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Peer socialization processes in the development of callous-unemotional traits

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Abstract

Callous-unemotional (CU) traits represent a risk factor for persistent, severe levels of externalizing problems. Irritability may predict the development of CU traits for some individuals, who are thought to acquire them in reaction to negative environmental experiences. Models on the development of CU traits have emphasized the socializing role of harsh parenting to the neglect of negative peer experiences. The present study 1) tested primary and alternative models of physical and relational peer victimization as socialization agents in relations between irritability and CU traits; and 2) considered hypothalamic-pituitary-adrenal-axis functioning as a moderator of these associations. Gender moderation was also considered. Aims were tested from middle childhood to adolescence using data from the NICHD Study of Early Child Care and Youth Development, which includes a large national sample ($N = 1{,}077$) and multiple methods and informants for the constructs of interest. Positive associations between irritability, peer victimization, and CU traits were supported, with indirect effects on CU traits supported specifically from peer victimization through increases in irritability. Associations between relational victimization, irritability, and CU traits may be particularly salient for females, whose experiences have been neglected to date. However, effects were small, and replication efforts are needed.

Keywords: irritability; peer victimization; callous-unemotional traits; salivary cortisol

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Introduction

Callous-unemotional (CU) traits consist of a combination of deficits in guilt, remorse, and empathy; shallow or superficial emotional expression; and disturbances in contingency learning (Frick et al., 2014; Waller & Wagner, 2019). Individuals high on CU traits also show deficits in emotion (especially fear) processing and insensitivity to punishment cues, which are thought to impair rule internalization processes against aggressive and violent behavior and limiting the development of moral emotions (Wakschlag et al., 2018). CU traits exist on a continuum from indifference to others' emotions and outcomes to intentional, goal-oriented provocation of distress; however, no level of CU traits or behaviors is considered developmentally normative (Wakschlag et al., 2018). CU traits and behaviors (as they are referred to in young populations for ethical purposes, see Waller & Hyde, 2018) may be meaningfully measured as young as early childhood. However, prior work has demonstrated the presence of increasing trajectories of CU traits through middle childhood into adolescence, with environmental factors contributing to this trajectory (Byrd et al., 2018; Fontaine et al., 2010, 2018). Understanding mechanisms by which CU traits develop during this developmental period may lead to critical prevention and intervention efforts with significant potential public health impact. To this end, the present study examines whether childhood irritability may promote the development of CU traits in adolescence through negative peer experiences (i.e., peer victimization) during a developmental period in which the peer context becomes increasingly salient (Sroufe, 2013).

Irritability and CU traits

A substantial body of work on the development of CU traits has focused on delineating two phenotypically distinct etiological pathways to the development of psychopathy, first proposed by Karpman (1941): 1) a primary pathway characterized by biologically-based, genetically predisposed hypo-arousal and low affiliative tendencies; and 2) a secondary pathway whereby youth with initially high levels of negative emotionality and reactivity become callous and uncaring in reaction to negative social experiences and trauma (e.g., Craig et al., 2021; Kimonis, 2023; Yildirim & Derksen, 2015). For this second group of individuals, negative emotional reactivity is thought to make the experiences of negative life events more emotionally and physiologically aversive than for those with lower sensitivity. In time, with particularly prolonged negative experiences and/or without the proper support to learn to cope with these experiences, some individuals may become "overloaded" and emotionally and empathically disengage to cope with this distress, reflected in CU traits (Kimonis, 2023). In short, some children who perceive the world as threatening and frustrating and are reactive to these threats may find challenging

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experiences particularly aversive and develop callousness to cope, which presents as CU traits (Kimonis, 2023).

Irritability may represent an especially important facet of this emotional sensitivity construct (which includes negative affectivity and emotional reactivity) for understanding developmental pathways from higher emotionality to CU traits, particularly after early childhood (Kimonis, 2023). Operationalized as an increased propensity to experience anger and frustration relative to peers (Brotman et al., 2017; Chen et al., 2021; Evans et al., 2024), irritability is distributed continuously in the population. Although specific definitions vary, irritability can generally be considered a persistent mood state characterized by a tendency toward reactive negative affectivity coupled with increased orientation toward and approach to perceived threat, higher appetitive reward tendencies, and temper outbursts (Brotman et al., 2017; Deveney et al., 2019; Leibenluft & Stoddard, 2013). These characteristics together create a construct with both tonic (i.e., persistent irritable mood state) and phasic (i.e., temper outbursts) components (Copeland et al., 2015). Given the increased threat sensitivity and reactivity characteristic of irritability, and the negative reactions to irritability by others (e.g., parents, peers) described further below, irritability in particular may serve as a risk factor for CU traits developing in response to environmental experiences of threat and frustration.

Historically, empirical programs of research specifically examining irritability and CU traits have largely proceeded independently from one another. For instance, CU traits and dysregulated negative affect (of which irritability is a component) have been considered key characteristics of two distinct pathways to engaging in externalizing behavior (e.g., Frick & Morris, 2004; Wakschlag et al., 2018; Waschbusch et al., 2020). Nevertheless, increasing empirical evidence supports the importance of considering these two constructs in tandem. For instance, in a high-risk sample of preschool children, a network analysis found that links between oppositional defiant disorder (ODD) and conduct disorder (CD) symptoms were largely driven by associations between irritability and callousness, and callousness served as the strongest bridge between irritability and CU traits (Bansal et al., 2021). Additionally, a recent study found that teacher- and parent-reported childhood irritability and CU traits interacted to predict higher levels of both general aggression (i.e., aggression measured without consideration of forms or collapsed across forms) and impairment (Waschbusch et al., 2020). Furthermore, a latent class analysis of children in Spain found that children belonging to a group consisting of high irritability and CU traits from ages 3 to 12 showed poorer psychological functioning (e.g., greater number of internalizing and externalizing disorders, higher overall psychopathology) and worse functional impairment than children on a trajectory involving high irritability without CU traits, or high CU traits without irritability (Ezpeleta et al., 2022a). There is also emerging evidence that irritability in middle childhood and early adolescence serves as a risk factor for the development of CU traits. For example, among boys aged 7 to 14, childhood-onset persistent irritability predicted higher levels of psychopathy features, including callousness, in adulthood, especially when co-occurring with poor cognitive control (Hawes et al., 2016). By contrast, among a Spanish sample, only the teacher-reported headstrong/defiant dimension of ODD, and not irritability, was reciprocally associated with teacher-reported CU traits from ages 3 to 12 years (Ezpeleta et al., 2022b).

Although there is theoretical and initial empirical support for the importance of understanding the overlap among irritability and CU traits, there remains limited work investigating developmental mechanisms involved in these pathways. Recent work has called for additional investigations on the overlap between irritability and CU traits, particularly applying a longitudinal, multi-method approach and a developmental psychopathology lens (e.g., Bansal et al., 2021; Kimonis, 2023; Waschbusch et al., 2020). This study aims to answer this call by examining the predictive role of irritability on the development of adolescent CU traits and examining peer victimization as a promotive socializing factor in these developmental links.

Developmental models of CU traits

Historically, work on the development of CU traits focused on demonstrating the downward extension of the psychopathy construct and predicting antisocial and psychopathic traits in adulthood (Craig et al., 2021; Waller & Wagner, 2019). Recently, researchers have been placing increased emphasis on how CU traits emerge from a developmental psychopathology perspective, including working to identify testable developmental mechanisms across biological, emotional, and contextual levels (e.g., Fonagy & Luyten, 2018; Frick et al., 2014; Kimonis, 2023; Wakschlag et al., 2018). One such model which has been garnering significant attention is the Sensitivity to Threat and Affiliative Reward (STAR) model (Waller & Wagner, 2019). The STAR model highlights two psychobiological and mechanistic factors, low affiliative reward (i.e., physical and nonphysical pleasure from interpersonal closeness) and low threat sensitivity (i.e., fearlessness), that together are theorized to together produce the behavioral presentation of CU traits (Waller & Wagner, 2019). Interestingly, although irritability alone is characterized by increased threat sensitivity (Brotman et al., 2017), among adolescents, high levels of co-occurring irritability and CU traits were actually characterized by hyposensitivity to threat in an fMRI study (Zhang et al., 2021), consistent with the profile predicted by the STAR model.

Although the STAR model hypothesizes genetic and biological factors, it also emphasizes the role of environmental influences in the development of both low affiliative reward and fearlessness, including for individuals who do not initially present with these characteristics (Waller & Wagner, 2019). For example, a lack of warm or affiliative inputs from the environment disrupts the development and expression of affiliative reward in children due to lack of modeling and opportunities to experience rewarding interactions with others (Waller & Wagner, 2019). Furthermore, overly harsh and threatening environments are thought to desensitize children to experiences of threat, leading to greater fearlessness and insensitivity to punishment over time. In both of these pathways, the STAR model and CU trait literature generally (e.g., Kimonis, 2023) emphasize the role of harsh parenting contexts. Specifically, the model theorizes that overly harsh and punitive parenting exacerbates risk by "eliciting anger or pain, direct modeling of aggression and fearless behavior, or reducing feelings of self-control," which may lead children to "become desensitized to any cues of threat or harshness in the environment, thus displaying a profile that appears even more fearless or callous" (Waller & Wagner, 2019, p. 663).

Indeed, the role of a harsh, negative early caregiving environment as a predictor of CU traits is well-established, and positive early environments appear to serve as a protective factor (e.g., Javakhishvili & Vazsonyi, 2022). However, although the STAR model acknowledges that children with CU traits have disrupted peer relationships (Waller & Wagner, 2019), this model

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and the broader literature to date have generally not considered whether harsh treatment by peers (i.e., peer victimization) could represent a similarly salient socialization force in the development of CU traits. Specifically, individuals experiencing higher levels of peer victimization may become desensitized to cues of interpersonal threat, find interpersonal relationships less rewarding, and begin to believe that others are insensitive to their needs, making them less sensitive to others' needs over time (Fontaine et al., 2014; Malti et al., 2010). This is consistent with socialization processes within the caregiving environment highlighted in the STAR model. Furthermore, these effects may be particularly impactful over middle childhood to adolescence as the peer context becomes more developmentally salient over this time (Sroufe, 2013).

Peer victimization

Peer victimization involves the receipt of aggression (i.e., actions intended to hurt or harm another) perpetrated by a child not related to the victim (e.g., excludes victimization by a sibling), and can take physical and relational forms (Ostrov & Kamper, 2015). Physical victimization includes the receipt of physical aggression, which involves physical force intended to hurt or harm another (e.g., hitting, kicking; Crick & Grotpeter, 1996; Ostrov & Kamper, 2015). Relational victimization involves the receipt of relational aggression, which uses removal or threat of removal of a relationship as the mechanism of harm (e.g., social exclusion, threats of friendship withdrawal; Crick & Grotpeter, 1996; Ostrov & Kamper, 2015). Relational aggression/peer victimization is considered the modal form for girls, whereas physical forms are modal for boys (Ostrov & Godleski, 2010; Ostrov & Kamper, 2015).

Children with higher levels of irritability are at a greater risk for peer problems, including peer victimization, due to their negative emotionality and aggressive tendencies (Barker & Salekin, 2012; Stringaris & Goodman, 2009). For instance, in a large cross-sectional study of elementary school children (i.e., ages 5–12), irritability was associated with higher levels of both physical and relational victimization, as well as peer rejection (i.e., dislike among the peer group), within the classroom context as reported by teachers (Evans et al., 2016). Another recent cross-sectional study of youth aged 8 – 18 found that self-reported irritability was positively associated with self-reported peer victimization, above and beyond effects of co-occurring anxiety (Chen et al., 2021).

CU traits are also associated with peer relationship problems, including peer victimization. For instance, CU traits predicted poorer peer relations, including lower social competence, greater loneliness, and lower friendship quality in middle childhood (Haas et al., 2018). Additionally, a recent meta-analysis found that individuals scoring high on peer victimization had greater odds of also scoring high on CU traits relative to non-victimized children (Zych et al., 2019). A more recent longitudinal study using a sample from the United States found that CU traits at the start of 5th grade, but not co-occurring conduct problems, were associated with greater levels of peer victimization as assessed using peer nominations across 5th and 6th grade (Wagner et al., 2020). Finally, a longitudinal study from middle childhood to adolescence found that physical and relational forms of peer victimization were each associated with greater likelihood of belonging to high-stable and increasing trajectories of CU traits from middle childhood to adolescence (Fontaine et al., 2018). However, only those on the increasing CU traits trajectory showed higher levels of both forms of victimization (Fontaine et al., 2018). Notably, associations between CU traits and peer victimization have not always been consistent, (e.g., Fanti et al., 2019; Fanti & Kimonis, 2012; Fite et al., 2021), and it has been suggested that children with CU traits would be *less* likely to be victimized by their peers due to their lack of guilt and empathy (Pellegrini et al., 1999). Nevertheless, most work to date, especially when considering forms of victimization and in community samples, points to a positive association between CU traits and negative peer relations, including peer victimization, in youth.

One study directly considered the role of peer victimization in the association between irritability and CU traits (Barker & Salekin, 2012). Through an extension of the Social Failure Model (Patterson & Capaldi, 1990), Barker and Salekin (2012) suggested that higher levels of irritability may lead individuals to be victimized and rejected by peers, which would in turn promote the development of a more callous worldview along with deficits in empathy and moral reasoning (Barker & Salekin, 2012). They found that both irritability and peer victimization predicted increases in one another and in CU traits from ages 8 to 13. However, contrary to hypotheses, only indirect effects from victimization to CU traits through irritability were significant (Barker & Salekin, 2012). Despite calls for replication (Fontaine et al., 2018), this remains the sole longitudinal test of this model. Furthermore, this study did not distinguish between physical and relational forms of victimization.

The first aim of the current study (**Aim 1**) was to extend this prior work by examining physical and relational forms of peer victimization as socializing agents in irritability's longitudinal associations with CU traits from middle childhood to adolescence. It was hypothesized that children with higher levels of irritability would experience higher levels of physical and relational victimization, and these victimization experiences would desensitize children to others' needs, leading to increases in CU traits consistent with theorized effects of repeated negative social experiences per the STAR model (Waller & Wagner, 2019). This pattern of effects — irritability provokes negative interpersonal relations in response to which children ultimately develop callousness to cope — is also consistent with the broader irritability literature (Stringaris & Goodman, 2009) and proposed developmental pathways from initial emotional sensitivity (of which irritability is a component) to later CU traits (Kimonis, 2023). Therefore, this was the primary theoretical model of interest. However, the only previous study to examine the role of peer victimization in longitudinal associations between irritability and CU traits (Barker & Salekin, 2012) found evidence of a reverse pathway (i.e., victimization predicts increases in irritability, which in turn predicts increases in CU traits). Although less consistent with the STAR model, this alternative direction of effects may reflect a process by which children who are victimized by their peers become increasingly frustrated before ultimately becoming desensitized and disengaged from these experiences, reflected by increases in CU traits. Therefore, this reverse alternative model was also tested to be consistent with prior work.

Primary and alternative models were tested independently in order to provide a targeted test of direct and indirect paths of interest. Physical and relational victimization were included as separate variables in both models, and moderation by gender was considered. Specifically, although some prior work has not found a moderating role of gender or sex on the association between CU traits and forms of peer victimization (Fanti & Kimonis, 2012; Fontaine et al., 2018), theory and the larger peer victimization literature suggest that relational victimization's effects would be particularly salient for girls (Crick & Bigbee, 1998; Ettekal & Ladd,

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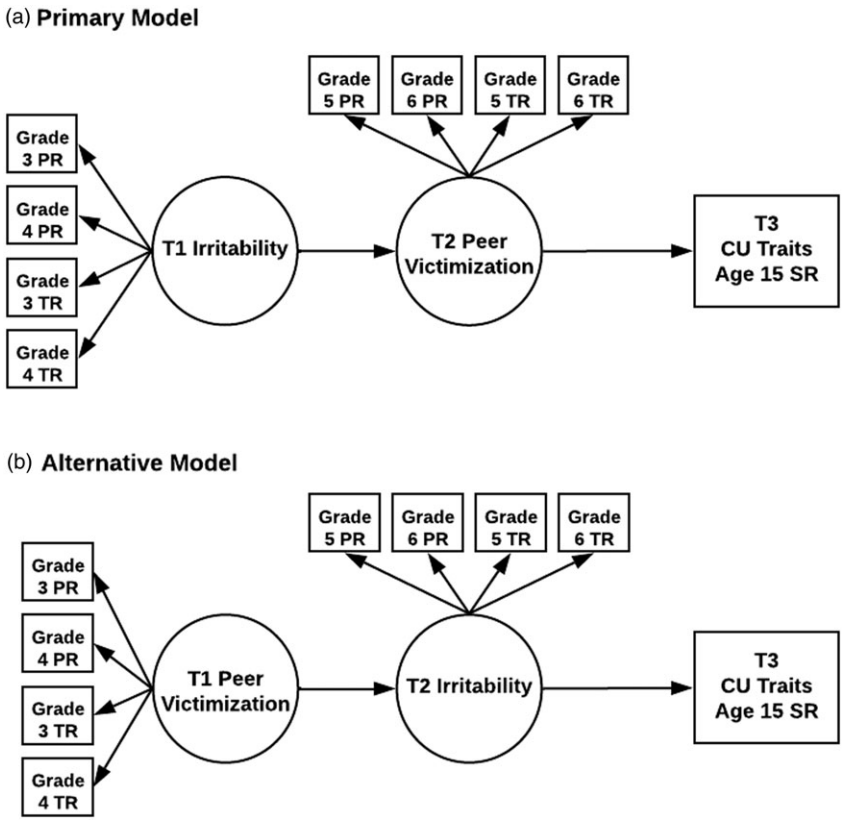

Figure 1. Aim 1 Conceptual models. *Note.* PR = parent report, TR = teacher report, SR = self-report, T1 = Time 1, T2 = Time 2, T3 = Time 3, CU = callous-unemotional; physical and relational peer victimization models were conducted separately; covariates, direct paths between T1 and T3, and residuals not depicted for ease of communication.

2020; Ostrov & Godleski, 2010; Ostrov & Kamper, 2015). Therefore, relational victimization may be more likely to lead to insensitivity to threat and affiliative reward, and therefore increases in CU traits, for girls relative to boys. As such, it was expected that relational victimization paths would be stronger for girls relative to boys. These theoretical models are depicted in Figure 1.

Moderation by HPA-axis functioning

The HPA axis is a critical component of the mammalian stress response and is crucial for maintaining homeostasis across a number of physiological systems (Lightman & Conway-Campbell, 2010). Its activity is commonly indexed through salivary assays of the hormone cortisol as assessed using basal levels, diurnal patterns, or reactivity to stress (Gunnar & Quevedo, 2007; Klimes-Dougan et al., 2001). Hypoactive HPA-axis functioning has been proposed as a biological marker of risk for the development of antisocial behavior associated with CU traits (Hawes et al., 2009). Theoretically, HPA axis hypoactivity is thought to be related to stimulation seeking or fearlessness (Blair, 1999; Coren, 1999; Quay, 1965). The STAR model in particular proposes genetic variation in the availability of corticotropin-releasing hormone as a biologically mediated individual difference factor involved in the development of low threat sensitivity (Waller & Wagner, 2019). Indeed, lower levels of HPA axis reactivity to stress in middle childhood (Stadler et al., 2011), blunted cortisol awakening response in early adolescence (von Polier et al., 2013), and lower basal salivary cortisol levels in adolescence (Loney et al., 2006) have all been associated with elevated CU traits in youth.

Importantly for the present study, the STAR model suggests that this biological predisposition toward fearlessness may interact with environmental inputs to shape the development of CU traits (Waller & Wagner, 2019). In particular, blunted HPA axis activity may partially underly failures in fear learning which ultimately produce deficits in conscience development and insensitivity to punishment cues in the environment. When faced with a high number of harsh environmental inputs (e.g., high levels of peer victimization), these individuals may be less likely to experience these as punishing and adjust their behavior accordingly. Instead, they may become further desensitized to threat and punishment cues and, through a cascade of failures in fear learning, be socialized to respond in a similarly harsh manner, reflected in increased CU traits (Waller & Wagner, 2019). Blunted cortisol diurnal slope has also been proposed as a moderating mechanism for irritability's associations with externalizing relative to internalizing problems (Kessel et al., 2021; Klein et al., 2021). It may be that the typically punishing environmental responses to irritability (e.g., peer victimization) which may promote the development of internalizing problems for many individuals, instead socialize the development of escalating externalizing problems in the context of low punishment and threat sensitivity (Kessel et al., 2021; Klein et al., 2021).

Together, this theory and evidence suggests that irritability and peer victimization may lead to the development of CU traits specifically for individuals who are also biologically predisposed to develop them, as reflected in hypoactive HPA-axis functioning. Accordingly, the second aim of the current study (**Aim 2**) tested whether HPA-axis functioning, as indexed through wake-time

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basal salivary cortisol at age 15, moderates the mediation models proposed in Aim 1. Specifically, the study tested the hypothesis (Hypothesis 2) that peer victimization's socialization effects to CU traits will be present only for those with low basal cortisol levels. Specifically, The NICHD Study on Early Child Care and Youth Development (SECCYD), the dataset for the current study, includes an assessment of basal cortisol upon awakening at age 15. Basal cortisol has historically been conceptualized as a relatively stable, trait-like indicator of HPA-axis functioning (Klimes-Dougan et al., 2001). Morning basal cortisol levels show inter-individual stability (Wüst et al., 2000), including moderate stability over three years from middle childhood to adolescence (ICC = 0.65; Kuhlman et al., 2019). However, there is also evidence for mean-level increases in basal salivary cortisol from middle childhood into adolescence (Gunnar et al., 2009), as well as potential recalibration of HPA-axis functioning to the environment across puberty (DePasquale et al., 2021). As the only available measure of cortisol in this dataset was collected simultaneously with the CU traits outcome variable, we are not able to account for these potential developmental changes that may unfold over the course of the mediation models being tested. Therefore, we consider this aim to present an initial, preliminary examination of the potential moderating role of HPA-axis activity in longitudinal associations between irritability, peer victimization, and CU traits, which will require replication and further examination.

Current study

In sum, the current study examined longitudinal associations between irritability and CU traits, and considered peer victimization as a potential socializing force in these paths, consistent with theorized effects of harsh social environments within the STAR model which has so far been limited to considering caregiving experiences (Waller & Wagner, 2019). First, **(Aim 1)** it aimed to test whether irritability may predict increases in CU traits through increases in physical and/or relational peer victimization. An alternative model whereby victimization instead predicted increases in CU traits through irritability was also tested given prior work which supported this direction of effects (Barker & Salekin, 2012). Second (**Aim 2**), it considered HPA-axis functioning as a moderator of these associations, as hypoactive HPA-axis functioning has been proposed as a biologically-based predisposing factor which may interact with harsh environmental experiences to promote the development of CU traits (Hawes et al., 2009; Waller & Wagner, 2019).[1]

These aims answer calls for additional work on the overlap between irritability and CU traits (Waschbusch et al., 2020), investigations into the role of peer victimization in the development of CU traits (Fontaine et al., 2018), and integration of biopsychological and social processes within a developmental framework in the CU trait literature (Frick et al., 2014). The aims were tested using three time points in three developmental periods - middle childhood (i.e., grades 3 – 4), pre-adolescence (i.e., grades 5 – 6) and adolescence (i.e., age 15). This maps onto the developmental periods in which peer victimization may become increasingly influential, due to increases in the relative salience of the peer relative to home environment (Sroufe, 2013). Importantly, across all aims, both physical and relational forms of victimization were considered, which is crucial for capturing girls' peer experiences (Crick & Bigbee, 1998; Ostrov & Kamper, 2015), and proposed pathways were examined for moderation by gender.

[1]The dissertation from which this paper is derived included a third aim examining moderation by co-occurring aggression. Results were considered less reliable due to model estimation difficulties. Results from this aim are presented in supplemental materials, and additional details are available from the first author upon request.

Method

Participants and procedures

The present study involves a secondary analysis of the NICHD SECCYD, which is a longitudinal study of child development that began in 1991 and followed children from birth through age 15, with data collection completed in 2007. The initial aims of the project were to examine how different child care experiences and other contextual and socialization factors were related to developmental outcomes across domains (NICHD Early Child Care Research Network [ECCRN], 2005). Associations between anger and externalizing problems are well-established within this dataset (e.g., Kim & Deater-Deckard, 2011; Runions & Keating, 2010), and prior work with the dataset have included important findings on the role of early caregiving experiences and HPA-axis functioning in the development of CU traits (e.g., Beaver et al., 2015; Fanti & Kimonis, 2017; Flexon & Encalada, 2021; Haltigan et al., 2011; Javakhishvili & Vazsonyi, 2022). Nevertheless, the current study represents a novel use of the SECCYD dataset, in that it aims to test the roles of irritability, forms of peer victimization, and HPA-axis functioning in the development of CU traits.

The NICHD SECCYD conducted data at multiple time points across four phases. The current study primarily uses data from Phase 3, which collected data when participants were in 2nd through 6th grade, and Phase 4, which followed participants from ages 13 to 15. Initially, 1,364 (48.3% female) participants were recruited from 24 hospitals across ten locations in the United States in 1991 (for details see NICHD ECCRN, 2004). At Phase 3, 1,077 participants remained enrolled (79% retention from birth); 958 participants (70% of the initial sample) were still engaged with the study in Phase 4 (Vandell et al., 2010). A total of 873 participants (91% of total Phase 4 sample) completed the cortisol collection process (Roisman et al., 2009). Participants lost to attrition by Phase 4 were more likely to be male, have lower maternal education levels, and have demonstrated lower quality parenting; there were no differences by attrition on income-to-needs ratio, race/ethnicity, or a number of additional child and parent socio-emotional variables, including externalizing problems (for details see Vandell et al., 2010).

The present study included participants with data on any of the measures of interest in Phase 3 (grades 3 – 6) or Phase 4 (ages 13 – 15). Specifically, the present study used latent variables comprised of teacher and parent reports on questionnaires from grades 3 and 4 as Time 1 (T1), from grades 5 and 6 as Time 2 (T2), and self-report and salivary cortisol at age 15 as Time 3 (T3). These time points were chosen to reflect distinct developmental periods (i.e., middle childhood, early adolescence, adolescence), and potentially different school contexts, as most U.S. children transition from elementary to middle school in grades 5 and 6. Of note, although parent report on CU behaviors at 36 months were included as a covariate, participants for which this was the only data available (i.e., did not continue to participate in Phases 3 or 4) were not included in the current sample. This resulted in a total sample of 1,103 (49.5% female) for the current study. The racial and ethnic makeup of the current sample was similar to the overall sample (77.2% non-Hispanic white, 11.5% non-Hispanic Black, 6.2% Hispanic, 5.1% Asian, Pacific-Islander, Native American, or other races/ethnicities).

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Measures

Irritability

Irritability was measured at grades 3 through 6 by parent report on the Child Behavior Checklist (CBCL) and teacher report on the Teacher Report Form from the Achenbach System of Empirically Based Assessments (ASEBA; Achenbach & Rescorla, 2001). Specifically, three items (e.g., "Stubborn, sullen, or irritable") previously shown to be valid and reliable indicators of irritability within this age range (Evans et al., 2020; Perhamus et al., 2024) were averaged at each time point within reporter. Items are rated on a 0 (*Not true*) to 2 (*Very true or often true*) scale. Both teacher report (Cronbach's αs = .78 – .83; McDonald's ωs = .79 – .84) and parent report (Cronbach's αs = .70 –.73; McDonald's ωs = .70 – .74) showed adequate internal consistency within the current study. Parent and teacher reports at the same grade were weakly but significantly correlated ($rs = .15 – .21$, $ps < .001$).

Physical and relational peer victimization

Physical and relational victimization were indexed by parent report on "My Child's Behavior with Other Children" and teacher report on the "Relationships with Peers: Part E" questionnaires at grades 3 through 6. These questionnaires are adapted from prior measures (Kochenderfer & Ladd, 1996a, 1996b; Ladd & Profilet, 1996). Parents were asked to reflect upon their children's behaviors with peers generally, and teachers were asked to reflect upon the participating child's experiences with peers as they have observed in the school setting. Three items from a 43-item scale measured relational victimization (e.g., "Peers say negative things about him/her to other children"), whereas two items measured physical victimization (e.g., "Is hit or kicked by others"). Items were scored on a 0 (*Not true*) to 2 (*Often true*) scale. Relational victimization items were averaged to create a composite at each grade for each reporter. All composites for teacher report (Cronbach's αs = .81 – .82; McDonald's ωs = .83 – .84) and parent report (Cronbach's αs = .76 – .79; McDonald's ωs = .76 – .80) demonstrated adequate internal consistency. Teacher and parent report of relational victimization within timepoint were moderately and significantly correlated ($rs = .31 – .35$, $ps < .001$). The two items measuring physical victimization were initially intended to be averaged to form composites as done with relational victimization. However, internal consistency was below acceptable levels for teacher report (Spearman-Brown $\rho s = .50 – .66$), although at marginally acceptable levels for parent report (Spearman-Brown $\rho s = .67 – .74$). Given these concerns surrounding internal consistency with the physical victimization subscale composite, physical victimization was measured at the item level. However, teacher report composites at grade 3 were used when controlling for initial levels of physical victimization.

CU traits

CU traits at age 15 were measured using self-report on the Youth Psychopathic Traits Inventory (YPI; Andershed et al., 2002). The full YPI includes 50 items across three scales assessing three dimensions of psychopathy: grandiose-manipulative, impulsive-irresponsible, and callous-unemotional. The current sample reported just on the 15-item CU scale, which is itself comprised of three subscales – callousness (e.g., "I think that crying is a sign of weakness, even if no one sees you"), unemotionality (e.g., "I usually feel calm when other people are scared"), and remorselessness (e.g., "To feel guilt and regret when you have done something wrong is a waste of time"). All items are scored on a 1 (*Does not apply at all*) to 4 (*Applies very well*) scale, with higher scores indicating higher levels of psychopathy. Main analyses for the current project used the full CU scale, scored as an average across all 15 items. This full scale showed good internal consistency in the current study (Cronbach's α = .82; McDonald's ω = .82) and has been used in prior work examining hypotheses related to the CU traits as conceptualized by the STAR model within this developmental period (Miron et al., 2020).

Early CU behaviors were assessed using caregiver report on the CBCL at age 36 months and included as a covariate. Consistent with prior work with this dataset (Willoughby et al., 2014) and others (e.g., Wagner et al., 2018), 5 items were used to assess CU behaviors (e.g., "Does not seem to feel guilty after misbehaving"), scored on a 0 (*Not* true) to 2 (*Very true or often true*) scale, and were averaged. The internal consistency of this subscale was low (Cronbach's α = .48, McDonald's ω = .47), and a review of the items indicated that none of their removal would improve reliability. Despite this low internal consistency, prior work in this dataset has demonstrated that this subscale represents a reliable factor distinguishable from other dimensions of disruptive behavior (i.e., ADHD and ODD) and predicted later levels of aggressive behavior (Willoughby et al., 2014). Furthermore, when included in a latent profile analysis, this measure of CU behaviors at 36 mos. showed evidence for stability to age 15 CU traits within the NICHD SECCYD (Fanti & Kimonis, 2017). It also shows evidence for stability in the current study, as parent-reported CU behaviors at age 36 mos. were weakly, but significantly, correlated with self-reported CU traits at age 15 ($r = .10$, $p = .003$). Therefore, given the importance of controlling for earlier CU behaviors to the current study and the evidence for validity despite low internal consistency of this scale within the current dataset, the measure was retained and used with caution in the current study.

Salivary cortisol

Salivary cortisol was collected at home during the age-15 assessment. Adolescents collected saliva using a salivette, which they placed in their mouth for three minutes on three consecutive school days upon awakening. Participants and their parents were trained on collection procedures during a home visit, and adolescents completed a "daily diary" record of the date and time of collection, awakening time, any medications taken, and the previous night's sleep quality (Roisman et al., 2009). All participants were considered eligible for saliva collection, and descriptive analyses found no significant differences in cortisol levels by medication use across 26 classes of medication, smoking, or pregnancy status (Roisman et al., 2009). After collection, participants stored samples in their freezer until they were either picked up by the study team or brought to the lab during a subsequent lab visit. Upon arrival at a research site, samples were stored in ultra-low freezers (-80°C) then shipped for assay on dry ice to Salimetrics (State College, PA). Samples were assayed in duplicate, and the average across assays was used as the cortisol sample value for each day. Daily average values were moderately correlated across days ($rs = .38 – .52$, $ps < .001$). Full details on collection, assay, and descriptive statistics are provided in Roisman et al. (2009).

Considered covariates

Family socioeconomic status (SES) calculated as income-to-needs ratio (Campbell et al., 2006, 2010; Gazelle & Spangler, 2007; Roisman et al., 2009) and child age at the grade 3 assessment were

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considered as covariates across models. In Aim 2 models, body mass index and the difference between cortisol collection time and the child's typical wake time (i.e., time since awakening) were also considered as covariates. Ultimately, variables were not substantially associated with key variables and so were not included. Details are presented in supplemental materials.

Analytic plan

Confirmatory factor analyses (CFAs) were conducted within Mplus Version 8 (Muthén & Muthén, 1998 – 2023) to test the utility of latent factors of irritability, and physical and relational peer victimization at each time point, as well as age-15 cortisol. Overall model fit was assessed using a likelihood χ^2 test, with $p > .05$ indicating close model fit. The comparative fit index (CFI) of which values greater than .95 suggest good fit and above .90 suggest acceptable fit, the standardized root mean square residual (SRMR), where values less than .08 represent adequate fit and less than .05 represent good fit (Hu & Bentler, 1999), and the root mean square error of approximation (RMSEA; Steiger, 1990), where values less than .08 represent mediocre fit and less than .06 indicate close fit (Browne & Cudeck, 1992; MacCallum et al., 1996) were also considered. Modification indices (MIs) were examined in the event that overall model fit was lower than conventional levels (Hu & Bentler, 1999).

Structural equation models (SEM) were conducted separately for the two competing Aim 1 models – one in which T2 peer victimization mediates the association between T1 irritability and T3 CU traits (Figure 1a), and a second in which T2 irritability mediated the relation between T1 peer victimization and T3 CU traits (Figure 1b). Models were conducted separately by form of peer victimization, with grade 3 levels of the alternative form (i.e., physical victimization in relational models, relational victimization in physical models) included as a covariate. Significance of indirect effects for Aim 1 models were tested using the Mplus Model Indirect test with 5000 bootstrap samples and 95% bias-corrected confidence intervals in conjunction with standardized effect estimates (Hayes & Preacher, 2010; Preacher & Kelley, 2011).

Aim 2 tested moderation of Aim 1 models by T3 cortisol levels to test whether paths from mediators to CU traits was moderated by concurrent HPA-axis functioning. Specifically, in primary models, an interaction term between victimization and cortisol was added; in alternative models, an interaction term between irritability and cortisol was added. Analyses followed the two-step process outlined by Maslowsky et al. (2015) for estimating latent variable interactions using the latent moderated structured equations method. Specifically, models were first estimated without latent interactions to provide model fit statistics, followed by models including latent variable interactions to provide a test of the significance of the interaction term and final model estimates. Significant moderation of direct effects was probed at ± 1 SD from the mean using Model Constraint (Aiken & West, 1991). Moderated mediation was tested by examining indirect effects at ±1 SD from the mean of the moderator (Hayes, 2015; Preacher et al., 2007) using Monte Carlo generated confidence intervals (Selig & Preacher, 2008).

Gender moderation was tested across all models. First, measurement invariance of CFA models was examined by comparing configural measurement models in which all paths were free across gender with metric models where factor loadings were constrained to equivalence across gender. Partial invariance was tested through the sequential use of MIs, with values > 3.84 indicating significant improvement in model fit if a path were to be freed across gender (Whittaker, 2012; Yoon & Millsap, 2007). Structural models were then run separately for each gender due to model complexity, and paths were examined for differences by gender.

Results

Preliminary analyses

Analyses were first conducted to examine patterns of missing data and attrition. Descriptive data were then obtained, and outliers were winsorized to ± 3 standard deviations from the mean (Kline, 2016). Descriptive statistics of key variables are presented in Table 1. Bivariate correlations are presented in supplemental materials. Variable distributions were within normal limits as defined by Kline (2016) with the exception of slight elevations on teacher report of physical victimization at grade 5 and grade 6. Given these elevations, maximum likelihood with robust standard errors estimation was used for models that included physical victimization. All other models were conducted with maximum likelihood estimation.

Data were then examined for systematic missingness. Data was expected to be missing at random (MAR) given that missingness was not randomly assigned within the study design, which is necessary to achieve data missing completely at random (i.e., MCAR; Baraldi & Enders, 2010). Demographic correlates of attrition were known from prior work with the dataset and are described above. Rates of missingness on study variables ranged from 7% (Grade 3 parent report measures) to 23% (Grade 6 teacher report measures). Nearly half of the sample (45.5%) had complete data, and 77% were missing 6 or fewer study variables (out of >40 total variables). Dummy coded variables were created reflecting presence or absence of any missing data on key variables at each time point, and these variables were examined for associations with child age, SES, and grade 3 levels of parent— and teacher—reported irritability and victimization. Details are presented in supplemental materials. Evidence was found for the MAR assumption and missing data was accommodated using full information maximum likelihood (FIML; Little, 2013).

Gender differences were also examined. Females showed higher levels of parent-reported irritability at grade 5 ($t[1008.42] = -2.28$, $p = .02$; Cohen's $d = -.14$) and grade 6 ($t[1013.68] = -2.48$, $p = .01$; Cohen's $d = -.16$). By contrast, males showed higher levels of teacher-reported irritability across all grades ($ts[790.61 – 955.07] = 2.13 – 3.46$, $ps < .001$ - .03; Cohen's $ds = .14 – .24$). Males showed higher levels of physical victimization ($ts[735.99 – 985.48] = 2.23 – 4.46$, $ps < .001$ - .03; Cohen's $ds = .14 – .28$) per both teachers and parents across all time points. Teachers also reported significantly higher levels of relational victimization among males in grades 4 and 6 ($ts[826.00 – 901.57] = 2.14 – 3.14$, $ps = .002 – .03$; Cohen's $ds = .14 – .22$), although there were no significant differences per parent report ($ps = .08 – .53$). There were no significant differences in parent-reported CU behaviors at age 36 months ($p = .11$), but males self-reported higher levels of CU traits at age 15 ($t[954] = 15.19$, $p < .001$; Cohen's $d = 0.98$) compared to females. Finally, females had higher average cortisol levels ($t[840.92] = -4.31$, $p < .001$; Cohen's $d = -.29$) relative to males.

CFA's were run for each latent construct (i.e., irritability, physical victimization, relational victimization, and cortisol) at each relevant time point. Models provided close or adequate fit to

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Table 1. Summary descriptive statistics

	PR Irr	TR Irr	PR RVic	TR RVic	PR PVic	TR PVic	PR CU 36 mos.	SR CU Age 15	Cortisol Age 15
M	0.33 – 0.37	0.14 – 0.18	0.19 – 0.26	0.29 – 0.31	0.29 – 0.31	0.05 – 0.07	0.27	1.95	0.36
SD	0.39 – 0.43	0.31 – 0.35	0.34 – 0.38	0.44 – 0.46	0.44 – 0.46	0.15 – 0.18	0.25	0.46	0.18
Skew	0.96 – 1.08	2.11 – 2.22	1.29 – 1.65	1.36 – 1.48	1.36 – 1.48	2.73 – 3.12	0.87	0.39	1.09
Kurtosis	0.04 – 0.24	3.50 – 3.91	0.37 – 1.53	0.84 – 1.39	0.84 – 1.39	5.20 – 8.10	0.36	−0.29	1.61
Range	0.00 – 1.68	0.00 – 1.37	0.00 – 1.46	0.00 – 2.00	0.00 – 1.12	0.00 – 0.77	0.00 – 1.06	1.00 – 3.36	0.01 – 1.12
Possible Range	0.00 – 2.00	0.00 – 2.00	0.00 – 3.00	0.00 – 3.00	0.00 – 3.00	0.00 – 3.00	0.00 – 2.00	1.00 – 4.00	–

Note. PR = parent report, TR = teacher report, SR = self-report, Irr = irritability, RVic = relational victimization, PVic = physical victimization, CU = callous-unemotional. Irritability and victimization values are presented as ranges across grades 3, 4, 5, and 6.

the data for all constructs and timepoints in the full sample and each gender, with the exception of the T2 physical victimization model which did not converge for females. Details and figures for these analyses are presented in supplemental materials.

Primary analyses

Aim 1

Initially, combined models including both physical and relational victimization latent factors in the same model were conducted. However, due to high correlation among these variables and measurement concerns (e.g., poor model fit, requiring cross-construct correlated residuals), models were divided by form. Grade 3 levels of the alternative form was included as a covariate. Results from initial combined models are presented in supplemental materials.

Aim 1 Primary Models. Standardized regression coefficients for these models are presented in Table 2. Both the relational victimization ($\chi^2[41] = 139.37$, $p < .001$; RMSEA = .05; CFI = .96; SRMR = .04) and physical victimization ($\chi^2[88] = 187.62$, $p < .001$; RMSEA = .03; CFI = .94; SRMR = .05) models provided adequate to close fit to the data. In both models, consistent with hypotheses, there were positive direct effects of irritability on relational and physical victimization and CU traits. However, there were no significant effects of victimization on CU traits. Likewise, indirect effects in the relational victimization (β = −.04, 95% CI [−.17, .03]) and physical victimization (β = .004, 95% CI [−.04, .05]) models were nonsignificant.

Models were run separately for males and females to test gender differences. Standardized regression coefficients for these models are presented in Table 2. The relational victimization models provided close fit to the data for males ($\chi^2[41] = 65.63$, $p < .001$; RMSEA = .03; CFI = .98; SRMR = .03) and adequate fit for females ($\chi^2[41] = 108.53$, $p < .001$; RMSEA = .06; CFI = .94; SRMR = .05). Consistent with the full sample model, irritability significantly predicted increases in relational victimization for both genders, although the effect was stronger for females (i.e., 95% CIs did not overlap across gender), consistent with hypotheses. However, the direct effect of irritability on CU traits was not significant for either gender. Likewise, relational victimization did not predict increases in CU traits for either gender, and indirect effects were nonsignificant for both males (β = −.04, 95% CI [−.14, .01]) and females (β = −.09, 95% CI [−3.64, .14]). For physical victimization, the models again provided close fit to the data for males [$\chi^2(88) = 116.33$, $p = .02$; RMSEA = .02; CFI = .97; SRMR = .05] and adequate fit to the data for females [$\chi^2(16) = 53.29$, $p < .001$; RMSEA = .07; CFI = .92; SRMR = .05]. For both genders and consistent with the full sample model, irritability predicted increases in physical victimization, but physical victimization did not predict increases in CU traits. Irritability directly predicted increases in CU traits for females only. Indirect effects were nonsignificant for both males (β = −.02, 95% CI [−.12, .02]) and females (β = −.006, 95% CI [−.11, .03]).

Aim 1 Alternative Models. Standardized regression coefficients for alternative models are presented in Table 3. The relational victimization model provided adequate fit to the data ($\chi^2[41] = 188.80$, $p < .001$; RMSEA = .06; CFI = .93; SRMR = .05). Relational victimization predicted increases in irritability, which marginally ($p = .08$) predicted increases in CU traits. The indirect effect was small but significant (β = .06, 95% CI [.001, .19]). The physical

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Table 2. Aim 1 primary: Standardized regression coefficients for relational and physical victimization models

	Full Sample	Males	Females
Irr ➔ Rel Vic	.44 (.09) ***	.28 (.11) **	.71 (.16) ***
Rel Vic ➔ CU	−.10 (.09)	−.13 (.10)	−.13 (.92)
Irr ➔ CU (direct)	.26 (.09) **	.15 (.10)	.37 (.88)
	Full Sample	Males	Females
Irr ➔ Phys Vic	.37 (.10) ***	.29 (.13) *	.28 (.11) *
Phys Vic ➔ CU	.01 (.06)	−.08 (.09)	−.05 (.21)
Irr ➔ CU (direct)	.21 (.07) **	.14 (.09)	.68 (.11) *

Note. Standardized regression coefficients, standard errors in parentheses. Irr = Irritability, Rel = Relational, Phys = Physical, Vic = Victimization, CU = Callous unemotional. *$p < .05$, **$p < .01$, ***$p < .001$.

Table 3. Aim 1 alternative: Standardized regression coefficients for relational and physical victimization models

	Full Sample	Males	Females
Rel Vic ➔ Irr	.42 (.10) ***	.36 (.13) **	.50 (.14) **
Irr ➔ CU	.15 (.09) $^{+}$	.07 (.14)	.22 (.11) *
Rel Vic ➔ CU (direct)	−.001 (.08)	−.03 (.10)	.06 (.12)
	Full Sample	Males	Females
Phys Vic ➔ Irr	.26 (.09) **	.33 (.13) *	.12 (.13)
Irr ➔ CU	.13 (.07) $^{+}$	.05 (.14)	.25 (.08) **
Phys Vic ➔ CU (direct)	.06 (.06)	.00 (.09)	−.05 (.08)

Note. Standardized regression coefficients, standard errors in parentheses. Irr = Irritability, Rel = Relational, Phys = Physical, Vic = Victimization, CU = Callous unemotional. $^{+}p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

victimization model also provided adequate fit to the data ($\chi^2[88] = 243.09$, $p < .001$; RMSEA = .04; CFI = .91; SRMR = .05). Similarly, physical victimization significantly predicted increases in irritability, and the effect of irritability on CU traits remained marginally significant ($p = .08$). A small but significant indirect effect (β = .03, 95% CI [.002, .11]) emerged.

Standardized regression coefficients for each gender are presented in Table 3. Relational victimization models provided close fit to the data for males ($\chi^2[41] = 80.48$, $p < .001$; RMSEA = .04; CFI = .95; SRMR = .04) and adequate fit for females ($\chi^2[41] = 103.18$, $p < .001$; RMSEA = .05; CFI = .92; SRMR = .06). Consistent with the full sample model, relational victimization predicted increases in irritability for both genders. However, irritability significantly predicted increases in CU traits specifically for females. Likewise, the indirect effect from relational victimization to CU traits through increases in irritability was significant for females (β = .11, 95% CI [.006, .41]) but not males (β = .02, 95% CI [−.08, .28]), consistent with hypotheses. Physical victimization models provided borderline adequate fit for males ($\chi^2[88] = 191.25$, $p < .001$; RMSEA = .05; CFI = .89; SRMR = .05) and adequate fit for females ($\chi^2[88] = 162.75$, $p < .001$; RMSEA = .04; CFI = .91; SRMR = .06); these models were retained with caution. Interestingly, physical victimization predicted increases in irritability for males only, whereas the effect of irritability on CU traits was significant for females only. Indirect effects were nonsignificant for both males (β = .02, 95% CI [−.09, .21]) and females (β = .03, 95% CI [−.03, .16]).

Overall, results from this aim speak to potentially reciprocal associations between irritability and peer victimization, as well as direct effects of irritability on CU traits, consistent with hypotheses. Associations with relational victimization were stronger for females, also consistent with hypotheses. Unexpectedly, indirect effects were supported for the alternative model only, such that peer victimization predicted increases in irritability, which in turn predicted increases in CU traits, although effects were small.

Aim 2

Aim 2 Primary Models. The primary physical victimization model including a main effect of cortisol on CU traits provided adequate fit to the data ($\chi^2[131] = 260.02$, $p < .001$; RMSEA = .03; CFI = .95; SRMR = .05). The main effect of T3 cortisol on T3 CU traits was nonsignificant (β = −.05, SE = .04, $p = .19$). Estimating an interaction between physical victimization and cortisol did not significantly improve model fit ($\chi^2[1] = 0.03$, $p = .85$) and its path predicting CU traits was nonsignificant (β = −.004, SE = .04, $p = .93$). When run separately for each gender (with T2 physical victimization measured using a manifest variable for females as in other models), the main effects model provided close fit to the data for males ($\chi^2[131] = 166.43$, $p = .02$; RMSEA = .02; CFI = .97; SRMR = .05) and females ($\chi^2[38] = 72.34$, $p < .001$; RMSEA = .04; CFI = .95; SRMR = .04). The main effect of cortisol remained nonsignificant for both genders (βs = .01, $ps = .83$ – .84). Likewise, estimating the interaction did not significantly improve model fit ($\chi^2[1] = 0.66$ – 0.95, $ps = .33$ – .42) and remained nonsignificant (βs = −.06 – .05, SEs = .05 – .06, $ps = .20$ – .41) for both genders.

The relational victimization measurement model demonstrated close fit to the data ($\chi^2[72] = 168.32$, $p < .001$; RMSEA = .04; CFI = .97; SRMR = .04). As in the physical model, the main effect of cortisol on CU traits was nonsignificant (β = −.05, SE = .04,

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$p = .21$). Likewise, the estimation of the relational victimization x cortisol interaction did not improve model fit ($\chi^2[1] = 0.24$, $p = .62$) and was nonsignificant ($\beta = -.02$, SE = .05, $p = .62$). The pattern was the same for the male specific model – the main effect model provided close fit to the data ($\chi^2[72] = 92.35$, $p = .05$; RMSEA = .02; CFI = .99; SRMR = .03), which was not improved by the estimation of the interaction ($\chi^2[1] = 0.22$, $p = .64$). Neither the main effect of cortisol ($\beta = -.004$, SE = .02, $p = .88$) nor its interaction with relational victimization ($\beta = .03$, SE = .06, $p = .63$) significantly predicted CU traits. For females, the main effect model also provided close fit to the data ($\chi^2[72] = 130.71$, $p < .001$; RMSEA = .04; CFI = .96; SRMR = .04) and the main effect of cortisol remained nonsignificant ($\beta = .03$, SE = .06, $p = .66$). However, unlike previous models, the estimation of the interaction significantly improved model fit [$\chi^2(1) = 4.58$, $p = .03$] and its prediction of CU traits was significant ($\beta = -.16$, SE = .08, $p = .04$). Despite the significant interaction, the simple slopes of effects of relational victimization on CU traits did not reach significance at low (i.e., −1 SD; $B = .10$, SE = .96, $p = .92$) or high (i.e., +1 SD; $B = -1.15$, SE = .96, $p = .23$) levels of cortisol. Similarly, the indirect effect was nonsignificant at low (95% CI [−1.97, 1.86]), mean (95% CI [−2.59, 1.14]), or high (95% CI [−3.36, 0.64]) levels of cortisol. Overall, this indicates that although relational victimization's effect on CU traits differs at varying levels of salivary cortisol for females, the direction and exact nature of these effects remains uncertain and requires replication.

Aim 2 Alternative Models. The alternative physical victimization model with a main effect of T3 cortisol added provided adequate fit to the data ($\chi^2[131] = 305.59$, $p < .001$; RMSEA = .04; CFI = .93; SRMR = .04). Consistent with the primary models, the direct effect of cortisol on CU traits remained nonsignificant ($\beta = -.05$, SE = .04, $p = .17$). The estimation of the interaction between irritability and cortisol did not improve model fit ($\chi^2[1] = 0.02$, $p = .88$) and did not significantly predict CU traits ($\beta = .01$, SE = .04, $p = .80$). The pattern of effects was the same when the model was examined separately by gender. The main effects model provided adequate fit to the data for males ($\chi^2[131] = 235.41$, $p < .001$; RMSEA = .04; CFI = .92; SRMR = .05) and close fit for females ($\chi^2[38] = 69.67$, $p = .001$; RMSEA = .04; CFI = .95; SRMR = .04). Of note, a manifest variable was used for T1 physical victimization for the female model due to estimation difficulties with the latent factor (i.e., nonsignificant factor loadings). Consistent with the full sample model, the main effect of cortisol was nonsignificant for both groups (βs = .002 –.008, ps = .88 – .97). The interaction did not improve model fit [$\chi^2(1) = 0.52$ – 1.46, $p = .23$ – .47) and was nonsignificant (βs = −.08 – .05, ps = .23 – .46) for both genders.

Finally, the alternative relational model including the main effect of T3 cortisol provided adequate fit ($\chi^2[72] = 219.81$, $p < .001$; RMSEA = .04; CFI = .94; SRMR = .04), and the main effect of cortisol on CU traits remained nonsignificant ($\beta = -.06$, SE = .04, $p = .14$). Estimating the interaction did not improve model fit ($\chi^2[1] = -0.03$, $p = .86$) and was not significant ($\beta = .006$, SE = .04, $p = .89$). This pattern of effects held for both genders. Main effects models demonstrated close fit for males ($\chi^2[72] = 126.15$, $p < .001$; RMSEA = .04; CFI = .96; SRMR = .04) and adequate fit for females ($\chi^2[72] = 146.53$, $p < .001$; RMSEA = .04; CFI = .94; SRMR = .05), and the main effect of cortisol on CU remained nonsignificant (βs = .01, ps = .80 – .87). The estimation of the interaction did not significantly improve model fit ($\chi^2[1] = 0.50$ – 1.88, $p = .17$ – .48) and effects were nonsignificant for both genders (βs = −.09 – .04, ps = .18 – .47).

Together, findings from Aim 2 generally did not support the hypothesized role of HPA-axis functioning in these developmental pathways to CU traits. However, the direct effect of relational victimization significantly interacted with salivary cortisol for females, although simple slopes were not significant. This suggests HPA-axis functioning may be influencing effects of relational victimization for females, although additional work is necessary.

Robustness tests

Two sets of robustness tests were conducted with Aim 1 models – 1) removing the unemotional subscale from the CU traits outcome measure, and 2) controlling for effects of harsh parenting. Details on these analyses are presented in supplemental materials. Broadly, results were considered robust to both the change in CU traits measure and the inclusion of effects of harsh parenting. However, although the magnitude of effects estimates remained virtually identical, in alternative models controlling for harsh parenting the indirect effects were no longer significant in full sample or gender-specific models. The lower tail of the confidence intervals for these effects hovered around cut-points for significance vs. non-significance in both original and these robustness test models. Therefore, interpreting effects based solely on significance vs. non-significance would overstate the amount of change seen in the models controlling for harsh parenting. Instead, as the magnitude of the indirect effects estimates are virtually identical to those seen in the original models, they are considered generally robust to the addition of harsh parenting. Changes in the confidence intervals that result in a nonsignificant interpretation underscore that these effects are quite small and require replication.

Discussion

The present study examined the role of negative peer experiences in longitudinal associations between irritability and CU traits from middle childhood to adolescence. This work aimed to extend theory and literature supporting effects of harsh social experiences in these developmental links (e.g., Kimonis, 2023; Waller & Wagner, 2019), which has focused on negative caregiving experiences to the neglect of the potential impact of peer relations. First (Aim 1), two conceptual models were tested regarding physical and relational peer victimization as socializing agents in irritability's longitudinal associations with CU traits. These included a primary model, consistent with the STAR model (Waller & Wagner, 2019), in which irritability was expected to predict increases in CU traits through increases in victimization; and a reverse alternative model consistent with prior findings (Barker & Salekin, 2012) in which victimization predicted increases in irritability, which then predicted increases in CU traits. Second (Aim 2), HPA-axis functioning, indexed using basal salivary cortisol, was considered as a moderator, with increases in CU traits expected to be present especially for those with low basal cortisol levels. Gender differences were also examined, with effects of relational victimization expected to be stronger for females. Overall, the present study provides novel evidence for a role of peer victimization in the development of CU traits and provides insights into potentially important gender differences in these developmental links.

Aim 1 Findings

First, direct effects supported hypothesized longitudinal associations between irritability and CU traits. Specifically, T1 irritability

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directly predicted increases in CU traits in the full sample in primary Aim 1 models. However, when examined separately by gender, this effect did not emerge for either group in relational models and was significant only for females in physical models. In alternative models, T2 irritability predicted increases in CU traits specifically for females in both the relational and physical models. These effects provide broad support for hypothesized positive developmental associations between irritability and CU traits from middle childhood to adolescence, consistent with prior work (e.g., Barker & Salekin, 2012; Hawes et al., 2016). This is also consistent with theory on the development of secondary CU traits, in which children present with high levels of negative emotional reactivity (including irritability), and then detach over time (i.e., develop callousness) to cope with the high levels of distress they experience in response to negative events (Karpman, 1941; Kimonis, 2023; Waller & Wagner, 2019). Despite theory highlighting potential developmental links between irritability and CU traits, there have been few empirical studies examining these pathways, and there have been recent calls for additional work in this area (Bansal et al., 2021; Waschbusch et al., 2020). The present study adds to this limited literature and provides empirical support for the importance of continued investigations examining associations between irritability and CU traits.

This study also found support for hypothesized positive associations between irritability and physical and relational victimization. Consistent with hypotheses, T1 irritability predicted increases in both physical and relational peer victimization at T2 in primary models. This is consistent with prior work which has demonstrated that children with higher levels of irritability are at risk for peer problems, including peer victimization (Barker & Salekin, 2012; Chen et al., 2021; Evans et al., 2016; Stringaris & Goodman, 2009). Both physical and relational victimization at T1 also predicted increases in irritability at T2, suggesting potential reciprocal associations between irritability and peer victimization, consistent with prior work in the same developmental period (Barker & Salekin, 2012). Importantly, effects of irritability on relational victimization were stronger for females relative to males. This was consistent with hypotheses, as well as prior work and theory suggesting that relational victimization may reflect a more salient threat and negative socialization experience for girls due to gender norms and gender-informed schemas placing greater emphasis on social status and relationships (Crick & Bigbee, 1998; Ostrov & Godleski, 2010; Ostrov & Kamper, 2015).

Support for the hypothesized socializing role of peer victimization in the development of CU traits was more mixed. Direct effects of both physical and relational victimization on CU traits were not significant in primary or alternative full sample or gender-specific Aim 1 models. Indirect effects were also not supported in primary models. However, small but significant positive indirect effects were detected for both physical and relational victimization predicting increases in CU traits through increases in irritability in alternative models. This could suggest that, although irritability also predicts increases in victimization, a pathway to the development of CU traits is characterized by harsh peer experiences first predicting increases in frustration and negative emotionality, which shifts to disengagement and callousness over time. That only this alternative indirect pathway was significant despite apparent reciprocal effects between irritability and peer victimization is consistent with the sole prior empirical study to examine longitudinal associations between irritability, peer victimization, and CU traits (Barker & Salekin, 2012). However, this finding was counter to both that prior study's and the present study's hypotheses, both of which emphasized the primary direction of effects. These alternative effects may be considered less consistent with theory presented within the STAR model (Waller & Wagner, 2019) and the development of secondary CU traits (Kimonis, 2023). These theoretical models emphasize that individual predispositions toward reactivity are shaped by harsh environmental experiences to promote the development of CU traits, rather than these experiences first prompting increases in emotional sensitivity. It may be that increases in irritability in response to peer victimization is an indicator that these experiences are salient enough to increase the emotional allostatic load for these individuals, who in time may disengage emotionally to cope. In this way, this study's findings could be considered consistent with recent theory on the importance of allostatic (over) load for the development of CU traits in emotionally sensitive individuals (Kimonis, 2023). As this is the second study which has supported this direction of effects, this warrants further investigation.

Importantly, this study suggests that associations between irritability, relational victimization, and CU traits may be stronger for girls to boys. That irritability was a stronger predictor for the development of CU traits for girls is consistent with prior work, including from middle childhood to adolescence, demonstrating positive associations between borderline personality disorder symptoms (including irritability) and CU traits among girls specifically (Kimonis, 2023). Furthermore, in gender-specific analyses the indirect effect of relational victimization on CU traits through irritability was significant for females only. This provides novel evidence that social exclusion may lead to the development of callousness through irritability for females specifically, consistent with theory that relational victimization may be particularly salient for girls (Crick & Bigbee, 1998; Ostrov & Godleski, 2010; Ostrov & Kamper, 2015). This also suggests that relational victimization may be a mechanism contributing to the over-representation of females within secondary CU trait phenotypes (Kimonis, 2023). Although prior work has suggested that environmental factors may have particularly impact on the development of CU traits for girls (Fontaine et al., 2010), studies have not yet identified specific mechanisms that may explain this difference (Kimonis, 2023), and this potential discrepancy is not accounted for in the STAR model (Waller & Wagner, 2019). Therefore, the evidence found in this study is novel and has potentially important theoretical and prevention implications.

However, it is critical to note that all significant indirect effects were small, and only just met criteria for statistical significance. Indeed, these indirect effects shifted to non-significance with the addition of harsh parenting as a covariate (although the magnitude of effects were consistent) and the physical victimization indirect effect was not significant for either gender when examined separately. Therefore, these effects require replication and further study before conclusions are drawn regarding their robustness and potential clinical significance.

Aim 2 Findings

The predicted role of HPA-axis functioning was generally not supported in Aim 2 models. Basal salivary cortisol was not correlated with CU traits at the bivariate level and did not directly predict change in CU traits in any model. Although an interaction between basal salivary cortisol and relational victimization predicted change in CU traits for females specifically, no simple slopes were significant. This makes the nature of this effect difficult

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to interpret. Nevertheless, this study provides initial evidence that the direct effects of relational victimization on CU traits may be influenced by HPA-axis functioning for females. As this may further elucidate a female-specific pathway of risk for the development of CU traits involving relational victimization (as suggested by Aim 1 models), this is an important area for future investigation.

It is also important to note that, due to the single assessment of HPA-axis functioning available within the SECCYD dataset, that this was a measure of momentary basal cortisol, and that this occurred at age 15 simultaneously with the outcome variable measurement, the present study serves as only initial and limited examination of the potential role of HPA-axis functioning in these paths. These analyses were included in the current manuscript given the proposed importance of blunted HPA-axis functioning in the development of CU traits, including that it may serve as a biologically-based, genetically mediated predisposing factor (i.e., moderation; Hawes et al., 2009; Waller & Wagner, 2019). However, change in HPA axis activity in response to environmental factors may instead play a mediating role in the development of CU traits. Despite moderate stability in basal cortisol levels, environmental and social factors can "get under the skin" to impact HPA-axis functioning (e.g., Koss & Gunnar, 2018; Vaillancourt et al., 2013). Specifically, chronic activation of the stress system through early interpersonal stressors is thought to ultimately lead to lower levels of basal cortisol (Susman, 2006). Indeed, HPA axis activity in infancy and toddlerhood may be positively associated with CU behaviors (Mills-Koonce et al., 2015; Schoorl et al., 2016; Wagner et al., 2019), reflecting an initially greater level of biological sensitivity to inputs from the environment, which would ultimately lead to greater allostatic load, resulting in hypoactivity over time (McEwen, 1998; Mills-Koonce et al., 2015). This is a major tenet of recent theoretical models of the development of CU traits for initially emotionally sensitive presentations, which emphasize the role of hypoactivity developing in response to allostatic overload (Kimonis, 2023). Furthermore, prior work has demonstrated that peer victimization may serve as a chronic interpersonal stressor significant enough to result in hypoactivity of the HPA axis (Calhoun et al., 2014; Ouellet-Mourin et al., 2011; Vaillancourt et al., 2013). Therefore, future work should examine whether HPA axis activity may function in a mediating, rather than moderating, role in associations between irritability, peer victimization, and CU traits.

In addition to this alternative role, future work could consider additional aspects of HPA-axis functioning. Many findings related to HPA axis activity and CU traits in youth samples have focused on reactivity (Stadler et al., 2011) or the cortisol awakening response (Gotisha et al., 2014; Stadler et al., 2011; von Polier et al., 2013). Prior studies on basal cortisol's associations with adolescent CU traits are limited in number and have shown mixed results (Feilhauer et al., 2013). Therefore, it may be that more dynamic aspects of HPA-axis functioning are implicated in these developmental pathways, rather than basal levels. Recently, investigators have also considered the ratio of cortisol to dehydroepiandrosterone (DHEA), rather than cortisol alone (Kimonis, 2013). DHEA is thought to aid in returning the stress response system to homeostasis, and high cortisol-to-DHEA ratios are thought to represent increased chronic stress with harmful effects on mental and physical health (Kamin & Kertes, 2017). For instance, high cortisol-to-DHEA ratios have been associated with greater maltreatment experiences and higher levels of secondary CU traits among adolescents (Kimonis, 2023).

Overall, the findings from this study as well as prior theoretical and empirical work point to the importance of considering the role of HPA-axis functioning in the development of CU traits. The present study adds to the somewhat equivocal literature surrounding the role of basal salivary cortisol. Additional work is needed in this area using a variety of methods (e.g., allostatic load, reactivity, diurnal slope, DHEA ratio) to further elucidate these potential effects.

Limitations

The current study has a number of strengths, including theory-driven hypotheses, use of a longitudinal design with multiple informants across multiple geographic areas of the U.S., a large sample providing adequate power for testing hypotheses, and the use of an SEM framework allowing for improved disaggregation of variance related to measurement error and constructs of interest. Nevertheless, these results need to be interpreted within the context of limitations.

First, given the nature of secondary data analysis, aspects of several of the measures were not ideally designed for testing the constructs of interest. Although used previously within this dataset, the measure of physical victimization was comprised of just two items, and these items did not demonstrate adequate internal consistency for use as a composite. These concerns were somewhat mitigated by the use of latent factors constructed with item-level indicators rather than with composite indicators as used for other constructs. However, composites of teacher report on physical victimization at grade 3 were used when controlling for this construct. Measurement error within this composite may have limited our ability to adequately account for earlier levels of physical victimization in primary physical models, or to account for effects of physical victimization as a covariate within relational models. In addition, restricted range for physical victimization resulted in stronger-than-expected correlations among constructs of interest, which required all models to be run as form-specific rather than combined. The measures available in this study also did not allow for a test of verbal victimization unique from physical and relational forms. This is a common limitation in peer victimization research (Ostrov & Kamper, 2015), and future work should examine the extent to which verbal forms may contribute to the development of irritability and CU traits. Similarly, the measure of caregiver report on CU behaviors at 36 months demonstrated low internal consistency and was collected substantially earlier than the grade 3 assessment which was the earliest timepoint for other constructs of interest. Importantly, prior work within this dataset has provided evidence for the discriminant and predictive validity of this subscale at this timepoint (Fanti & Kimonis, 2017; Willoughby et al., 2014). Furthermore, this measure was positively correlated (albeit weakly) with age-15 self-reported CU traits in the current study, despite the large temporal gap and change in both reporter and measure. This was also the latest measure of CU traits in the SECCYD dataset prior to the age-15 timepoint. This measure was therefore the most proximal assessment available and was considered sufficiently able to capture earlier levels of CU traits, although caution is warranted.

There was also some concern regarding restricted range given the use of a community sample. For instance, a T2 physical victimization latent factor did not converge for females within a univariate CFA due to several indicators being dichotomous, driven by a lack of endorsement of higher levels of physical victimization for females. Future work could consider including

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self- and/or peer-reports to capture experiences of victimization to which parents and teachers may not be privy (Cullerton-Sen & Crick, 2005; Holt et al., 2008; Putallaz et al., 2007). Additionally, theorized pathways to the development of CU traits may be better assessed within clinical or at-risk samples given the low prevalence of CU traits within the general population across developmental periods (Colins et al., 2021; Wakschlag et al., 2018). Future work examining the role of negative peer experiences in the development of CU traits within clinical and/or at-risk samples is needed and may better be able to capture these processes.

That cortisol was only available at age 15 within the dataset also presents conceptual challenges related to the current study's test of moderated mediation. Temporal precedence is not required for tests of moderation (Baron & Kenney, 1986), and prior work has examined later basal salivary cortisol as a moderator of earlier developmental processes (Laurent et al., 2013). Furthermore, basal cortisol, and morning basal cortisol in particular, has demonstrated moderate stability over three years from ages 11 to 14, which approximates that used in the current study (ICC = .65; Kuhlman et al., 2019). Nevertheless, basal salivary cortisol is far from perfectly stable, especially when measured at a single moment in time rather than more dynamic processes such as the diurnal curve or awakening response (Kuhlman et al., 2019; Shirtcliff et al., 2012), and unmodeled longitudinal change in basal cortisol levels across the study could have masked effects. Furthermore, it may be conceptually difficult to understand the effect of a variable at an earlier timepoint being dependent on levels of another variable measured several years later. Therefore, as described above, this aim is an initial test of the role of HPA-axis functioning in longitudinal associations between irritability, peer victimization, and CU traits. Additional research is needed to understand the potential role of HPA-axis functioning in these pathways.

Finally, although the analytical strategy employed provided robust targeted tests of paths of interest and allowed us to construct latent variables that captured the perspectives of multiple reporters across multiple years, it was limited to examining unidirectional between-person change across distinct timepoints. Hypothesized effects of negative peer experiences are described as predicting increases in CU traits over time, which may be better captured within a growth modeling framework. Furthermore, the models imply within-person change, suggesting the need to disaggregate within-person processes from mean-level and between-person change (Curran et al., 2014). Results of this study and theorized effects also suggest the presence of bidirectional associations between irritability and peer victimization, potentially within a developmental cascade, which may be better captured by modeling cross-lagged or reciprocal associations, rather than the unidirectional tests used in the current study (Curran et al., 2014; Masten & Cicchetti, 2010). These models would also allow for an explicit test of the comparative strength of indirect effects tested within the present study's primary and alternative models. Future work is needed using models that allow for modeling of growth trajectories over time (e.g., latent trajectory analysis, growth curve modeling), and/or distinguishing between- and within-person effects (e.g., latent curve models with structured residuals [LCM-SR]; Curran et al., 2014). The present study's analytic strategy was chosen given the focus on testing specifically the primary direction of effects, the limited number of measurement occasions for CU traits within the SECCYD which precluded this variable from being included within a cross-lagged model framework, and our interest in testing moderation of mediation processes by cortisol (and aggression as described within the supplemental materials). Nevertheless, we encourage this work, including within the NICHD SECCYD, which is publicly available. For instance, although tests involving paths to CU traits may be limited by the lack of measurement occasions for this construct, this dataset could be used to test potential reciprocal associations between irritability and peer victimization in more depth.

Conclusions & future directions

Despite these limitations, the present study has a number of important theoretical implications with potential clinical impact. First, this study provides novel evidence for positive developmental links between irritability, peer victimization, and CU traits within a large community sample. Prior work has largely focused on broader emotional sensitivity and negative emotionality constructs as risk factors for CU traits, and more work is needed to determine whether irritability may represent an especially significant facet of these components for the development of CU traits. Findings also speak to potential reciprocal associations between irritability and peer victimization with indirect paths to CU traits significant specifically from peer victimization through increases in irritability. This is consistent with the only known prior study directly examining associations between irritability, peer victimization, and CU traits (Barker & Salekin, 2012). Specifically, these findings speak to a pathway whereby higher levels of negative peer experiences, which first provoke increases in frustration and negative emotional reactivity, ultimately results in a detachment from social relationships (i.e., low affiliative reward) and desensitization to threat, reflected in increased CU traits. This suggests that pathways involving negative peer experiences should be integrated into developmental psychopathology models of CU traits, including the STAR model (Waller & Wagner, 2019) and models specific to secondary CU traits (Kimonis, 2023). Furthermore, findings from the present study suggest that associations between relational victimization, irritability, and CU traits may be particularly relevant for girls, whose experiences remain understudied in the CU traits literature.

Although the present study was conceptually informed by the STAR model, it did not investigate the mechanisms purported by the model to be underlying the development of CU traits – reductions in sensitivity to threat and affiliative reward (Waller & Wagner, 2019). Additional work is needed to determine the extent to which negative peer relations may impact these proposed mechanisms. Future work could also consider whether effects of peer victimization may be impacted by changes in the school context. For instance, peer victimization and aggression tend to increase around school transitions due to shifts in the social dominance hierarchy (Pellegrini & Long, 2002). Therefore, these transitions could represent periods of temporarily increased risk of peer victimization and may be particularly impactful in developmental paths to CU traits. Likewise, although the NICHD SECCYD data was collected before the rise of social media, future work could consider the online context to capture additional peer aggression and victimization experiences with potential differential or additive effects to those of in-person experiences (Kowalski et al., 2014). Finally, it is critical to note that harsh parenting experiences also predicted increases in irritability and CU traits, and the small indirect effects of peer victimization did not remain significant with the addition of this construct. It will be important for future work to determine the extent to which effects of peer victimization provide incremental information above and beyond well-established effects of harsh parenting (Kimonis, 2023;

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Waller & Wagner, 2019), and whether these socialization effects may be more impactful in different developmental periods or represent potentially cascading cumulative risk. Nevertheless, the presence of peer victimization effects even within a relatively low-risk community sample, and evidence that the magnitude of effects were broadly robust to the inclusion of harsh parenting as a covariate, suggests that peer processes may play an impactful role in the development of adolescent CU traits independent from parenting.

Clinically, current treatments for CU traits focus primarily on improving emotional skills and/or parent-child relationships (Kimonis, 2023). The present study provides initial evidence that peer functioning, including experiences of both physical and relational victimization and aggression, should be integrated into these efforts. Furthermore, relational victimization experiences may be particularly important to target for females. As the direction of indirect effects of victimization on CU traits through increases in irritability has now been demonstrated in two studies (Barker & Salekin, 2012), targeting increases in anger and frustration among victimized children may specifically aid in reducing risk for the development of CU traits. However, given the small effect sizes in the present study and reliance on a community rather than clinical sample, this study's findings require replication before clinical utility can be determined. With replication, this study's findings have the potential to inform critical intervention and prevention efforts for the development of CU traits, which would have important public health implications.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579424001846.

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References

Achenbach, T. M., & Rescorla, L. (2001). *Manual for the ASEBA school-age forms & profiles: An integrated system of multi-informant assessment*. University of Vermont, Research Center for Children, Youth, & Families.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Sage Publications, Inc.

Andershed, H., Kerr, M., Stattin, H., & Levander, S. (2002). Psychopathic traits in non-referred youths: Initial test of a new assessment tool. In E. Blaauw, & L. Sheridan (Eds.), *Psychopaths: Current international perspectives* (pp. 131–158). Elsevier.

Bansal, P. S., Goh, P. K., Eng, A. G., Elkins, A. R., Thaxton, M., Smith, T. E., & Martel, M. M. (2021). Identifying the inter-domain relations among ODD, CD, and CU traits in preschool children using network analysis. *Research On Child and Adolescent Psychopathology*, *49*(10), 1289–1301. https://doi.org/10.1007/s10802-021-00836-7

Baraldi, A. N., & Enders, C. K. (2010). An introduction to modern missing data analyses. *Journal of School Psychology*, *48*(1), 5–37. https://doi.org/10.1016/j.jsp.2009.10.001

Barker, E. D., & Salekin, R. T. (2012). Irritable oppositional defiance and callous unemotional traits: Is the association partially explained by peer victimization? *Journal of Child Psychology and Psychiatry*, *53*(11), 1167–1175. https://doi.org/10.1111/j.1469-7610.2012.02579.x

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*(6), 1173–1182. https://doi.org/10.1037/0022-3514.51.6.1173

Beaver, K. M., Hartman, S., & Belsky, J. (2015). Differential susceptibility to parental sensitivity based on early-life temperament in the prediction of adolescent affective psychopathic personality traits. *Criminal Justice and Behavior*, *42*(5), 546–565.

Blair, R. J. R. (1999). Responsiveness to distress cues in the child with psychopathic tendencies. *Personality and Individual Differences*, *27*(1), 135–145.

Brotman, M. A., Kircanski, K., Stringaris, A., Pine, D. S., & Leibenluft, E. (2017). Irritability in youths: A translational model. *The American Journal of Psychiatry*, *174*(6), 520–532. https://doi.org/10.1176/appi.ajp.2016.16070839

Browne, M. W., & Cudeck, R. (1992). Alternative ways of assessing model fit. *Sociological Methods & Research*, *21*(2), 230–258. https://doi.org/10.1177/0049124192021002005

Byrd, A. L., Hawes, S. W., Loeber, R., & Pardini, D. A. (2018). Interpersonal callousness from childhood to adolescence: Developmental trajectories and early risk factors. *Journal of Clinical Child & Adolescent Psychology*, *47*(3), 467–482. https://doi.org/10.1080/15374416.2016.1144190

Calhoun, C., Helms, D., W., S., Heilbron, N., Rudolph, K., Hastings, D., P., D., Prinstein, M., & **J** (2014). Relational victimization, friendship, and adolescents' hypothalamic–pituitary–adrenal axis responses to an in vivo social stressor. *Development and Psychopathology*, *26*(3), 605–618. https://doi.org/10.1017/S0954579414000261

Campbell, S. B., Spieker, S., Burchinal, M., Poe, M. D. & **The NICHD Early Child Care Research Network** (2006). Trajectories of aggression from toddlerhood to age 9 predict academic and social functioning through age 12: Outcomes of aggression trajectories. *Journal of Child Psychology and Psychiatry*, *47*(8), 791–800. https://doi.org/10.1111/j.1469-7610.2006.01636.x

Campbell, S. B., Spieker, S., Vandergrift, N., Belsky, J.,Burchinal, M. & **The NICHD Early Child Care Research Network** (2010). Predictors and sequelae of trajectories of physical aggression in school-age boys and girls. *Development and Psychopathology*, *22*(1), 133–150. https://doi.org/10.1017/S0954579409990319

Chen, H. W. B., Gardner, E. S., Clarkson, T., Eaton, N. R., Wiggins, J. L., Leibenluft, E., & Jarcho, J. M. (2021). Bullying perpetration and victimization in youth: Associations with irritability and anxiety. *Child Psychiatry and Human Development*, *53*(5), 1075–1082. https://doi.org/10.1007/s10578-021-01192-9

Colins, O. F., Fanti, K. A., & Andershed, H. (2021). The DSM-5 limited prosocial emotions specifier for conduct disorder: Comorbid problems, prognosis, and antecedents. *Journal of the American Academy of Child & Adolescent Psychiatry*, *60*(8), 1020–1029. https://doi.org/10.1016/j.jaac.2020.09.022

Copeland, W. E., Brotman, M. A., & Costello, E. J. (2015). Normative irritability in youth: Developmental findings from the great smoky mountain study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*(8), 635–642. https://doi.org/10.1016/j.jaac.2015.05.008

Coren, S. (1999). Arousal predisposition as a predictor of antisocial and delinquent behavior. *Personality and Individual Differences*, *27*(5), 815–820.

Craig, S. G., Goulter, N., & Moretti, M. M. (2021). A systematic review of primary and secondary callous-unemotional traits and psychopathy variants in youth. *Clinical Child and Family Psychology Review*, *24*(1), 65–91. https://doi.org/10.1007/s10567-020-00329-x

Crick, N. R., & Bigbee, M. A. (1998). Relational and overt forms of peer victimization: A multi-informant approach. *Journal of Consulting and Clinical Psychology*, *66*(2), 337–347. https://doi.org/10.1037/0022-006X.66.2.337

Crick, N. R., & Grotpeter, J. K. (1996). Children's treatment by peers: Victims of relational and overt aggression. *Development and Psychopathology*, *8*(2), 367–380. https://doi.org/10.1017/S0954579400007148

Cullerton-Sen, C., & Crick, N. R. (2005). Understanding the effects of physical and relational victimization: The utility of multiple perspectives in predicting social-emotional adjustment. *School Psychology Review*, *34*(2), 147–160. https://doi.org/10.1080/02796015.2005.12086280

Curran, P. J., Howard, A. L., Bainter, S., Lane, S. T., & McGinley, J. S. (2014). The separation of between-person and within-person components of individual change over time: A latent curve model with structured residuals. *Journal of Consulting and Clinical Psychology*, *82*(5), 879–894. https://doi.org/10.1037/a0035297

DePasquale, C. E., Herzberg, M. P., & Gunnar, M. R. (2021). The pubertal stress recalibration hypothesis: Potential neural and behavioral consequences. *Child Development Perspectives*, *15*(4), 249–256. https://doi.org/10.1111/cdep.12429

Deveney, C. M., Stoddard, J., Evans, R. L., Chavez, G., Harney, M., & Wulff, R. A. (2019). On defining irritability and its relationship to affective traits and social interpretations. *Personality and Individual Differences*, *144*, 61–67. https://doi.org/10.1016/j.paid.2019.02.031

Ettekal, I., & Ladd, G. W. (2020). Development of aggressive-victims from childhood through adolescence: Associations with emotion dysregulation, withdrawn behaviors, moral disengagement, peer rejection and friendships. *Development and Psychopathology*, *32*(1), 271–291. https://doi.org/10.1017/S0954579419000063

Evans, S. C., Bonadio, F. T., Bearman, S. K., Ugueto, A. M., Chorpita, B. F., & Weisz, J. R. (2020). Assessing the irritable and defiant dimensions of youth oppositional behavior using CBCL and YSR items. *Journal of Clinical Child & Adolescent Psychology*, *49*(6), 804–819. https://doi.org/10.1080/15374416.2019.1622119

Evans, S. C., Karlovich, A. R., Khurana, S., Edelman, A., Buza, B., Riddle, W., & López Sosa, D. (2024). Evidence base update on the assessment of irritability, anger, and aggression in youth. *Journal of Clinical Child & Adolescent Psychology*, *53*(2), 277–308. https://doi.org/10.1080/15374416.2023.2292041

Evans, S. C., Pederson, C. A., Fite, P. J., Blossom, J. B., & Cooley, J. L. (2016). Teacher-reported irritable and defiant dimensions of oppositional defiant disorder: Social, behavioral, and academic correlates. *School Mental Health*, *8*(2), 292–304. https://doi.org/10.1007/s12310-015-9163-y

Ezpeleta, L., Penelo, E., Navarro, J. B., de la Osa, N., & Trepat, E. (2022a). Co-developmental trajectories of defiant/headstrong, irritability, and prosocial emotions from preschool age to early adolescence. *Child Psychiatry & Human Development*, *53*(5), 908–918. https://doi.org/10.1007/s10578-021-01180-z

Ezpeleta, L., Penelo, E., Navarro, J. B., de la Osa, N., Trepat, E., & Wichstrøm, L. (2022b). Reciprocal relations between dimensions of Oppositional defiant problems and callous-unemotional traits. *Research on Child and Adolescent Psychopathology*. https://doi.org/10.1007/s10802-022-00910-8

Fanti, K. A., & Kimonis, E. (2017). Heterogeneity in externalizing problems at age 3: Association with age 15 biological and environmental outcomes. *Developmental Psychology*, *53*(7), 1230–1241. https://doi.org/10.1037/dev0000317

Fanti, K. A., & Kimonis, E. R. (2012). Bullying and victimization: The role of conduct problems and psychopathic traits. *Journal of Research On Adolescence*, *22*(4), 617–631. https://doi.org/10.1111/j.1532-7795.2012.00809.x

Fanti, K. A., Kokkinos, C. M., Voulgaridou, I., & Hadjicharalambous, M. (2019). Investigating the association between callous-unemotional traits with relational bullying and victimization: A cross-national study. *Social Development*, *28*(4), 854–872. https://doi.org/10.1111/sode.12381

Feilhauer, J., Cima, M., Korebrits, A., & Nicolson, N. A. (2013). Salivary cortisol and psychopathy dimensions in detained antisocial adolescents. *Psychoneuroendocrinology*, *38*(9), 1586–1595. https://doi.org/10.1016/j.psyneuen.2013.01.005

Fite, P. J., Williford, A., Griffith, R. L., & Parker, K. (2021). Peer victimization among detained youth: The impact of callous-unemotional traits. *Child & Youth Care Forum*, *50*(4), 569–585. https://doi.org/10.1007/s10566-020-09593-y

Flexon, J. L., & Encalada, T. M. (2021). Antecedents to secondary-like psychopathy in noninstitutionalized youth. *The Journal of Forensic Psychiatry & Psychology*, *32*(4), 535–559. https://doi.org/10.1080/14789949.2020.1867620

Fonagy, P., & Luyten, P. (2018). Conduct problems in youth and the RDoC approach: A developmental, evolutionary-based view. *Clinical Psychology Review*, *64*, 57–76. https://doi.org/10.1016/j.cpr.2017.08.010

Fontaine, N. M. G., Hanscombe, K. B., Berg, M. T., McCrory, E. J., & Viding, E. (2018). Trajectories of callous-unemotional traits in childhood predict different forms of peer victimization in adolescence. *Journal of Clinical Child & Adolescent Psychology*, *47*(3), 458–466. https://doi.org/10.1080/15374416.2015.1105139

Fontaine, N. M. G., Rijsdijk, F. V., McCrory, E. J. P., & Viding, E. (2010). Etiology of different developmental trajectories of callous-unemotional traits. *Journal of the American Academy of Child & Adolescent Psychiatry*, *49*(7), 656–664. https://doi.org/10.1016/j.jaac.2010.03.014

Fontaine, R. G., Fida, R., Paciello, M., Tisak, M. S., & Caprara, G. V. (2014). The mediating role of moral disengagement in the developmental course from peer rejection in adolescence to crime in early adulthood. *Psychology, Crime & Law*, *20*(1), 1–19. https://doi.org/10.1080/1068316X.2012.719622

Frick, P. J., & Morris, A. S. (2004). Temperament and developmental pathways to conduct problems. *Journal of Clinical Child & Adolescent Psychology*, *33*(1), 54–68. https://doi.org/10.1207/S15374424JCCP3301_6

Frick, P. J., Ray, J. V., Thornton, L. C., & Kahn, R. E. (2014). Annual research review: A developmental psychopathology approach to understanding callous-unemotional traits in children and adolescents with serious conduct problems. *Journal of Child Psychology and Psychiatry*, *55*(6), 532–548. https://doi.org/10.1111/jcpp.12152

Gazelle, H., & Spangler, T. (2007). Early childhood anxious solitude and subsequent peer relationships: Maternal and cognitive moderators. *Journal of Applied Developmental Psychology*, *28*(5-6), 515–535. https://doi.org/10.1016/j.appdev.2007.06.006

Gostisha, A. J., Vitacco, M. J., Dismukes, A. R., Brieman, C., Merz, J., & Shirtcliff, E. A. (2014). Beyond physiological hypoarousal: The role of life stress and callous-unemotional traits in incarcerated adolescent males. *Hormones and Behavior*, *65*(5), 469–479. https://doi.org/10.1016/j.yhbeh.2014.03.016

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*(1), 145–173. https://doi.org/10.1146/annurev.psych.58.110405.085605

Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology*, *34*(7), 953–967. https://doi.org/10.1016/j.psyneuen.2009.02.010

Haas, S. M., Becker, S. P., Epstein, J. N., & Frick, P. J. (2018). Callous-unemotional traits are uniquely associated with poorer peer functioning in school-aged children. *Journal of Abnormal Child Psychology*, *46*(4), 781–793. https://doi.org/10.1007/s10802-017-0330-5

Haltigan, J. D., Roisman, G. I., Susman, E. J., Barnett-Walker, K., Monahan, K. C. & **The National Institute of Child Health and Human Development Early Child Care Research Network** (2011). Elevated trajectories of externalizing problems are associated with lower awakening cortisol levels in midadolescence. *Developmental Psychology*, *47*(2), 472–478. https://doi.org/10.1037/a0021911

Hawes, D. J., Brennan, J., & Dadds, M. R. (2009). Cortisol, callous-unemotional traits, and pathways to antisocial behavior. *Current Opinion in Psychiatry*, *22*(4), 357–362. https://doi.org/10.1097/YCO.0b013e32832bfa6d

Hawes, S. W., Perlman, S. B., Byrd, A. L., Raine, A., Loeber, R., & Pardini, D. A. (2016). Chronic anger as a precursor to adult antisocial personality features: The moderating influence of cognitive control. *Journal of Abnormal Psychology*, *125*(1), 64–74. https://doi.org/10.1037/abn0000129

Hayes, A. F. (2015). An index and test of linear moderated mediation. *Multivariate Behavioral Research*, *50*(1), 1–22. https://doi.org/10.1080/00273171.2014.962683

Hayes, A. F., & Preacher, K. J. (2010). Quantifying and testing indirect effects in simple mediation models when the constituent paths are nonlinear. *Multivariate Behavioral Research*, *45*(4), 627–660. https://doi.org/10.1080/00273171.2010.498290

Holt, M. K., Kantor, G. K., & Finkelhor, D. (2008). Parent/child concordance about bullying involvement and family characteristics related to bullying and peer victimization. *Journal of School Violence*, *8*(1), 42–63. https://doi.org/10.1080/15388220802067813

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*(1), 1–55. https://doi.org/10.1080/10705519909540118

Javakhishvili, M., & Vazsonyi, A. T. (2022). Empathy, self-control, callous-unemotionality, and delinquency: Unique and shared developmental antecedents. *Child Psychiatry & Human Development*, *53*(2), 389–402. https://doi.org/10.1007/s10578-021-01137-2

Kamin, H. S., & Kertes, D. A. (2017). Cortisol and DHEA in development and psychopathology. *Hormones and Behavior*, *89*, 69–85. https://doi.org/10.1016/j.yhbeh.2016.11.018

Karpman, B. (1941). On the need of separating psychopathy into two distinct clinical types: The symptomatic and the idiopathic. *Journal of Criminal Psychopathology*, *3*, 112–137.

Kessel, E. M., Frost, A., Goldstein, B. L., Black, S. R., Dougherty, L. R., Carlson, G. A., & Klein, D. N. (2021). Developmental pathways from preschool irritability to multifinality in early adolescence: The role of diurnal cortisol. *Psychological Medicine*, *51*(5), 761–769. https://doi.org/10.1017/S0033291719003684

Kim, J., & Deater-Deckard, K. (2011). Dynamic changes in anger, externalizing and internalizing problems: Attention and regulation. *Journal of Child Psychology and Psychiatry*, *52*(2), 156–166. https://doi.org/10.1111/j.1469-7610.2010.02301.x

Kimonis, E. R. (2023). The emotionally sensitive child-adverse parenting experiences-allostatic (Over)Load (ESCAPE-AL) model for the development of secondary psychopathic traits. *Clinical Child and Family Psychology Review*, *26*(4), 1097–1114. https://doi.org/10.1007/s10567-023-00455-2

Klein, D. N., Dougherty, L. R., Kessel, E. M., Silver, J., & Carlson, G. A. (2021). A transdiagnostic perspective on youth irritability. *Current Directions in Psychological Science*, *30*(5), 437–443. https://doi.org/10.1177/09637214211035101

Klimes-Dougan, B., Hastings, P. D., Granger, D. A., Usher, B. A., & Zahn, C. (2001). Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. *Development and Psychopathology*, *13*(3), 695–719. https://doi.org/10.1017/S0954579401003157

Kline, R. B. (2016). *Principles and practice of structural equation modeling* (4th ed.), Guilford Publications.

Kochenderfer, B. J., & Ladd, G. W. (1996a). Peer victimization: Cause or consequence of school maladjustment? *Child Development*, *67*(4), 1305–1317. https://doi.org/10.2307/1131701

Kochenderfer, B. J., & Ladd, G. W. (1996b). Peer victimization: Manifestations and relations to school adjustment in kindergarten. *Journal of School Psychology*, *34*(3), 267–283. https://doi.org/10.1016/0022-4405(96)00015-5

Koss, K. J., & Gunnar, M. R. (2018). Early adversity, the hypothalamic-pituitary–adrenocortical axis, and child psychopathology. *Journal of Child Psychology and Psychiatry*, *59*(4), 327–346. https://doi.org/10.1111/jcpp.12784

Kowalski, R. M., Giumetti, G. W., Schroeder, A. N., & Lattanner, M. R. (2014). Bullying in the digital age: A critical review and meta-analysis of cyberbullying research among youth. *Psychological Bulletin*, *140*(4), 1073–1137. https://doi.org/10.1037/a0035618

Kuhlman, K. R., Robles, T. F., Dickenson, L., Reynolds, B., & Repetti, R. L. (2019). Stability of diurnal cortisol measures across days, weeks, and years across middle childhood and early adolescence: Exploring the role of age, pubertal development, and sex. *Psychoneuroendocrinology*, *100*, 67–74. https://doi.org/10.1016/j.psyneuen.2018.09.033

Ladd, G. W., & Profilet, S. M. (1996). The child behavior scale: A teacher-report measure of young children's aggressive, withdrawn, and prosocial behaviors. *Developmental Psychology*, *32*(6), 1008–1024. https://doi.org/10.1037/0012-1649.32.6.1008

Laurent, H. K., Leve, L. D., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Fisher, P. A., Marceau, K., Harold, G. T., & Reiss, D. (2013). Effects of parental depressive symptoms on child adjustment moderated by hypothalamic pituitary adrenal activity: Within- and between-family risk. *Child Development*, *84*(2), 528–542. https://doi.org/10.1111/j.1467-8624.2012.01859.x

Leibenluft, E., & Stoddard, J. (2013). The developmental psychopathology of irritability. *Development and Psychopathology*, *25*(4pt2), 1473–1487. https://doi.org/10.1017/S0954579413000722

Lightman, S. L., & Conway-Campbell, B. L. (2010). The crucial role of pulsatile activity of the HPA axis for continuous dynamic equilibration. *Nature Reviews Neuroscience*, *11*(10), 710–718. https://doi.org/10.1038/nrn2914

Little, T. D. (2013). *Longitudinal structural equation modeling*. Guilford Press.

Loney, B. R., Butler, M. A., Lima, E. N., Counts, C. A., & Eckel, L. A. (2006). The relation between salivary cortisol, callous-unemotional traits, and conduct problems in an adolescent non-referred sample. *Journal of Child Psychology and Psychiatry*, *47*(1), 30–36. https://doi.org/10.1111/j.1469-7610.2005.01444.x

MacCallum, R. C., Browne, M. W., & Sugawara, H. M. (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods*, *1*(2), 130–149. https://doi.org/10.1037/1082-989X.1.2.130

Malti, T., Perren, S., & Buchmann, M. (2010). Children's peer victimization, empathy, and emotional symptoms. *Child Psychiatry & Human Development*, *41*(1), 98–113. https://doi.org/10.1007/s10578-009-0155-8

Maslowsky, J., Jager, J., & Hemken, D. (2015). Estimating and interpreting latent variable interactions: A tutorial for applying the latent moderated structural equations method. *International Journal of Behavioral Development*, *39*(1), 87–96. https://doi.org/10.1177/0165025414552301

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(3), 491–495. https://doi.org/10.1017/S0954579410000222

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*(1), 33–44. https://doi.org/10.1111/j.1749-6632.1998.tb09546.x

Mills-Koonce, W. R., Wagner, N. J., Willoughby, M. T., Stifter, C., Blair, C., Granger, D. A. & **Family Life Project Key Investigators** (2015). Greater fear reactivity and psychophysiological hyperactivity among infants with later conduct problems and callous-unemotional traits. *Journal of Child Psychology and Psychiatry*, *56*(2), 147–154. https://doi.org/10.1111/jcpp.12289

Miron, C. D., Satlof-Bedrick, E., & Waller, R. (2020). Longitudinal association between callous-unemotional traits and friendship quality among adjudicated adolescents. *Journal of Adolescence*, *81*(1), 19–26. https://doi.org/10.1016/j.adolescence.2020.03.010

Muthén, L. K., & Muthén, B. O. (1998-2023). *Mplus (version 8.4 – 8.7)*, [computer software]. Los Angeles, CA: Author.

NICHD Early Child Care Research Network, (2004). Trajectories of physical aggression from toddlerhood to middle childhood: predictors, correlates, and outcomes. *Monographs of the Society for Research in Child Development*, *69*(4), vii–129. https://doi.org/10.1111/j.0037-976x.2004.00312.x.

NICHD Early Child Care Research Network 2005). *Child care and child development: Results from the NICHD study of early child care and youth development*. Guilford Press.

Ostrov, J. M., & Godleski, S. A. (2010). Toward an integrated gender-linked model of aggression subtypes in early and middle childhood. *Psychological Review*, *117*(1), 233–242. https://doi.org/10.1037/a0018070

Ostrov, J. M., & Kamper, K. E. (2015). Future directions for research on the development of relational and physical peer victimization. *Journal of Clinical Child & Adolescent Psychology*, *44*(3), 509–519. https://doi.org/10.1080/15374416.2015.1012723

Ouellet-Morin, I., Danese, A., Bowes, L., Shakoor, S., Ambler, A., Pariante, C. M., Papadopoulos, A. S., Caspi, A., Moffitt, T. E., & Arseneault, L.

https://doi.org/10.1017/S0954579424001846 Published online by Cambridge University Press

(2011). A discordant monozygotic twin design shows blunted cortisol reactivity among bullied children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(6), 574–582.e3. https://doi.org/10.1016/j.jaac.2011.02.015

Patterson, G. R., & Capaldi, D. M. (1990). A mediational model for boys' depressed mood. In J. E. Rolf, A. S. Masten, D. Cicchetti, K. H. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 141–163). Cambridge University Press.

Pellegrini, A. D., Bartini, M., & Brooks, F. (1999). School bullies, victims, and aggressive victims: Factors relating to group affiliation and victimization in early adolescence. *Journal of Educational Psychology*, *91*(2), 216–224. https://doi.org/10.1037/0022-0663.91.2.216

Pellegrini, A. D., & Long, J. D. (2002). A longitudinal study of bullying, dominance, and victimization during the transition from primary school through secondary school. *British Journal of Developmental Psychology*, *20*(2), 259–280. https://doi.org/10.1348/026151002166442

Perhamus, G. R., Ostrov, J. M., & Murray-Close, D. (2024). Aggression and irritability in middle childhood: Between- and within-person associations. *Journal of Clinical Child and Adolescent Psychology*, *53*(2), 184–198. https://doi.org/10.1080/15374416.2023.2272941

Preacher, K. J., & Kelley, K. (2011). Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychological Methods*, *16*(2), 93–115. https://doi.org/10.1037/a0022658

Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research*, *42*(1), 185–227. https://doi.org/10.1080/00273170701341316

Putallaz, M., Grimes, C. L., Foster, K. J., Kupersmidt, J. B., Coie, J. D., & Dearing, K. (2007). Overt and relational aggression and victimization: Multiple perspectives within the school setting. *Journal of School Psychology*, *45*(5), 523–547. https://doi.org/10.1016/j.jsp.2007.05.003

Quay, H. C. (1965). Psychopathic personality as pathological stimulation-seeking. *American Journal of Psychiatry*, *122*(2), 180–183.

Roisman, G. I., Susman, E., Barnett-Walker, K., Booth-LaForce, C., Owen, M. T., Belsky, J., Bradley, R. H., Houts, R., Steinberg, L. & The NICHD Early Child Care Research Network (2009). Early family and child-care antecedents of awakening cortisol levels in adolescence. *Child Development*, *80*(3), 907–920. https://doi.org/10.1111/j.1467-8624.2009.01305.x

Runions, K. C., & Keating, D. P. (2010). Anger and inhibitory control as moderators of children's hostile attributions and aggression. *Journal of Applied Developmental Psychology*, *31*(5), 370–378. https://doi.org/10.1016/j.appdev.2010.05.006

Schoorl, J., Rijn, S.van, Wied, M.de, van Goozen, S., & Swaab, H. (2016). The role of anxiety in cortisol stress response and cortisol recovery in boys with oppositional defiant disorder/conduct disorder. *Psychoneuroendocrinology*, *73*, 217–223. https://doi.org/10.1016/j.psyneuen.2016.08.007

Selig, J. P., & Preacher, K. J. (2008), Monte Carlo method for assessing mediation: An interactive tool for creating confidence intervals for indirect effects [Computer software]. Available from: https://quantpsy.org/

Shirtcliff, E. A., Allison, A. L., Armstrong, J. M., Slattery, M. J., Kalin, N. H., & Essex, M. J. (2012). Longitudinal stability and developmental properties of salivary cortisol levels and circadian rhythms from childhood to adolescence. *Developmental Psychobiology*, *54*(5), 493–502. https://doi.org/10.1002/dev.20607

Sroufe, L. A. (2013). The promise of developmental psychopathology: Past and present. *Development and Psychopathology*, *25*(4pt2), 1215–1224. https://doi.org/10.1017/S0954579413000576

Stadler, C., Kroeger, A., Weyers, P., Grasmann, D., Horschinek, M., Freitag, C., & Clement, H.-W. (2011). Cortisol reactivity in boys with attention-deficit/hyperactivity disorder and disruptive behavior problems: The impact of callous unemotional traits. *Psychiatry Research*, *187*(1), 204–209. https://doi.org/10.1016/j.psychres.2010.05.004

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research*, *25*(2), 173–180. https://doi.org/10.1207/s15327906mbr2502_4

Stringaris, A., & Goodman, R. (2009). Three dimensions of oppositionality in youth. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *50*(3), 216–223. https://doi.org/10.1111/j.1469-7610.2008.01989.x

Susman, E. J. (2006). Psychobiology of persistent antisocial behavior: Stress, early vulnerabilities and the attenuation hypothesis. *Neuroscience and Biobehavioral Reviews*, *30*(3), 376–389. https://doi.org/10.1016/j.neubiorev.2005.08.002

Vaillancourt, T., Hymel, S., & McDougall, P. (2013). The biological underpinnings of peer victimization: Understanding why and how the effects of bullying can last a lifetime. *Theory Into Practice*, *52*(4), 241–248. https://doi.org/10.1080/00405841.2013.829726

Vandell, D. L., Belsky, J., Burchinal, M., Steinberg, L., Vandergrift, N., & **NICHD Early Child Care Network** (2010). Do effects of early child care extend to age 15 years? Results from the NICHD study of early child care and youth development. *Child Development*, *81*(3), 737–756. https://doi.org/10.1111/j.1467-8624.2010.01431.x

von Polier, G. G., Herpertz-Dahlmann, B., Konrad, K., Wiesler, K., Rieke, J., Heinzel-Gutenbrunner, M., Bachmann, C. J., & Vloet, T. D. (2013). Reduced cortisol in boys with early-onset conduct disorder and callous-unemotional traits. *BioMed Research International*, *2013*, 1–9. https://doi.org/10.1155/2013/349530

Wagner, N. J., Bowker, J. C., & Rubin, K. H. (2020). Associations between callous-unemotional traits and peer-rated social-behavioral outcomes in elementary and middle school. *Journal of Abnormal Child Psychology*, *48*(6), 757–769. https://doi.org/10.1007/s10802-020-00636-5

Wagner, N. J., Hastings, P. D., & Rubin, K. H. (2018). Callous-unemotional traits and autonomic functioning in toddlerhood interact to predict externalizing behaviors in preschool. *Journal of Abnormal Child Psychology*, *46*(7), 1439–1450. https://doi.org/10.1007/s10802-017-0374-6

Wagner, N. J., Mills-Koonce, W. R., Willoughby, M. T., Cox, M. J., & **Investigators, F. L. P. K** (2019). Parenting and cortisol in infancy interactively predict conduct problems and callous-unemotional behaviors in childhood. *Child Development*, *90*(1), 279–297. https://doi.org/10.1111/cdev.12900

Wakschlag, L. S., Perlman, S. B., Blair, R. J., Leibenluft, E., Briggs-Gowan, M. J., & Pine, D. S. (2018). The neurodevelopmental basis of early childhood disruptive behavior: Irritable and callous phenotypes as exemplars. *The American Journal of Psychiatry*, *175*(2), 114–130. https://doi.org/10.1176/appi.ajp.2017.17010045

Waller, R., & Hyde, L. W. (2018). Callous-unemotional behaviors in early childhood: The development of empathy and prosociality gone awry. *Current Opinion in Psychology*, *20*, 11–16. https://doi.org/10.1016/j.copsyc.2017.07.037

Waller, R., Hyde, L. W., Baskin-Sommers, A. R., & Olson, S. L. (2017). Interactions between callous unemotional behaviors and executive function in early childhood predict later aggression and lower peer-liking in late-childhood. *Journal of Abnormal Child Psychology*, *45*(3), 597–609. https://doi.org/10.1007/s10802-016-0184-2

Waller, R., & Wagner, N. (2019). The sensitivity to threat and affiliative reward (STAR) model and the development of callous-unemotional traits. *Neuroscience & Biobehavioral Reviews*, *107*, 656–671. https://doi.org/10.1016/j.neubiorev.2019.10.005

Waschbusch, D. A., Baweja, R., Babinski, D. E., Mayes, S. D., & Waxmonsky, J. G. (2020). Irritability and limited prosocial emotions/callous-unemotional traits in elementary-school-age children. *Behavior Therapy*, *51*(2), 223–237. https://doi.org/10.1016/j.beth.2019.06.007

Whittaker, T. A. (2012). Using the modification index and standardized expected parameter change for model modification. *The Journal of Experimental Education*, *80*(1), 26–44. https://doi.org/10.1080/00220973.2010.531299

Willoughby, M. T., Mills-Koonce, W. R., Gottfredson, N. C., & Wagner, N. J. (2014). Measuring callous unemotional behaviors in early childhood: Factor structure and the prediction of stable aggression in middle childhood. *Journal of Psychopathology and Behavioral Assessment*, *36*(1), 30–42. https://doi.org/10.1007/s10862-013-9379-9

Wüst, S., Federenko, I., Hellhammer, D. H., & Kirschbaum, C. (2000). Genetic factors, perceived chronic stress, and the free cortisol response to

https://doi.org/10.1017/S0954579424001846 Published online by Cambridge University Press

awakening. *Psychoneuroendocrinology*, *25*(7), 707–720. https://doi.org/10.1016/S0306-4530(00)00021-4

Yildirim, B. O., & Derksen, J. J. L. (2015). Clarifying the heterogeneity in psychopathic samples: Towards a new continuum of primary and secondary psychopathy. *Aggression and Violent Behavior*, *24*, 9–41. https://doi.org/10.1016/j.avb.2015.05.001

Yoon, M., & Millsap, R. E. (2007). Detecting violations of factorial invariance using data-based specification searches: A Monte Carlo study. *Structural Equation Modeling: A Multidisciplinary Journal*, *14*(3), 435–463.

Zhang, R., Bashford-Largo, J., Lukoff, J., Elowsky, J., Carollo, E., Schwartz, A., Dobbertin, M., Bajaj, S., Blair, K. S., Leibenluft, E., & Blair, R. J. R. (2021). Callous-unemotional traits moderate the relationship between irritability and threatening responding. *Frontiers in Psychiatry*, *12*, 617052. https://doi.org/10.3389/fpsyt.2021.617052

Zych, I., Ttofi, M. M., & Farrington, D. P. (2019). Empathy and callous-unemotional traits in different bullying roles: A systematic review and meta-analysis. *Trauma, Violence, & Abuse*, *20*(1), 3–21. https://doi.org/10.1177/1524838016683456

https://doi.org/10.1017/S0954579424001846 Published online by Cambridge University Press