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# Female primary and secondary psychopathic variants show distinct endocrine and psychophysiological profiles

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ABSTRACT

Research with predominantly male samples supports primary and secondary developmental pathways to psychopathy that are phenotypically indistinguishable on aggressive and antisocial behavior. The aim of this study was to examine whether female variants of psychopathy show divergent endocrine (i.e., cortisol, dehydroepiandrosterone [DHEA], testosterone, and their ratios) and psychophysiological (i.e., heart rate variability [HRV]) reactivity to social provocation. We also tested whether variants differed on reactive aggression when performing a competitive reaction time task against the fictitious participant who previously insulted them. Latent profile analyses on 101 undergraduate women oversampled for high psychopathic traits identified a high-anxious, maltreated secondary variant ($n = 64$) and a low-anxious primary variant ($n = 37$). Although variants did not differ on aggression, secondary variants showed higher cortisol, testosterone, cortisol-to-DHEA ratios, and HRV following social provocation relative to primary variants. Findings suggest that the neurobiological mechanisms underpinning aggression in psychopathy may differ between women on primary versus secondary developmental pathways.

## 1. Introduction

Interest in the neurobiological underpinnings of antisocial behavior has typically focused on the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis. However, as emphasized in recent reviews, the association between antisocial behavior and physiological indices of stress and emotional responding may be more complex than previously thought—perhaps explaining the divergent findings in research to date (Alink et al., 2008; Fanti, 2018). Growing recognition of heterogeneity among antisocial individuals has helped to refine developmental models of antisocial behavior in recent years. Research guided by the construct of psychopathy has been particularly informative in this regard (Frick et al., 2014). Antisocial individuals scoring high on psychopathic traits exhibit a particularly severe and chronic trajectory of antisocial behavior, in addition to abnormalities across a range of physiological indices relative to other antisocial individuals (Fanti, 2018). Within individuals with psychopathic traits, the construct of anxiety has further clarified distinct neurobiological correlates, suggesting multiple developmental pathways to both antisocial behavior and psychopathic traits. In recent years, this research has fueled interest in primary and secondary subtypes, or variants, of psychopathic traits that is rooted in the writing of early theorists such as Benjamin Karpman (1941).

Primary psychopathy is thought to be underpinned by a genetic constitution or dispositional deficits in emotional responsivity (Cleckley et al., 1976), whereas experiences of social and environmental adversity are central to theories of secondary psychopathy (Karpman, 1941). The dominant strategy for identifying psychopathy variants in contemporary subtyping research is clustering methods and the indicators tend to include psychopathy facets (Hicks et al., 2012; Mokros et al., 2015) in combination with childhood maltreatment and/or anxiety symptoms (Kahn et al., 2013; Kimonis et al., 2011). Psychopathy variant groups are then validated against theoretically and empirically relevant variables (Goulter et al., 2017; Kimonis et al., 2012). An accumulating body of largely descriptive research on psychopathy variants among predominately-male samples has revealed remarkably similar findings across development and population setting. Secondary psychopathy variants are distinguished from primary variants by their greater histories of psychological distress and depression, post-traumatic stress disorder (PTSD) and borderline personality disorder (BPD)

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symptomatology, and their absence of emotional deficits that are observed among primary variants (Kimonis et al., 2012; Skeem et al., 2007).

Despite these many differences, psychopathy variants are phenotypically indistinguishable with respect to observable characteristics of callous and antisocial behaviors (Karpman, 1941; Kimonis et al., 2012). Research comparing psychopathy variants on aggression is inconsistent, however. Some studies find that reactive aggression (i.e., impulsive, retaliatory, typically associated with high emotion and physiological arousal) is more common among secondary than primary psychopathy variants for whom both proactive and reactive aggression is more common (Poythress et al., 2010). Other studies find that community and undergraduate individuals with secondary psychopathy score higher than those with primary psychopathy on both proactive (i.e., premeditated, not typically preceded by an emotional reaction or high levels of physiological arousal) and reactive aggression (Falkenbach et al., 2008), or report no differences between clinic-referred variants on aggression behavior (Kahn et al., 2013). However, this literature overwhelmingly relies on self-report measures of aggression. Laboratory paradigms that have proven useful for objectively examining aggression may offer additional insights into how primary versus secondary variants respond to provoking stimuli (Chester and Lasko, 2018). Research has found that these tasks also elicit changes in endocrine and psychophysiological responding (Denson et al., 2013; Memedovic et al., 2010). Given that the persistent aggressive behavior of psychopathic individuals is attributed to their deficits in emotional processing that interfere with moral socialization, and evidence that secondary variants fail to show such deficits (Kimonis et al., 2012, 2017), it would stand to reason that primary and secondary psychopathy variants may react differently to social provocation.

Beyond the aforementioned failure to distinguish important heterogeneity in antisocial individuals on the basis of psychopathic traits and anxiety levels, equivocal findings in neurobiological mechanisms of aggression may also be due in part to past research failing to examine sex differences. For example, women scoring high on psychopathy do not show the blunted cortisol reactivity response to a stressor compared to high scoring men (e.g., O'Leary et al., 2007). Additionally, dysregulation to stress may be more accurately captured by examining multiple hormones in concert. Chronic exposure to stressors, as is common among secondary psychopathy variants, disrupts the ability of the stress response system to maintain homeostasis, resulting in hypertrophy of the adrenal glands and system dysregulation (McEwen, 1998). In particular, dehydroepiandrosterone (DHEA) has emerged as a promising hormonal biomarker that parallels and interacts with cortisol to influence risk and resilience. DHEA follows a similar but less pronounced diurnal rhythm to cortisol and is co-released with cortisol under stress. It has a protective anti-glucocorticoid effect by buffering the HPA axis from the neurotoxic effects of prolonged high cortisol levels, and aiding the system in returning to homeostasis (Young et al., 2002). A high basal cortisol-to-DHEA ratio is thought to index increased chronic stress exposure and is associated with poor mental health (Cicchetti et al., 2015; Markopoulou et al., 2009). For example, aggressive adolescent secondary psychopathy variants had higher basal cortisol-to-DHEA ratios and more comorbid psychopathology than aggressive and nonaggressive primary variants who were characterized by low to average psychopathology and high basal levels of DHEA (Kimonis et al., 2016). Research examining tactics for modeling coordinated effects of multiple salivary analytes have found that ratios, which emphasize the relative strength of the two hormones, are most common in the field, better predict psychopathology than interactive effects that emphasize absolute hormone levels, and useful when it is hypothesized that two analytes have opposing effects (Chen et al., 2015; Kamin and Kertes, 2017).

Whereas cortisol-to-DHEA ratios are implicated in the experience of chronic adversity and are associated with stress-related psychopathology (i.e., anxiety, depression), the testosterone-to-cortisol ratio is associated with aggression (Montoya et al., 2012). Testosterone is a steroid hormone secreted by the hypothalamic-pituitary-gonadal (HPG) axis in both sexes (Mazur and Booth, 1998). According to the testosterone-to-cortisol ratio hypothesis (Terburg et al., 2009), imbalanced levels of testosterone to cortisol increase risk for aggressive behavior. Consistent with this model, total psychopathy scores were associated with an increased testosterone-to-cortisol ratio to a social stressor, controlling for sex (Glenn et al., 2011). However, some research finds sex differences in the effect of interacting HPA-HPG hormones on aggression. To illustrate, high testosterone levels predicted greater reactive aggression when cortisol concentrations were high but not when concentrations were low-average in a study of female undergraduates (Denson et al. (2013). The authors posited that high cortisol concentrations may reduce dominant behavior in men but have the opposite effect in women by increasing negative emotionality, in turn, increasing reactive aggression. This study, however, examined the interaction of testosterone and cortisol (i.e., moderation), and it is important to extend these findings to testosterone-to-cortisol ratios given research supporting the use of endocrine ratios in the prediction of antisocial behavior (Chen et al., 2015). Despite their importance to models of aggression, this is the first study to comprehensively examine both HPA and HPG hormones among psychopathy variants exposed to a provocative social stressor.

Like the endocrine system, the ANS has also been implicated in stress and emotion regulatory processes. Heart rate variability (HRV) is the interaction of SNS and parasympathetic nervous system (PNS) arms of the ANS on cardiac function. In response to a stressor the SNS readies the individual for fight, flight, or freeze by producing catecholamine mediated responses inducing changes to target organs (Cacioppo et al., 1998). In contrast, PNS activation serves to restore homeostatic control of organ function and counteract excitation (Porges, 2007). High HRV (i.e., low arousal) indexes autonomic flexibility, which is characterized by successful adaptation to environmental and physiological demands. By contrast, low HRV (i.e., high arousal) is associated with system dysregulation (Thayer et al., 2012). Reduced HRV is implicated as a biomarker of a number of problems relevant to secondary psychopathy (e.g., anxiety, depression, PTSD, and childhood maltreatment; Meyer et al., 2016), but high HRV is associated with interpersonal psychopathic traits, perhaps due to their negative association with anxiety and psychopathology (Hansen et al., 2007).

While extensive research has found associations between dysregulated reactivity of the HPA axis and ANS and negative outcomes, less research has examined the responsivity of both highly interconnected systems (Del Giudice et al., 2012). The stress response system is hierarchically organized with the most immediate response from the PNS, followed by the SNS and the HPA axis (Del Giudice et al., 2011). In contrast to sympathetic mediation, parasympathetic effects are fast (milliseconds versus seconds) and it is the parasympathetic system that influences the beat-to-beat changes of the heart known as HRV. Not all systems activate in response to a stressor and typically, where parasympathetic activation is insufficient in coping with the demand, the second layers of stress response will activate. Research examining multisystem models have typically focused on the opposing arms of the ANS (i.e., SNS and PNS; El-Sheikh et al., 2013) or the HPA axis and the SNS in response to laboratory stressors or among individuals exposed to family conflict (e.g., Gordis et al., 2008; Koss et al., 2014). However, examining both endocrine and parasympathetically mediated HRV in concert may provide a more complete understanding of differences in the psychobiological bases of aggression between primary and secondary psychopathy variants.

### 1.1. The present study

This study aimed to uniquely contribute to the field by examining endocrine and psychophysiological biomarkers associated with stress and aggression among a young adult female community population oversampled for psychopathic traits. While there is a heavy focus in the literature on adult forensic populations, research suggests the psychopathy construct is dimensional rather than taxonic in nature (Edens et al., 2006; Guay et al., 2007). Thus, the disorder is best understood as the extreme of a continuum of psychopathic personality traits that are evident in both community and forensic populations, and research is increasingly using community samples to gain insights into the disorder (e.g., Dindo and Fowles, 2011; Heritage and Benning, 2013; Maes and Brazil, 2015). In line with past research (Kahn et al., 2013; Kimonis et al., 2011), latent profile analysis of psychopathy scores, maltreatment history, and anxiety was used to identify groups, which were then validated against known correlates, namely high psychopathology (depression, PTSD, BPD symptomatology) and low parental warmth for secondary psychopathy variants relative to primary variants. It was hypothesized that secondary psychopathy variants would show higher cortisol, testosterone, and cortisol-to-DHEA ratios and lower testosterone-to-cortisol ratios, coupled with lower HRV indicative of system dysregulation at baseline and post-provocation, relative to primary variants that would show higher DHEA concentrations. It was further hypothesized that secondary psychopathy variants would show greater reactive aggression during a competitive reaction time task (CRTT) aggression paradigm performed against the provocateur.

## 2. Method

### 2.1. Participants

Participants included 101 community and undergraduate women ($M$ age = 19.02 years, $SD$ = 1.50) oversampled for high psychopathic traits via online prescreening questionnaires (28 items). These questionnaires included abbreviated versions of the Inventory of Callous-Unemotional Traits (ICU, items: 1, 3, 5, 6, 8, 13, 15, 16, 17, 24; Frick, 2004) selected from four and nine item criterion sets considered for the DSM-5 limited prosocial emotions (LPE) specifier to conduct disorder on the basis of factor analytic research (see Kimonis et al., 2015), and the meanness scale of the Triarchic Psychopathy Measure (TriPM, items 6, 8, 11, 14, 17, 20, 23, 26, 29, 33, 36, 39, 40, 42, 45, 48, 52, 55; Patrick, 2010). Items are rated on a four-point scale (ICU; 0 '*not at all true*', 1 '*somewhat true*', 2 '*very true*', 3 '*definitely true*'; TriPM, 0 '*false*', 1 '*somewhat false*', 2 '*somewhat true*', 3 '*true*'), and participants had to endorse a mean score of > 1 across both measures to be eligible.[1] Exclusion criteria included; oral infections, taking hormonal birth control, pregnant or breastfeeding; using certain medications (e.g., immunosuppressive agents, glucocorticoids, beta-blockers); or any cancer, immune, cardiovascular, metabolic, or kidney disorders; due to the impact these disorders have on the HPA axis. Participants were also asked to refrain from strenuous exercise and consuming any food or liquid (except water) two hours prior to the study. The majority of the participants identified as Chinese ($n$ = 45; 44.1%) and White Australian ($n$ = 14; 13.7%), while the remainder identified as other Asian ($n$ = 19), Indian ($n$ = 5), European ($n$ = 4), African ($n$ = 2), Middle Eastern ($n$ = 2), or Pacific Islander ($n$ = 2). Nine participants did not report their ethnicity. Participants completed the study as part of their psychology course requirements or were compensated with AUD$20 if recruited from the university wide participant pool.

[1] Participant's prescreening scores were significantly greater than the screening sample of ineligible undergraduates: ICU, participants, $M$ = 1.48, $SD$ = .36, range = .50-2.10; screening sample, $M$ = .82, $SD$ = .32, range = .00-1.70, $t$ (1318) = 19.29, $p$ < .001, $d$ = 1.06; TriPM, participants, $M$ = 1.17, $SD$ = .34, range = .30-2.10; screening sample, TriPM, $M$ = .39, $SD$ = .22, range = .00-1.20, $t$ (1319) = 22.53, $p$ < .001, $d$ = 1.24; mean score across both measures, participants $M$ = 1.32, $SD$ = .29, range = .50-1.90, screening sample $M$ = .60, $SD$ = .21, range = .00-1.05, $t$ (1317) = 24.32, $p$ < .001, $d$ = 1.34.

### 2.2. Procedure

Approval for all study procedures was obtained from the university ethics committee. Informed written consent was obtained from all participants prior to data collection. Data collection occurred during a two hour session, in which the participant remained seated the entire duration, between the hours of 13:00–19:00 to permit the flattening of hormone levels and minimize the influence of circadian rhythm on findings (Nicolson, 2007). Participants were given approximately 20 min to acclimate to the laboratory environment while they read over ethical approval forms and provided information on possible exclusionary criteria before the first salivary sample was taken. Next, participants provided the first of two 3 ml saliva samples using the passive drool method—one prior to data collection and one approximately 60 min later, which equates to approximately 20 min post-provocation (i.e., negative feedback)/10 min after completion of the full post-provocation procedure. Research finds that, for most individuals, salivary cortisol reaches peak levels at approximately 10–30 min post stressor, DHEA at approximately 20 min post stressor, and testosterone between 10–40 minutes post stressor (Bedgood et al., 2014; Denson et al., 2013; Kirschbaum and Hellhammer, 1994; Lennartsson et al., 2012). Thus, 20 min post negative feedback was chosen to most accurately capture reactive levels of cortisol, DHEA, and testosterone. Participants then completed questionnaires on a computer, including a pre-provocation angry affect checklist (Positive and Negative Affect Scale, PANAS; Watson et al., 1988). Participants were then connected to the psychophysiological equipment, which collected data for the remainder of the study session. Participants' skin below each clavicle and on the back of their neck was cleaned using isopropyl alcohol swaps. Disposable Ag/AgCl electrodes were connected to snap lead wires connected to a Dual Bio amplifier. The ground electrode was placed on the back of the participants' neck, and two electrodes were placed below each of the clavicle bones. The experimenter collected five minutes of resting electrocardiography (ECG) data (Shaffer and Ginsberg, 2017). Then participants completed the social provocation and competitive reaction time task (CRTT). Finally, participants completed the second post-provocation PANAS checklist, demographic information, and provided the final post-task saliva sample. All participants were then debriefed on the social provocation deception. Salivary samples were stored immediately in the freezer at -20 ° Celsius. The experimenters were all female.

### 2.3. Measures

#### 2.3.1. Questionnaires

*2.3.1.1. Demographic information.* Participants self-reported their age, race/ethnicity, and whether English was their first and best language.

*2.3.1.2. Angry affect.* Angry affect was assessed with eight items from the self-report PANAS (Watson et al., 1988) administered before and after social provocation. Participants rated to what extent they felt the emotions 'at this moment' on a five-point scale (1 '*very slightly*' to 5 '*extremely*'). Internal consistency of anger scores was good to excellent (α = .84–91).

#### 2.3.2. Grouping variables

*2.3.2.1. Psychopathic traits.* Psychopathic traits were assessed with the self-report Psychopathic Personality Inventory-Short Version (PPI-SV; Vaughn et al., 2008). The 56-item PPI-SV is based on the full 187-item PPI, and items are rated on a four-point scale (1 '*false*' to 4 '*true*'). Factor analyses suggest that PPI measures are composed of an affective-

interpersonal factor (F1) and an impulsivity factor (F2). Internal consistency was acceptable for PPI total scores (α = .72), affective-interpersonal (α = .79) and impulsivity (α = .76) subscales.

*2.3.2.2. Childhood maltreatment.* The Childhood Trauma Questionnaire (CTQ; Bernstein and Fink, 1998) is a 28-item self-report measure and items are rated on a five-point scale from '*never true*' to '*very often true*'. Internal consistency was good (α = .89). Using Walker et al. (1999) thresholds for determining whether women have been exposed to a clinically meaningful degree of childhood maltreatment, 29.8% of this sample selected for high psychopathic traits reported physical abuse, 40.7% physical neglect, 52.5% emotional abuse, 17.9% emotional neglect, and 13% sexual abuse.

*2.3.2.3. Anxiety*. Participants self-reported anxiety using the trait form of the State-Trait Anxiety Inventory (STAI-T; Spielberger, 1983). The STAI-T consists of 20 items assessing dimensions of excessive worry, tension, low self-esteem, and demoralization. The respondent is asked to reflect on 'how you generally feel' in regards to each item and endorse '*almost never*' to '*almost always*' on a four-point scale. Internal consistency was good (α = .89).

*2.3.3. Validating variables*

*2.3.3.1. Parental warmth.* The Parental Bonding Instrument-Care Scale (PBI; Parker, 1990) measures maternal ($n$ = 25 items) and paternal ($n$ = 25 items) warmth on a four-point scale (0 '*very unlike*' to 3 '*very like*'). A combined score was created, with excellent internal consistency (α = .90).

*2.3.3.2. Depression symptoms*. The Brief Symptom Inventory (BSI; Derogatis, 1993) was used to assess depression. Participants rated on a five-point (0 '*not at all*' to 5 '*extremely*') scale how relevant each item is to their experience in the past seven days. Internal consistency for BSI depression was good (α = .84).

*2.3.3.3. Post-traumatic stress symptoms*. PTSD symptoms were assessed with the PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 1993). Respondents indicated how distressed they were by each symptom in the past month (1 '*not at all*' to 5 '*extremely*'). PCL-5 total scores demonstrated excellent internal consistency (α = .94).

*2.3.3.4. Borderline personality symptoms*. Borderline personality features were assessed with the Minnesota Borderline Personality Disorder Scale (MBPD; Bornovalova et al., 2011). The MBPD is a 19-item scale measuring stress reaction, alienation, control, aggression, well-being, and absorption on a four-point scale (1 '*definitely false*' to 4 '*definitely true*'). BPD total scores demonstrated good internal consistency (α = .84).

*2.3.4. Aggression variables*

*2.3.4.1. Aggression questionnaire.* Participants' self-report of aggression was measured using the Peer Conflict Scale (PCS; Marsee et al., 2011). The PCS is a 40-item self-report scale that includes items measuring reactive (relational and overt) and proactive (relational and overt) aggression. Items are rated on a four-point scale (0 '*not at all true*' to 3 '*definitely true*'). Internal consistencies were good in the present study (α = .84–.93). Proactive and reactive aggression scores were skewed and square-root transformed for analyses (pre-transformation values: proactive, $M$ = 6.76, $SD$ = 8.52; reactive, $M$ = 7.52, $SD$ = 7.50; post-transformation values are reported in Table 1.

*2.3.4.2. Provocation procedure*. The provocation procedure is described in more detail elsewhere (Denson et al., 2013) but in brief, participants were given 10 min to prepare a 2-minute speech. After the speech the participant was ostensibly verbally insulted by another fictitious participant. Subsequently, participants completed a competitive reaction time task (CRTT) in which they could deliver blasts of white noise to the same 'participant' who previously insulted them. In line with past research using this task (Bushman and Baumeister, 1998; Konijn et al., 2007), although the procedure involves 25 trials, the first trial provides the best measure of reactive aggression to the social provocation as participants had recently been insulted by the fictitious participant but had not yet received a noise blast from her. After this first trial, the participant matches her response to her partner's, consistent with research demonstrating reciprocation norms in aggressive behavior (Axelrod, 1984). The intensity and duration of the noise blasts are found to be strongly correlated ($r$ = .94, $p$ < .001), so reactive aggression was operationalized as the composite of intensity and duration scores (0–10) selected by participants on the first trial. Several reviews and meta-analyses have examined the validity of the social provocation paradigm and support its use as a valid and reliable laboratory measure of reactive aggression (Anderson and Bushman, 1997; Chester and Lasko, 2018; Giancola and Parrott, 2008; Giancola and Zeichner, 1995).

*2.3.5. Salivary hormone determination*

Salivary cortisol (in micrograms/decilitre [mg/dL]), DHEA (in picograms/millilitre [pg/ml]), and testosterone (in pg/ml) were assayed using commercially available enzyme immunoassay kits (Stratech Scientific). All samples (baseline [T1] and post-provocation [T2]) from each subject were assayed in the same run and were tested in duplicate. The cortisol test used 25 µl of saliva and has a lower limit of sensitivity of 0.012 µg/dl, the DHEA test used 50 µl of saliva and has a minimum detection limit of 10.2 pg/ml, the testosterone test used 25 µl of saliva and has a lower limit of sensitivity of 6.1 pg/ml. Average intra- and interassay coefficients of variation were required to be less than 10% and 15%, respectively. Cortisol values in mg/dL, and DHEA and testosterone values in pg/ml were transformed into nanomoles/litre values (conversion factors are x 27.59 and x 3.47, respectively) prior to computing hormone ratios, as per guidelines provided by Sollberger and Ehlert (2016). Cortisol and DHEA variables were skewed and were square-root transformed before ratios were calculated (pre-transformation values: cortisol T1, $M$ = 239.56, $SD$ = 138.84; cortisol T2, $M$ = 175.19, $SD$ = 128.95; DHEA T1, $M$ = 960.42, $SD$ = 333.09; DHEA T2, $M$ = 950.94, $SD$ = 321.99; post-transformation values are reported in Table 1).

*2.3.6. Psychophysiological data acquisition and reduction*

ECG signals were recorded digitally at a sampling rate of 1000 Hz during the five-minute rest period, the speech task, the feedback, and the CRTT, and processed offline using LabChart 8 software (ADInstruments Inc., Bella Vista, NSW, Australia). Electronic markers were included in the task program so psychophysiological data could be extracted accurately for each task component. Data from each participant were visually inspected for erroneous interbeat intervals and errors were corrected in LabChart 8. HRV frequency indices provide information on the overall variance from oscillations of heart rate at various frequencies, the time domain indexes either the intervals between heart beats or the lengths of adjacent cycles (Stein et al., 1994). The Root Mean Square of the Successive Differences (RMSSD) time variable represents parasympathetically mediated HRV. RMSSD is one of the most commonly used variables to assess HRV autonomic

functioning, with high scores indexing flexibility to environmental stress and low scores being associated with emotion dysregulation (Thayer et al., 2012). LabChart 8 HRV software computes RMSSD indices from raw ECG data. Distributions of RMSSD were significantly skewed, as is typical with psychophysiological data and values were log transformed for analyses (pre-transformation values: resting, $M = 60.64$, $SD = 29.89$; speech, $M = 84.36$, $SD = 35.95$; feedback, $M = 60.73$, $SD = 39.92$; CRTT, $M = 68.35$, $SD = 37.44$; post-transformation values reported in Table 1).

### 2.4. Statistical analyses

For all analyses, missing data (BPD, 21 cases; laboratory reactive aggression, 8 cases; hormone data, 10 cases; HRV data, 11 cases) were estimated in SPSS using the multiple imputation method with 10 imputations (Newgard and Haukoos, 2007). To confirm that the social provocation manipulation induced anger, as measured by the PANAS (Watson et al., 1988) administered before and after provocation, a one-way repeated measures ANOVA was conducted. Correlational analyses were used to examine the inter-relationships.

Latent Profile Analysis (LPA) using Mplus 8 statistical software (Muthén and Muthén, 2017) was used to identify distinct groups of individuals based on their Z-scores on PPI affective-interpersonal and impulsivity subscales, maltreatment and anxiety total scores. The Bayesian information criterion (BIC), Akaike information criterion (AIC), Lo-Mendel-Rubin (LMR) statistic, posterior probabilities, and entropy values were used as statistical criteria to identify the optimal number of groups to retain. Average posterior probabilities and entropy values equal to or greater than 0.70 indicate clearer classification and greater power to predict class membership, and lower BIC and AIC values are preferred; however, a non-significant chi-square value ($p > .05$) for the LMR statistic suggests that a model with one fewer class is optimal.[2]

A series of independent samples t-tests were used to compare LPA groups on validating (i.e., questionnaires) and outcome variables (i.e., self-reported and laboratory aggression, hormone levels at baseline [time 1 (T1)] and post-provocation [time 2 (T2)], and HRV indices), using Holm's correction for multiple comparisons given Bonferroni correction can be too conservative for simple t-tests (Armstrong, 2014). Cohen's *ds* for between group comparisons are reported. Finally, a series of one-way ANCOVAs were conducted to examine group differences between post-provocation hormone levels (T2), controlling for baseline levels (T1) and covariates (i.e., hours since waking; time of testing; days since first day of menstrual cycle; blood in the sample, $n = 19$), and speech, feedback, and CRTT RMSSD HRV controlling for baseline RMSSD HRV; partial eta squares are reported.[3]

## 3. Results

### 3.1. Manipulation check

Participants reported an increase in angry affect from baseline ($M = 10.26$, $SD = 4.44$) to post-provocation ($M = 15.03$, $SD = 7.56$) suggesting that the manipulation was effective ($F$ [1, 100] = 48.83, $p = 0.001$, $\eta^2 = .33$). Secondary psychopathy variants scored significantly higher than primary variants on baseline angry affect (primary, $M = 9.27$, $SD = 2.79$; secondary, $M = 11.97$, $SD = 6.02$; $t(99) = -3.07$, $p = 0.013$, $d = .62$) but not post-provocation angry affect (primary, $M = 14.39$, $SD = 7.19$; secondary, $M = 16.14$, $SD = 8.12$; t(99) = -1.12, $p = .264$, $d = .23$).

With regard to hormones, for the full-sample, cortisol decreased from baseline ($M = 14.80$, $SD = 4.55$) to post-provocation ($M = 12.26$, $SD = 5.02$; $F$ [1, 100] = 23.54, $p = 0.001$, $\eta^2 = 0.19$); and DHEA (baseline, $M = 30.52$, $SD = 5.40$; post-provocation, $M = 30.39$, $SD = 5.26$; $F$ [1, 100] = 0.10, $p = .752$, $\eta^2 = 0.00$) and testosterone remained unchanged (baseline, $M = 363.84$, $SD = 108.19$; post-provocation, $M = 355.58$, $SD = 103.78$; $F$ [1, 100] = 1.36, $p = 0.246$, $\eta^2 = 0.01$). On average, HRV did not change from resting to negative feedback for the full sample (resting, $M = 1.74$, $SD = 0.20$; feedback, $M = 1.72$, $SD = 0.24$; $F$[1, 100] = .94, $p = .335$, $\eta^2 = 0.01$). As detailed below, this pattern appeared to be driven by opposing effects for primary and secondary psychopathic variants.

### 3.2. Descriptives and correlations

Means and standard deviations for the questionnaires and the laboratory measure of reactive aggression are as follows; grouping variables: PPI affective/interpersonal ($M = 76.64$, $SD = 11.02$), PPI impulsivity ($M = 49.52$, $SD = 7.87$), childhood maltreatment ($M = 41.14$, $SD = 11.85$), anxiety ($M = 42.41$, $SD = 9.31$); validating variables: PPI total ($M = 142.31$, $SD = 13.61$; this score was 1.5 SD greater than a sample of undergraduate women not selected for high psychopathic traits; Lilienfeld and Hess, 2001), parental warmth ($M = 47.60$, $SD = 12.95$), depression ($M = 12.75$, $SD = 5.14$), PTSD symptoms ($M = 19.00$, $SD = 14.97$), BPD symptoms ($M = 42.30$, $SD = 8.52$); aggression variables: PCS proactive aggression ($M = 2.24$, $SD = 1.332$), PCS reactive aggression ($M = 2.44$, $SD = 1.26$), laboratory reactive aggression ($M = 5.91$, $SD = 2.55$; this score was greater than a sample of undergraduate women not selected for psychopathic traits; $M = 4.07$, $SD = 2.73$; Denson et al., 2013).

As reported in Table 1, correlations between the main study variables showed that self-reported proactive and reactive aggression were uncorrelated with laboratory reactive aggression. With respect to hormone levels, baseline DHEA was negatively correlated with laboratory aggression. Baseline and post-provocation testosterone-to-cortisol ratios were negatively correlated with PPI impulsivity. Post-provocation testosterone was positively correlated with anxiety, depression, and PTSD symptoms. Post-provocation cortisol was positively correlated with PPI impulsivity, anxiety, depression, BPD symptoms, and proactive aggression, and negatively correlated with PPI affective/interpersonal and parental warmth. Post-provocation cortisol-to-DHEA ratio was positively correlated with PPI impulsivity, anxiety, and negatively correlated with PPI affective/interpersonal, and parental warmth. With respect to HRV variables, resting RMSSD HRV was positively correlated PPI impulsivity, maltreatment, PPI total, PTSD and BPD symptoms, and negatively correlated with PPI affective/interpersonal, and laboratory reactive aggression. Speech RMSSD was positively correlated with PPI impulsivity and total scores, maltreatment, PTSD and BPD symptoms. Feedback and speech RMSSD were positively correlated with BPD symptoms and negatively correlated with PPI affective/interpersonal. Hormone and HRV variables were uncorrelated.

[2] Given the sample size ($N = 101$) and oversampling of individuals with high psychopathic traits, we also created variant groups using a median split of the anxiety measure (median = 42) such that those below the median value were classified into the primary psychopathy group ($n = 53$) and those above were classified into the secondary group ($n = 48$), as per prior studies in the field (e.g., Cecil, McCrory, Barker, Guiney, & Viding, 2017; Sharf, Kimonis, & Howard, 2014). Findings remained mostly consistent with LPA results with the exception of: primary variants scored higher than secondary variants on parental warmth, and the difference in feedback RMSSD reduced to non significance (parental warmth, p$M = 51.34$, $SD = 12.48$, s$M = 43.48$, $SD = 12.30$, $t(99) = 3.18$, $p = .002$; feedback RMSSD, p$M = 1.66$, $SD = .23$, s$M = 1.78$, $SD = .24$, $F(98) = .80$, $p = .372$).

[3] Levene's test for equality of variances ($p = .10$-$.85$) supported homogeneity of variance for all variables except depression ($p < .001$) and PTSD ($p = .001$). Thus, for these variables we repeated analyses using nonparametric Kruskal-Wallis H test and findings remained the same (depression, $X^2(1) = 27.90$, $p = < .001$; PTSD, $X^2(1) = 29.47$, $p = < .001$).

**Table 1**
Correlations between Questionnaires, Laboratory Reactive Aggression and Hormone and HRV Variables; and Descriptive Statistics for Hormone and HRV Variables.

| | 1. Cortisol T1 | 2. Cortisol T2 | 3. DHEA T1 | 4. DHEA T2 | 5. Test T1 | 6. Test T2 | 7. Cortisol: DHEA T1 | 8. Cortisol: DHEA T2 | 9. Test: Cortisol T1 | 10. Test: Cortisol T2 | 11. Resting RMSSD | 12. Speech RMSSD | 13. Feedback RMSSD | 14. CRTT RMSSD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Grouping Variables* | | | | | | | | | | | | | | |
| 1. PPI affective/interpersonal | −.12 | −.20* | −.05 | −.10 | −.09 | −.17 | −.07 | −.20* | .06 | .01 | −.21* | −.14 | −.30** | −.22* |
| 2. PPI impulsivity | .19 | .26** | .18 | .14 | .02 | .06 | .09 | .21* | −.23* | −.21* | .26* | .24* | .13 | .14 |
| 3. Childhood maltreatment | .09 | .11 | −.01 | .10 | −.04 | −.00 | .11 | .06 | −.16 | −.08 | .14 | .22* | .07 | .14 |
| 4. Anxiety | .11 | .25* | .03 | .08 | .02 | .21* | .10 | .24* | −.15 | −.10 | .22* | .18 | .17 | .21* |
| *Validating Variables* | | | | | | | | | | | | | | |
| 5. PPI total | .14 | .10 | .08 | .03 | .07 | .07 | .09 | .06 | −.13 | −.03 | .22* | .23* | .08 | .16 |
| 6. Parental warmth | −.17 | −.29** | −.06 | −.18 | −.07 | −.15 | −.19 | −.26** | .13 | .18 | −.08 | −.18 | −04 | −.09 |
| 7. Depression symptoms | .09 | .21* | .06 | .16 | .05 | .24* | .05 | .15 | −.08 | −.04 | .19 | .14 | .11 | .14 |
| 8. PTSD symptoms | .17 | .15 | .07 | .05 | .11 | .23* | .15 | .15 | −.07 | −.04 | .28** | .27** | .19 | .19 |
| 9. BPD symptoms | .19 | .25* | .06 | .11 | .04 | .19 | .16 | .20* | −.18 | −.15 | .30** | .35*** | .21* | .36*** |
| *Aggression Variables* | | | | | | | | | | | | | | |
| 10. PCS proactive aggression | .07 | .21* | .05 | .10 | −.02 | .06 | .02 | .16 | −.06 | −.11 | .15 | .16 | .06 | .10 |
| 11. PCS reactive aggression | .10 | .18 | .02 | .04 | −.03 | .04 | .10 | .18 | −.11 | −.11 | .16 | .12 | .05 | .08 |
| 12. Laboratory aggression | −.16 | −.12 | −.20* | −.11 | −.16 | −.13 | −.02 | −.07 | .03 | .10 | −.33** | −.01 | −.14 | −.15 |
| *Descriptives* | | | | | | | | | | | | | | |
| *M* | 14.80 | 12.26 | 30.52 | 30.39 | 363.83 | 355.58 | .48 | .40 | 25.99 | 35.06 | 1.74 | 1.88 | 1.72 | 1.77 |
| *SD* | 4.55 | 5.02 | 5.40 | 5.26 | 108.19 | 103.78 | .13 | .15 | 8.77 | 24.48 | .20 | .21 | .24 | .24 |
| Range | 2.39-26.22 | 1.41-23.50 | 15.46-46.62 | 16.97-44.35 | 108.26-616.56 | 104.88-596.17 | .13-1.03 | .05-.80 | 9.05-62.02 | 9.32-198.90 | 1.28-2.30 | 1.33-2.27 | 1.23-2.36 | 1.16-2.28 |

*Note.* *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. T1 = baseline, T2 = stress-reactive/post-provocation, DHEA = dehydroepiandrosterone, Test =. testosterone, RMSSD = root mean square successive R-R interval differences, CRTT = competitive reaction time task.

**Table 2**
Model Fit Statistics for the Latent Profile Analysis with Z-scored PPI Subscales, CTQ Total Score and STAIT Total Score.

| Classes | BIC | AIC | Entropy | LMR |
|---|---|---|---|---|
| 1 | 1154.14 | 1158.48 | NA | NA |
| **2** | **1125.40** | **1132.46** | **.641** | **.046** |
| 3 | 1112.76 | 1122.54 | .738 | .723 |
| 4 | 1103.78 | 1116.27 | .754 | .282 |

*Note.* Bolded text indicates the best-fitting model. BIC = Bayesian information criterion; AIC = Akaike information criterion; LMR = Lo-Mendal-Rubin test.

### 3.3. Grouping variables

Four LPAs were conducted to identify the optimal number of classes to retain (see Table 2). BIC and AIC values continued to decrease through all four models. The model with two classes had a significant LMR chi-square value, this fell out of significance for the three and four class models. Thus, the two-class solution was selected as the optimal fit to the data. This model revealed a primary psychopathy variant (high PPI affective/interpersonal, and low PPI impulsivity, maltreatment, and anxiety; $n = 64$), and a high-anxious secondary psychopathy variant (low PPI affect/interpersonal, and high PPI impulsivity, maltreatment, and anxiety; $n = 37$).

### 3.4. Outcome variables

Psychopathy variant groups differed as predicted on the grouping and validating variables (see Fig. 1, and supplemental Tables 1 and 2). Primary psychopathy variants scored higher than secondary variants on PPI affective/interpersonal. Secondary variants scored higher than primary variants on PPI impulsivity, childhood maltreatment, anxiety, depression, PTSD and BPD symptoms. There were no significant group differences on PPI total score or on self-reported or laboratory aggression.

### 3.5. Hormone and psychophysiology variables

As shown in Table 3 and Fig. 2, secondary psychopathy variants scored higher than primary variants on post-provocation cortisol, testosterone and cortisol:DHEA ratio, controlling for baseline concentrations. There were no significant group differences on post-provocation DHEA and testosterone:cortisol ratio.

Controlling for resting RMSSD HRV, secondary psychopathy variants scored significantly higher than primary variants on RMSSD HRV during feedback. There were no significant group differences on speech and CRTT RMSSD HRV.

## 4. Discussion

Accumulating research supports that psychopathy is a heterogeneous construct, but the majority of prior studies are with male samples. This study aimed to examine whether female psychopathy variants showed divergent psychobiological patterns prior to and post-social provocation while engaged in an aggression paradigm with the provocateur. Despite similar phenotypic expression of aggression using both self-reported and laboratory measures, female psychopathy variants differed in their underlying endocrine and psychophysiological responses. In contrast to past research with incarcerated and clinical samples (Kimonis et al., 2016; van Voorhees et al., 2014), we did not find a significant difference between women with primary and secondary psychopathic traits on resting hormone levels. However, secondary variants showed higher cortisol-to-DHEA ratios after social provocation—this appears to be driven by changes in cortisol (i.e., post-provocation cortisol but not DHEA was significantly greater for secondary relative to primary variants). This finding suggests that among secondary variants, DHEA is not being optimally released in response to a stressor to counter the negative effects of cortisol and restore homeostasis (Young et al., 2002). Our examination of stress-reactive hormone concentrations is more likely to capture fluctuations in this stress hormone (cortisol as the end-product of the HPA axis responsible for the fight-or-flight response) than are resting measurements. These findings suggest that an imbalance of cortisol to DHEA may be a reliable biomarker differentiating subtypes across demographic and contextual factors; however, our novel post-provocation findings (with small to medium effect sizes) require replication before strong inferences can be made. In light of reviews finding the link between antisocial traits and cortisol to be mixed at best (Alink et al., 2008), we argue for continued focus on examining hormone ratios toward elucidating psychobiological mechanisms underpinning developmental pathways to psychopathy. Importantly, the imbalanced high cortisol-to-DHEA ratio that reflects HPA dysfunction (i.e., low levels of DHEA are unable to

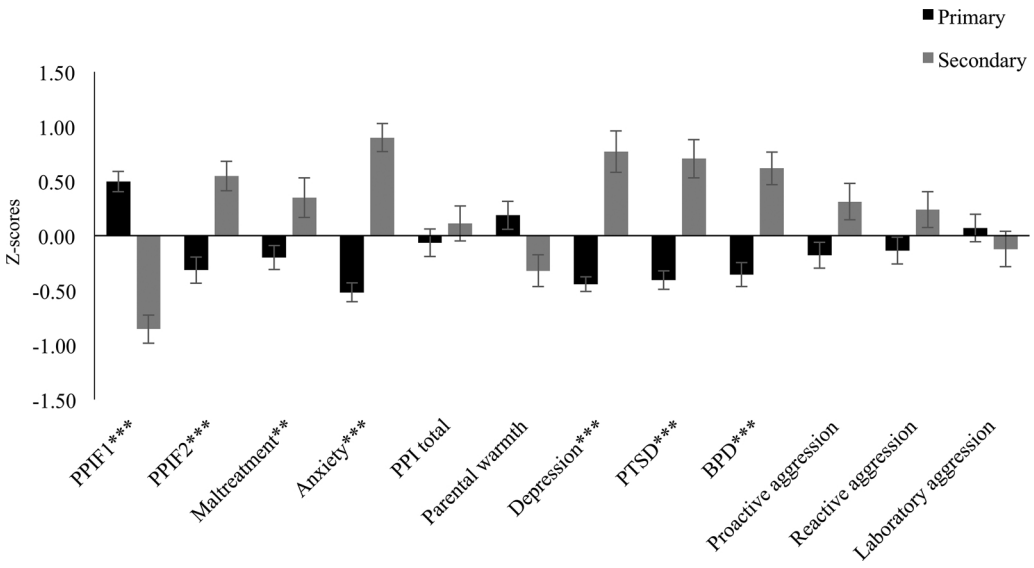


**Fig. 1.** Comparisons between Primary and Secondary Psychopathy Variants on Questionnaire and Behavioral Variables.
*Note.* *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$

**Table 3**
Comparisons between Primary and Secondary Psychopathy Variants on Post-Provocation Hormone and HRV Variables controlling for Baseline Levels.

| Variable | Primary ($n$ = 64) | Secondary ($n$ = 37) | $F$ | $df$ | $p$ | $\eta^2$ |
|---|---|---|---|---|---|---|
| *Hormone Concentrations* | | | | | | |
| Cortisol T2 | 11.12 (4.73) | 14.23 (4.95) | 6.66 | 94 | .011 | .07 |
| DHEA T2 | 29.53 (5.35) | 31.87 (4.81) | 2.83 | 94 | .096 | .03 |
| Testosterone T2 | 335.21 (102.43) | 390.82 (97.69) | 12.03 | 94 | .001 | .12 |
| Cortisol: DHEA T2 | .37 (.14) | .45 (.15) | 6.04 | 94 | .016 | .06 |
| Testosterone: Cortisol T2 | 37.10 (27.03) | 31.52 (19.10) | .35 | 94 | .559 | .00 |
| *HRV Variables* | | | | | | |
| Speech RMSSD | 1.85 (.21) | 1.93 (.21) | .262 | 98 | .610 | .00 |
| Feedback RMSSD | 1.66 (.23) | 1.82 (.21) | 5.51 | 98 | .021 | .05 |
| CRTT RMSSD | 1.72 (.24) | 1.86 (.21) | 3.43 | 98 | .067 | .03 |

*Note.* T2 = stress-reactive/post-provocation, DHEA = dehydroepiandrosterone. RMSSD = root mean square successive R-R interval differences, CRTT = competitive reaction time task.

counteract the negative effects of high cortisol levels) is consistent with developmental models of secondary psychopathy that emphasize chronic exposure to stressful adverse life events and downstream effects of poor mental and physical health.

Our finding that secondary psychopathy variants had significantly higher post-provocation testosterone levels than primary variants (with a large effect size) is consistent with prior research finding a relationship between high testosterone and the impulsivity dimension of psychopathy, but not interpersonal/affective traits (Yildirim and Derksen, 2012). These authors posited that the development of interpersonal/affective features of psychopathy results from an interaction between high testosterone levels and other biological or social (e.g., early life adversity) risk factors consistent with theories of secondary psychopathy (Karpman, 1941). Extensive research has linked testosterone with antisocial behavior, but some suggest it is critical to consider this relationship in the context of HPA functioning, prompting interest in the testosterone–to-cortisol ratio (Terburg et al., 2009). Sex differences may explain past research findings (e.g., Glenn et al., 2011) in the testosterone-cortisol relationship; whereas androgen production mainly stems from the HPG axis among males, approximately half of the androgen production stems from the adrenal cortex—the final component of the HPA axis—among females (Burger, 2002). In this study of young women, we hypothesized that we would find a lower testosterone-to-cortisol ratio among secondary psychopathy variants, linked with their dysregulated emotional profiles, compared with primary variants. We failed to find a significant difference in testosterone-to-cortisol ratios between psychopathy variants, but our negative bivariate correlations between the impulsivity dimension and the testosterone-to-cortisol ratio provides partial support for hypotheses. However, we are hesitant to make inferences from correlational data given they do not account for baseline hormone concentrations, and thus, future research should examine post-provocation testosterone-to-cortisol ratios among men versus women differentiated into primary and secondary psychopathic variants.

The finding of higher HRV, which is thought to indicate flexible emotion regulation, at rest and during social provocation among secondary psychopathy variants relative to primary variants was at odds with predictions. Whereas *low* HRV has been consistently associated with anxious and depressive disorders, childhood adversity, and reactive aggression, all characteristic of secondary psychopathy variants, research finds that high HRV is associated with positive mental health and proactive aggression (Hansen et al., 2007). The present study is the first to examine stress-reactive HRV among psychopathy variants; therefore, these findings are in need of replication before strong inferences can be made. However, one possible explanation is that high HRV (i.e., low arousal) among secondary psychopathy variants that experience high levels of adversity could be an adaptive mechanism. Karpman (1941) suggested that secondary variants adopt psychopathic traits as a means of coping with adverse and unpredictable social environments, and psychopathic traits are linked with high HRV (Hansen et al., 2007); thus, high HRV among secondary variants may allow them to better predict and respond to instances of acute threat as a survival mechanism. It would, therefore, be important for future research into the psychophysiology of psychopathy variants to include anxious populations who typically show low HRV as this may provide insights into what differentiates individuals with secondary psychopathy from those with clinical anxiety, since both groups show elevated and often

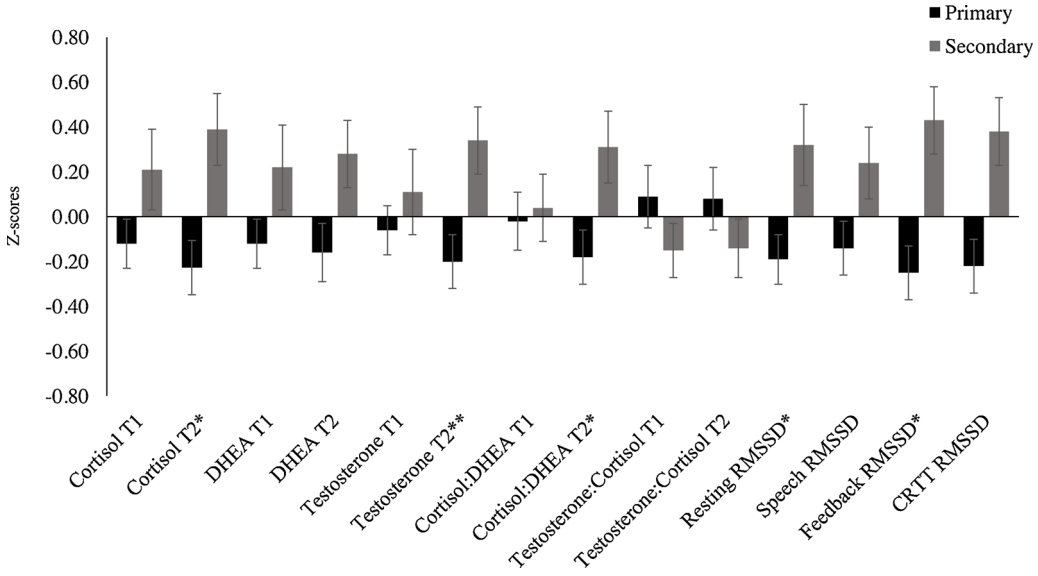


**Fig. 2.** *Comparisons between Primary and Secondary Psychopathy Variants on Hormone and Psychophysiological Variables.*
*Note.* *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$

undifferentiated levels of anxiety symptoms. For example, a longitudinal study of 1167 children followed from toddlerhood to adolescence found that low maternal sensitivity in the first two years of life best differentiated externalizing adolescents with secondary psychopathic traits from low-psychopathic individuals with equivalently high anxiety levels (Fanti and Kimonis, 2017). Thus, the moderating effect of early caregiving on psychobiological patterns of responding in psychopathic variants will also be important to examine in future research.

Relative to primary variants, secondary psychopathy variants displayed a seemingly incongruent pattern of high HRV during insult together with heightened post-provocation hormone concentrations of cortisol and testosterone. According to Del Giudice and colleagues (2011) study testing their Adaptive Calibration Model of stress responsivity, children from chronically stressful environments were characterized as 'vigilant' or 'unemotional' and showed deactivation of the PNS and activation of the HPA axis. However, another group exposed to moderate environmental stress showed moderate arousal across both physiological systems. In the present study, although our secondary psychopathy group scored higher than the primary group on histories of early life maltreatment, the level of maltreatment may be only considered 'moderate' among this high functioning undergraduate sample. Thus, it will be important for future research to examine cross-system activation among psychopathy variants with more severe experiences of early life stress. Further, among high functioning student samples, coactivation of these systems may also manifest as risky sexual behaviors, eating problems, or substance abuse—behaviors more commonly found among college populations. However, we did not include such measures in the present study and so future research should examine risky behavioral profiles related to coactivation of the stress systems in the context of primary and secondary traits among high functioning populations.

Methodological strengths of the present study include our focus on the understudied population of female psychopathy variants and our multi-method approach to examining aggression and emotional reactivity. This study was the first to examine differences between psychopathy variants in their aggressive behavior during a social provocation laboratory paradigm, extending prior research beyond questionnaire measures. It is also the first to examine interactions between hormones at baseline and post-provocation (stress-reactive) between psychopathy variants, which is important given inconsistencies in the field.

The present study also had limitations that must be considered in interpreting results. First, it focused on an undergraduate sample oversampled for high psychopathic traits of which a majority (63%) identified as Asian. Scant research has examined the psychopathy construct among Asian populations but preliminary results from factor analytic studies suggest a similar factor structure to majority populations for common psychopathy measures (Fung et al., 2009).[4] Research also supports the assessment of psychopathic traits in college populations and finds similar correlates to incarcerated populations (e.g., Falkenbach et al., 2008), but there is a need to replicate findings in future criminal samples especially those that were novel and not guided by a priori predictions. Further, future research should include a male comparison group and a low psychopathic control group to understand whether hormone patterns are atypical. In line with this, while past research supports the timing of our post-provocation salivary sample (20 min) to most accurately assess all three hormones (Bedgood et al., 2014; Kirschbaum and Hellhammer, 1994; Lennartsson et al., 2012), variability has been found between hormones and between individuals, and thus, future research should undertake a more robust multiple sampling approach for measuring salivary analytes, and include diurnal measures, to further elucidate endocrine patterns associated with psychopathy. Second, only self-report measures of psychopathic traits were used, and while they represent a valid method for assessing psychopathic traits (Lilienfeld and Andrews, 1996) and correlate moderately with the Psychopathy Checklist-Revised (Hare, 2003), findings should be replicated with such clinical interview measures within an antisocial population. Finally, while a strength of the current study was the inclusion of a behavioral aggression measure, the laboratory task included only examined reactive aggression and it was uncorrelated with our questionnaire measure of aggression. Future research should examine behavioral proactive aggression between psychopathy variants as well. Other research has found significant associations between this laboratory paradigm and physical aggression, but not verbal aggression that is more common to women and girls (e.g., Denson et al., 2011). These findings have led some to suggest a need for aggression paradigms that assess relational aggression (Denson et al., 2018). However, meta-analytic findings suggest that while men are more aggressive than women unprovoked, when provoked this sex difference attenuates (Bettencourt and Miller, 1996)—supporting the validity of laboratory aggression paradigms among men and women (Chester and Lasko, 2018). Our questionnaire measure of aggression was administered prior to provocation, and thus, future research should examine correlates of self-report and behavioral reactive aggression post-provocation. Additionally, given the sequence of events it is possible that group differences may reflect a more general stress response that is not specific to provocation.

While the concept of multiple developmental pathways to psychopathic traits was proposed some time ago (Karpman, 1941), empirical research on psychopathy variants is relatively recent and has been dominated by descriptive research with male samples. The present study was the first to examine stress-reactive endocrine and autonomic indices among women divided into primary and secondary variants of psychopathy, providing support for divergent psychobiological functioning. Secondary psychopathy variants showed greater HPA dysregulation but greater HRV regulation compared with primary variants. These significant and novel findings are in need of replication, but provide preliminary psychobiological support for Karpman's (1941) theory that secondary variants develop psychopathic traits to survive unpredictable and threatening environments.

## Author contributions

NG and EK developed the study concept. All authors contributed to the study design. NG carried out data collection. NG and DB performed data analyses and all authors contributed to data interpretation. NG and EK drafted the paper, and TD and DB provided critical revisions. All authors approved the final version of the paper for submission.

## Conflicts of interest

none.

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## Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi: https://doi.org/10.1016/j.psyneuen0.2019.02.011.

[4] Analyses were repeated controlling for ethnicity and findings remained mostly the same with the exception of parental warmth, which was significantly greater among primary than secondary variants, and proactive aggression which was significantly greater among secondary than primary variants (parental warmth, $F(2, 98) = 6.66$, $p = .011$; proactive aggression, $F(2, 98) = 5.87$, $p = .017$).

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