

PSYCHOPHYSIOLOGICAL CORRELATES OF PRIMARY AND  
TRAUMA-RELATED ACQUIRED CALLOUSNESS  
IN A SAMPLE OF DETAINED YOUTH

by

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## ABSTRACT

This study investigated psychophysiological responding comparing youth with primary, acquired, and lower callous-unemotional (CU) traits in a sample of 361 detained adolescents (265 boys, 96 girls). Mixture modeling using posttraumatic stress symptoms (PTSS) to delineate groups resulted in two groups of youth high in CU traits that were consistent with primary and secondary, or acquired, CU variants. Compared to youth classified in the primary group, youth classified as acquired-CU self-reported higher levels of PTSS, trauma exposure, anxiety, and emotion dysregulation, consistent with previous studies. Psychophysiological responses, specifically electrodermal activity (EDA) and respiratory sinus arrhythmia (RSA), were measured during baseline, in response to a video task, and during recovery. Results of multilevel models indicated no differences between the primary- and acquired-CU youth in RSA, although the acquired CU group evidenced a less steep recovery slope in EDA. The results of the current study have implications for our understanding of the pathways underlying the development of CU traits as well as for informing interventions with youth with these characteristics. Future directions for research on the development of CU traits are discussed.

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## INTRODUCTION

Recent research on the development of delinquency has focused on the role of callous-unemotional (CU) traits, a construct related to adult psychopathy that is thought to characterize a subgroup of juvenile offenders with the most stable, severe, and aggressive trajectories (Frick & White, 2008). CU is defined by low levels of empathy and remorse, lack of response to punishment, and deficits in emotion processing (Frick & Marsee, 2006). In a testament to the wealth of research that has substantiated differences between youth who are high versus low in CU traits, a specifier has been added to the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013)* in order to distinguish CU as a subtype of conduct disorder, and growing attention has been placed on the need to develop better strategies for identifying and intervening with these youth.

Emerging theory suggests that there may, in fact, be two groups of youth high in CU traits who arrive at the same outcome through different pathways. According to psychopathy theorists (e.g., Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Cleckley, 1941), primary callousness is characterized by a genetically-based deficit in emotion processing that results in a deficit of anxiety. The lack of responsiveness to others' negative emotional cues demonstrated by individuals with classic psychopathy or CU is believed to contribute to diminished sensitivity to others' distress and deficits in the experience of self-conscious emotions such as guilt, remorse, and empathy (Blair, 1995;



1999). In contrast, the concept of “secondary psychopathy” (Karpman, 1941) or “acquired callousness” (Kerig & Becker, 2010) proposes that CU also might arise through the result of environmental factors. Porter (1996) further developed this model, positing that individuals who have experienced trauma may enact a learned emotional detachment as a method of self-protection, resulting in a callous presentation. Porter theorized that, in contrast to the emotional deficits inherent in primary-CU, individuals with acquired-CU traits have the capacity for a full range of emotions but their responsiveness to others is inhibited by attempts to avoid experiencing their own posttraumatic reactions. Similarly, Ford, Chapman, Mack, and Pearson (2006) proposed that youth who have been chronically victimized may develop a tough façade of defiance and callousness as a form of “survival coping.” Ford’s notion has been confirmed empirically, with numerous studies demonstrating that youth typologized as secondary-CU report higher levels of trauma exposure (Tatar, Cauffman, Kimonis, & Skeem, 2012; Vaughn, Edens, Howard, & Smith, 2009), posttraumatic stress symptoms (PTSS) (Bennett & Kerig, 2014; Krischer & Sevecke, 2008; Sink & Kerig, 2011; Tatar et al., 2012), and emotion dysregulation (Bennett & Kerig, 2014; Gill & Stickle, 2015) in comparison to low-anxious youth typologized as primary-CU or youth from normative samples, suggesting that youth with acquired-CU may be better characterized as “callous and emotional” rather than “callous unemotional” (Gill & Stickle, 2015).

Traumatized youth, including those likely to be classified as acquired-CU, are not rare in the juvenile justice (JJ) population. Teplin, Abram, McClelland, Dulcan, and Mericle (2002) found that nearly 70% of boys and 75% of girls in the JJ system met diagnostic criteria for at least one psychiatric disorder, and posttraumatic stress disorder

(PTSD) is among the most prevalent psychiatric disorders among youth in the JJ system. Similarly, Abram and colleagues (2004) found that over 92% of youth in a juvenile detention sample had experienced at least one traumatic event, with youth reporting an average of 14 distinct traumas, and as many as 50% of detained girls and 30% of detained boys meeting full criteria for PTSD (see Kerig & Becker, 2012 for a review). Further, PTSD symptoms are associated with a greater likelihood of recidivism (Becker, Kerig, Lim, & Ezechukwu, 2012; Sadeh & McNiel, 2014), and thus these symptoms likely maintain youths' involvement in the JJ system. Therefore, traumatized youth—which includes the subset with acquired-CU traits—represent a group of youth at high risk for both ongoing mental health problems and persistent offending.

Despite the critical need to identify and intervene with traumatized youth who have acquired-CU traits, little research to date has elucidated how these youth might differ from primary-CU youth underneath their mask of callousness. Psychophysiological research allows for the study of psychological reactions that cannot be gleaned from behavioral observations alone, as well as those that are not consciously processed, and thus unavailable to self-report. The inclusion of physiological measures in studies of primary- and acquired-CU youth promises to help us to determine whether these two subtypes represent two routes to the same destination, or whether they are different phenomena at their core. To this end, the present study examined psychophysiological reactions among youth typologized as primary-, acquired-, and lower-CU.

#### Differentiating Primary- and Acquired-CU

According to theory, the experience of anxiety is antithetical to the concept of psychopathy (Cleckley, 1941), and self-reported trait anxiety has been the variable most

commonly identified as the differentiating feature between high- and low-CU groups to date (e.g., Krischer & Sevecke, 2008; Lee, Salekin, & Iselin, 2010; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). However, previous research has determined that not all high-CU youth are low in anxiety (Dolan & Rennie, 2007), and most studies have failed to distinguish between primary- and acquired-CU variants. Studies that have differentiated between primary- and acquired-CU variants have generally used anxiety as the key differentiator, with the expectation that youth high in CU traits and high in anxiety are of the acquired variant. However, empirical justification for using anxiety as the sole basis for differentiating these groups is not well-established. For example, Skeem and colleagues (2003) reviewed the literature and concluded that there is little compelling evidence that primary and secondary variants of either children or adults reliably differ on measures of anxiety, and a number of studies comparing anxiety levels among these variants have found null results (e.g., Schmitt & Newman, 1999).

Given the mixed empirical support for identifying subgroups on the basis of anxiety, a better strategy might be to differentiate groups using tenets central to the theory of acquired-CU. Porter's (1996) theory of secondary psychopathy and the work of Ford and colleagues (2006) suggest that youth who develop PTSS in response to trauma are at heightened risk to develop callousness and engage in delinquency as a result. Research to date using the traditional differentiator of anxiety confirms that detained youth with secondary-CU traits do exhibit higher levels of PTSS than do youth with primary traits or nondetained comparison samples (Krischer & Sevecke, 2008; Sink & Kerig, 2011; Tatar et al., 2012). Bennett and Kerig (2014) recently conducted a study that differentiated primary- and acquired-CU youth on the basis of PTSS. Differentiating

youth on this basis resulted in groupings that were in agreement with previous research using the traditional differentiator of anxiety, while also being more consistent with theory in that the acquired-CU group evidenced higher levels of anxiety, higher levels of trauma exposure, and more difficulties with emotion regulation. Therefore, the current study utilized this theoretically derived and empirically supported method for identifying primary- and acquired-CU variants on the basis of PTSS.

### The Contribution of Physiological Measurements to Understanding Secondary Callousness

Most studies distinguishing between primary and acquired psychopathy to date have utilized self-report or observer report alone. This methodology is inherently limited, given that psychopathy is associated with a tendency toward deceit and manipulation of others (Cleckley, 1941). Another potential limitation associated with reliance on self-report is that individuals with secondary-CU traits may mask their distress through emotional detachment, and thus may self-report inaccurately (Kalisch et al., 2005). Youth with acquired-CU traits in particular have difficulty identifying and labeling their own emotional states, which also may hinder accurate self-reporting (Bennett & Kerig, 2014). Use of psychophysiological measurements, which are largely unsusceptible to impression management, provides the potential to determine whether or not acquired-CU youth are truly “callous” under the surface.

The psychophysiological measurements that may be most relevant to understanding acquired-CU individuals involve the autonomic nervous system (ANS), comprised of the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS). The ANS is an instrumental component of the stress response system, and

therefore, is directly relevant to understanding individuals who have experienced trauma and posttraumatic reactions. When the “fight or flight” response is initiated, one of the tasks carried out by the SNS is the activation of the eccrine sweat glands, which has the evolutionary purpose of increasing palmar grip. One of the most common and noninvasive measures of SNS activity is electrodermal activity (EDA), an index of the activity of the eccrine sweat glands (Dawson, Schell, & Fillion, 2007). EDA is considered to be a marker of arousal and emotional reactivity (Dawson et al., 2007; Fowles, 1993) which, in the context of negative emotionality, would indicate an anxious or overall negatively aroused state. EDA also may indicate the extent to which self-control resources have been allocated, with higher EDA indicating greater allocation of emotional control resources (Crider, 2008; Sheppes, Catran, & Meiran, 2009; Wegner & Gold, 1995). However, EDA is also susceptible to becoming attenuated with repeated exposure to negative or strong emotions across many years (e.g., Danese & McEwen, 2012). Thus, this measure may be especially relevant to understanding youth with acquired-CU traits. These youths’ EDA may be elevated due to conscious attempts to convey a “mask” of callousness despite internal anxiety and self-conscious emotions during emotional stress, or these youth might demonstrate attenuated EDA as a result of chronic trauma exposure, management of posttraumatic reactions, and negative emotionality.

In turn, respiratory sinus arrhythmia (RSA), the most common and noninvasive measure of PNS activity, is an index of heart rate (HR) fluctuations across the respiration cycle, and represents the influence of the vagus nerve on the sinoatrial node and nucleus ambiguus (Berntson, Quigley, & Lozano, 2007; Frazier, Strauss, & Steinhauer, 2004). In

times of stress, when experiencing a fight or flight response, mammals react with attentional engagement, which requires activation of the vagus nerve to inhibit HR, or the individual opts to flee the situation, which involves vagal withdrawal (decreases in RSA) to facilitate large sympathetically-driven increases in HR by reducing the inhibitory effects of the vagus nerve (see Beauchaine, 2001). The influence of the vagus nerve varies across the respiratory cycle, and the extent of the variability predisposes an individual's readiness to react in situations. Through fibers that receive information into the central nervous system and fibers that send impulses to limbs and organs, the vagus nerve communicates with the brain continuously to regulate cardiac functioning (e.g., Porges et al., 1996). Lower RSA is found among individuals with a variety of both internalizing and externalizing disorders and is indicative of emotional lability, whereas higher RSA indicates greater variability in HR during the respiratory cycle and, under certain conditions with an emotion-laden stimulus, is associated with greater emotion regulation (e.g., Appelhans & Luecken, 2006; Beauchaine, 2001). Individuals' baseline RSA measurement is thought to represent their capability of coping with environmental stress, where higher baseline RSA indicates that an organism is able to respond more flexibly to the environment, and is associated with lower levels of internalizing psychopathology, for example, in studies of youth with a history of child abuse (McLaughlin, Rith-Najarian, Dirks, & Sheridan, 2015). A decrease in RSA from baseline during an emotionally evocative stressor task, which indicates allocation of resources toward addressing the stressor, is considered to be adaptive. However, patterns of low resting RSA and especially large decreases in RSA are considered to be biomarkers of emotional lability and are associated with a variety of internalizing and externalizing

disorders (see Beauchaine, 2015, for a review). Additionally, individuals with comorbid internalizing and externalizing symptoms are more likely to have larger reductions in RSA during an emotionally evocative lab task (Calkins, Graziano, & Keane, 2007; Pang & Beauchaine, 2013). Consequently, children with higher baseline RSA and moderate RSA reactivity to a stressor, a more adaptive response profile, tend to be protected either partially or fully from the adverse effects associated with many negative life events (as described in Zisner & Beauchaine, in press).

### Psychophysiological Correlates of PTSD

Given that the construct of acquired callousness involves both CU traits and PTSS, physiological response patterns of individuals with PTSD can inform hypotheses about how individuals with acquired-CU traits may present physiologically. The diagnostic criteria for PTSD inherently involve physiological activity, given that the arousal and intrusion clusters each contain symptoms assessing autonomic dysregulation (Blechert, Michael, Grossman, Lajitman, & Wilhelm, 2007; Orr, Metzger, Miller, & Kaloupek, 2004). Therefore, it is likely that youth with higher levels of PTSD symptoms differ physiologically from youth with fewer symptoms of PTSD.

Overall, the literature on psychophysiological correlates of PTSD in adults has provided inconsistent results. One review (Southwick, Krystal, Johnson, & Charney, 1998) concluded that adults with PTSD demonstrate higher basal SNS activity than nontraumatized controls or traumatized controls without PTSD, although results are mixed. Similarly, some scholars have suggested that weaker PNS activity is observed among individuals with PTSD (see Orr et al., 2004, for a review), whereas others have suggested that PNS responding does not differ between individuals who meet criteria for

PTSD and traumatized controls, and therefore differences may be a function of trauma exposure rather than PTSS (Sahar, Shalev, & Porges, 2001). However, an innovative study of male twins, all of whom were trauma-exposed combat veterans, found that men with PTSD had lower PNS activity (measured by heart rate variability) than their trauma-exposed brothers without PTSD (Shah et al., 2013). The authors found that remitted PTSD was not associated with autonomic dysregulation, suggesting that levels of PTSS at the time of physiological measurement are important to understanding psychophysiological response profiles above and beyond any changes in reactivity associated with trauma exposure alone. Therefore, it is likely that youth who have current PTSS, such as youth in the acquired-CU group, may present as more physiologically dysregulated than youth in the primary-CU or comparison groups, even those who may have experienced trauma but who do not have current PTSS. Further, according to a meta-analysis, although individuals with PTSD tend to exhibit elevated psychophysiological activity, especially at rest and as measured by greater increases in heart rate during a stressor task, the differences in effect sizes are much larger when comparing individuals with PTSD to those without a history of trauma, whereas effect sizes are less strong when comparing those with PTSD to trauma-exposed individuals without PTSD (Pole, 2007). Effect sizes are even larger when the PTSD group has chronic symptoms lasting longer than 12 years (Buckley & Kaloupek, 2001). In summary, the existing literature suggests that the acquired-CU group is likely to present as the most dysregulated physiologically based on their history of trauma exposure and current PTSS, as compared to the primary-CU group, although the magnitude of the expected difference is unclear given that trauma exposure itself is also associated with



some degree of physiological dysregulation. These reviews and meta-analyses also highlight a number of limitations in the extant research, including the need for studies that utilize a wider range of participants beyond male veteran samples, as well as research that parses out PNS and SNS influences. Unfortunately, the state of our current knowledge about differences in psychophysiological profiles between trauma-exposed individuals with and without PTSS is incomplete, especially with regard to children and adolescents.

One important consideration regarding the psychophysiological profiles of individuals with acquired-CU involves the theory as to how callousness emerges across development. The theory underlying acquired-CU emphasizes that attempts to disengage from one's own negative emotional states is a key mechanism in the acquisition of callousness (Ford et al., 2006; Karpman, 1941; Porter, 1996). This theory has also found some support in the empirical literature, with self-reported emotional numbing acting as a mechanism linking CU traits to trauma exposure (Kerig, Bennett, Thompson, & Becker, 2012). Further, the use of emotional disengagement strategies may play an important role in the transition from physiological hyperreactivity to later hyporeactivity (Nugent, Christopher, & Delahanty, 2006), suggesting that emotional numbing may be manifest physiologically as well. Consequently, there is reason to believe that the acquired-CU subgroup of youth may not only self-report as callous but may also demonstrate distinctive psychophysiological differences as compared to youth who do not evidence the same patterns of emotional numbness. Despite the evidence suggesting that there is a developmental process underlying posttraumatic acquired callousness, research on the physiological processes involved is limited. Therefore, the goal of the present study was

to examine differences in psychophysiological responding between primary- and acquired-CU groups of detained youth.

### Psychophysiological Correlates of CU Traits

Both theory and research support the hypothesis that there is a connection between trauma exposure and the development of CU traits. However, little is known about the biological underpinnings of this pathway, and our understanding of the psychophysiological manifestations of CU traits is also limited. There are several theories offering explanations for the development of aggression, including the underarousal theory of aggression (Raine, 2002; van Goozen, Fairchild, Snoek, & Harold, 2007) and overarousal theories (e.g., Keller & El-Sheikh, 2009; Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2009; van Goozen et al., 1998), which have each found support in the literature given that aggression has been associated with both physiological hypo- and hyperreactivity (Rudolph, Troop-Gordon, & Granger, 2010). Although this literature is mixed, there is also compelling evidence that CU is associated with an entirely different pattern of responding than aggression or other types of externalizing disorders, and understanding the psychophysiological presentation of individuals with CU traits is integral to identifying potential differences between those of the primary- and acquired-CU variants.

A number of reviews have focused attention specifically on psychopathy and CU traits. Lorber (2004) found different physiological correlates of CU as compared to more general conduct problems, noting that higher levels of psychopathy were typically associated with lower SNS reactivity. However, conclusions about PNS responding and CU traits are less clear. Hinnant and El-Sheikh (2013) proposed that low baseline RSA

combined with low RSA reactivity may be related to both emotional disengagement and CU traits, whereas low baseline RSA combined with high RSA reactivity may be related to emotion dysregulation, hypervigilance, and anxiety. However, studies to date have not proposed a psychophysiological pattern for individuals who are high in both CU traits and emotion dysregulation. Because lower RSA is associated with poorer emotion regulation and greater impulsivity, and youth classified as acquired-CU demonstrate poorer self-reported emotion regulation capabilities than primary-CU youth or lower-CU controls (Bennett & Kerig, 2014), primary- and acquired-CU youth may differ in RSA. Therefore, it is important for research to examine differences in both sympathetic and parasympathetic nervous system differences between youth with primary- versus acquired-CU traits, and the present study addresses this limitation in the extant research with the first known investigation of RSA among variants of high-CU youth.

Although the literature on the psychophysiological patterns associated with CU traits continues to grow, it has a number of clear limitations and areas requiring attention from researchers. Youth with CU traits, who demonstrate a specific constellation of behavioral and affective characteristics, make up only a subset of youth with conduct problems and youth involved in the JJ system. Unfortunately, little research has examined individuals high in CU traits independently from broader externalizing or antisocial behavior. A further limitation of the extant research on CU traits is that the majority of studies utilize normative populations, for example, college students, rather than detained samples that presumably would include individuals at the extreme end of the continuum who are most likely to exhibit pathology (Vasey, Kotov, Frick, & Loney, 2005). This discrepancy may be even more pronounced in studies of youth. For example, in a review,

Frick and White (2008) noted that only 5 of 27 studies on correlates of CU traits included adjudicated youth, although there has been some increase in focus on this population since Frick and White published their work. Therefore, research on JJ-involved youth, who are more likely to exhibit pathological levels of CU than other segments of the population, is essential to understanding the role of psychophysiological response patterns.

#### Studies Distinguishing Between Primary- and Secondary-CU

Few studies have sought to bridge the extant literatures on the psychophysiological underpinnings of CU and PTSD that would be relevant to understanding primary and acquired groups of high-CU individuals, as the majority of research to date does not distinguish between high-CU variants. Hare (1968) was among the first to call for research differentiating primary and secondary variants of psychopathy. He made the argument that previous studies measuring EDA among adults with psychopathy did not differentiate between subtypes, and therefore, the high-CU group could not be considered “pure.” He conducted the first study comparing subgroups, typologizing 51 maximum-security inmates as primary, secondary, or nonpsychopathic and measuring physiological responding. He found no significant differences in HR across groups, although he concluded that the primary group may be underreactive compared to the secondary group and controls. Despite this early foray into physiological research comparing groups, very few studies since have heeded Hare’s advice to distinguish between variants. Kimonis, Frick, Cauffman, Goldweber, & Skeem (2012) differentiated between primary- and secondary-CU subgroups of juvenile offenders ages 14 to 17 on the basis of trait anxiety. They found that the primary group was less engaged

by emotionally distressing pictures than the secondary group as measured by an autonomic attention task. However, neither variant of high-CU youth processed stimuli significantly differently from lower-CU controls. Gostisha and colleagues (2014) identified a distinct pattern in diurnal cortisol levels characterized by steeper diurnal rhythms for youth high in both CU traits and with a history of prior stress exposure as compared to one or the other, suggesting that youth with higher trauma exposure and CU traits may be a distinct group. A handful of other studies have differentiated between primary and secondary psychopathy on the basis of anxiety, although these studies have neglected to include between-variant comparisons. Zeier, Maxwell, and Newman (2009) categorized Caucasian inmates into high and low psychopathy groups but focused their analyses on a comparison of the primary psychopathy (high psychopathy, low anxiety) group versus the remainder of the sample, concluding that the primary psychopathy group was less sensitive to contextual information during an attention task, which may place them at risk for missing environmental cues that are important to self-regulation. Another study found that high levels of anxiety, regardless of psychopathic traits, was associated with larger and more frequent skin conductance responses in response to punishment among Caucasian inmates (Arnett, Howland, Smith, & Newman, 1993). As this review suggests, the literature on physiological differences between primary- and acquired-CU is inherently limited by the types of methods used, infrequent comparisons between variants, and the more common focus on comparing primary-CU groups to low-CU with little attention to the acquired variant.

Similar limitations are evident in the literature on adolescents with CU traits. For example, one heavily cited study indicated that adolescents with psychopathic traits

report reduced physiological responses to fear (Marsh et al., 2011). However, this study utilized only 18 participants and included only self-reported symptoms of sympathetic and parasympathetic reactions rather than actually measuring those reactions physiologically. Not only did the authors neglect to separately analyze the variants of high-CU youth, but they also excluded individuals with psychiatric disorders, including anxiety disorders and PTSD. The exclusion of youth with anxiety or PTSD thus eliminated the acquired variant altogether from this study. Without further research differentiating between subtypes of CU and investigation of patterns of autonomic responding measured physiologically among groups of high-CU youth, group differences beneath the mask of callousness remain largely unknown.

#### The Current Study

The current study investigated detained youths' physiological reactivity (EDA and RSA) at baseline, in response to an emotionally evocative stimulus, and during a recovery period. Self-report measures were used to identify two distinct subgroups of youth high in CU traits, consistent with the theory of primary and acquired variants, on the basis of PTSS. The questions addressed in the present study were as follows:

1. Does membership in the primary- versus acquired-CU group help to explain variance in average EDA and RSA? Youth in the acquired-CU group, based on greater difficulties in emotion regulation, are expected to evidence lower RSA across baseline, task, and recovery periods as compared to primary-CU youth. Two competing hypotheses were tested regarding EDA among youth in the acquired-CU group: Based on the conceptualization of this group as having greater emotional distress than the primary-CU group, higher EDA was expected.

- However, given that these youth are presumed to have had chronic trauma exposure and posttraumatic distress over an extended period of time, an alternative hypothesis was that these youth would demonstrate attenuated EDA that does not differ significantly from the primary-CU youth.
2. Does group membership help to explain variance in the slopes of EDA and RSA across the video task period and recovery period? It was hypothesized that acquired-CU youth would have a steeper increase in EDA during the video task as well as a less steep slope during recovery compared to primary-CU youth. Expected findings for group differences in RSA were less clear based on review of the literature. Given the limited evidence to date, it was expected that the acquired-CU youth would evidence a steeper decrease in RSA across the video task period, and a less steep slope during recovery, as compared to the primary-CU group.

## METHOD

### Sample and Participant Selection

Participants included 361 youth (265 boys, 96 girls) recruited from a juvenile detention center in the United States. Youth ranged in age from 12 to 18 ( $M = 15.99$ ,  $SD = 1.30$ ); 54.0% were White/Caucasian, 4.7% Black/African American, 25.5% Hispanic/Latino, 3.6% Native American/Alaskan Native, 5.0% Pacific Islander/Native Hawaiian, 6.1% Multiracial, 0.3% Asian/Asian American, and 0.8% identified as “Other.” Descriptive statistics are displayed in Table 1.

### Procedure

#### Youth Questionnaires and Interview

All youth measures were administered via a computer survey tool resident on a laptop computer in interview format due to poor reading skills common among youth in this population and the sensitive nature of many questionnaires. After completing the questionnaires, youth completed the physiological procedure in a separate meeting, generally within 48 hr.

#### Youth Physiological Procedures

Youth were seated comfortably in a private room at the detention center with a research assistant to monitor them. Electrodes were placed in a standardized Einthoven’s triangle configuration with one sensor on the shoulder and one on each side of the torso



for RSA measurement, and two sensors on the palm of the hand for EDA measurement. RSA and EDA were measured using Biopac's MP150 system during a three minute vanilla baseline period (during which youth viewed PowerPoint slides with nature photos), followed by a three minute video clip from the movie *The Champ* (1979), in which a young child witnesses and reacts to the death of his father, and finally during a two minute vanilla recovery period (with youth again viewing nature photos). This film clip was selected as the stimuli because film clips are standardized and effective at using visual and auditory stimuli to evoke emotional responses, and require no reading ability. This film clip has been demonstrated to evoke sadness, with over 94% of the normative sample indicating that they felt this emotion (see Gross & Levenson, 1995). *The Champ* has been shown to result in physiological reactivity, including changes in RSA and EDA, which are consistent with individual differences in emotion regulation abilities (e.g., Crowell et al., 2005).

### Measures

#### Callous-Unemotional Traits

The *Inventory of Callous Unemotional Traits* (ICU; Frick, 2004) is a 24-item self-report measure that was developed to provide an efficient, reliable, and valid assessment of CU traits in samples of youth. The measure contains three subscales (i.e., Uncaring, Callous, and Unemotional) that sum to create a higher-order callous-unemotional dimension, the total score for which was used in the present analyses. Sample items include "I am concerned about the feelings of others" (reverse scored) and "I do not show my emotions to others." Youth report on a 0 (*not at all true*) to 3 (*definitely true*) scale. Cronbach's alpha for the total scale in the present sample was .71.

## Trauma Exposure and Posttraumatic Stress Symptoms

The *University of California at Los Angeles Posttraumatic Stress Disorder Reaction Index for DSM-IV—Adolescent Version* (PTSD-RI; Steinberg, Brymer, Decker, & Pynoos, 2004) is a well-validated brief screening measure used to assess exposure to traumatic events and symptoms of PTSD. First, youth are asked whether or not they have experienced any of 20 different types of traumatic events, resulting in a summed score for total trauma exposure. In the second portion of the questionnaire, youth report on 17 PTSD symptoms as dictated by DSM-IV-TR criteria using a 5-point scale (0 = *none of the time* to 4 = *most of the time*), and a total sum score is derived. The PTSD-RI has demonstrated good convergent validity with other diagnostic measures, as well as high internal and high test-retest reliability over a period of 7 days (Steinberg et al., 2004). Cronbach's alpha for these PTSD symptoms in the current sample was .89.

## Anxiety

The Revised Children's Manifest Anxiety Scale, 2<sup>nd</sup> Edition (RCMAS-2; Reynolds & Richmond, 2008) is a 49-item self-report measure validated for youth ages six to nineteen, and assesses both the level and nature of youths' anxiety. This measure includes scales for physiological anxiety, worry, social anxiety, defensiveness, and an inconsistent responding index, and there is also a total anxiety score, which was used for analyses in the current study. Cronbach's alpha in the current sample was .91.

## Electrodermal Responding

EDA, a measure of sympathetic nervous system activation, indexes the activity of the eccrine sweat glands on the palmar and plantar regions of the hand by measuring the

electric current passed between a pair of electrodes placed on the surface of the skin. EDA was assessed from the amplitude of nonspecific fluctuations (NSFs) in skin conductance. The EDA signal was collected using two 0.8 cm<sup>2</sup> Ag-AgCl electrodes with saline gel. Electrodes were secured to the participant's nondominant hand using adhesive masking collars. The number of nonspecific responses were counted across 30-s epochs during baseline, video task, and recovery. Movement artifacts in electrodermal responding were not scored. Research assistants were trained to differentiate true responses from artifacts, and artifacts were flagged during data collection and removed during scoring.

#### Respiratory Sinus Arrhythmia

RSA was assessed from the ECG signal using spectral analysis. Spectral power is typically divided into low- to mid-frequency variability (below 0.15 Hz), and high-frequency variability (above 0.15 Hz). Parasympathetic influences on heart rate, such as RSA, can be observed in the high-frequency range. High-frequency spectral densities were calculated in 30-s epochs via fast Fourier analysis using the Mindware software package. During scoring, movement and other artifacts were corrected after examining each interbeat interval (IBI) file for discrepancies. Discrepancies were detected in instances where the physiological software program incorrectly identified movement as a heart beat (resulting in an IBI that was shorter than expected) or the program failed to identify a heart beat that existed (resulting in an IBI that was longer than expected). The raw data was examined to confirm these errors, and artifactual heart beats were removed. Average RSA was measured across 30-s epochs during baseline, video task, and recovery.

### Medication and Medical Considerations

Legal guardians were asked if their child was taking any medications at the time of admission into the detention center, or if their child had a pacemaker, insulin pump, or history of a heart murmur. At time of interview, youth were also asked if they had a pacemaker, insulin pump, or history of a heart murmur. If guardians reported that their child was taking medications or if they were unsure, guardians were asked to provide the names of the medications and give permission for research assistants to verify names of medications with nursing staff at the detention center, given that some medications are known to interact with physiological measurement, such as those with anticholinergic effects (Dawson, Nuechterlein, Schell, & Mintz, 1992). In this sample, many youth were taking psychotropic medications, including mood stabilizers ( $n = 14$ ), stimulants ( $n = 19$ ), antihistamines ( $n = 9$ ), selective serotonin reuptake inhibitors (SSRIs,  $n = 25$ ), or other psychotropic/antipsychotic medications ( $n = 18$ ), as determined by guardian report and/or verification by nursing staff at the detention center; for a further 28 youth, specific medication information was unavailable. An additional 25 youth had a heart murmur, insulin pump, or pacemaker according to youth self-report or their guardian's report.

### Analytic Plan

To determine group membership, first a threshold score was used to select youth scoring higher in CU traits relative to the remainder of the sample, consistent with methods employed by previous investigators (e.g., Bennett & Kerig, 2014; Finger et al., 2008; Hicks, Vaidyanathan, & Patrick, 2010; Kimonis et al., 2012; Tatar et al., 2012; Vitale et al., 2005). Because there is no established clinical cut-off score for the ICU measure, we identified those youth scoring in the top third of the sample as high-CU,

consistent with previous research (Bennett & Kerig, 2014; Murrie & Cornell, 2002; Skeem, Johansson, Andershed, Kerr, & Loudon, 2007). In the current sample, those youth scoring equal to or above a score of 31 ( $n = 139$ ) were identified as high-CU following the above guidelines, and the remaining youth scoring below 31 on the ICU ( $n = 222$ ) were retained as a lower-CU comparison group. Mixture modeling was used to classify the 139 high-CU youth into two subgroups based on their PTSS, consistent with the theory of primary- and acquired-CU. Mplus version 6.11 (Muthén & Muthén, 1998-2011) was therefore programmed to create two classes, allowing factor means for PTSS scores to vary across groups. Mixture modeling utilizes full information maximum likelihood and expectation-maximization to handle missing data. Average latent class probabilities for most likely latent class membership were .90 and .92, respectively, with off-diagonal probabilities of .10 and .08 indicating the degree of misclassification. Descriptive statistics for the total sample as well as each subgroup are displayed in Table 1. Of the 139 high-CU youth, 85 were placed in the first class, labeled ‘primary,’ and 54 were placed in the second class, labeled ‘acquired.’ Using the most likely class assignment for each individual, independent-samples *t*-tests and chi square analyses were used to assess group differences.

To address all remaining study aims, models were run using HLM version 7 (Raudenbush, Bryk, & Congdon, 2011). For Aim 1, a series of two-level models were run, such that Level 1 contained three scores that represented mean physiological activity across all epochs of baseline, across all epochs of the video task, and across all epochs of the recovery period. Separate models were run for EDA and RSA data. At Level 1, dummy codes were used to draw comparisons between the baseline, video task, and

recovery periods to examine relative differences, and separate models were run to examine each of these contrasts by adjusting the reference period. At Level 2, group membership (using the most likely group membership determined by the mixture model) was included as a predictor using dummy-coded variables to compare the primary-, acquired-, and lower-CU comparison groups, with separate models run to allow for each different contrasts by adjusting the reference group. In all models, a random effect for the intercept was included at Level 2. Because EDA was measured using the sum of the number of nonspecific electrodermal responses an individual had during a given time period, it was treated as a count variable and a Poisson distribution (constant exposure) was used for models using EDA as the outcome. The general equations were as follows:

$$L1: \beta_{0j} + \beta_{1j}*(\text{time period}_{ij}) + \beta_{2j}*(\text{time period}_{ij}) + r_{ij}$$

$$L2: \beta_{0j} = \gamma_{00} + \gamma_{01}*(\text{group}_j) + \gamma_{02}*(\text{group}_j) + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{group}_j) + \gamma_{12}*(\text{group}_j)$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}*(\text{group}_j) + \gamma_{22}*(\text{group}_j)$$

where  $i$  is time and  $j$  is person; the time period variables were dummy coded to indicate either baseline, video task, or recovery period as the reference; and group variables were also dummy coded to indicate either the primary-, acquired-, or lower-CU comparison group as the reference. These models allowed for investigation of relative change among baseline, video task, and recovery periods across the three groups, as well as investigation of differences among groups at any one time period. Robustness of effects were examined by running additional models excluding youth taking psychotropic medications or with relevant medical conditions, excluding statistical outliers, and controlling for age, ethnicity, and gender, which have been demonstrated to affect physiological responding

(e.g., Anderson & McNeilly, 1991; Boucsein et al., 2012), and results of these alternative models are presented in the Appendix.

Aim 2 was also addressed using a series of two-level models in HLM. Level 1 contained scores for each 30-s epoch in physiological measurement either across the video task (6 epochs) or the recovery period (4 epochs). Simple slopes were examined by centering the time variable to adjust the time period of reference in analyses. Thus, analyses were run in a way that allowed for examination effects at the start of the time period (with the 0 point at the first epoch of measurement), in the middle of the period (between epochs 3 and 4 for the video task, or between epochs 2 and 3 for the recovery period), and at the end of the time period (with the 0 point at the last epoch of measurement). At Level 2, group membership was included as a predictor using dummy codes, similar to the method in Aim 1, and separate models were run to test each of the group contrasts. At Level 2, individuals' average score at baseline (grand mean centered) was also included as a predictor for the intercept, and a random effect for the intercept was also included. As with Aim 1, the models using EDA as the outcome were run using a Poisson distribution (constant exposure) because scores were the count of nonspecific responses an individual had in each epoch. Additionally, the fit of both linear and quadratic models were examined for these models. The general equations for the linear models were as follows:

$$L1: \beta_{0j} + \beta_{1j} * (\text{time period}_{ij}) + r_{ij}$$

$$L2: \beta_{0j} = \gamma_{00} + \gamma_{01} * (\text{group}_j) + \gamma_{02} * (\text{group}_j) + \gamma_{03} * (\text{baseline score}) + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11} * (\text{group}_j) + \gamma_{12} * (\text{group}_j)$$

The general equations for the quadratic models were as follows:

$$L1: \beta_{0j} + \beta_{1j}*(\text{time period}_{ij}) + \beta_{2j}*(\text{time period}_{ij})^2 + r_{ij}$$

$$L2: \beta_{0j} = \gamma_{00} + \gamma_{01}*(\text{group}_j) + \gamma_{02}*(\text{group}_j) + \gamma_{03}*(\text{baseline score}) + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{group}_j) + \gamma_{12}*(\text{group}_j) + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}*(\text{group}_j) + \gamma_{22}*(\text{group}_j) + \mu_{2j}$$

where i is time and j is person, and baseline score is grand centered. These models indicated whether or not there were differences between groups in slopes of physiological responses across epochs during the video task and the recovery period, relative to the individual's baseline score. Robustness of effects for the results were examined by running additional models excluding youth taking psychotropic medications or with relevant medical conditions, excluding statistical outliers, and controlling for age, ethnicity, and gender, and results of these alternative models are presented in the Appendix.



Table 1  
*Means and Standard Deviations for the Total Sample, Lower-CU Comparison Group, and Primary and Acquired Groups*

	Total Sample		Lower-CU		Primary-CU		Acquired-CU	
	(N = 361)		(n = 222)		(n = 85)		(n = 54)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	15.99	1.30	15.98 <sub>a</sub>	1.34	16.07 <sub>a</sub>	1.21	15.92 <sub>a</sub>	1.28
Total CU	28.02	7.62	23.78 <sub>a</sub>	5.38	34.67 <sub>b</sub>	3.49	39.94 <sub>b</sub>	7.68
Trauma Exposure	6.62	3.93	6.41 <sub>a</sub>	3.99	5.75 <sub>a</sub>	3.41	8.85 <sub>b</sub>	3.73
PTSS	26.94	14.40	25.47 <sub>a</sub>	14.38	19.83 <sub>b</sub>	8.08	43.98 <sub>c</sub>	7.39
Anxiety	12.44	8.87	11.80 <sub>a</sub>	8.47	10.37 <sub>a</sub>	7.84	18.45 <sub>b</sub>	9.66
ED	85.00	21.86	83.69 <sub>a</sub>	20.68	77.39 <sub>b</sub>	18.95	103.00 <sub>c</sub>	21.79
RSA- baseline	6.81	1.27	6.81 <sub>a</sub>	1.36	6.87 <sub>a</sub>	1.05	6.70 <sub>a</sub>	1.21
RSA- video task	6.48	1.14	6.49 <sub>a</sub>	1.16	6.50 <sub>a</sub>	1.10	6.37 <sub>a</sub>	1.12
RSA- recovery	6.67	1.21	6.68 <sub>a</sub>	1.26	6.69 <sub>a</sub>	1.11	6.58 <sub>a</sub>	1.15
EDA- baseline	4.20	4.95	4.21 <sub>a</sub>	5.11	4.62 <sub>a</sub>	5.18	3.53 <sub>a</sub>	3.76
EDA- video task	6.59	7.02	6.36 <sub>a</sub>	6.73	7.59 <sub>a</sub>	7.90	5.92 <sub>a</sub>	6.69
EDA- recovery	3.09	3.77	3.23 <sub>a</sub>	3.94	2.92 <sub>a</sub>	3.43	2.80 <sub>a</sub>	3.67

*Note.* Scores in the same row that do not share subscripts differ significantly ( $p < .05$ ) based on independent samples  $t$ -tests comparing primary-, acquired-, and lower-CU youth. ED = Emotion Dysregulation.

## RESULTS

### Cluster Validation

In order to ensure that the groups differed from one another in ways that acquired-CU theory would predict, comparisons were made on relevant variables. The acquired-CU group self-reported significantly higher levels of PTSS,  $t(136) = 17.71, p < .001$ , anxiety,  $t(90.07) = 5.03, p < .001$ , emotion dysregulation,  $t(132) = 7.17, p < .001$ , and trauma exposure,  $t(137) = 5.03, p < .001$ , as compared to the primary group. The acquired group ( $n = 18$  girls) included a significantly higher proportion of girls than the primary group ( $n = 13$  girls),  $\chi^2(1) = 6.20, p = .013$ , although the two groups did not differ in age, ethnicity, or CU traits.

When compared to the lower-CU group, primary-CU youth self-reported lower levels of PTSS,  $t(260.76) = 4.29, p < .001$ , and emotion dysregulation,  $t(300) = 2.42, p = .02$ , but these groups did not differ by age, trauma exposure, or anxiety. When compared to the lower-CU group, acquired-CU youth reported higher levels of trauma exposure,  $t(274) = -4.08, p < .001$ , PTSS,  $t(163.39) = -9.16, p < .001$ , anxiety,  $t(261) = -4.90, p < .001$ , and emotion dysregulation,  $t(268) = -5.94, p < .001$ . There were no differences in age. The acquired group was the most likely to meet DSM-IV criteria for PTSD,  $\chi^2(2) = 54.55, p < .001$  (47% of the acquired group vs. 15% of the comparison sample and 0% of the primary group meeting full criteria).

### Aim 1

For models with RSA as the outcome, it was hypothesized that youth in the acquired-CU group would demonstrate lower RSA across the baseline, video task, and recovery periods as compared to primary-CU youth. However, results indicated that the primary-, acquired-, and lower-CU comparison group did not significantly differ from one another at baseline, during the video task, or during the recovery period in mean RSA. Additionally, within-group results indicated that mean scores significantly varied between the baseline, video, and recovery periods for both the primary-CU and lower-CU groups. The acquired group's scores differed from baseline to video and from video to recovery, although the difference between baseline and recovery was not significant. Finally, there were no between-group differences in the magnitude of changes between baseline and video task, video task to recovery, or baseline to recovery. Results for the model with the video task as the reference period and the acquired-CU group as the reference group are displayed in Table 2, and mean scores for each group at baseline, video task, and recovery are displayed in Figure 1.

For models with EDA as the outcome, it was hypothesized that the acquired-CU group might evidence either higher or lower EDA than the primary group, related to competing theories suggesting greater likelihood of either hyper- or hyporeactivity. Results indicated that the primary-, acquired-, and lower-CU comparison groups did not significantly differ from one another at baseline, during the video task, or during the recovery period in mean number of nonspecific electrodermal responses, which was not consistent with either hypothesis. Within-group results indicated that each group's mean scores significantly varied between the baseline, video, and recovery periods, suggesting

that they each increased in EDA from baseline to video and then decreased in EDA during the recovery period. Additionally, a number of between-group discrepancies emerged in the magnitude of differences between time periods. The change between the video and recovery periods was significantly greater for youth in the primary-CU group as compared to youth in the lower-CU group,  $B = .29$ ,  $SE = .08$ ,  $p < .001$ , which was a finding that was not anticipated in the a priori hypotheses. The difference between the baseline and recovery periods was also significantly greater for youth in the primary-CU group as compared to youth in the lower-CU group,  $B = .19$ ,  $SE = .07$ ,  $p = .013$ . The acquired group did not differ significantly from either the primary- or lower-CU group in their rate of change between any of the time periods, which was inconsistent with the hypothesized effects. Results for the model with the video task as the reference period and the acquired-CU group as the reference group are displayed in Table 2, and mean scores for each group are displayed in Figure 2.

## Aim 2

For models with RSA as the outcome, it was hypothesized that youth in the acquired-CU group would evidence steeper reactivity during the video task. Results investigating the slope across the video task indicated that baseline RSA was a significant predictor of RSA score at the beginning, middle, and end of the video task period for each group of youth. Additionally, for each group, the slope at the beginning, middle, and end of the video task period was significantly different from zero, with each group having a negative slope, indicating that RSA decreased across the video task period for all groups of youth. However, there were no significant group differences at the beginning, middle, or end of the video task period, nor were there group differences in slope across

the video task period. When examining a model with time as a quadratic rather than linear slope, a difference of 17 in the BIC (Bayesian information criterion) values of the two models indicated that the linear model was a significantly better fit for the data across the video task period.

For models with RSA as the outcome, it was hypothesized that youth in the acquired-CU group would evidence a less steep slope across the recovery period. Results examining the slope across the recovery period indicated that baseline RSA was significant predictor of RSA score at the beginning, middle, and end of the recovery period for each group of youth. Additionally, for the primary- and lower-CU groups, the slope at the beginning, middle, and end of the recovery period was significantly different from zero. However, for the acquired group, the slope was not significantly different from zero, indicating that there was no meaningful change in RSA from the start to the end of the recovery period for the this group. There were no significant between-group differences in mean RSA at the beginning, middle, or end of the recovery period, nor were there any significant between-group differences in slope across the recovery period, which was inconsistent with hypothesized effects. When examining a model with time as a quadratic rather than linear slope, a difference of 7 in the BIC (Bayesian information criterion) values of the two models indicated that the linear model was a significantly better fit for the data for the video task period. Results for the linear models with the acquired-CU group as the reference group and the midpoint of the video task and recovery task as the reference period, respectively, are displayed in Table 3. Mean scores for each group are displayed in Figure 3.

For models with EDA as the outcome, it was hypothesized that acquired-CU

youth would have a steeper increase in EDA during the video task as well as a less steep recovery compared to primary-CU youth. Results investigating the slope for the video task indicated that baseline EDA was a significant predictor of EDA at the beginning, middle, and end of the video period for each group. Additionally, for the primary group only, the slope at the beginning, middle, and end of the video task was significantly different from zero, indicating a significant increase in EDA across the video task, whereas the acquired- and lower-CU comparison groups did not evidence a significant increase or decrease in EDA across the video task. There were no significant group differences between groups in mean EDA at either the beginning, middle, or end of the video task period, nor were there any significant differences in slope across the video task period, a pattern that was not consistent with the hypothesis. As a deviance statistic is not calculated for models using the Poisson distribution, the Bayesian information criterion (BIC) value could not be calculated to compare the linear and quadratic models. However, inclusion of a quadratic term for time did not result in any additional significant effects, and thus, the linear model was maintained for parsimony.

For models with EDA as the outcome, results examining the slope for the recovery period indicated that baseline EDA was a significant predictor of EDA at the beginning, middle, and end of the recovery period for all groups. Additionally, for the primary- and lower-CU groups, the slope was significantly different from zero at the beginning, middle, and end of the recovery period, whereas for the acquired group the slope was only significantly different from zero at the beginning of the recovery period. Although the EDA scores of the groups did not differ from one another at the beginning, middle, or end of the recovery period, there were between-group differences in slope.

Specifically, the primary-CU group evidenced a steeper declining slope than the acquired group,  $B = -0.17$ ,  $SE = .08$ ,  $p = .03$ , consistent with the hypothesized pattern. There were no significant differences between the lower-CU group and either the primary or acquired groups in EDA slope during the recovery period. Again, the BIC value to compare the linear and quadratic models could not be calculated due to use of the Poisson distribution, but inclusion of a quadratic term for time resulted in a loss of the majority of the significant effects for the recovery period, and the limited number of significant effects remaining indicated that a linear model should be retained. Results for the model with the acquired-CU group as the reference group and the midpoint of the video task and recovery task as the reference periods, respectively, are displayed in Table 3. Mean scores for each group are displayed in Figure 4.

Table 2  
*Final Estimation of Fixed Effects (with Robust Standard Errors) Across Models for Aim 1 with the Video Task and Acquired-CU Group as Reference, for RSA and EDA as Outcomes*

	RSA	EDA
For INTERCEPT, $\beta_0$		
Intercept, $\gamma_{00}$	6.38 (.16)***	1.82 (.13)***
Primary vs. Not, $\gamma_{01}$	0.12 (.21)	0.24 (.15)
Lower-CU vs. Not, $\gamma_{02}$	0.13 (.18)	0.05 (.14)
For BASELINE slope, $\beta_1$		
Intercept, $\gamma_{10}$	0.32 (.08)***	-0.52 (.09)***
Primary vs. Not, $\gamma_{11}$	0.03 (.10)	0.01 (.10)
Lower-CU vs. Not, $\gamma_{12}$	-0.04 (.09)	0.11 (.10)
For RECOVERY slope, $\beta_2$		
Intercept, $\gamma_{20}$	0.21 (.08)*	-0.76 (.11)***
Primary vs. Not, $\gamma_{21}$	-0.02 (.10)	-0.18 (.13)
Lower-CU vs. Not, $\gamma_{22}$	-0.004 (.09)	0.11 (.12)

*Note.* All models for EDA as the outcome were run using a Poisson distribution and population-average model results are displayed.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

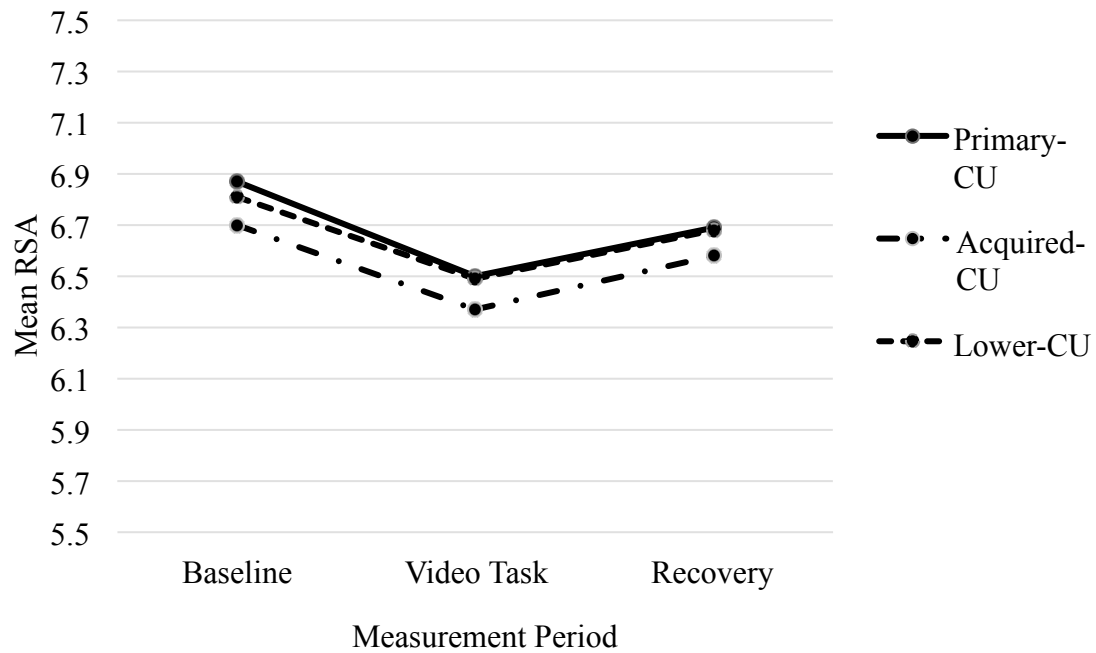


Table 3  
*Final Estimation of Fixed Effects (with Robust Standard Errors) Across Models for Aim 2 for the Video Task (Top) and Recovery Period (Bottom) with the Acquired-CU Group and Time Centered at Midpoint for RSA and EDA as Outcomes*

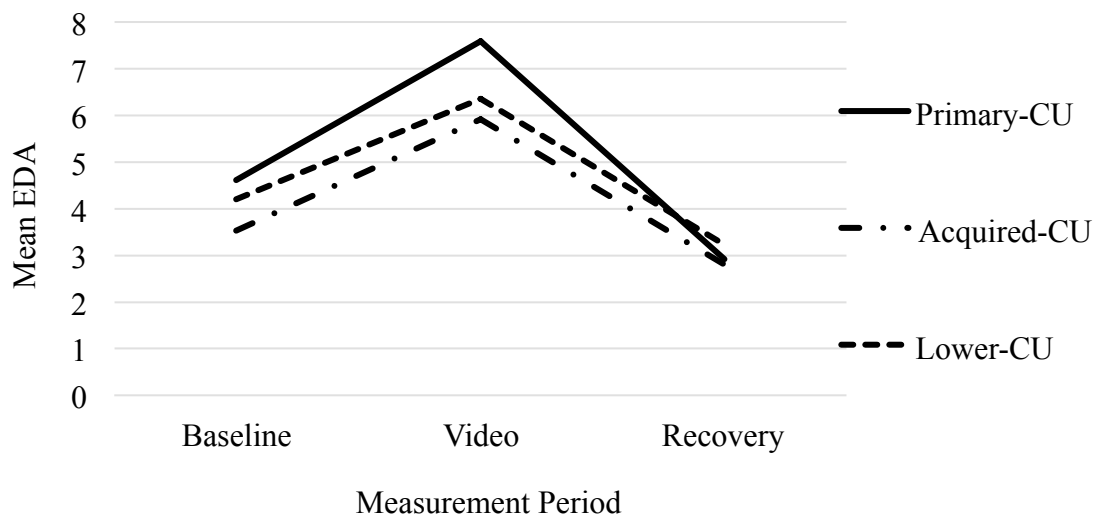
	RSA	EDA
For INTERCEPT, $\beta_0$		
Intercept, $\gamma_{00}$	6.45 (.08)***	-0.16 (.14)
Primary vs. Not, $\gamma_{01}$	0.02 (.10)	0.03 (.16)
Lower-CU vs. Not, $\gamma_{02}$	0.05 (.09)	-0.06 (.15)
Baseline Mean, $\gamma_{03}$	0.78 (.04)***	0.16 (.01)***
For Slope at Midpoint, $\beta_1$		
Intercept, $\gamma_{10}$	-0.07 (.02)**	-0.02 (.03)
Primary vs. Not, $\gamma_{11}$	0.01 (.03)	0.01 (.04)
Lower-CU vs. Not, $\gamma_{12}$	0.02 (.03)	-0.01 (.03)
For INTERCEPT, $\beta_0$		
Intercept, $\gamma_{00}$	6.67 (.09)***	-0.46 (.17)**
Primary vs. Not, $\gamma_{01}$	-0.03 (.11)	-0.26 (.19)
Lower-CU vs. Not, $\gamma_{02}$	0.002 (.10)	-0.09 (.18)
Baseline Mean, $\gamma_{03}$	0.81 (.06)***	0.16 (.01)***
For Slope at Midpoint, $\beta_1$		
Intercept, $\gamma_{10}$	-0.08 (.05)	0.01 (.07)
Primary vs. Not, $\gamma_{11}$	-0.01 (.06)	-0.16 (.08)*
Lower-CU vs. Not, $\gamma_{12}$	-0.01 (.05)	-0.08 (.07)

*Note.* All models for EDA as the outcome were run using a Poisson distribution and population-average model results are displayed. Mean baseline score was grand mean centered.

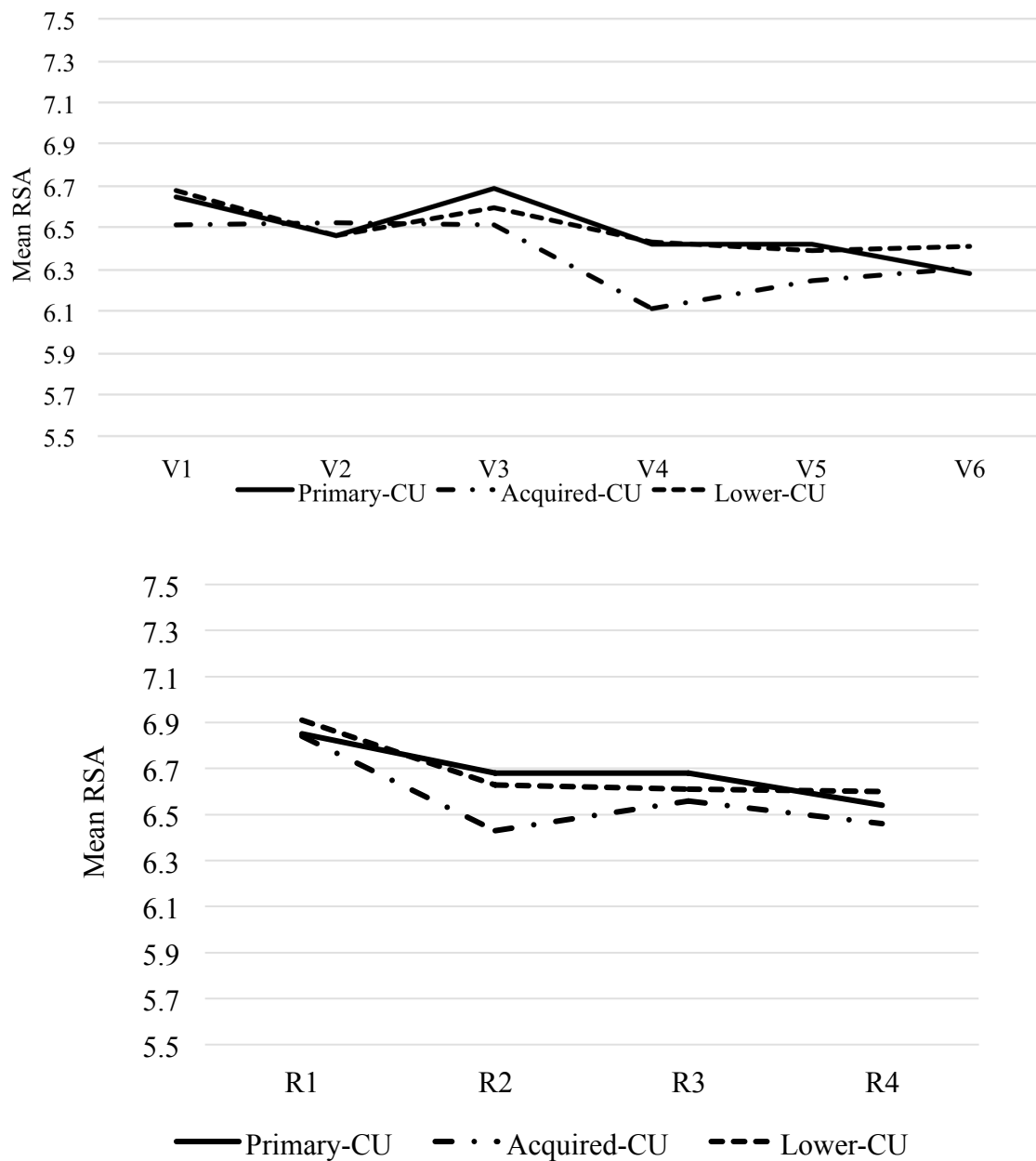
\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .



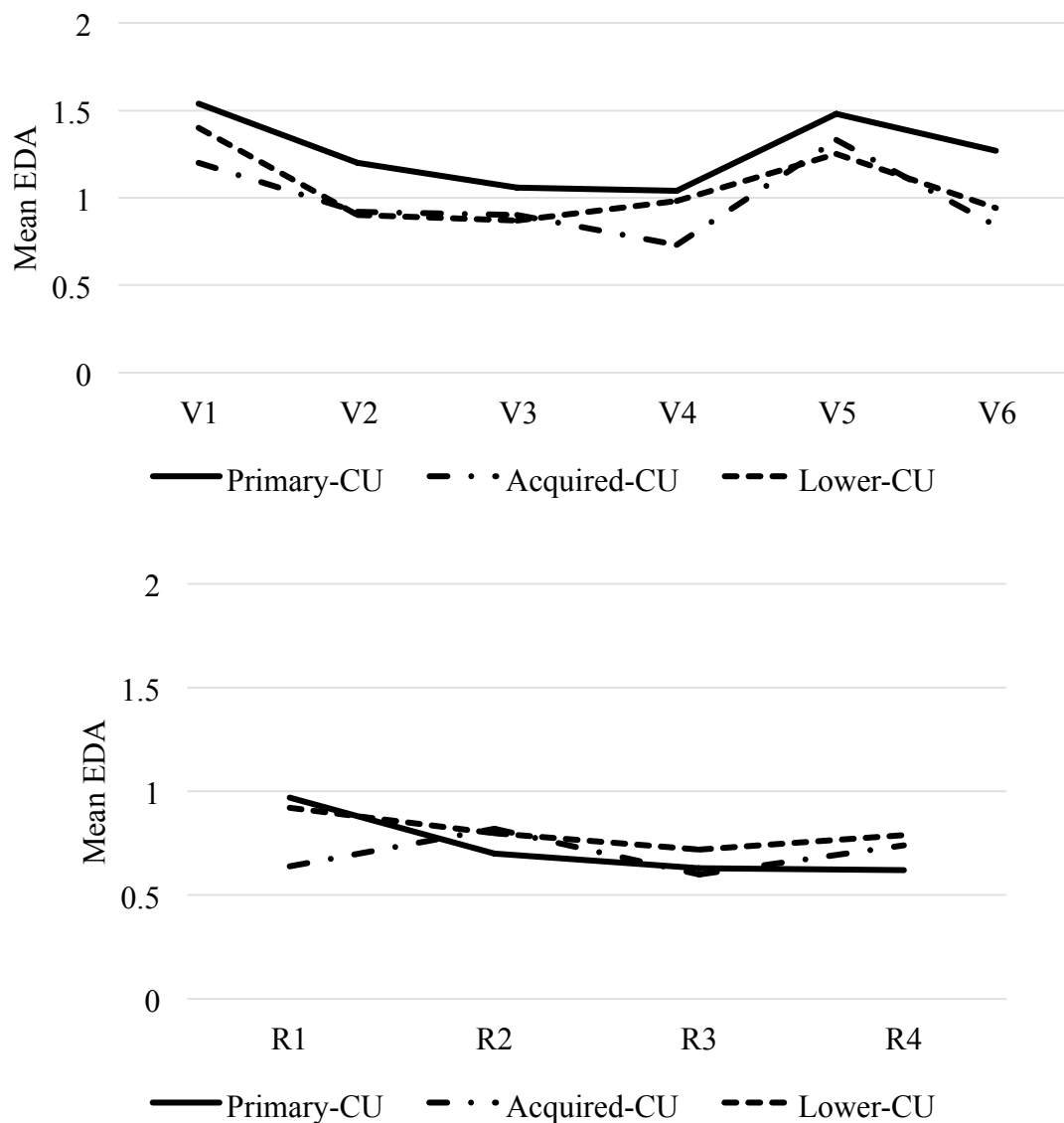
*Figure 1.* Mean RSA at baseline, during the video task, and during the recovery period for lower-CU, primary-CU, and acquired-CU groups. No significant group differences were observed at discrete time periods or in the change between time periods, as assessed for Aim 1.



*Figure 2.* Mean EDA (as measured by the mean across group members in the number of nonspecific responses during that period) at baseline, during the video task, and during the recovery period for lower-CU, primary-CU, and acquired-CU groups. No significant group differences were observed at discrete time points, although the primary group evidenced a greater change from baseline to recovery and from video to recovery as compared to the lower-CU comparison group, as assessed in Aim 1.



*Figure 3.* Mean RSA across each of the six epochs during the video task (top) and across each of the four epochs of the recovery period (bottom) for the low-CU, primary-CU, and acquired-CU groups. No significant group differences were observed at the beginning, middle, or end of each measurement period, nor were there any differences between groups in slope at those points, as assessed in Aim 2.



*Figure 4.* Mean EDA across each of the six epochs during the video task (top) and across each of the four epochs of the recovery period (bottom) for the low-CU, primary-CU, and acquired-CU groups. No significant group differences were observed at the beginning, middle, or end of each measurement period, although the primary-CU group evidenced a steeper declining slope across the recovery period than the acquired-CU group, as assessed in Aim 2.

## DISCUSSION

The present study was the first known to examine differences in both EDA and RSA activity among detained youth with primary- and acquired-CU traits as well as a lower-CU comparison group. This study used a method of differentiating the primary and acquired variants that is relatively novel in the field, and remains true to the theory of acquired-CU by using PTSS as the key differentiator (see Bennett & Kerig, 2014). The acquired-CU group as compared to the primary-CU group evidenced higher levels of PTSS, as specified by the model, as well as self-reported anxiety, trauma exposure, and emotion dysregulation. Notably, the primary-CU group did not differ significantly from the lower-CU control group on measures of trauma exposure or anxiety, consistent with previous research suggesting that using anxiety as a differentiator between high- and low-CU groups may be ineffective given the lack of consistency in group differences on this variable (e.g., Dolan & Rennie, 2007; Schmitt & Newman, 1999; Skeem et al., 2003). The use of PTSS, which is more consistent with the theory of acquired-CU (Karpman, 1941; Porter, 1996), may help clarify inconsistent between-group findings and warrants continued replication in future studies.

The main goal of the current study was to examine psychophysiological differences between groups in response to an emotionally evocative video task, during baseline, and during a recovery period, with a particular focus on mean group differences at discrete time points and differences in the slope across time among groups. Results

indicated that the primary- and acquired-CU groups did not differ in mean RSA at any discrete time point examined nor in their rates of change over time during the measurement periods. However, although the mean number of electrodermal responses at discrete time points generally did not differ between groups, the primary-CU group demonstrated a greater increase between the baseline and video task periods in EDA as compared to the lower-CU group, and also evidenced a steeper decline from the video task to the recovery period compared to the lower-CU group. The acquired-CU group did not differ from the other groups when looking at average EDA across the baseline, video task, or recovery period. However, when examining the slopes within the recovery period in particular, the acquired-CU group evidenced a flatter slope in returning to baseline than did those in the primary-CU group. These findings suggest that the primary-CU youth evidenced a more rapid decline from the stressor task toward their initial baseline. Although the flatter recovery slope for acquired-CU youth may indicate poorer recovery toward baseline, this conclusion is tempered by the fact that the acquired-CU youth did not evidence a significant increase in EDA during the video task (as evidenced by a slope not significant different from zero), whereas the primary-CU youth did have a significant increase in EDA. Although these differences in slope of EDA during the video task were not significantly different between groups, they provide some indication that the primary-CU group, as a whole, may have been more reactive to the video task given the lack of significant slope for the acquired-CU group, and thus, the significant differences in recovery slope must be interpreted with that context in mind.

The lack of significant group differences in RSA was inconsistent with hypothesized patterns, although they provide an important addition to a very limited

literature. To date, studies have reported mixed results regarding how PTSD is related to PNS functioning (see Southwick, Krystal, Johnson, & Charney, 1998, for a review), and no known studies have examined PNS functioning among variants of CU traits or even among high- versus low-CU groups. Although not significantly different, the pattern of responses was such that, consistent with hypotheses, the acquired-CU group evidenced lower RSA at baseline, during the video task, and during recovery. Notably, the lack of between-group findings for RSA indicates that when both PTSS and CU traits are taken into account, youth with higher levels of PTSS do not differ from other youth in RSA, which is consistent with previous research indicating that it may not be PTSS but rather other life experiences, such as trauma exposure, that are responsible for differences in RSA (Sahar et al., 2001). Had the present study included a nontraumatized normative sample, there would be an additional context in which to interpret group differences.

There are multiple potential explanations as to why the groups did not significantly differ in patterns of RSA. Given all three groups of youth were detained at the time of the study (suggesting some level of functional impairment) and nearly all youth reported having experienced traumatic stressors, it is possible that all three groups evidenced some degree of physiological dysregulation, hence, the lack of significant differences. However, because the youth in the acquired-CU group self-reported significantly higher levels of emotion dysregulation than their peers, it seems unlikely that the groups are equivalently dysregulated. Moreover, because the groups differed on self-reported emotion dysregulation, the lack of observed differences in RSA is also meaningful in that dysynchrony between behavioral and psychophysiological responses may be indicative of emotion dysregulation in itself (Beauchaine, 2005). It is possible that either the acquired-



CU youth are subjectively reporting a higher-than-actual level of dysregulation, or the primary-CU youth may be subjectively experiencing a lower-than-actual level of dysregulation, the latter of which has some empirical support. Specifically, Kahn and colleagues (2013) found that youth classified as primary-CU were perceived by blind raters to be less credible reporters than youth in acquired- or lower-CU groups. Further, Kahn and colleagues noted that youth in the primary-CU cluster also had a tendency to underreport their impulsivity, externalizing behavior, and behavioral inhibition relative to their parents' report of those constructs. Although Kahn and colleagues' results should be replicated in future studies, if this pattern holds true, it may be relevant to understanding the mismatch between physiological and self-report measures seen in the present study. Another possible explanation for the absence of significant group differences in RSA is that the emotionally evocative task used in the present study, a video clip from *The Champ* (1979), was not effective in eliciting a parasympathetic response from the youth sufficient enough to result in detectable group differences; however, the overall pattern of results indicated that each group, on average, decreased from baseline to the video task, indicating that vagal activity was affected, and within-group changes between each period were significantly different from zero. Future studies should continue to examine group differences in RSA with different stressor tasks, and also examine the correspondence between physiological and self-report measures to aid in interpreting findings. Future studies would also benefit from the inclusion of nontraumatized control groups to further elucidate group differences, or lack thereof, as the majority of youth in all three groups in the current study had some history of trauma exposure, which may limit the generalizability of findings in the current study.

Although no significant between-group differences emerged in the slopes across the video task and recovery periods for RSA, there was one significant difference when examining patterns of EDA between groups. Results indicated differences in the slope across recovery between the primary and acquired groups, consistent with the hypothesized pattern. This group difference in recovery slope in particular is consistent with previous meta-analytic findings comparing individuals with and without PTSD (Pole, 2007), and the lack of significant group differences at specific time points, although in contrast to the proposition that a lack of anxiety is the hallmark feature of primary-CU (Frick & Marsee, 2006), is consistent with at least one prior study that failed to observe differences in electrodermal responses between groups that were similar to primary- and acquired-CU variants (Munoz, Frick, Kimonis, & Aucoin, 2008). Given the limited research to date examining EDA among high-CU variants, it is difficult to determine at present whether the current pattern of findings could be related to the measurement used (EDA and RSA in response to an emotionally evocative stimuli) or the methodology in which groupings were derived (formed on the basis of PTSS rather than trait anxiety). In the present study, the primary-CU group appeared to have the steepest increase from baseline to video task, and the steepest decrease from the video task to recovery. It is possible that the acquired-CU youth, and perhaps to a lesser extent the lower-CU comparison sample, have more attenuated reactions than youth in the primary-CU group, allowing the primary-CU group to appear more reactive in comparison. Without inclusion of a nontraumatized control group, it is difficult to interpret the pattern of results for the primary-CU group. However, the between-group differences observed in the current study between the primary- and acquired-CU groups lend some support to

the theory of allostatic load (AL), which suggests that physiological response systems are adjusted in contexts of extreme or prolonged stress to maintain stability for the individual (Sterling & Eyer, 2008), resulting in attenuated reactivity to stimuli over time. Most of the literature on AL focuses on the hypothalamic-pituitary-adrenal (HPA) axis activity rather than the PNS or SNS, although evidence suggests that a broad range of biological and neurological systems are affected by AL processes (Beauchaine, Neuhaus, Zalewski, Crowell & Potapova, 2011). Further longitudinal research is needed to test the hypothesis that these group differences, if replicated, may be indicative of attenuated responding as a result of cumulative wear and tear on the body due to repeated stress reactions over time (Beauchaine et al., 2011) for youth in the acquired-CU group in particular.

The present study had a number of strengths, including the use of a detained sample for whom the presence of high levels of CU traits is clinically meaningful and has potentially deleterious consequences. Additionally, youth in the present sample reported levels of trauma exposure and PTSS that were higher than those of youth in the general population. Studies of detained youth with CU traits and high levels of PTSS are critical given that clinical and normative samples may display different patterns of physiological responding (e.g., Beauchaine, 2015), and therefore, additional studies are needed that focus specifically on samples of high-CU youth, such as the detained sample studied here, rather than community samples. Additionally, this was the first known study to examine both PNS and SNS activity among high-CU variants of detained adolescents, and utilized a relatively new method for differentiating the high-CU variants that is consistent with the theory of acquired-CU. The current study also highlights a number of complications that arise while conducting research with this population. First, nearly one

third of the sample was taking some form of psychotropic medication that may interfere with physiological responding. However, this is likely characteristic of detained youth more generally, with previous studies demonstrating that as many as two-thirds of detained boys meet diagnostic criteria for at least one psychiatric disorder and that the majority of diagnosed youth in detention facilities are taking psychotropic medications (see Desai et al., 2006, for a review); thus, excluding youth who are taking psychotropic medications from this research would involve excluding an important subset of the detained population. Analyses in the present study examined patterns of results with and without medicated youth included (presented in the Appendix), and controlled for several key demographic variables known to influence psychophysiological responding (including age, ethnicity, and gender). Age, gender, and ethnicity were each related to physiological responses in certain analyses, although the pattern of between-group differences remained largely unchanged once these variables were included. However, when youth taking medications or with relevant medical conditions were completely excluded, the significant between-group differences disappeared, suggesting that decisions to include or exclude the subset of medicated youth may have important consequences for conclusions drawn. Additionally, there are variables known to affect physiological responding, such as BMI or recency of exercise, that were not accounted for in the present analyses, in part, due to difficulty accessing such information with youth in a detained setting. As well, the current study relied on youth self-report to gather information about trauma history, PTSS, and CU traits. Although reliance on self-report for those measures is a limitation of the present study, given that many detained youth have had inconsistent caregiving histories, reports from others may not be as feasible to

obtain or as informative as would be ideal. However, inclusion of records maintained by child welfare organizations may be beneficial in future studies as corroborating information to self-report data from youth.

The results of the current study highlight a number of additional future directions. In addition to including control variables known to affect physiological measurement, as discussed above, the inclusion of other variables of interest into models like those tested in the present study would help to further illuminate differences between primary- and acquired-CU variants of youth. First, gender should be considered as a moderating variable rather than simply a covariate in future studies of physiological responding among high-CU youth. Research to date on CU traits has paid little attention to gender, although one study indicated that higher levels of CU traits were associated with lower EDA for boys but not girls (Isen et al., 2010). Additionally, research has consistently demonstrated that women and girls report higher levels of trauma exposure and PTSS (e.g., Wood et al., 2002), and in the present sample, we found that girls were more likely to be classified into the acquired-CU as compared to primary-CU group. Results of models in the present study that included gender as a covariate (described in Appendix) indicated significant effects of gender on physiological activity. Therefore, gender warrants additional attention as a variable of interest and not just a covariate in future studies. Additionally, a number of trauma-related variables should be examined in future research. Previous studies have suggested that age of onset of trauma, chronicity of exposure, type of stressor experienced, and perceived controllability of the stressor each relate to physiological responses (see Miller, Chen, & Zhou, 2007, for a review). Exposure to chronic and repeated stressors may create a “floor effect” that undermines

the individual's capacity for self-regulation and influences the stress response system (Hinnant, El-Sheikh, Keiley, & Buckhalt, 2013). However, there is some evidence that there may be a recalibration period during puberty in which response systems can be reset following early adversity if exposure does not continue into adolescence (Doom & Gunnar, 2013), and those with attenuated responses after puberty are thought to have been in chronically high-stress environments that continue into adolescence (Badanes, Watamura, & Hankin, 2011). Without accounting for these aspects of the participants' trauma history, we are left unclear as to whether differences between high-CU variants are related to PTSS, other trauma-related considerations, or a combination thereof.

Ultimately, longitudinal studies are necessary for understanding the psychophysiological differences in responding evidenced by youth with primary- versus acquired-CU traits. Between-group differences in cross-sectional studies may be more difficult to identify and interpret given that the acquired-CU group in particular is likely to be heterogeneous when viewed at a single point in time. The theory of acquired-CU proposes that the formation of a callous presentation is a developmental process that emerges over time, and therefore, depending on where each individual is in that process, there may be considerable within-group heterogeneity among youth in the acquired-CU group. For this study in particular, group differences may be masked by this within-group heterogeneity, such that some acquired-CU youth may look physiologically similar to their primary-CU counterparts, whereas others who are earlier in the process may appear quite different. Longitudinal studies examining youth from early childhood through adolescence and into adulthood will help elucidate whether or not these two groups of youth actually have separate pathways to the same destination, as is marginally suggested

in the results of the current study, and will also help to determine the points in development at which that destination is most similar between groups. Longitudinal studies also provide the ability to include relevant control variables, such as age of onset of trauma and chronicity of exposure (described above), to better understand how each of these details may relate to the development of CU traits. In support of these ideas, theorists have recently suggested that there may be a continuum between primary and secondary psychopathy along axes of self-control and emotional reactivity, thus proposing connections between primary and secondary psychopathy and a variety of psychiatric disorders, including borderline personality disorder, antisocial personality disorder, and narcissistic personality disorder (Yildirim & Derksen, 2015). Examination of the development of CU traits across time may illuminate whether primary- and acquired-CU should be viewed on such a continuum by elucidating how overall group presentation is similar to, or different from, that of different psychiatric disorders (e.g., whether the acquired-CU group as a whole compared to the primary-CU group more closely resembles the borderline personality disorder presentation), and how within-group heterogeneity is relevant to the potential continuum of disorders (e.g., whether some individuals within the acquired-CU group have more borderline personality features than others).

Longitudinal studies also have the potential to examine how certain theories, such as the adaptive calibration model (ACM; Del Giudice, Ellis, & Shirtcliff, 2011), might apply to the physiological profiles of high-CU youth, as well to offer a better understanding of why some youth exposed to high levels of trauma and with PTSS develop a hyporeactive physiological response pattern whereas others do not. ACM

theory proposes an interaction between a person's sensitivity to the environment and the demands of the environment across development, with a focus on adjustments to the stress response system that may be adaptive for the individual's survival and functioning. Examination of psychophysiology among high-CU youth through the lens of models such as the ACM would also allow for the integration of genetic or epigenetic influences in stress responding, and place results of various studies, which may have inconsistent findings, into a broader context that may help clarify conclusions drawn about these complex developmental processes.

Another possibility for clarifying the present results that should be investigated in future studies is the coordination between parasympathetic and sympathetic responses. Measurement of a single system increases the likelihood that reactivity may be under or overestimated in people who may be more responsive in one system than another (Orr et al., 2004). Although one strength of the present study is the inclusion of two separate measures of ANS activity, these response systems were analyzed independently. Coordinated patterns of parasympathetic and sympathetic responding can represent vulnerability or protective factors for youth. Berntson, Cacioppo, and colleagues' taxonomy for classifying individuals' parasympathetic and sympathetic responding during stress (Berntson et al., 1996; Berntson, Cacioppo, & Quigley, 1991, 1993; Cacioppo, Uchino, & Berntson, 1994) propose four profiles of coordinated responding, some of which may be more adaptive than others. Although their taxonomy has great utility, it has not been widely used in the empirical literature, and therefore, examination of these coordinated system profiles among high-CU youth classified as primary and acquired variants would make a strong contribution to the CU literature by providing a



finer-tuned examination of group differences. Another approach to examining physiological data would be to cluster youth based on their response patterns first, perhaps using the coordinated response profiles proposed by Berntson and colleagues, and then examine how the physiologically-derived groups differ on CU traits, PTSD symptoms, and other self-reported or behavioral variables of interest. Although this methodological approach would answer different questions than what were proposed in the current study, examination of data in this way would help to identify how much variability in constructs such as PTSD or CU traits is seen among youth at extreme ends of the continuum of physiological responding, and when combined with studies such as the present study, could provide a broader picture of psychophysiological patterns among high-CU youth.

Overall, the results of the present study indicate that the primary and acquired variants of CU, when examined using a cross-sectional design during adolescence, present quite similarly in terms of EDA and RSA in response to an emotionally evocative video clip, although they may differ in their recovery following exposure to a stressor. These results, if replicated by future studies, may support the notion that high-CU youth, although potentially through different pathways, arrive at the same destination regarding both self-reported and physiological callousness. However, despite their similar physiological profiles, the two high-CU groups of youth may still benefit from different clinical interventions. There is some evidence that individuals with remitted PTSS do not evidence the same autonomic dysregulation present among those with active PTSS (Shah et al., 2013). Similarly, studies of maltreated children have demonstrated that trauma-informed treatment during the preschool years can normalize cortisol responses (Fisher,

Gunnar, Chamberlain, & Reid, 2000) and result in increased (as opposed to hyporeactive) EDA by age eleven (Raine et al., 2001). Although this research is limited and little is known about the effects of trauma-focused treatments on the physiological response patterns of adolescents, these findings suggest that there may be potential for alterations to psychophysiological reactivity following treatment for PTSD, and thus, the physiologically callous presentation of acquired-CU youth may be alleviated through trauma treatment as well. Notably, although theorists have suggested that it may be difficult for youth who down-regulate their autonomic reactions to engage in trauma-focused interventions, an increased sense of safety gained in treatment may actually help them to better regulate their ANS responses (Ford, Fraleigh, Albert, & Connor, 2010). Therefore, it is possible that youth in the acquired-CU group may benefit from trauma-focused treatment, whereas individuals with primary-CU traits may benefit from different intervention strategies, such as the use of pharmacological interventions (e.g., stimulants) or behavioral therapy, which have some limited support for reducing aggression among high-CU children according to the literature (see Newcorn, 2013, for a review). Given the increasing reliance on the juvenile justice system to provide mental health care to youth (Desai et al., 2006), further attention to the issues facing high-CU youth as a heterogeneous group with varying levels and types of psychopathology is necessary to better direct treatment interventions and prevention efforts.

## APPENDIX

Additional analyses were conducted for each aim as sensitivity analyses, to examine the robustness of the results of the original models. The first set of models involved running analyses excluding youth who were taking psychotropic medications or who had medical considerations (pacemaker, insulin pump, or heart murmur) ( $n = 113$ ). The second set of models involved running analyses excluding youth who were considered statistical outliers relative to the remainder of the sample ( $n = 10$  for EDA,  $n = 5$  for RSA). The equations for the models excluding youth on the basis of medication/medical conditions or statistical outlier status were identical to the equations for the initial models. Finally, models were run controlling for youth age, ethnicity, and gender. Specifically, these variables were added as predictors to each of the Level 2 equations. The general equations for Aim 1 that included age, ethnicity, and gender as covariates were as follows:

$$L1: \beta_{0j} + \beta_{1j}*(\text{time period}_{ij}) + \beta_{2j}*(\text{time period}_{ij}) + r_{ij}$$

$$L2: \beta_{0j} = \gamma_{00} + \gamma_{01}*(\text{gender}) + \gamma_{02}*(\text{ethnicity}) + \gamma_{03}*(\text{age}) + \gamma_{04}*(\text{group}_j) + \\ \gamma_{05}*(\text{group}_j) + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{gender}) + \gamma_{12}*(\text{ethnicity}) + \gamma_{13}*(\text{age}) + \gamma_{14}*(\text{group}_j) + \\ \gamma_{15}*(\text{group}_j)$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}*(\text{gender}) + \gamma_{22}*(\text{ethnicity}) + \gamma_{23}*(\text{age}) + \gamma_{24}*(\text{group}_j) + \\ \gamma_{25}*(\text{group}_j)$$

where, as with the initial models,  $i$  is time and  $j$  is person, the time period variables were dummy coded to indicate either baseline, video task, or recovery period as the reference, and group variables were also dummy coded to indicate either the primary-, acquired-, or lower-CU comparison group as the reference. The general equations for Aim 2 that included age, ethnicity, and gender as covariates were as follows:

$$L1: \beta_{0j} + \beta_{1j} * (\text{time period}_{ij}) + r_{ij}$$

$$L2: \beta_{0j} = \gamma_{00} + \gamma_{01} * (\text{gender}) + \gamma_{02} * (\text{ethnicity}) + \gamma_{03} * (\text{age}) + \gamma_{04} * (\text{group}_j) + \\ \gamma_{05} * (\text{group}_j) + \gamma_{06} * (\text{baseline score}) + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11} * (\text{gender}) + \gamma_{12} * (\text{ethnicity}) + \gamma_{13} * (\text{age}) + \gamma_{14} * (\text{group}_j) + \\ \gamma_{15} * (\text{group}_j)$$

where  $i$  is time and  $j$  is person, and baseline score is grand centered.

### Aim 1

For models with RSA as the outcome, group differences did not emerge in subsequent models excluding outliers, excluding youth with relevant medication or medical considerations, or when controlling for age, ethnicity, and gender, although in the latter model, within-group changes across periods were also no longer significant. Additionally, gender emerged as a significant main effect on individuals' average RSA during the video task for all three groups, as well as the change from video to recovery. Overall, the results of these alternative models indicated that model results did not change on the basis of medication or medical conditions, outliers, or the inclusion of age, gender, and ethnicity.

For models with EDA as the outcome, results diverged more from those found in the original models. When outliers were excluded, only the video to recovery difference

between primary- and lower-CU youth remained significant. The same pattern was observed for a model excluding youth with relevant medications and medical considerations, and the primary group's change from baseline to video was also larger than the lower-CU group's change in that period,  $B = .21$ ,  $SE = .08$ ,  $p = .009$ . When controlling for age, ethnicity, and gender, the significant difference between the primary- and lower-CU groups in the difference between baseline and recovery remained significant, as did the difference between the primary- and lower-CU groups in the slope from video to recovery. These alternative models also revealed main effects for both gender and ethnicity on baseline EDA, a main effect of ethnicity on the slope from baseline to video, and a main effect of gender on recovery EDA, for all three groups. Overall, results from these alternative models indicate that the patterns of results identified in the initial models are largely robust, although ethnicity and gender may also be related to EDA. Results for these alternative models for EDA and RSA, with time centered at the video task and the acquired-CU group as reference, are displayed in Table 4.

### Aim 2

For models with RSA as the outcome, group differences did not emerge in subsequent models excluding outliers, excluding youth with relevant medication or medical considerations, or when controlling for age, ethnicity, and gender. For the models without medications and medical considerations as well as when controlling for age, ethnicity, and gender, group slopes across video and recovery did not significantly differ from zero. Gender, but not age or ethnicity, emerged as a significant main effect on the mean RSA at the midpoint of the video task for all three groups, and of mean RSA at

the end of the video task for the primary group only. Ethnicity, but not age or gender, emerged as a significant main effect on slope at the midpoint of the recovery period for all three groups. Results for these alternative models, with time centered at the midpoint of the video and recovery periods, respectively, and the acquired-CU group as the reference group are presented in Table 5. Notably, an additional alternative model was run to examine differences between groups in mean scores as well as in simple slopes across the recovery period that controlled for the individual's mean score during the video task rather than during the baseline period. The results of this model with RSA as the outcome also did not result in any significant group differences.

For models with EDA as the outcome examining the video task period, group differences did not emerge in models excluding outliers, excluding youth with relevant medication or medical considerations, or when controlling for age, ethnicity, and gender. When youth with medication and medical considerations were excluded, the slope of time was no longer significant for any groups at the beginning, midpoint, or end of the video task period. The slope of time also failed to be significantly different from zero for certain models excluding youth who were identified as statistical outliers. When controlling for age, ethnicity, and gender, age emerged as a significant main effect on mean EDA at baseline for all groups at the beginning of the video period task whereas ethnicity emerged as a significant main effect on mean EDA at the end of the video task period. Age and ethnicity both also significantly influenced the slope as measured at the beginning, midpoint, and end of the video period. No significant effects for gender were observed. For models with EDA in the recovery period as the outcome, the initial results in terms of group differences were maintained when excluding statistical outliers. The

model controlling for age, gender, and ethnicity also did not result in any changes in group differences as compared to the initial model, although the slopes for each group were no longer significantly different from zero at the beginning, midpoint, or end of the recovery period. When excluding youth taking medications or with medical considerations, no significant group differences were observed, suggesting that the results of the initial model were not robust when medication and medical issues were considered. Results for these alternative models, with time centered at the midpoint of the video and recovery periods, respectively, and the acquired-CU group as the reference group are presented in Table 6. Notably, an additional alternative model was run to examine differences between groups in mean scores as well as in simple slopes across the recovery period that controlled for the individual's mean score during the video task rather than during the baseline period. The results of this model with EDA as the outcome indicated that the group differences identified when controlling for baseline EDA were maintained when controlling for video EDA instead.

To summarize, gender, age, or ethnicity were each related to physiological measurements in at least one analytic model tested in the present study, and thus warrant consideration in future studies of this nature. However, inclusion of these covariates generally did not alter between-group differences identified in the initial models, suggesting that these variables may influence RSA and EDA independently of CU group membership. The results of these alternative models also indicate that overall, the group differences identified in the initial models are largely robust to outlier influence, as models excluding youth identified as statistical outliers continued to evidence significant group differences. Generally, the exclusion of youth who were taking psychotropic

medications or had relevant medical considerations did change results. Notably, these models excluded roughly one third of the full sample, thus changing the makeup of the sample significantly. Further examination of how youth in the juvenile justice system who are medicated differ from those who are not is warranted, as are further studies devoted to understanding the effects of including versus excluding these youth in future studies of psychophysiological responding.



Table 4  
*Additional Analyses for Aim 1: Final Estimation of Fixed Effects (with Robust Standard Errors) Across Models with the Video Task and Acquired-CU Group as Reference, for RSA (Top) and EDA (Bottom) as Outcome*

	Model 1	Model 2	Model 3
For INTERCEPT			
Intercept	6.53 (.18)***	6.45 (.14)***	5.37 (.82)***
Gender			0.35 (.14)*
Ethnicity			0.002 (.04)
Age			0.03 (.05)
Primary vs. Not	-0.07 (.23)	0.10 (.18)	0.06 (.20)
Lower-CU vs. Not	0.11 (.20)	0.08 (.16)	0.12 (.17)
For BASELINE slope			
Intercept	0.32 (.14)*	0.34 (.08)***	0.76 (.47)
Gender			-0.09 (.07)
Ethnicity			-0.01 (.03)
Age			-0.01 (.03)
Primary vs. Not	0.02 (.16)	0.01 (.11)	0.04 (.11)
Lower-CU vs. Not	-0.06 (.15)	-0.04 (.10)	-0.03 (.10)
For RECOVERY slope			
Intercept	0.13 (.11)	0.20 (.09)*	0.63 (.43)
Gender			-0.14 (.07)*
Ethnicity			0.04 (.03)
Age			-0.02 (.03)

Table 4 cont.

	Model 1	Model 2	Model 3
Primary vs. Not	0.04 (.12)	-0.02 (.11)	0.02 (.11)
Lower-CU vs. Not	0.07 (.12)	0.02 (.10)	0.02 (.10)
For INTERCEPT			
Intercept	1.97 (.16)***	1.73 (.12)***	0.81 (.61)
Gender			0.19 (.10)
Ethnicity			-0.003 (.03)
Age			0.04 (.03)
Primary vs. Not	0.19 (.18)	0.23 (.15)	0.21 (.14)
Lower-CU vs. Not	-0.10 (.17)	0.07 (.13)	0.03 (.14)
For BASELINE slope			
Intercept	-0.45 (.10)	-0.48 (.09)***	-0.03 (.34)
Gender			0.07 (.08)
Ethnicity			-0.07 (.02)**
Age			-0.02 (.02)
Primary vs. Not	-0.16 (.12)	-0.04 (.11)	-0.03 (.10)
Lower-CU vs. Not	0.05 (.11)	0.05 (.10)	0.07 (.09)
For RECOVERY slope			
Intercept	-0.66 (.14)***	-0.73 (.11)***	-0.72 (.56)
Gender			0.14 (.14)
Ethnicity			-0.03 (.02)

Table 4 cont.

	Model 1	Model 2	Model 3
Age			-0.01 (.03)
Primary vs. Not	-0.29 (.16)	-0.21 (.13)	-0.22 (.12)
Lower-CU vs. Not	0.06 (.16)	0.02 (.12)	0.08 (.12)

*Note.* All models for EDA as the outcome were run using a Poisson distribution and population-average model results are displayed. Model 1 excluded youth with relevant medical conditions or psychotropic medications, Model 2 excluded youth whose data evidenced statistical outliers, and Model 3 controlled for age, ethnicity, and gender.

Table 5  
*Final Estimation of Fixed Effects (with Robust Standard Errors) Across Models for Aim 2 for the Video Task (Top) and Recovery Period (Bottom) with the Acquired-CU Group and Time Centered at Midpoint for RSA as the Outcome*

	Model 1	Model 2	Model 3
For INTERCEPT			
Intercept	6.54 (.13)***	6.48 (.08)***	6.06 (.45)***
Gender			0.15 (.06)*
Ethnicity			0.01 (.06)
Age			0.01 (.03)
Primary vs. Not	0.0001 (.15)	0.03 (.10)	-0.01 (.11)
Lower-CU vs. Not	0.07 (.14)	0.06 (.09)	0.05 (.09)
Baseline Mean	0.76 (.06)***	0.81 (.03)***	0.78 (.04)***
For Slope at Midpoint			
Intercept	-0.06 (.03)*	-0.06 (.02)**	0.03 (.14)
Gender			0.004 (.02)
Ethnicity			-0.01 (.01)
Age			-0.01 (.01)
Primary vs. Not	0.04 (.04)	0.001 (.03)	0.01 (.03)
Lower-CU vs. Not	0.01 (.04)	0.01 (.03)	0.02 (.03)
For INTERCEPT			
Intercept	6.71 (.11)***	6.68 (.09)***	6.78 (.43)***
Gender			-0.01 (.06)
Ethnicity			0.05 (.02)*
Age			-0.02 (.03)

Table 5 cont.

	Model 1	Model 2	Model 3
Primary vs. Not	-0.03 (.14)	-0.01 (.11)	-0.002 (.11)
Lower-CU vs. Not	0.07 (.13)	0.04 (.09)	0.02 (.10)
Baseline Mean	0.75 (.10)***	0.86 (.03)***	0.81 (.06)***
For Slope at Midpoint			
Intercept	-0.16 (.06)*	-0.08 (.05)	0.03 (.26)
Gender			-0.01 (.05)
Ethnicity			0.01 (.02)
Age			-0.01 (.01)
Primary vs. Not	0.03 (.07)	-0.01 (.06)	-0.002 (.06)
Lower-CU vs. Not	0.07 (.07)	-0.02 (.05)	-0.01 (.05)

*Note.* Mean baseline score was grand mean centered. Model 1 excluded youth with relevant medical conditions or psychotropic medications, Model 2 excluded youth whose data evidenced statistical outliers, and Model 3 controlled for age, ethnicity, and gender. \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

Table 6  
*Final Estimation of Fixed Effects (with Robust Standard Errors) Across Models for Aim 2 for the Video Task (Top) and Recovery Period (Bottom) with the Acquired-CU Group and Time Centered at Midpoint for EDA as the Outcome*

	Model 1	Model 2	Model 3
<hr/> For INTERCEPT			
Intercept	-0.15 (.14)	-0.26 (.14)	-1.01 (.58)
Gender			-0.02 (.12)
Ethnicity			0.05 (.04)
Age			0.04 (.03)
Primary vs. Not	0.20 (.17)	0.07 (.17)	0.06 (.16)
Lower-CU vs. Not	-0.02 (.17)	-0.01 (.16)	-0.03 (.14)
Baseline Mean	0.15 (.01)***	0.17 (.01)***	0.16 (.001)***
<hr/> For Slope at Midpoint			
Intercept	0.003 (.03)	-0.03 (.03)	0.33 (.14)*
Gender			-0.05 (.03)
Ethnicity			0.03 (.01)**
Age			-0.02 (.01)**
Primary vs. Not	-0.001 (.04)	0.01 (.04)	0.04 (.04)
Low-CU vs. Not	-0.03 (.04)	-0.01 (.03)	0.01 (.04)
<hr/> For INTERCEPT			
Intercept	-0.26 (.20)	-0.53 (.18)**	-1.29 (.71)
Gender			0.12 (.13)
Ethnicity			0.03 (.04)
Age			0.03 (.04)

Table 6 cont.

	Model 1	Model 2	Model 3
Primary vs. Not	-0.22 (.23)	-0.24 (.21)	-0.27 (.19)
Low-CU vs. Not	-0.16 (.22)	-0.10 (.19)	-0.08 (.18)
Baseline Mean	0.15 (.01)***	0.17 (0.01)***	0.16 (.01)***
For Slope at Midpoint			
Intercept	-0.02 (.08)	0.03 (.07)	0.54 (.29)
Gender			-0.04 (.05)
Ethnicity			-0.03 (.05)
Age			-0.02 (.08)
Primary vs. Not	-0.10 (.10)	-0.19 (.08)*	-0.17 (.08)*
Low-CU vs. Not	-0.05 (.09)	-0.09 (.08)	-0.08 (.07)

*Note.* Models were run using a Poisson distribution and population-average model results are displayed. Mean baseline score was grand mean centered. Model 1 excluded youth with relevant medical conditions or psychotropic medications, Model 2 excluded youth whose data evidenced statistical outliers, and Model 3 controlled for age, ethnicity, and gender.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

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