table 3 displays the full parameter estimates for the visit 1 model fitting. Dropping C from the model (AE model; model II) did not result in a

significant deterioration in model fit ( $p = 1$ ). However, dropping A (CE model; model III) produced significant model deterioration ( $p = .017$ ). These results indicate that an AE model best fits the ICU data for visit 1. The genetic factor accounted for approximately 39% (95% CIs: .22–.56) of the variance in parent-report ICU, with the remaining 61% (95% CIs: .49–.78) of variance due to the unique environmental factor.

**Visit 2**—Our smaller visit 2 sample ( $N = 120$  twin pairs) lacks sufficient power to detect significant genetic and environmental effects. For this reason, visit 2 data was only used in the overall measurement model.

### Measurement Model

A common factor measurement model, as described above, was used to estimate the full model displayed in Figure 2 (model I in Table 4). Dropping A from the model (CE model; model III) resulted in a significant deterioration in model fit ( $p = .014$ ). However, dropping C from the model (AE model; model II) did not result in significant model deterioration ( $p = 1.00$ ). Based on this information, the AE model was further adjusted by constraining  $\delta_1$  to equal  $\delta_2$  and  $\lambda_1$  to equal  $\lambda_2$  (model V), and this model did not result in a significant deterioration in fit ( $p = .674$ ). Figure 3 displays the final, best-fitting, most parsimonious, SEM (model V). In this model, sex was significantly associated with mean ICU score, such that females had, on average, a score that was 1.41 (95% CIs:  $-2.41$ – $.41$ ) units less than males. The genetic factor accounted for approximately 47% (95% CIs: .29–.62) of the variance in the latent liability to callous-unemotional traits, with the remaining 53% (95% CIs: .38–.71) variance in the latent liability due to the unique environmental factor. This latent liability contributed 79% (95% CIs: .68–.90) of the variance in the measured parent-report ICU at visit 1 and visit 2. The remaining 21% (95% CIs: .18–.26) of the variance in the measured phenotype was due to time-specific error.

### Discussion

This study sought to examine the reliability and heritability of callous-unemotional traits in children as measured by the parent-report ICU. CFA models examining the factor structure of this instrument demonstrated that a bifactor model, with a general factor and two specific factors relating to callous/uncaring and unemotional subscales, fit the data best. The traditional callous and uncaring subscales are very highly correlated (.94), indicating that the items within these subscales are likely indexing a single, broader underlying construct. It is also worth noting that item #10 did not load well on any factor, and therefore future studies may consider removal of this item.

We found reasonably high test-retest reliability for the parent-report ICU total ( $r = .84$ ) in 9–14 year olds over a mean interval of 23 days. Slightly lower test-retest reliabilities were found when examining the uncaring, unemotional, and callousness subscales separately ( $r = .81$ ,  $.75$ , and  $.67$ , respectively). These reliability estimates, the first reported for the parent-report ICU, are somewhat higher than those described for the self-report ICU total in individuals aged 8–20 ( $r = .72$ ) (Feilhauer et al. 2012). Current results suggest that the ICU completed by parents is a reliable measure of their offspring's CU traits.



Our results also indicate that callous-unemotional traits, as measured by the parent-report ICU, are significantly influenced by both additive genetic and unique environmental factors. At visit 1 an AE model fit the data best, indicating 39% of the variance in parent-report ICU was due to the latent genetic factor A, with the remaining 61% variance due to the unique environmental factor E. The heritability for visit 1 of the current study appears lower than some previously reported estimates. Earlier twin studies of CU traits reported heritability estimates ranging from 40–79% (Bezdjian et al. 2011; Viding and McCrory 2012; Humayun et al. 2014; Mann et al. 2015; Henry et al. 2016). However, the highest estimates stem from studies that examined the heritability of specific ICU factors (Henry et al. 2016) or focused on individuals with extreme CU traits (Humayun et al. 2014). Only one study examined the heritability of CU traits within a community sample of children, and this study reported heritabilities of 49% and 69% for males and females, respectively (Bezdjian et al. 2011). However, this study used a measurement instrument that has not been widely validated for the assessment of CU traits. It is therefore likely that the point estimate of heritability of CU traits within a community sample of children is within the range reported here.

This estimate of CU trait heritability in children is similar to estimates obtained in adolescent and young adult samples indicating that the phenotype is 40–45% heritable in samples ranging from 16–24 years old (e.g., Taylor et al. 2003; Blonigal et al. 2005; Blonigan et al. 2006; Larsson et al. 2006). Taken together, this suggests that heritability may remain quite stable across middle childhood, adolescence and young adulthood. However, the various instruments used in these reports make it difficult to compare estimates across studies. Therefore, potential age effects on the heritability of CU traits in community samples remains an important area of future research.

This study also was able to parse out the effects of time-specific measurement error, finding that a latent liability to CU traits contributes 79% of the variance in parent-report ICU scores at multiple time points. In the current sample, 21% of the phenotypic variance of CU traits was due to time-specific measurement error, suggesting that the accuracy of heritability estimates can be greatly improved by including more than one time-point in future assessments. This latent liability to CU traits was found to be under moderate genetic control (47%), with the remaining variance in this latent dimension due to unique environmental effects (53%). No significant effect of common/shared environment was found.

These ACE estimates are consistent with the vast majority of complex traits, most of which display a similar pattern of moderate genetic and unique environmental influences with no significant influence of common environment (Polderman et al. 2015). It is worth noting that one exception to this general pattern of ACE estimates for complex traits is antisocial behavior. In a meta-analysis of 51 studies, Rhee and Waldman (2002) found that antisocial behavior is influenced by a mild amount of common environmental influences (16%). However, they also found that the specific operationalization of the phenotype significantly moderated the ACE estimates. Taken together with this and other research indicating that psychopathy and CU traits are minimally influenced by common environment (for a review see Viding and McCrory 2012), it is reasonable to suggest that the common environmental influences on antisocial behavior are driven by symptoms that are not within the psychopathic domain.

The magnitude of heritability of CU traits found in the current study is also within the range of previous reported estimates from community samples (e.g., Taylor et al. 2003; Blonigan et al. 2005; Blonigan et al. 2006; Larsson et al. 2006; Mann et al. 2015), as well as estimates for complex traits in general (Polderman et al. 2015). Although most studies indicate a general heritability range of 40–50% for CU traits, the search for molecular genetic mechanisms has not produced promising results. Several candidate genes have been tentatively implicated in CU traits, including COMT, MAOA, and 5-HTTLPR (e.g., Fowler et al. 2009; Sadeh et al. 2010). However, there have been no robustly replicated effects, and the percentage of variance accounted for is small. Given the noted heterogeneity within the antisocial behavior phenotype, it is likely that very large sample sizes and advanced genetic techniques will be necessary in the search for molecular genetic mechanisms with replicable and meaningful effects.

Although the current study was able to parse out the effect of time-specific measurement error, which is usually included in the estimate of E, the influence of unique environment was still large (53%). Some of the most researched environmental influences on CU traits are the parenting and peer environments. Deviant peer association and harsh/inconsistent parenting have been repeatedly reported as influences on CU traits (for a review see Frick et al. 2014). Because only three parameters can be estimated in the classical twin model (Neale and Cardon 1992), specific environmental influences are rarely included in such studies. However, given the insubstantial influence of common environment on CU traits, future biometric studies should consider modeling the effects of specific unique environments. Such inclusion would allow for the estimation of these specific environmental effects while controlling for the influence of genetic factors.

This study has several notable strengths. First and foremost, this is the first study to examine the heritability of CU traits with multiple assessments and using a common factor measurement model. This approach allows one to account for measurement error and more accurately estimate the components of variance that are influencing the latent liability to CU traits. In addition, this is the first study to examine the test-retest reliability of the parent-report ICU and only the second to examine the heritability of the parent-report version. Lastly, this study is the first to directly examine the heritability of CU traits in a general population sample of children via parent-report ICU. Therefore, this study contributes substantially to literature on CU traits in children and supports the use of the parent-reported ICU as a reliable measure for assessing such traits.

Despite the significant strengths of the current study, the results must be interpreted in light of several limitations. First, the entirety of our sample was Caucasian, which may limit the generalizability of the current findings. Second, our sample size for visit 2 was too small for biometrical analyses, although that data meaningfully contributed to our analyses of the latent liability to CU traits via the measurement model. Finally, the sample size precludes our ability to test for potential quantitative or qualitative sex differences in the heritability of CU traits. There is some evidence that sex may influence the heritability of CU traits (Bezdjian et al. 2011), and the DZOS correlations for this sample also suggest potential qualitative sex effects. Therefore, formally examining qualitative and quantitative sex-effects in psychopathy and CU traits is an important future direction. Despite these limitations, this

study is the first to account for measurement error while examining the heritability of CU traits in a community sample of children. This study is therefore an important further step in better understanding the etiology of callous-unemotional and psychopathic traits in children.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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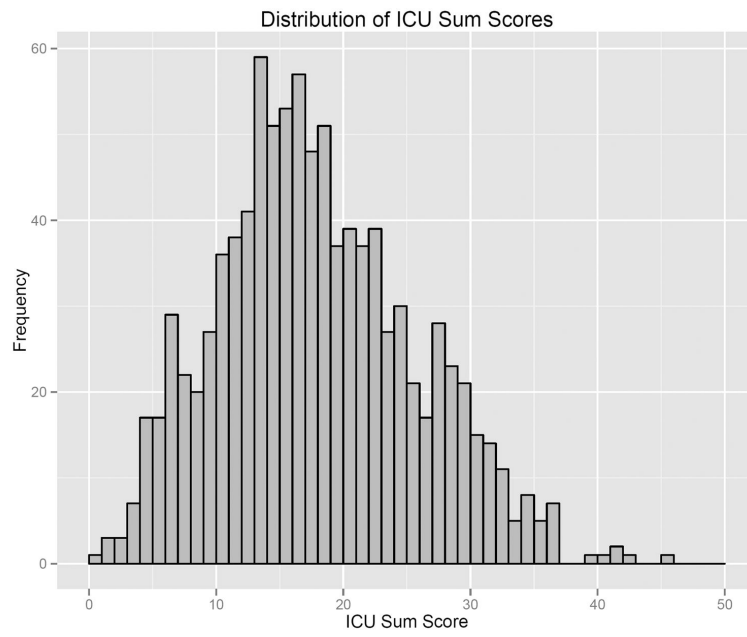
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\*includes data from both visit 1 and visit 2

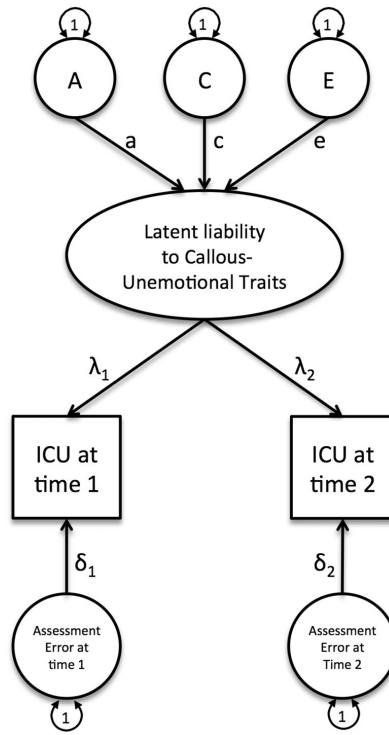
**Figure 1.**  
Distribution of ICU Sum Scores in the Total Sample\*

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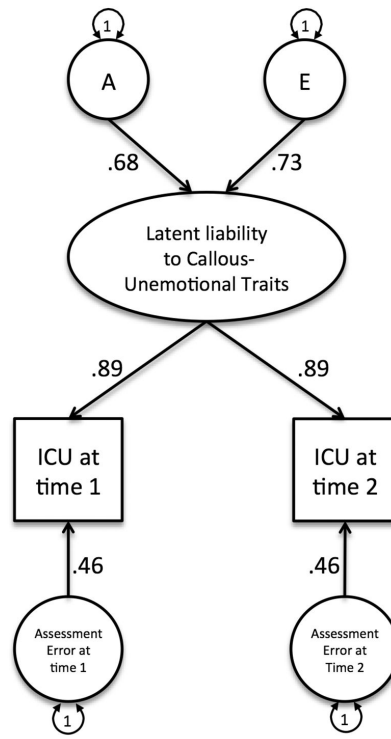
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\*Model depicted for 1 twin only

**Figure 2.**  
Measurement Model



\*Model depicted for 1 twin only

**Figure 3.**  
Best-Fit Measurement Model Estimates

**Table 1**

Cross-twin cross-time correlations for ICU sum score for MZ and DZ twin pairs

		Visit 1		Visit 2	
		Twin 1	Twin 2	Twin 1	Twin 2
MZ Twin Pairs					
Visit 1	Twin 1	1	-	-	-
	Twin 2	.390*	1	-	-
Visit 2	Twin 1	.726*	.409*	1	-
	Twin 2	.281	.880*	.371*	1
DZ Twin Pairs					
Visit 1	Twin 1	1	-	-	-
	Twin 2	.160*	1	-	-
Visit 2	Twin 1	.833*	.058	1	-
	Twin 2	.059	.755*	.093	1

ICU = Inventory of Callous Unemotional Traits; MZ = monozygotic; DZ = dizygotic.

\* = significant at  $\alpha = .05$ 

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**Table II**

Twin correlations for ICU sum score overall and by individual zygosity group

Zygosity	Visit 1	Visit 2
	<i>r</i>	<i>r</i>
MZ	.390*	.371*
MZF	.338*	.470*
MZM	.453*	.423
DZ	.160*	.105
DZF	.285*	.169
DZM	.221*	.470*
DZOS	.054	-.029

ICU = Inventory of Callous-Unemotional Traits; MZ = monozygotic; MZF = monozygotic female; MZM = monozygotic male; DZ = dizygotic; DZF = dizygotic female; DZM = dizygotic male; DZOS = dizygotic opposite sex.

\* = significant at  $\alpha = .05$

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**Table III**

## Model Fit Statistics and Parameter Estimates for ICU at Visit 1

Model	Estimated Parameters	-2 LL	df	AIC	$\chi^2$	df	p	$\beta$ (sex)	a <sup>2</sup>	c <sup>2</sup>	e <sup>2</sup>
I. ACE	5	4631.822	671	3289.822	-	-	-	-1.48	.39	.00	.61
II. AE*	4	4631.822	672	3287.822	$1 \times 10^{-11}$	1	1.00	-1.40	.39	-	.61
III. CE	4	4637.506	672	3293.506	5.6833	1	.017	-1.37	-	.23	.77
IV. E	3	4656.535	673	3310.535	24.7125	2	$4 \times 10^{-6}$	-1.47	-	-	1.00

Fits of sub-models are compared to base model I. All parameter estimates have been standardized and squared to reflect the percentages of variance accounted for.

\* = best fitting model



**Table IV**

**Model Fit Statistics and Parameter Estimates for ICU Measurement Model**

Model	Estimated Parameters	-2 LL	df	AIC	$\chi^2$	df	p	$\beta$ (sex)	a <sup>2</sup>	c <sup>2</sup>	e <sup>2</sup>	$\lambda_1$	$\lambda_2$	$\delta_1$	$\delta_2$
I. ACE	10	6211.391	924	4365.391	-	-	-	-1.42	.45	.00	.55	.84	.74	.16	.26
II. AE	9	6211.391	925	4363.391	$4 \times 10^{-10}$	1	1.00	-1.41	.45	-	.55	.84	.74	.16	.26
III. CE	9	6217.470	925	4369.470	6.0786	1	.014	-1.42	-	.23	.77	1.00	.62	.00	.38
IV. E	8	6235.774	926	4385.774	24.3823	2	$5 \times 10^{-6}$	-1.42	-	-	1.00	.80	.78	.20	.22
V. AE ( $\delta_1 = \delta_2 / \lambda_1 = \lambda_2$ )*	7	6212.926	927	4360.926	1.5352	3	.674	-1.41	.47	-	.53	.79	.79	.21	.21

Fits of sub-models are compared to base model I. All parameter estimates have been standardized and squared to reflect the percentages of variance accounted for.

\* = best fitting model.