Journal of Crime and Justice

ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/rjcj20

Hormones, autonomic nervous system activity, and criminal behavior

Todd A. Armstrong, Danielle L. Boisvert, Jessica Wells, Richard H. Lewis, Eric M. Cooke, Matthias Woeckener, Nicholas Kavish, Nicholas Vietto & James M. Harper

To cite this article: Todd A. Armstrong, Danielle L. Boisvert, Jessica Wells, Richard H. Lewis, Eric M. Cooke, Matthias Woeckener, Nicholas Kavish, Nicholas Vietto & James M. Harper (03 Aug 2024): Hormones, autonomic nervous system activity, and criminal behavior, Journal of Crime and Justice, DOI: 10.1080/0735648X.2024.2382985

To link to this article: https://doi.org/10.1080/0735648X.2024.2382985

JOURNAL OF CRIME AND JUSTICE
https://doi.org/10.1080/0735648X.2024.2382985

Hormones, autonomic nervous system activity, and criminal behavior

Todd A. Armstrong[a], Danielle L. Boisvert[b], Jessica Wells[c], Richard H. Lewis[d], Eric M. Cooke[e], Matthias Woeckener[f], Nicholas Kavish[g], Nicholas Vietto[a] and James M. Harper[h]

[a]School of Criminology and Criminal Justice, University of Nebraska, Omaha, NE, USA; [b]Department of Criminal Justice and Criminology, Sam Houston State University, Huntsville, TX, USA; [c]Department of Criminal Justice, Boise State University, Boise, ID, USA; [d]Department of Criminal Justice, University of Arkansas at Little Rock, Little Rock, AR, USA; [e]Criminal Justice Program, Bowling Green State University, Bowling Green, OH, USA; [f]Department of Criminal Justice, University of Nebraska at Kearney, Kearney, NE, USA; [g]Department of Neurological Sciences, University of Nebraska Medical Center, Omaha, NE, USA; [h]Department of Biological Sciences, Sam Houston State University, Huntsville, TX, USA

ABSTRACT

Evidence that hormones and autonomic nervous system activity exert a joint influence on antisocial behavior suggests that they may work together to explain risk for criminal behavior. Thus, the current study explored the interactive effects of hormones (testosterone, cortisol) and ANS activity (heart rate, skin conductance) on impulsive and violent criminal behavior in a large sample of university students ($n = 495$). Initial analyses found positive direct associations between impulsive and violent crime and both testosterone and cortisol and a negative direct association between heart rate reactivity and impulsive and violent crime. Subsequent analyses revealed a statistically significant interaction between testosterone and heart rate in association with impulsive and violent criminal behavior. The positive association between testosterone and crime became statistically significant at −.29 standard deviations below the mean (HR = 76.44) and grew stronger as heart rate decreased from this point. The interaction between testosterone and heart rate remains statistically significant after the inclusion of controls for constructs that feature prominently in contemporary criminological theories. Other interactions between hormones and ANS activity were present but were not consistent across alternative model specifications.

ARTICLE HISTORY
Received 14 February 2024
Accepted 16 July 2024

KEYWORDS
Testosterone; cortisol; heart rate; skin conductance; crime

Introduction

While research indicates that both hormones and autonomic nervous system (ANS) activity play a role in the etiology of crime (Ling, Umbach, and Raine 2019; Moffitt, Ross, and Raine 2011), studies have yet to consider their joint influence on risk for criminal behavior. The potential moderation of associations between hormones such as testosterone and cortisol and risk for criminal behavior by ANS activity is directly indicated by evidence for interactions between ANS activity and cortisol in association with youth behavioral problems (Jones, Rohleder, and Schreier 2020). In parallel, reciprocal associations between testosterone and stress systems, of which the ANS is a major component, suggest that ANS activity may moderate the influence of testosterone on risk for criminal

CONTACT Todd A. Armstrong toddarmstrong@unomaha.edu School of Criminology and Criminal Justice, University of Nebraska—Omaha, 6001 Dodge St, Omaha, NE 68182, USA
Supplemental data for this article can be accessed online at https://doi.org/10.1080/0735648X.2024.2382985

behavior (Handa and Weiser 2014). Thus, our understanding of biological contributions to risk for criminal behavior may be further specified by a consideration of interactions between hormones and indicators of ANS activity including heart rate and skin conductance. The implications of such an approach for models of the etiology of criminal behavior would be maximized by attention to the known individual and socio-environmental correlations of crime.

Background

Hormones and crime

A large body of research suggests that the hormones testosterone and cortisol are associated with antisocial behavior including aggression (Fairchild, Baker, and Eaton 2018; Geniole et al. 2020; van Goozen et al. 2007). This work is complemented by a smaller group of studies showing that these associations extend to crime (Booth and Osgood 1993; Dabbs et al. 1987; Johnson et al. 2015). Testosterone, a hormone product of the Hypothalamic Pituitary Gonadal (HPG) axis, is widely known for its role in reproductive physiology and behavior (Wingfield et al. 1990) and is thought to lead to increased antisocial and aggressive behaviors through associations with dominance and status seeking (Mazur and Booth 1998). Cortisol is a steroid hormone released as part of the Hypothalamic Pituitary Adrenal (HPA) axis stress response (Chrousos and Gold 1992). As part of the response to physiological or psychological stressors, cortisol is associated with an array of effects including the mobilization of energy, immune suppression, and cardiovascular changes (Sapolsky, Romero, and Munck 2000). The cumulative work on the relationship between stress system activity and antisocial, aggressive, and criminal behavior points to a bifurcated pattern of association with an increased risk through negative affect (e.g., anger and depression) when stress system activity is upregulated and cortisol levels are high (Kemeny and Shestyuk 2008; Lykken 1995; Patrick, Bradley, and Lang 1993) and an increased risk through callousness and lack of affect when stress system activity is inhibited and cortisol levels are low (Lykken 1995; Patrick, Bradley, and Lang 1993).

ANS and crime

A robust body of evidence finds that reduced ANS activity is associated with antisocial behavior including crime (De Looff et al. 2022; Lorber 2004; Portnoy and Farrington 2015). Studies relating ANS activity to antisocial behavior commonly measure ANS activity with heart rate and skin conductance. Skin conductance is influenced by the sympathetic nervous system (SNS) branch of the ANS, while heart rate is influenced by both the SNS and the parasympathetic branch of the ANS. SNS activity is commonly characterized as potentiating the 'fight or flight' response to stress including cardiovascular changes, while parasympathetic nervous system (PNS) activity is largely associated with changes in support of a return to homeostasis after stress, sometimes referred to as the 'rest and digest' state.

The association between low-resting heart rate and measures of criminal behavior including convictions for violence, convictions for serious offenses, and officially recorded delinquency has been established by prospective longitudinal studies (Latvala et al. 2015; Murray et al. 2016; Raine, Venables, and Williams 1990, 1995). A lesser but still substantive research base points to an association between resting skin conductance and both antisocial and aggressive behaviors (De Looff et al. 2022; Lorber 2004). Work in this area also shows that the negative association between resting skin conductance and antisocial behavior extends to crime (Raine, Venables, and Williams 1990).

In addition to work demonstrating an association between ANS activity at rest (baseline ANS) and antisocial behavior, research has also assessed the association between ANS reactivity to stress and antisocial behavior. Early meta-analyses of work in this area identified negative associations between heart rate reactivity and antisocial behavior (Ortiz and Raine 2004) and between skin conductance reactivity and psychopathy/sociopathy but not aggression (Lorber 2004). However, a recent meta-

analysis found that heart rate reactivity and skin conductance reactivity were not associated with conduct disorder or physical aggression (De Loof, et al. 2022). In work specific to criminal behavior, De Vries-Bouw et al. (2011) found a negative association between heart rate reactivity and re-offending in juvenile males.

Reduced ANS activity may be associated with increased antisocial behavior through sensation seeking enacted to increase physiological arousal or through a lack of fear response to the aversive aspects of antisocial behavior (Lykken 1957; Quay 1965; Raine 2002). Studies considering the role of traits in the association between ANS activity and antisocial behavior have found evidence in support of the hypothesis that low heart rate is related to increased aggression through sensation seeking (Hammerton et al. 2018; Portnoy et al. 2014), but no support for the suggestion that fearlessness mediates the association between ANS activity and antisocial behavior (Portnoy et al. 2014).

Interactions between biological substrates

While work examining the effects of either hormone or ANS activity on criminal behavior is important, the accurate specification of the association between each of these biological substrates and criminal behavior depends on the consideration of the other. The potential utility of considering both hormones and ANS activity when attempting to parse the role of biology in criminal behavior was delineated in Bauer, Quas, and Boyce (2002) seminal work.

Bauer, Quas, and Boyce (2002) reviewed research showing that the SNS and HPA axis work together to enact physiological and behavioral responses to stress and the restoration of homeostasis. The authors then argued that a simultaneous consideration of both systems would 'allow a more thorough understanding of the physiological correlates of behavior problems' (Bauer, Quas, and Boyce 2002, 104). This simultaneous consideration of both SNS and HPA axis activity was thought to be important due to interconnectivity between the SNS and the HPA axis (Chrousos and Gold 1992) and due to the interactive associations that may stem from this interconnectivity (Bauer, Quas, and Boyce 2002). The two systems may have additive interaction effects when hyper-arousal across systems leads to risk for behavior problems through negative effects, or when coordinated hypo-arousal leads to problem behavior through under-arousal and a lack of fear (Kagan, Reznick, and Snidman 1988). Alternatively, negative interactions may occur as a result of dysregulation and asymmetrical activity in the SNS and HPA axis (Bauer, Quas, and Boyce 2002). While Bauer's arguments regarding the virtue of considering multiple biological substrates in the explanation of problem behavior have garnered attention with regard to the explanation of youth behavior problems, they have yet to be applied to the explanation of criminal behavior. The potential utility of the consideration of such interactions for models of the role of biology in risk of criminal behavior is indicated by a growing body of work finding additive and interactive associations between SNS activity and cortisol in the explanation of youth behavior problems (Jones, Rohleder, and Schreier 2020). For example, Chen, Raine, and Granger (2015) found increased externalizing and internalizing behaviors among boys with reduced basal cortisol and reduced basal SNS activity indexed by salivary alpha amylase (sAA).

While Bauer, Quas, and Boyce (2002) arguments are specific to the joint influence of the HPA axis and SNS, there is also reason to anticipate that ANS activity may moderate the association between testosterone and problem behaviors including crime. Androgens such as testosterone influence HPA axis function at multiple levels including the adrenal gland, pituitary gland, and the hypothalamus (Handa and Weiser 2014). In turn, activity in these areas is associated with indicators of ANS activity including heart rate variability (Stauss 2003). Thus, moderation of the association between testosterone and crime by ANS activity may occur as a result of reciprocal interconnections between HPG axis and stress system activity (Chichinadze and Chichinadze 2008; Kutlikova et al. 2020). It is also possible that interactions occur as a result of the cumulative or sequential action of testosterone and ANS activity on reward seeking. Research has shown that sensation seeking mediates the association

between heart rate and aggression (Hammerton et al. 2018; Portnoy et al. 2014; Sijtsema et al. 2010). Concurrently, studies indicate testosterone is associated with increased preference for risk as indicated by sensation seeking, impulsivity, and direct measures of risk preference (Kurath and Mata 2018). Should associations between testosterone and risk preference be compounded by increased sensation seeking at lower level of heart rate, we may anticipate that any positive relationship between testosterone and criminal behavior would be strengthened when heart rate is low.

The current study

The current study explores interactions between hormone products of the endocrine system (i.e., testosterone and cortisol) and indicators of ANS activity (i.e., skin conductance and heart rate). Initial analyses focus on associations between criminal behavior and the direct and interactive effects of hormones and ANS measures. Subsequent models contextualize these associations within causal mechanisms central to contemporary criminological theory by including controls for individual differences in risk for criminal behavior and measures of the socio-environmental correlations of crime including parents, peers, and romantic partners.

While the current literature suggests that hormones and ANS activity exert a joint influence on criminal behavior, the analyses presented here are nonetheless exploratory. The results of studies on the role of neuroendocrine coordination in youth behavior problems provide evidence for both additive and interactive associations between cortisol and SNS activity as measured by sAA (Jones, Rohleder, and Schreier 2020). Collectively, the studies reviewed by Jones, Rohleder, and Schreier (2020) suggest that work attempting to specify the role of biological variation in risk for criminal behavior should consider both measures of ANS activity and cortisol. However, this work stops short of providing a research base that would lead to specific expectations regarding the nature of the joint influence of ANS measures and hormones on variation in crime. Similarly, the positive associations between testosterone and risk preference (Kurath and Mata 2018) may be compounded by increased sensation seeking at lower levels of heart rate (Hammerton et al. 2018; Portnoy et al. 2014; Sijtsema et al. 2010) thus contributing to a stronger association between testosterone and criminal behavior when heart rate is low. However, direct evidence for increased risk preference with testosterone at lower heart levels is lacking. Therefore, the current analyses are framed as an initial exploration of the potential interactions between hormones and ANS activity in relation to risk for criminal behavior.

Methods

Data were gathered in 2016 as part of a larger project on the role of biology in the etiology of antisocial, aggressive, and criminal behavior conducted at a university in the southern United States. The current study builds directly on earlier work with these data (*citation redacted*). Analyses presented in this earlier work explored the joint effects of testosterone and cortisol on a measure of impulsive and violent criminal behavior and a measure of income generating crime. Results indicated that testosterone had a positive direct association with impulsive and violent crime. Hormones were not directly related to income generating crime, but the association between testosterone and income generating crime was positive and statistically significant when cortisol was below −0.70 SD under the mean and negative and statistically significant when cortisol was above 1.669 over the mean. We frame our initial exploratory analysis of the potential role of ANS activity in associations between hormones and crime around the direct association between testosterone and impulsive and violent crime while also considering potential interactions between cortisol and ANS activity as indicated by work reviewed earlier (Bauer, Quas, and Boyce 2002; Jones, Rohleder, and Schreier 2020). The analysis is framed in this manner to balance the complexity inherent in interaction models with disciplinary standards (and journal limitations) regarding manuscript length. To our knowledge,

the current study is the first to consider interactions between hormones and ANS in association with risk for criminal behavior. Analyses begin by considering the effects of hormones and ANS activity on the criminal behavior network of demographic controls after which models incorporate control measures for the socio-environmental correlations of crime and criminal propensity.

Sample

The study protocol was approved by the IRB of the institution where the study was administered. Participants were recruited for convenience from large Criminology and Criminal Justice classes. After obtaining informed consent, participants completed an in-class survey including measures of the socio-environmental correlates of crime, trait measures, and criminal behavior measures (n = 872). Following survey completion, participants were referred to a laboratory measurement protocol where ANS and hormone measures would be gathered. Subjects were asked to schedule a time for the laboratory protocol through signupgenius.com, an online scheduling tool. A total of 567 students attended the laboratory protocol. The analytic sample is comprised of 495 of these participants that provided complete measures of hormones and ANS, demographic information, and self-reported criminal behavior. The self-identified race/ethnicity of these participants was 12.7% African American, 1.0% Asian, 37.2% Caucasian, 40.0% Hispanic, and 9.1% other. Participants in the analysis sample were 66.5% females and averaged 20.35 years of age (SD = 3.16).

Hormone and ANS quantification

Analyses included measures of testosterone and cortisol taken at rest and measures of heart rate and skin conductance taken at rest and in response to a social stressor. Measures were gathered between the hours of 0800 and 1830. Participants were instructed to refrain from a variety of activities that may have affected testosterone and cortisol levels (e.g., smoking, eating, and exercise) for at least one hour prior to reporting to the lab. Upon arrival at the lab, participants were seated comfortably. Next, compliance with activity restrictions was confirmed, and informed consent was verbally reaffirmed. Saliva samples for baseline hormone assays were gathered after a 30 second rest period with Salimetrics LLC Saliva Collection Aids. Each sample contained at least 1.5 mL of saliva. Prior to the analysis, samples were stored in a freezer at −20 degree Celsius. Saliva samples were analyzed using materials from and following established protocols for salimetric testosterone and cortisol enzyme immunoassay kits. All samples were tested in duplicates. Testosterone concentrations were recorded in pg/mL and cortisol concentrations in μg/dL. The mean intra-assay coefficients of variation for testosterone and cortisol were 5.98% and 11.01%, respectively, and the mean inter-assay coefficients of variation for testosterone and cortisol were 7.95% and 5.91%, respectively.

After gathering the sample for hormone assays, sensors for the measurement of ANS activity were attached to the fingertips of the participant's right hand. Heart rate was measured using a plethysmograph-based NeuLog infrared LED transmitter/receptor sensor. The skin conductance level was measured using NeuLog galvanic skin response sensors. Values for both were recorded on a single laptop using the NeuLog data acquisition and analysis application. Sampling rates were set at one measurement every 2 seconds. After sensors were attached, participants rested for 3 minutes and were then told that they had 2 minutes to prepare a 2-minute speech on their biggest faults and weaknesses and that the speech would be recorded and later evaluated. The ANS recording continued for 30 seconds after the conclusion of the speech. ANS measureswere estimated for four time periods during the measurement protocol. These include a baseline measure across the last 30 seconds of the rest period before the announcement of the task, a second measure across the last 30 seconds of speech preparation, a third measure comprised of the full 2 minutes of speech delivery, and a follow-up measure across the 30 seconds after the conclusion of the speech. For each of the measurement periods, ANS measures were estimated as the averaged sum of measures gathered every 2 seconds during the measurement period. ANS reactivity to stress was estimated as area under the curve with respect to increase (AUC_i) during speech preparation, delivery, and

Table 1. Descriptive statistics ($n = 495$).

	M/%	*SD*	Min	Max
Dependent Variable				
Impulsive and Violent Crime	0.34	0.68	0.00	5.00
Biological Measures				
Testosterone	72.87	53.66	6.15	295.40
Cortisol	0.22	0.16	0.02	1.14
HR	80.16	12.84	49.36	119.87
SC	3.65	2.12	0.20	9.80
HRR	1166.52	1851.98	−4690.71	6987.83
SCR	229.47	219.82	−419.37	903.79
Control Measures				
GSCS	2.53	0.47	1.13	4.00
LSRPS	1.97	0.35	1.04	3.04
Parental Affection	3.59	0.63	1.00	4.00
Peer Delinquency	1.69	0.45	1.00	3.50
Relationship	0.43	0.50	0.00	1.00
Demographics				
Age	20.35	3.16	18.00	49.00
Female	66.5%	—	—	—
African American	12.7%	—	—	—
Asian	1.0%	—	—	—
White	37.2%	—	—	—
Hispanic	40.0%	—	—	—
Other	9.1%	—	—	—

HR = heart rate, SC = skin conductance, HRR = heart rate reactivity, SCR = skin conductance reactivity, GSCS = Grasmick self-control scale, LSRPS = Levenson self-report psychopathy scale. The possible min/max of scale measures correspond to the values of the Likert scale that was used to collect responses to items in the scale.

a short follow-up period (Pruessner et al. 2003). AUC_i represents the magnitude of change in the phenomenon of interest net of the starting value during the period under consideration. AUC_i has been used as a summary measure of changes in ANS activity in prior work attempting to understand the joint contribution of hormones and ANS activity to human behavior (Rimmele et al. 2007; Romero-Martínez et al. 2013). We quantify changes in ANS activity with AUC_i as it offers a single measure of change in ANS activity that incorporates all of the available ANS activity measurements. Descriptive statistics for the study of measures are presented Table 1 and correlations among ANS measures are presented in Supplementary Materials.

Survey measures

The self-reported survey included items measuring criminal behavior, measures of criminal propensity including self-control and psychopathy, and measures of the socio-environmental correlates of crime including delinquent peers, parental affection, and the presence of a romantic partner. A 10-item measure of impulsive and violent crime was derived from iterative principal component factor analyses of a 35-item self-report inventory of criminal behavior in the past year. Items in the self-reported inventory were factor analyzed in light of evidence indicating that associations between the biological substrates under consideration here (i.e., ANS activity and hormones) vary across antisocial and aggressive behavior types (Armstrong et al. 2021; Popma et al. 2007; Ruttle et al. 2011; van Bokhoven et al. 2005). During the factor analysis, items were discarded unless the largest loading for the item was on the largest factor. This process was repeated until the largest loading for each of the items was on the only factor with an eigenvalue over one. In the first and second factor models, there were 12 and 5 factors with eigenvalues over one, respectively. In the third iteration, a set of 10 items indicated a single factor with an eigenvalue over one. These items included indicators of vandalism, arson, assault, sexual assault, and petty theft and appeared to be reasonably well represented by the label impulsive and violent crime (Cronbach's $\alpha = .71$). Items were combined in a variety of scores, which increased by one for each different type of impulsive and violent crime that

a participant indicated that they had engaged in during the past year. Variety scores are preferable when scales included both serious and less serious forms of criminal behavior, as they are not overly influenced by the increased frequency of less serious forms of crime (Sweeten 2012). After visual inspection of the distribution of impulsive and violent crime scores, a single non-continuous outlier was re-scored as the next highest continuous value.

Self-control was measured with the 24-item Grasmick et al. (1993) self-control scale (GSCS; Cronbach's alpha = .84). Psychopathy was measured with the 26-item Levenson Self-Report Psychopathy Scale (LSRPS; Levenson, Kiehl, and Fitzpatrick 1995; Cronbach's alpha = .85). While there is evidence indicating the multidimensionality of GSCS and LSRPS (Brinkley et al. 2008; Conner, Stein, and Longshore 2009) here they are treated as indicators of general criminal propensity and analyses are based on aggregate scale scores.

Peer delinquency was assessed with 14 items asking respondents to specify the frequency with which their peers engaged in a variety of criminal activities ranging from substance use to property crime to violent crime (Cronbach's alpha = .84). Responses were recorded on a 5-item Likert scale ranging from '*none of them*' to '*all of them*.' Quantified in this way each item in the peer delinquency scale indicates the proportion of friends that have any engagement in a particular delinquent behavior and the sum of these items indicates the average proportion of friends that engage in delinquent behaviors. Scores for the peer delinquency measure and the remaining scales were estimated as the averaged sum of item scores. Parental affection was measured with four items inquiring as to whether or not the respondent's primary caregivers (e.g., mother/step-mother/foster-mother) loved and cared for the respondent before the age of 8 and from the age of 8 through age 16 (Cronbach's alpha = .77). Responses were recorded on a 4-point Likert scale ranging from '*Not at all*' to '*Very much*'. A dichotomous indicator of being in a romantic relationship was created from responses to a single item asking respondents to indicate their relationship status (married or not married, but in a relationship = 1; divorced, separated, not in a relationship = 0).

Analyses also included controls for age in years, race, and ethnicity as indicated by dummy variables for the two largest groups (Caucasian, 0 = no, 1 = yes; Hispanic, 0 = no, 1 = yes), and sex (0 = male, 1 = female).

Analytical strategy

Prior to the estimation of inferential statistics, the distributions of hormone and ANS measures were considered. Outliers were rescored to 3 SD from the mean (Wilcox 2010), and cortisol concentrations were log 10 transformed after the addition of a constant to address skewness. When measures exhibited sex differences, outliers were identified separately within sex. Details regarding this procedure are available in the Supplementary Materials.

Associations between hormones, ANS, and impulsive and violent criminal behaviors were tested with negative binomial regression models estimated with StataMP 15 (StataCorp 2017). Negative binomial regression models are preferable to Poisson models due to overdispersion and are specifically designed to accommodate the non-normal distribution present in count variables (Hilbe 2011). Zero inflated models were considered, but the proposed associations failed to meet the theoretical assumptions of this model (e.g., there is a different causal process for zeros; Allison 2012). To facilitate comparison of regression coefficients and the estimation of interaction terms, hormone, and ANS measures were standardized (z-scores). When hormone or ANS measures exhibited sex differences, standardization was done within sex. In the first regression model, the standardized hormone and ANS measures were regressed on impulsive and violent crime. Next, potential interactions between hormones and the ANS measures were tested by adding multiplicative interaction terms. Interaction terms modeled the interactive effects of each hormone measure with each type of ANS activity at both baseline and change. Finally, measures of criminal propensity (self-control and psychopathy) and controls for the effects of parents, peers, and romantic partners were added to the regression model. Potential multicollinearity was investigated with the

estat vif function after rerunning each model with Ordinary Least-Squares regression. The results indicated that multicollinearity was not an issue (all vifs < 2.1). All regression models included control variables representing the two largest race/ethnic groups in the sample (1 = Caucasian, 0 = other; 1 = Hispanic, 0 = other), sex (0 = male, 1 = female), age in years, and time of data collection represented with a whole number of hours on the 24-hour clock and fraction of minutes within an hour to two decimal places. Statistically significant interactions were visualized with Johnson-Neyman region of significance plots (D. J. Bauer and Curran 2005) generated with the tidyverse package (Wickham et al. 2019) in RStudio 4.2.1 (R Core Team 2020).

Prior to the specification of the analytic sample, patterns of missing data were assessed among all those participating in the laboratory protocol. The frequency of missing data across hormone measures, ANS measures, control measures, and criminal behavior ranged from 1.1% (crime measure) to 4.5% (heart rate responsivity). The frequency of missing data was similar for most of the theoretically motivated control measures ranging from 1.3% (romantic relationship) to 4.2% (self-control), with the exception of the psychopathy measure (11.2% missing data). Patterns of missing data were assessed with Little's test of missing completely at random implemented with the mcartest command (Li 2013; Little and Rubin 2002). Results indicated that data were not significantly different from missing completely at random for all analyses' variables ($\chi^2(541) = 547.27$, $p = .42$).

In the initial review of the results, all coefficients at the traditional $p < .05$ threshold were considered substantive. Following this, the implications of the Bonferroni correction for multiple comparisons are discussed (Benjamini and Hochberg 1995). This approach acknowledges both the importance of consideration multiple comparisons and concerns that corrections for multiple testing may stifle exploratory research when researchers deliberately limit the range of variables in data collection or analysis in order to increase the likelihood of meeting corrected thresholds for statistical significance (Nakagawa 2004). We also present coefficients and confidence intervals when reporting results for regression models. Nakagawa (2004) argues this approach is preferable to correction for multiple testing.

Results

Direct effects of hormone and ANS measures

Both testosterone and cortisol were positively associated with impulsive and violent crime (Table 2, Model 1; $b = 0.17$, $SE = 0.09$, $p = 0.046$, *95% CI* [0.00, 0.34]; $b = 0.21$, $SE = 0.09$, $p = 0.029$, *95% CI* [0.02, 0.40] respectively). Heart rate reactivity had a negative and statistically significant association with impulsive and violent crime ($b = -0.20$, $SE = 0.09$, $p = 0.034$, *95% CI* [−0.38, −0.02]), but the other ANS measures were not. Time of data collection was positively associated with the impulsive and violent crime measure ($b = 0.08$, $SE = 0.03$, $p = 0.022$, *95% CI* [0.01, 0.15]), while being female was associated with decreases in impulsive and violent crime ($b = -0.72$, $SE = 0.17$, $p = 0.000$, *95% CI* [−1.06, −0.38]).

Hormone and ANS measure interaction effects

The interaction between testosterone and baseline heart rate had a negative and statistically significant association with impulsive and violent crime (Table 2, Model 2; $b = -0.29$, $SE = 0.10$, $p = 0.003$, *95% CI* [−0.48, −0.10]), as did the interaction between testosterone and baseline skin conductance ($b = -0.20$, $SE = 0.09$, $p = 0.039$, *95% CI* [−0.38, −0.01]). Interactions between testosterone and ANS responsivity measures were not significantly associated with impulsive and violent crime. None of the interactions between cortisol and ANS measures were associated with impulsive and violent crime measures. Region of significance plots show that the association between testosterone and impulsive and violent crime is positive and statistically significant as baseline heart rate decreases from −0.29 standard deviations below the mean (heart rate = 76.44) and negative and statistically significant as baseline heart rate is over 1.54 standard deviations

Table 2. Negative binomial regression of impulsive and violent crime on hormone and autonomic nervous system measures with interaction terms ($n = 495$).

	Model 1			Model 2		
Measure	*b (SE)*	*p*	*95% CI*	*b(SE)*	*p*	*95% CI*
Time	0.08(0.03)	**0.022**	[0.01, 0.15]	0.06(0.03)	0.055	[−0.00, 0.13]
Caucasian	−0.19(0.23)	0.426	[−0.64, 0.27]	−0.18(0.23)	0.434	[−0.62, 0.27]
Hispanic	−0.06(0.22)	0.803	[−0.49, 0.38]	−0.05(0.22)	0.806	[−0.48, 0.38]
Age	−0.01(0.03)	0.801	[−0.07, 0.06]	−0.02(0.03)	0.634	[−0.08, 0.48]
Female	−0.72(0.17)	**0.000**	[−1.06, −0.38]	−0.62(0.17)	**0.000**	[−0.95, −0.28]
T	0.17(0.09)	**0.046**	[0.00, 0.34]	0.09(0.09)	0.292	[−0.08, 0.26]
C	0.21(0.09)	**0.029**	[0.02, 0.40]	0.18(0.09)	0.070	[−0.02, 0.37]
HR	−0.06(0.09)	0.526	[−0.24, 0.12]	−0.02(0.09)	0.868	[−0.20, 0.17]
HRR	−0.20(0.09)	**0.034**	[−0.38, −0.02]	−0.20(0.09)	**0.037**	[−0.38, −0.12]
SC	0.10(0.09)	0.257	[−0.07, 0.28]	0.05(0.09)	0.609	[−0.13, 0.22]
SCR	−0.04(0.09)	0.639	[−0.22, 0.13]	−0.06(0.09)	0.542	[−0.23, 0.12]
Interaction of Testosterone with ANS Measures						
TxHR	—	—	—	−0.29(0.10)	**0.003**	[−0.48, −0.10]
TxHRR	—	—	—	0.00(0.09)	0.959	[−0.16, 0.17]
TxSC	—	—	—	−0.20(0.09)	**0.039**	[−0.38, −0.01]
TxSCR	—	—	—	0.12(0.08)	0.120	[−0.03, 0.28]
Interaction of Cortisol with ANS Measures						
CxHR	—	—	—	0.08(0.09)	0.366	[−0.10, 0.27]
CxHRR	—	—	—	−0.12(0.09)	0.174	[−0.29, 0.05]
CxSC	—	—	—	0.04(0.10)	0.698	[−0.16, 0.24]
CxSCR	—	—	—	0.18(0.10)	0.062	[−0.01, 0.37]
	LR χ^2 (11, 484) = 37.19, $p = 0.001$, $R^2 = 0.050$			LR χ^2 (19, 476) = 60.46, $p = 0.000$, $R^2 = 0.081$		

T = Testosterone, C = Cortisol, HR = Heart rate, HRR = Heart rate reactivity, SC = Skin conductance, SCR = Skin conductance reactivity.

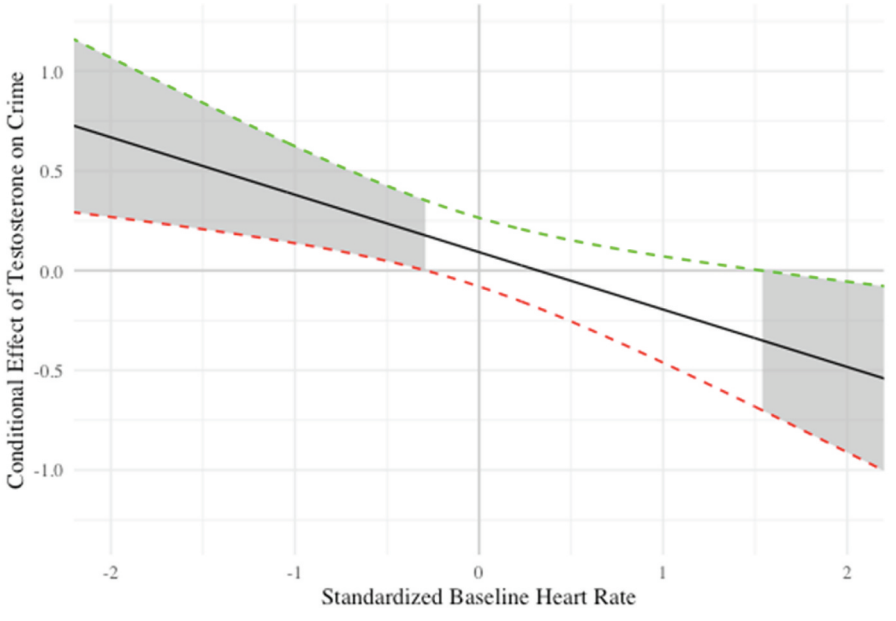
above the mean (heart rate = 99.93; Figure 1 Panel A). In parallel, the positive association between testosterone and impulsive and violent crime becomes significant when the baseline skin conductance decreases from −.42 standard deviations under the mean (skin conductance = 2.76; Figure 1 Panel B).

Hormone and ANS measure interaction effects with trait and socio-environmental controls

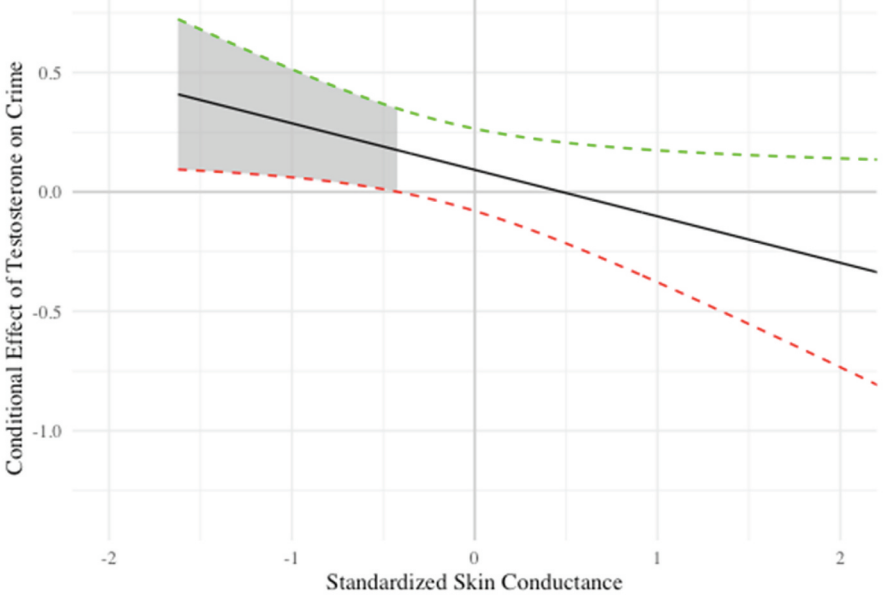
After the inclusion of measures quantifying traits and socio-environmental controls, the interaction of testosterone with baseline heart rate continued to have a negative and statistically significant association with impulsive and violent crime ($b = -0.22$, $SE = 0.10$, $p = 0.037$, *95% CI* [−0.42, −0.01]). The association between the interaction of testosterone with baseline skin conductance reactivity and impulsive and violent crime was no longer statistically significant, the interaction of cortisol with skin conductance reactivity emerged as statistically significant ($b = 0.22$, $SE = 0.10$, $p = 0.030$, *95% CI* [0.02, 0.41]). Full model results and region of significance plots of significant interactions are available as Supplemental Materials.

A consideration of multiple comparisons and the transformation of cortisol scores

The Bonferroni correction based on the number of direct and interactive associations between the measure of impulsive and violent crime and both hormone and ANS measures in each model resulted in a corrected *p* value of .00357 (.05/14). At this threshold, the interaction between testosterone and heart rate prior to the inclusion of theoretically motivated control variables remains statistically significant. All other associations fail to meet this threshold. While this highlights differences in the statistical significance of associations, we would suggest that given the novel nature of the analyses presented here all associations are strong candidates for replication. Variability in patterns of interaction between SNS and cortisol measures shown in research on childhood



Panel A Conditional effect of testosterone on crime by heart rate



Panel B Conditional effect of testosterone on crime by skin conductance

Figure 1. Region of significance plots for interactions between hormones and ANS measures.

behavior problems further highlights the need for replication and necessary caution when making theoretical inferences based on interactions between hormone and ANS measures (see Jones, Rohleder, and Schreier 2020).

The log 10 transformation of the cortisol measure undertaken prior to the estimation of inferential statistics is consistent with prominent studies on the role of hormones in the explanation of antisocial behaviors (e.g., Platje et al. 2015; Tackett et al. 2014). Nonetheless, we replicated all regression models with an untransformed baseline cortisol measure. In the direct effects model, the direction and statistical significance of all associations were the same. In the model with interactions and demographic controls, the interaction between testosterone and skin conductance was slightly attenuated (b =-0.17, SE = 0.09, p = 0.061, *95% CI* [−0.36, −0.01]). In the model with interactions and both demographic and theoretical control measures, the direct effect of HRR emerged as statistically significant b = −.20, SE = .10, p = .048 (b =-0.20, SE = 0.10, p = 0.048, *95% CI* [−0.39, <-0.01]).

Discussion

While strong conclusions await replication, the current results indicate that hormones and ANS activity interact to influence risk for impulsive and violent criminal behavior. Specifically, the positive association between testosterone and impulsive and violent crime became statistically significant as basal ANS activity decreased below its mean. This association is particularly robust for the interaction of testosterone with baseline heart rate (Table 2; Supplemental Table S2). In the broader literature informing associations between hormones, ANS activity, and antisocial behavior, there is some suggestion that the interaction of testosterone with heart rate may be mediated by sensation seeking resulting from increased reward salience. An association between testosterone and crime through sensation seeking is indicated by studies showing that testosterone influences activity in the brain's mesolimbic reward system (Laube, Lorenz, and van den Bos 2020; Op de Macks et al. 2011) and research showing a positive association between testosterone and risk preference (Kurath and Mata 2018). In addition, a small but growing group of studies show that the direct association between resting heart rate and crime is mediated by sensation seeking (Hammerton et al. 2018; Portnoy et al. 2014; Sijtsema et al., 2010). Thus, a positive association between testosterone and risk for impulsive and violent criminal behavior through increased reward salience may be amplified by increased sensation seeking at lower levels of heart rate. This potential explanation of the association between the interaction of testosterone with impulsive and violent criminal behavior is of course highly speculative. The search for traits with moderate associations between hormones and human behavior often result in weak associations and null findings (Sundin et al. 2021) though perhaps the consideration of multiple biological substrates may help to clarify relationships between biological substrates and traits.

Results also indicated that the positive association between cortisol and impulsive and violent crime was stronger at higher levels of skin conductance reactivity after the inclusion of controls for the trait and socio-environmental correlates of crime (Supplemental Table S2, Supplemental Figure S1 Panel B). This result is consistent with the increased risk for problem behavior as a function of additive interactions between ANS and HPA axis activity as hypothesized by Bauer, Quas, and Boyce (2002). At the trait level, elevated stress system activity indicated by the interaction of cortisol with skin conductance reactivity may result in increased risk for criminal behavior through emotional arousal including anger (Lobbestael, Arntz, and Wiers 2008; Moons, Eisenberger, and Taylor 2010). The weight given to this conjecture should be contextualized by the results of prior work exploring associations between youth problem behaviors and the interaction of cortisol with sAA, an alternative measure of SNS activity. A recent review of this study showed results varied with evidence of both positive and negative interactions between cortisol and SNS activity in association with youth problem behavior (Jones, Rohleder, and Schreier 2020). This variability suggests that inference

regarding the interactive associations between HPA-axis, SNS activity, and risk for criminal behavior should be regarded as tentative.

Heart rate reactivity had a negative direct association with impulsive and violent crime that survived the inclusion of interaction terms. The association between heart rate reactivity and impulsive and violent crime is to a certain extent anticipated by Ortiz and Raine (2004) who found the association between heart rate during a stressor and antisocial behavior ($d = -0.76$) was much larger in magnitude than the effect size for the association between heart rate at rest and antisocial behavior ($d = -0.44$). In the context of the current results, lower heart rate reactivity to stress may result in a tolerance for the aversive aspects of impulsive and violent crime. However, the negative direct association between heart rate reactivity and impulsive and violent crime did not extend to skin conductance reactivity, and both baseline measures of ANS activity did not have direct associations with the impulsive and violent crime measures. The lack of association between baseline heart rate and impulsive violent crime is particularly surprising. Meta analyses have confirmed an association between resting heart rate and general antisocial behavior (Portnoy and Farrington 2015, De Loof et al., 2022). It is possible that the lack of association is specific to the content of the impulsive and violent crime measure. Estimates of the association between measures of baseline heart rate and antisocial behavior show considerable heterogeneity across findings (De Loof et al., 2022).

Broadly speaking, the results herein suggest that biological substrates interact to influence risk for criminal behavior though the mechanism of this influence remains at issue. Booth and Osgood (1993) found the association between testosterone and a measure of adult deviance largely defined by criminal behavior was mediated by the inclusion of measures of social integration and prior criminal behavior. It is possible that the interaction between testosterone and heart rate is associated with criminal behavior in a similar fashion. This interaction may contribute to a behavioral disposition whose consequences accrete over the life-course, leading to increased risk for criminal behavior through state-dependent effects and by eroding social integration. A meaningful test of this proposed set of associations would require longitudinal measures of hormones and social integration. Similarly, the data used here lack the structure and measures necessary to capture interrelationships between aspects of biology and the proximal processes thought to directly influence patterns of development and behavior over the life-course. Nonetheless, these data do include measures of constructs featuring prominently in criminological theory. The measures capture major classes of the correlations of criminal behavior, including individual differences like self-control and psychopathy, and indicators of key aspects of the socio-environment, including parental affection and peer delinquency. Associations between criminal behavior and aspects of biology-like hormones and autonomic nervous system activity in models that include controls for these measures indicate that comprehensive theoretical accounts of risk for crime need to address individual differences including aspects of biology in addition to constructs featuring prominently in traditional criminological theories like parental affection and peer delinquency. These results also indicate that indicators of biological risk for criminal behavior do not operate in silos but instead may interact to influence behavioral dispositions, increasing risk for specific types of criminal behavior. The practical implications of the current work and for the broader literature on biological risk for criminal behavior remain remote as they require the development of a much more systematic knowledge base grounded in methodologically strong studies. However, it is possible that a better understanding of the role of biology in the explanation of risk for criminal behavior may facilitate better use of treatment and prevention resources through the matching of risk for specific types of criminal behavior to treatment and prevention modalities that are tailored towards ameliorating a given type of risk.

The implications of the current work should be contextualized by the characteristics of the sample under consideration and by other aspects of the research methodology. These concerns may be particularly salient with regard to the positive association between cortisol and impulsive and violent crime. As noted above, cortisol shows a bifurcated pattern of association with antisocial behavior including crime with negative associations often found in samples with high risk for criminal

behavior and in studies employing case–control methodologies (c.f., van Bokhoven et al. 2005; van Goozen et al. 2007). Thus, the positive associations between cortisol and crime found in this study both directly and in interaction with SCR may not generalize to samples with increased risk for criminal behavior, to alternative methods of analysis, or to general measures of criminal behavior.

The weight given results should also be conditioned by the statistical power of the analysis and by the retrospective prediction of past year criminality by biological measures. To contextualize the statistical power of the current study, we conducted a series of post hoc power analyses with G*Power. The first indicated that a sample size of 759 would have been necessary to detect the very small effect sizes ($d = .02$) with .80 power at the $p = .05$ level of statistical significance ($\alpha = .05$). While $d = .02$ is quite small, it is a reasonable benchmark for the magnitude of effect sizes found in moderation analyses. The required sample sizes for $d = .15$ and $d = .35$ were 109 and 53, respectively (.80 power at $\alpha = .05$). Collectively, results from the power analyses indicate that even though our sample size is very large compared to the strong majority of work considering associations between hormones and either traits or behaviors, it falls short of the mark for the detection of very small effect sizes at conventional levels of power and statistical significance. This may reduce the stability of statistical estimates and increase the importance of replication.

Analyses in the current study focused on associations between biological measures and retrospective measures of impulsive and violent crime. In support of this approach, we would note that studies testing the association between hormone measures and retrospective measures of antisocial behavior are not uncommon (e.g., Platje et al. 2015; Tackett et al. 2014) and that both hormone levels and antisocial behaviors including crime exhibit a fair amount of between individual stability over time (Nagin and Paternoster 2000; Zhang et al. 2017; Zmuda et al. 1997). Heart rate and skin conductance measurements taken both at rest and in response also show stability across time (Allen et al. 1987; El-Sheikh 2007; Hassellund et al. 2010). This stability appears to be stronger for heart rate than for skin conductance and there is some evidence that the stability of skin conductance reactivity is task specific (El-Sheikh 2007).

Nonetheless, stability in hormone and ANS measures is far from absolute and variation over time may introduce measurement error. The tendency to rely on retrospective behavioral measures is in part due to the costs of hormone analyses in both monetary terms and in time. While limited, this approach may be an efficient use of resources that provide initial findings that can serve to frame well-powered and prospective studies.

The results of the current study may also be influenced by aspects of the quantification of ANS function and hormone levels. The current study used plethysmography to measure heart rate. Plethysmography indexes arterial pulses to quantify heart rate. As an alternative to plethysmography, an electrocardiogram can provide a more accurate measure of heart rate by directly quantifying cardiac activity (Berntson et al. 1997). Results may also be influenced by diurnal variations in testosterone and cortisol (Brownlee, Moore, and Hackney 2005; Clow et al. 2010; Cumming, Quigley, and Yen 1983; Zhang et al. 2017). In order to account for diurnal variation, studies often collect samples for hormone assays in either the morning or afternoon, though there are studies with wider data collection windows (e.g., Welker et al. 2014). To account for the potential influence of the time of day on associations between hormones and crime, the time that salvia samples for hormone assays were collected was included as a control variable in regression models. However, a better understanding of the impact of time of day on associations between hormones and crime may come from randomization into data collection windows. Associations between hormone measures and criminal behavior in the current work may also be influenced by the use of enzyme immunoassays rather than tandem mass spectrometry. Comparisons of hormone values gathered with these respective methods are limited but predominantly show statistically significant correlations. However, in some cases, correlations between hormone values based on the respective methods lack statistical significance (Prasad et al. 2019; Welker et al. 2016).

Despite the concerns and caveats outlined above, the current work does show that the risk for criminal behavior is influenced by the interaction between distinct biological substrates. Specifically,

the interaction between hormones and ANS activity is associated with increased risk for impulsive and violent criminal behavior. While these results are preliminary and await replications, they demonstrate the potential utility of examining the interaction between biological substrates for attempts to specify the role of biology in the etiology of risk for criminal behavior. Future research in this direction should include the replication of the current results and the extension of the larger framework considering interactions between biological substrates in the explanation of risk for criminal behavior to other substrates such as brain structure and function. This study may also explore the extent to which the association between genetic variation and crime is conditioned by biological substrates known to influence risk for criminal behavior.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This work was supported by an Enhancement Grant for Professional Development from the Office of Research and Sponsored Programs at Sam Houston State University. Additional support was provided by an internal grant from the College of Criminal Justice at Sam Houston State University.

Notes on contributors

Todd, A. Armstrong, Ph.D., is a professor in the School of Criminology and Criminal Justice at the University of Nebraska Omaha. His research interests center on the nature and origins of criminal propensity with an emphasis on biological contributions to risk for criminal behavior.

Danielle L. Boisvert (Professor) is the Senior Associate Dean in the College of Criminal Justice at Sam Houston State University. Her key research interests include life-course/developmental criminology, biosocial criminology, and behavioral genetics.

Jessica Wells, Ph.D., is an Associate Professor in the Criminal Justice Program at Boise State University. Her research focuses on the underpinnings and impacts of trauma and abuse on antisocial behavior and psychological traits associated with antisocial behavior.

Richard H. Lewis, Ph.D., is an Assistant Professor at the University of Arkansas at Little Rock. His primary research interests are the role of biological and psychological factors in antisocial behavior and the consequences of trauma.

Eric M. Cooke, Ph.D., is an Assistant Professor in the Criminal Justice Program at Bowling Green State University. His primary research interests include life-course and developmental criminology, developmental processes and trajectories, biopsychosocial criminology, risky behaviors among adolescents and young adults, the effects that early life adversities have on later life outcomes, and victimology.

Matthias Woeckener, M.A., is an Assistant Professor in the Criminal Justice Program at the University of Nebraska at Kearney. His research interests include life-course and developmental criminology, biopsychosocial criminology, and attachment theory.

Nicholas Kavish, Ph.D., is a Neuropsychology Fellow in the Department of Neurological Sciences at the University of Nebraska Medical Center. His primary research interests include neurocognitive and personality assessment and psychometrics.

Nicholas Vietto, MS, is a Ph.D. student in the College of Criminology and Criminal Justice at the University of Nebraska-Omaha. His primary research interests are biopsychosocial criminology, quantitative methodology, and antisocial behavior.

James M. Harper, Ph.D., is an Associate Professor and the Graduate Coordinator in the Department of Biological Sciences at Sam Houston State University. His current research interests are in the characterization of speckled cockroaches as a new insect model for aging research, as well as quantifying the effect of plasticizing agents on physiological function in both insect and mammalian cell-line models. Dr. Harper also conducted collaborative research with psychologists and criminologists to study endocrine correlations of behavior in dogs and humans using salivary samples.

References

Allen, M. T., A. Sherwood, P. A. Obrist, M. D. Crowell, and L. A. Grange. 1987. "Stability of Cardiovascular Reactivity to Laboratory Stressors: A 2 1/2 Yr Follow-Up." *Journal of Psychosomatic Research* 31 (5): 639–645. https://doi.org/10.1016/0022-3999(87)90043-2.

Allison, P. 2012. "Do We Really Need Zero Inflated Models?" Statistical Horizons. http://statisticalhorizons.com/zero-inflated-models.

Armstrong, T., J. Wells, D. Boisvert, R. Lewis, E. Cooke, M. Woeckener, and N. Kavish. 2021. "An Exploratory Analysis of Testosterone, Cortisol, and Aggressive Behavior Type in Men and Women." *Biological Psychology* 161:108073. https://doi.org/10.1016/j.biopsycho.2021.108073.

Bauer, A. M., J. A. Quas, and W. T. Boyce. 2002. "Associations Between Physiological Reactivity and children's Behavior: Advantages of a Multisystem Approach." *Journal of Developmental & Behavioral Pediatrics* 23 (2): 102–113. https://doi.org/10.1097/00004703-200204000-00007.

Bauer, D. J., and P. J. Curran. 2005. "Probing Interactions in Fixed and Multilevel Regression: Inferential and Graphical Techniques." *Multivariate Behavioral Research* 40:373–400. https://doi.org/10.1207/s15327906mbr4003_5.

Benjamini, Y., and Y. Hochberg. 1995. "Controlling for the False Discovery Rate: A Practical and Powerful Approach to Multiple Testing." *Journal of the Royal Statistical Society: Series B (Methodological)* 57 (1): 289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x.

Berntson, G. G., J. T. Bigger, D. L. Eckberg, P. Grossman, P. G. Kaufmann, M. Malik, H. N. Nagaraja, et al. 1997. "Heart Rate Variability: Origins, Methods, and Interpretive Caveats." *Psychophysiology* 34 (6): 623–648. https://doi.org/10.1111/j.1469-8986.1997.tb02140.x.

Booth, A., and D. W. Osgood. 1993. "The Influence of Testosterone on Deviance in Adulthood: Assessing and Explaining the Relationship." *Criminology* 31 (1): 93–117. https://doi.org/10.1111/j.1745-9125.1993.tb01123.x.

Brinkley, C. A., P. M. Diamond, P. R. Magaletta, and C. P. Heigel. 2008. "Cross-Validation of Levenson's Psychopathy Scale in a Sample of Federal Female Inmates." *Assessment* 15 (4): 464–482. https://doi.org/10.1177/1073191108319043.

Brownlee, K. K., A. W. Moore, and A. C. Hackney. 2005. "Relationship Between Circulating Cortisol and Testosterone: Influence of Physical Exercise." *Journal of Sports Science & Medicine* 4 (1): 76–83.

Chen, F. R., A. Raine, and D. A. Granger. 2015. "Tactics for Modeling Multiple Salivary Analyte Data in Relation to Behavior Problems: Additive, Ration, and Interaction Effects." *Psychoneuroendocrinology* 51:188–200. https://doi.org/10.1016/j.psyneuen.2014.09.027.

Chichinadze, K., and N. Chichinadze. 2008. "Stress-Induced Increase of Testosterone: Contributions of Social Status and Sympathetic Reactivity." *Physiology & Behavior* 94 (4): 595–603. https://doi.org/10.1016/j.physbeh.2008.03.020.

Chrousos, G. P., and P. W. Gold. 1992. "The Concepts of Stress and Stress System Disorders: Overview of Physical and Behavioral Homeostasis." *JAMA* 267 (9): 1244–1252. https://doi.org/10.1001/jama.1992.03480090092034.

Clow, A., F. Hucklebridge, T. Stalder, P. Evans, and L. Thorn. 2010. "The Cortisol Awakening Response: More Than a Measure of HPA Axis Function." *Neuroscience & Biobehavioral Reviews* 35 (1): 97–103. https://doi.org/10.1016/j.neubiorev.2009.12.011.

Conner, B. T., J. A. Stein, and D. Longshore. 2009. "Examining Self-Control as a Multidimensional Predictor of Crime and Drug Use in Adolescents with Criminal Histories." *Journal of Behavioral Health Services & Research* 36 (2): 137–149. https://doi.org/10.1007/s11414-008-9121-7.

Cumming, D. C., M. E. Quigley, and S. S. C. Yen. 1983. "Acute Suppression of Circulating Testosterone Levels by Cortisol in Men." *Journal of Clinical Endocrinology & Metabolism* 57 (3): 671–673. https://doi.org/10.1210/jcem-57-3-671.

Dabbs, J. M., Jr., R. L. Frady, T. S. Carr, and N. F. Besch. 1987. "Saliva Testosterone and Criminal Violence in Young Adult Prison Inmates." *Psychosomatic Medicine* 49 (2): 174–182. https://doi.org/10.1097/00006842-198703000-00007.

De Looff, P. C., L. J. Cornet, C. H. De Kogel, B. Fernández-Castilla, P. J. Embregts, R. Didden, and H. L. Nijman. 2022. "Heart Rate and Skin Conductance Associations with Physical Aggression, Psychopathy, Antisocial Personality Disorder and Conduct Disorder: An Updated Meta-Analysis." *Neuroscience & Biobehavioral Reviews* 132:553–582. https://doi.org/10.1016/j.neubiorev.2021.11.003.

De Vries-Bouw, M., A. Popma, R. Vermeiren, T. A. Doreleijers, P. M. Van De Ven, and L. M. Jansen. 2011. "The Predictive Value of Low Heart Rate and Heart Rate Variability During Stress for Reoffending in Delinquent Male Adolescents." *Psychophysiology* 48 (11): 1597–1604. https://doi.org/10.1111/j.1469-8986.2011.01233.x.

El-Sheikh, M. 2007. "Children's Skin Conductance Level and Reactivity: Are These Measures Stable Over Time and Across Tasks?" *Developmental psychobiology* 49 (2): 180–186. https://doi.org/10.1002/dev.20171.

Fairchild, G., E. Baker, and S. Eaton. 2018. "Hypothalamic-Pituitary-Adrenal Axis Function in Children and Adults with Severe Antisocial Behavior and the Impact of Early Adversity." *Current Psychiatry Reports* 20 (10): 1–9. https://doi.org/10.1007/s11920-018-0952-5.

Geniole, S. N., B. M. Bird, J. S. McVittie, R. B. Purcell, J. Archer, and J. M. Carré. 2020. "Is Testosterone Linked to Human Aggression? A Meta-Analytic Examination of the Relationship Between Baseline, Dynamic, and Manipulated Testosterone on Human Aggression." *Hormones and Behavior* 123:104644. https://doi.org/10.1016/j.yhbeh.2019.104644.

Grasmick, H. G., C. R. Tittle, R. J. Bursik Jr, and B. J. Arneklev. 1993. "Testing the Core Empirical Implications of Gottfredson and Hirschi's General Theory of Crime." *The Journal of Research in Crime and Delinquency* 30 (1): 5–29. https://doi.org/10.1177/0022427893030001002.

Hammerton, G., J. Heron, L. Mahedy, B. Maughan, M. Hickman, and J. Murray. 2018. "Low Resting Heart Rate, Sensation Seeking and the Course of Antisocial Behaviour Across Adolescence and Young Adulthood." *Psychological Medicine* 48 (13): 2194–2201. https://doi.org/10.1017/S0033291717003683.

Handa, R. J., and M. J. Weiser. 2014. "Gonadal Steroid Hormones and the Hypothalamo–Pituitary–Adrenal Axis." *Frontiers in Neuroendocrinology* 35 (2): 197–220. https://doi.org/10.1016/j.yfrne.2013.11.001.

Hassellund, S. S., A. Flaa, L. Sandvik, S. E. Kjeldsen, and M. Rostrup. 2010. "Long-Term Stability of Cardiovascular and Catecholamine Responses to Stress Tests: An 18-Year Follow-Up Study." *Hypertension* 55 (1): 131–136. https://doi.org/10.1161/HYPERTENSIONAHA.109.143164.

Hilbe, J. M. 2011. *Negative binomial regression*. Cambridge, UK: Cambridge University Press.

Johnson, M. M., A. Mikolajewski, E. A. Shirtcliff, L. A. Eckel, and J. Taylor. 2015. "The Association Between Affective Psychopathic Traits, Time Incarcerated, and Cortisol Response to Psychosocial Stress." *Hormones and Behavior* 72:20–27. https://doi.org/10.1016/j.yhbeh.2015.04.010.

Jones, E. J., N. Rohleder, and H. M. Schreier. 2020. "Neuroendocrine Coordination and Youth Behavior Problems: A Review of Studies Assessing Sympathetic Nervous System and Hypothalamic-Pituitary Adrenal Axis Activity Using Salivary Alpha Amylase and Salivary Cortisol." *Hormones and Behavior* 122:104750. https://doi.org/10.1016/j.yhbeh.2020.104750.

Kagan, J., J. S. Reznick, and N. Snidman. 1988. "Biological Bases of Childhood Shyness." *Science* 240 (4849): 167–171. https://doi.org/10.1126/science.3353713.

Kemeny, M. E., and A. Shestyuk. 2008. "Emotions, the Neuroendocrine and Immune Systems, and Health." In *Handbook of Emotions*, edited by M. Lewis, J. M. Haviland-Jones, and L. F. Barrett, 661–675. New York: Guilford Press.

Kurath, J., and R. Mata. 2018. "Individual Differences in Risk Taking and Endogeneous Levels of Testosterone, Estradiol, and Cortisol: A Systematic Literature Search and Three Independent Meta-Analyses." *Neuroscience & Biobehavioral Reviews* 90:428–446. https://doi.org/10.1016/j.neubiorev.2018.05.003.

Kutlikova, H. H., J. B. Durdiakova, B. Wagner, M. Vlček, C. Eisenegger, C. Lamm, and I. Riečanský. 2020. "The Effects of Testosterone on the Physiological Response to Social and Somatic Stressors." *Psychoneuroendocrinology* 117:104693. https://doi.org/10.1016/j.psyneuen.2020.104693.

Latvala, A., R. Kuja-Halkola, C. Almqvist, H. Larsson, and P. Lichtenstein. 2015. "A Longitudinal Study of Resting Heart Rate and Violent Criminality in More Than 700 000 Men." *JAMA Psychiatry* 72 (10): 971–978. https://doi.org/10.1001/jamapsychiatry.2015.1165.

Laube, C., R. Lorenz, and W. van den Bos. 2020. "Pubertal Testosterone Correlates with Adolescent Impatience and Dorsal Striatal Activity." *Developmental Cognitive Neuroscience* 42:100749. https://doi.org/10.1016/j.dcn.2019.100749.

Levenson, M. R., K. A. Kiehl, and C. M. Fitzpatrick. 1995. "Assessing Psychopathic Attributes in a Noninstitutionalized Population." *Journal of Personality & Social Psychology* 68 (1): 151–158. https://doi.org/10.1037/0022-3514.68.1.151.

Li, C. 2013. "Little's Test of Missing Completely at Random." *The Stata Journal* 13 (4): 795–809. https://doi.org/10.1177/1536867X1301300407.

Ling, S., R. Umbach, and A. Raine. 2019. "Biological Explanations of Criminal Behavior." *Psychology Crime & Law* 25 (6): 626–640. https://doi.org/10.1080/1068316X.2019.1572753.

Little, R. J., and D. B. Rubin. 2002. *Statistical Analysis with Missing Data*. 2nd ed. New York: Wiley.

Lobbestael, J., A. Arntz, and R. W. Wiers. 2008. "How to Push someone's Buttons: A Comparison of Four Anger-Induction Methods." *Cognition & Emotion* 22 (2): 353–373. https://doi.org/10.1080/02699930701438285.

Lorber, M. F. 2004. "Psychophysiology of Aggression, Psychopathy, and Conduct Problems: A Meta-Analysis." *Psychological Bulletin* 130 (4): 531–552. https://doi.org/10.1037/0033-2909.130.4.531.

Lykken, D. T. 1957. "A Study of Anxiety in the Sociopathic Personality." *The Journal of Abnormal and Social Psychology* 55 (1): 6–10. https://doi.org/10.1037/h0047232.

Lykken, D. T. 1995. *The Antisocial Personalities*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Mazur, A., and A. Booth. 1998. "Testosterone and Dominance in Men." *Behavioral and Brain Sciences* 21 (3): 353–363. https://doi.org/10.1017/S0140525X98001228.

Moffitt, T. E., S. Ross, and A. Raine. 2011. "Crime and Biology." In *Crime and Public Policy*, edited by J. Q. Wilson, & J. Petersilia, 53–87.

Moons, W. G., N. I. Eisenberger, and S. E. Taylor. 2010. "Anger and Fear Responses to Stress Have Different Biological Profiles." *Brain, Behavior, and Immunity* 24 (2): 215–219. https://doi.org/10.1016/j.bbi.2009.08.009.

Murray, J., P. C. Hallal, G. I. Mielke, A. Raine, F. C. Wehrmeister, L. Anselmi, and F. C. Barros. 2016. "Low Resting Heart Rate is Associated with Violence in Late Adolescence: A Prospective Birth Cohort Study in Brazil." *International Journal of Epidemiology* 45 (2): 491–500. https://doi.org/10.1093/ije/dyv340.

Nagin, D., and R. Paternoster. 2000. "Population Heterogeneity and State Dependence: State of the Evidence and Directions for Future Research." *Journal of Quantitative Criminology* 16 (2): 117–144. https://doi.org/10.1023/A:1007502804941.

Nakagawa, S. 2004. "A Farewell to Bonferroni: The Problems of Low Statistical Power and Publication Bias." *Behavioral Ecology* 15 (6): 1044–1045. https://doi.org/10.1093/beheco/arh107.

Op de Macks, Z. A., B. G. Moor, S. Overgaauw, B. Güroğlu, R. E. Dahl, and E. A. Crone. 2011. "Testosterone Levels Correspond with Increased Ventral Striatum Activation in Response to Monetary Rewards in Adolescents." *Developmental Cognitive Neuroscience* 1 (4): 506–516. https://doi.org/10.1016/j.dcn.2011.06.003.

Ortiz, J., and A. Raine. 2004. "Heart Rate Level and Antisocial Behavior in Children and Adolescents: A Meta-Analysis." *Journal of the American Academy of Child and Adolescent Psychiatry* 43 (2): 154–162. https://doi.org/10.1097/00004583-200402000-00010.

Patrick, C. J., M. M. Bradley, and P. J. Lang. 1993. "Emotion in the Criminal Psychopath: Startle Reflex Modulation." *Journal of Abnormal Psychology* 102 (1): 82–92. https://doi.org/10.1037/0021-843X.102.1.82.

Platje, E., A. Popma, R. R. Vermeiren, T. A. Doreleijers, W. H. Meeus, P. A. van Lier, and L. M. Jansen. 2015. "Testosterone and Cortisol in Relation to Aggression in a Non-Clinical Sample of Boys and Girls." *Aggressive Behavior* 41 (5): 478–487. https://doi.org/10.1002/ab.21585.

Popma, A., R. Vermeiren, C. A. Geluk, T. Rinne, W. van den Brink, D. L. Knol, and T. A. Doreleijers. 2007. "Cortisol Moderates the Relationship Between Testosterone and Aggression in Delinquent Male Adolescents." *Biological Psychiatry* 61 (3): 405–411. https://doi.org/10.1016/j.biopsych.2006.06.006.

Portnoy, J., and D. P. Farrington. 2015. "Resting Heart Rate and Antisocial Behavior: An Updated Systematic Review and Meta-Analysis." *Aggression & Violent Behavior* 22:33–45. https://doi.org/10.1016/j.avb.2015.02.004.

Portnoy, J., A. Raine, F. R. Chen, D. Pardini, R. Loeber, and J. R. Jennings. 2014. "Heart Rate and Antisocial Behavior: The Mediating Role of Impulsive Sensation Seeking." *Criminology* 52 (2): 292–311. https://doi.org/10.1111/1745-9125.12038.

Prasad, S., B. Lassetter, K. M. Welker, and P. H. Mehta. 2019. "Unstable Correspondence Between Salivary Testosterone Measured with Enzyme Immunoassays and Tandem Mass Spectrometry." *Psychoneuroendocrinology* 109:104373. https://doi.org/10.1016/j.psyneuen.2019.104373.

Pruessner, J. C., C. Kirschbaum, G. Meinlschmid, and D. H. Hellhammer. 2003. "Two Formulas for Computation of the Area Under the Curve Represent Measures of Total Hormone Concentration versus Time-Dependent Change." *Psychoneuroendocrinology* 28 (7): 916–931. https://doi.org/10.1016/S0306-4530(02)00108-7.

Quay, H. C. 1965. "Psychopathic Personality as Pathological Stimulation-Seeking." *The American Journal of Psychiatry* 122 (2): 180–183. https://doi.org/10.1176/ajp.122.2.180.

Raine, A. 2002. "Annotation: The Role of Prefrontal Deficits, Low Autonomic Arousal, and Early Health Factors in the Development of Antisocial and Aggressive Behavior in Children." *Journal of Child Psychology and Psychiatry* 43 (4): 417–434. https://doi.org/10.1111/1469-7610.00034.

Raine, A., P. H. Venables, and M. Williams. 1990. "Relationships Between Central and Autonomic Measures of Arousal at Age 15 Years and Criminality at Age 24 Years." *Archives of General Psychiatry* 47 (11): 1003–1007. https://doi.org/10.1001/archpsyc.1990.01810230019003.

Raine, A., P. H. Venables, and M. Williams. 1995. "High Autonomic Arousal and Electrodermal Orienting at Age 15 Years as Protective Factors Against Criminal Behavior at Age 29 Years." *The American Journal of Psychiatry* 152 (11): 1595–1600.

R Core Team. 2020. "R: A Language and Environment for Statistical Computing." Vienna, Austria. R Foundation for Statistical Computing. https://www.R-project.org/.

Rimmele, U., B. C. Zellweger, B. Marti, R. Seiler, C. Mohiyeddini, U. Ehlert, and M. Heinrichs. 2007. "Trained Men Show Lower Cortisol, Heart Rate and Psychological Responses to Psychosocial Stress Compared with Untrained Men." *Psychoneuroendocrinology* 32 (6): 627–635. https://doi.org/10.1016/j.psyneuen.2007.04.005.

Romero-Martínez, A., M. Lila, R. K. Williams, E. González-Bono, and L. Moya-Albiol. 2013. "Skin Conductance Rises in Preparation and Recovery to Psychosocial Stress and Its Relationship with Impulsivity and Testosterone in Intimate Partner Violence Perpetrators." *International Journal of Psychophysiology* 90 (3): 329–333. https://doi.org/10.1016/j.ijpsycho.2013.10.003.

Ruttle, P. L., E. A. Shirtcliff, L. A. Serbin, D. B. D. Fisher, D. M. Stack, and A. E. Schwartzman. 2011. "Disentangling Psychobiological Mechanisms Underlying Internalizing and Externalizing Behaviors in Youth: Longitudinal and Concurrent Associations with Cortisol." *Hormones and Behavior* 59 (1): 123–132. https://doi.org/10.1016/j.yhbeh.2010.10.015.

Sapolsky, R. M., L. M. Romero, and A. U. Munck. 2000. "How Do Glucocorticoids Influence Stress Responses? Integrating Permissive, Suppressive, Stimulatory, and Preparative Actions." *Endocrine Reviews* 21 (1): 55–89. https://doi.org/10.1210/edrv.21.1.0389.

Sijtsema, J. J., Veenstra, R., Lindenberg, S., Van Roon, A. M., Verhulst, F. C., Ormel, J., Riese, H. "Mediation of sensation seeking and behavioral inhibition on the relationship between heart rate and antisocial behavior: The TRAILS study." *Journal of the American Academy of Child & Adolescent Psychiatry* 49 (5): 493–502.

StataCorp. 2017. *Stata Statistical Software: Release 15*. College Station, TX: StataCorp LLC.

Stauss, H. M. 2003. "Heart Rate Variability." *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology* 285 (5): R927–R931. https://doi.org/10.1152/ajpregu.00452.2003.

Sundin, Z. W., W. J. Chopik, K. M. Welker, E. Ascigil, C. M. Brandes, K. Chin, and J. L. Tackett. 2021. "Estimating the Associations Between Big Five Personality Traits, Testosterone, and Cortisol." *Adaptive Human Behavior and Physiology* 7 (3): 307–340. https://doi.org/10.1007/s40750-020-00159-9.

Sweeten, G. 2012. "Scaling Criminal Offending." *Journal of Quantitative Criminology* 28 (3): 533–557. https://doi.org/10.1007/s10940-011-9160-8.

Tackett, J. L., K. Herzhoff, K. P. Harden, E. Page-Gould, and R. A. Josephs. 2014. "Personality× Hormone Interactions in Adolescent Externalizing Psychopathology." *Personality Disorders Theory, Research, & Treatment* 5 (3): 235–246. https://doi.org/10.1037/per0000075.

van Bokhoven, I., S. H. M. Van Goozen, H. Van Engeland, B. Schaal, L. Arseneault, J. R. Séguin, and R. E. Tremblay. 2005. "Salivary Cortisol and Aggression in a Population-Based Longitudinal Study of Adolescent Males." *Journal of Neural Transmission* 112 (8): 1083–1096. https://doi.org/10.1007/s00702-004-0253-5.

van Goozen, S. H., G. Fairchild, H. Snoek, and G. T. Harold. 2007. "The Evidence for a Neurobiological Model of Childhood Antisocial Behavior." *Psychological Bulletin* 133 (1): 149–182. https://doi.org/10.1037/0033-2909.133.1.149.

Welker, K. M., B. Lassetter, C. M. Brandes, S. Prasad, D. R. Koop, and P. H. Mehta. 2016. "A Comparison of Salivary Testosterone Measurement Using Immunoassays and Tandem Mass Spectrometry." *Psychoneuroendocrinology* 71:180–188. https://doi.org/10.1016/j.psyneuen.2016.05.022.

Welker, K. M., E. Lozoya, J. A. Campbell, C. S. Neumann, and J. M. Carré. 2014. "Testosterone, Cortisol, and Psychopathic Traits in Men and Women." *Physiology & Behavior* 129:230–236. https://doi.org/10.1016/j.physbeh.2014.02.057.

Wickham, H., M. Averick, J. Bryan, W. Chang, L. D. McGowan, R. François, G. Grolemund, et al. 2019. "Welcome to the Tidyverse." *Journal of Open Source Software* 4 (43): 1686. https://doi.org/10.21105/joss.01686.

Wilcox, R. R. 2010. *Fundamentals of Modern Statistical Methods: Substantially Improving Power and Accuracy*. New York: Springer.

Wingfield, J. C., R. E. Hegner, A. M. Dufty Jr, and G. F. Ball. 1990. "The "Challenge Hypothesis": Theoretical Implications for Patterns of Testosterone Secretion, Mating Systems, and Breeding Strategies." *The American Naturalist* 136 (6): 829–846. https://doi.org/10.1086/285134.

Zhang, Q., Z. Chen, S. Chen, Y. Xu, and H. Deng. 2017. "Intraindividual Stability of Cortisol and Cortisone and the Ratio of Cortisol to Cortisone in Saliva, Urine and Hair." *Steroids* 118:61–67. https://doi.org/10.1016/j.steroids.2016.12.008.

Zmuda, J. M., J. A. Cauley, A. Kriska, N. W. Glynn, J. P. Gutai, and L. H. Kuller. 1997. "Longitudinal Relation Between Endogenous Testosterone and Cardiovascular Disease Risk Factors in Middle-Aged Men: A 13-Year Follow-Up of Former Multiple Risk Factor Intervention Trial Participants." *American Journal of Epidemiology* 146 (8): 609–617. https://doi.org/10.1093/oxfordjournals.aje.a009326.