Title
Examining the Psychobiology of Callous-Unemotional Traits

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Examining the Psychobiology of Callous-Unemotional Traits

DISSERTATION

submitted in partial satisfaction of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

in Psychological Science

by

Cortney Simmons

Dissertation Committee:
Professor Elizabeth Cauffman, Ph.D., Chair
Assistant Professor Kate Ryan Kuhlman, Ph.D.
Professor Jodi Quas, Ph.D.

2020

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ACKNOWLEDGMENTS

I would like to express the deepest appreciation and gratitude to my mentor and committee chair, Professor Elizabeth Cauffman. Without her guidance and persistent support, this dissertation and all that I have accomplished in graduate school would not have been possible. Beth, I cannot thank you enough for all that you have done for me.

I would also like to express sincere gratitude to my committee members, Professor Jodi Quas and Assistant Professor Kate Ryan Kuhlman, for providing helpful feedback on previous drafts and for their help in the design and framing of this project. I would also like to thank Professor Chuansheng Chen and Professor Michael Gottfredson for serving on my advancement committee and for their help in the design and conceptualization of this project. I am also grateful for my wonderful undergraduate research assistants and coordinator Anshuman Chiplunkar, without whom data collection would never have been possible. I also would like to thank the Crossroads study investigators, Professor Cauffman, Professor Paul J. Frick, and Professor Laurence Steinberg, for generously allowing me to use a portion of their data for this project.

Finally, I thank my parents, husband, and friends for their endless love, support, and encouragement over the past six years of graduate school. I would not have made it through without you all.

VITA

CORTNEY SIMMONS

EDUCATION

UNIVERSITY OF CALIFORNIA, IRVINE ***IRVINE, CA***

2014-	Doctoral Candidate in Psychological Science Specializations in Developmental Psychology, Psychology and the Law, and Quantitative Methods Advancement to Candidacy— May 2018 *Dissertation:* Examining the Psychobiology of Callous-Unemotional Traits
2017	Master of Arts in Social Ecology *Thesis*: The Differential Influence of Absent and Harsh Fathers on Juvenile Delinquency

RICE UNIVERSITY ***HOUSTON, TX***

2014	Bachelor of Arts with dual majors in Psychology and Kinesiology Academic Honors in Psychology

FELLOWSHIPS, GRANTS, AND AWARDS

2020	UCI Social Ecology Dean's Dissertation Writing Fellowship
2019	Society for Research on Adolescence (SRA) Emerging Scholar
2018	UC Consortium on the Developmental Science of Adolescence Seed Grant— $9960
2018	UCI Social Ecology Dean's Advancement Fellowship— $6683.67
2018	American Psychological Association (APA) Dissertation Research Award— $5000
2018	UCI Social Ecology Dean's Dissertation Data Collection Award— $1000
2018	EADP-EARA-SRA International Summer School— Junior Scholar
2018	Society for Research on Adolescence (SRA) Travel Award
2018	UCI Center for Psychology and Law Travel Award
2018	UCI DECADE Student Travel Award
2017	UC Consortium for Social Sciences and Law Summer Fellowship
2017	American Psychology-Law Society (AP-LS) Grant-in-Aid— $1000
2017	American Psychology-Law Society (AP-LS) Minority Affairs Committee Diversity Research Grant— $1000
2016-2019	National Science Foundation Graduate Research Fellow
2016-2018	UCI Center for Psychology and Law— Distinguished Student Fellow
2015-2019	UCI School of Social Ecology Graduate Mentoring Award
2015	Ford Foundation Graduate Research Fellowship- Honorable Mention
2014	UCI Provost Ph.D. Fellowship
2014	UCI Diversity Recruitment Fellowship

PUBLICATIONS

JOURNAL ARTICLES

Fine, A., **Simmons, C.**, Cavanagh, C., Rowan, Z., Frick, P.J., Steinberg, L., & Cauffman, E. (In press). Implications of Youths' Perceptions of the Police for the Cycle of Violence, Code of the Streets, and Violent Offending. *Psychology of Violence.*

Simmons, C., Fine, A., Knowles, A., Frick, P.J., Steinberg, L., & Cauffman, E. (2020). The

relation between callous-unemotional traits, psychosocial maturity, and delinquent behavior among justice involved youth. *Child Development.*

Simmons, C., Rowan, Z., Knowles, A., Steinberg, L., Frick., P.J., & Cauffman, E. (2019). A Life History Approach to Understanding Juvenile Offending and Aggression. *Aggression and Violent Behavior.*

Fine, A., Rowan, Z., & **Simmons, C.** (2019)**.** Do Politics Trump Race in Determining America's Youths' Perceptions of Law Enforcement? *Journal of Criminal Justice.*

Fine, A., **Simmons, C.**, Miltimore, S., Steinberg, L., Frick, P.J., & Cauffman, E. (2018). The school experiences of male adolescent offenders: Implications for academic performance and recidivism. *Crime & Delinquency, 64*(10), 1326-1350.

Simmons, C., Steinberg, L., Frick, P.J., & Cauffman, E. (2018). The differential influence of absent and harsh fathers on juvenile delinquency. *Journal of Adolescence, 62*, 9-17.

Mahler A., **Simmons, C**., Frick, P.J., Steinberg, L., & Cauffman, E. (2017). Aspirations, Expectations and Delinquency: The Moderating Effect of Impulse Control. *Journal of Youth and Adolescence*, *46*(7), 1503-1514.

Fine, A., Mahler, A., **Simmons, C.**, Chen, C., Moyzis, R., & Cauffman, E. (2016). Relations between three dopaminergic system genes, school attachment, and adolescent delinquency. *Developmental Psychology*, *52*(11), 1893-1903.

LAW REVIEWS

**Asterisks denote equal contribution from each author*

*Cauffman, E., *Fine, A., *Mahler, A., & ***Simmons, C**. (2018). How Developmental Science Influences Juvenile Justice Reform. *University of California, Irvine Law Review, 8*, 21-40.

PUBLICATIONS UNDER REVIEW

Simmons, C., Kan, E., Simpkins, S., Donley, S., Steinberg, L., Frick., P.J., & Cauffman, E. Assessing the association between participation in extracurricular activities and delinquent behavior among justice-involved youth.

OTHER WRITING

Fine, A., & **Simmons, C.** (2018). Moving justice-involved kids between schools may be good for their grades, but it may increase their reoffending. *Post for London School of Economics (LSE) US Center's blog on American Politics and Society*.

CONFERENCE PRESENTATIONS

Simmons, C., Frick, P.J., Steinberg, L., & Cauffman, E. (2020, March). *Examining the Age-Related Associations between Callous-Unemotional Traits, Impulsivity, and Delinquent Behavior* [Paper presentation]. Society for Research on Adolescence, San Diego, CA, United States. (Conference canceled).

Simmons, C., Kan, E., N., Steinberg, L., Frick, P.J., & Cauffman, E. (2020, March). *Examining the Influence of Justice System Involvement on Substance Use in Justice-Involved Youth* [Paper presentation]. Society for Research on Adolescence, San Diego, CA, United States. (Conference canceled).

Simmons, C., Knowles, A., Brown, C., Steinberg, L., Frick, P.J., & Cauffman, E. (2020, March). *The Psychological Consequences of Contact with the Justice System* [Paper presentation]. American Psychology and Law Society, New Orleans, LA, United States.

Simmons, C., Knowles, A., Duell, N., Fine, A., Steinberg, L., Frick, P.J., & Cauffman, E. (March

2019). *The Impact of Justice System Involvement on Adolescent Well-Being.* Paper presented at the Society for Research on Child Development Biennial Meeting in Baltimore, Maryland.

Simmons, C. & Cauffman, E. (March 2019). *Juveniles Offenders in Solitary Confinement* [Paper presentation]. American Psychology-Law Society (APLS) Conference, Portland, OR, United States.

Kan, E., **Simmons, C**., Simpkins, S., Steinberg, L., Frick, P.J., & Cauffman, E., (March 2019). *Unraveling bidirectionality between extracurricular activity participation and criminal behaviors* [Paper presentation]. American Psychology-Law Society (APLS) Conference in Portland, OR, United States.

Simmons, C., Fine, A., Mahler, A., Frick, P.J., Steinberg, L., & Cauffman, E. (2018). *The relation between callous-unemotional traits, psychosocial maturity, and delinquent behavior among justice involved youth* [Paper presentation]. American Psychology-Law Society (APLS) Conference, Memphis, TN, United States.

Cavanagh, C., Dalzell, E., **Simmons, C.,** & Cauffman, E. (2018). *CU Traits among Mothers and Juvenile Offenders* [Paper presentation]. American Psychology-Law Society (APLS) Conference, Memphis, TN, United States.

Craig-Welti, J., **Simmons, C.**, Nizkorodov, E., Heckmann, A., & Alvarez-Noli, K. (2018). *Addressing Graduate Student Wellness through Climate Committees*. [Workshop and presentation]. NASPA Student Affairs Administrators in Higher Education Annual Conference, Philadelphia, PA, United States.

Simmons, C., Frick, P.J., Steinberg, L., & Cauffman, E. (2017). *Bidirectional Effects of Callous-Unemotional Traits and Negative Parenting* [Poster presentation]. Society for Research on Child Development Biennial Meeting, Austin, TX, United States.

Donley, S., Fine, A., **Simmons, C.**, Cauffman, E., & Pluess, M. (2017). *Environmental Sensitivity Among Juvenile Offenders: Do Some Offenders Benefit from a Positive Home Environment More than Others?* [Paper presentation]. Society for Research on Child Development Biennial Meeting in Austin, TX, United States.

Simmons, C., Cavanagh, C., Cauffman, E., Frick, P.J., & Steinberg, L. (2016). *The influence of father- child relationship quality on adolescent delinquency* [Poster]. Society for Research on Adolescence Biennial Meeting, Baltimore, MD, United States.

Cauffman, E., Cavanagh, C., **Simmons, C.,** Frick, P.J., & Steinberg, L. (2016). *Substance use among first time juvenile offenders* [Paper presentation]. Society for Research on Adolescence Biennial Meeting, Baltimore, MD, United States.

Simmons, C., Cavanagh, C., & Cauffman, E. (2015). *Father-child relationship quality and adolescent offending* [Poster]. UC Consortium on Social Science and Law, Irvine, CA. United States.

Simmons, C. & Hebl, M. (2014). *The effect of challenging one's status on judgment of others and bullying.* Poster presented at the Psychology Undergraduate Research Conference at UCLA in Los Angeles, CA.

Simmons, C., Yeager, D. Lee, H., & Yang, M. (2014). *Sensitivity to hierarchy as a social motivator for facial recognition.* Poster presented at the biennial meeting of the Society for Research on Adolescence in Austin, TX.

RESEARCH EXPERIENCE

UNIVERSITY OF CALIFORNIA, IRVINE ***IRVINE, CA***

2019-2020 UC Irvine Center for Psychology and Law— Graduate Researcher
-Organize speaking events pertaining to topics on psychology and law for

students, faculty, and community members. Plan networking opportunities for graduate students and faculty members. Facilitate student relationships with practitioners to strengthen the application of research to community problems.

2014-2020 Development, Disorder, and Delinquency Lab— Graduate Researcher
Mentor: Elizabeth Cauffman, Ph.D.
- Investigated the long-term effects of juvenile justice system involvement on adolescent developmental outcomes, accounting for contextual, emotional and psychosocial factors. Responsible for conducting field interviews with juvenile offenders, training and supervising undergraduate research assistants, cleaning and analyzing data, and publishing and presenting findings at national conferences.

RICE UNIVERSITY ***HOUSTON, TX***

2012-2014 Industrial and Organizational Psychology Research Lab— Research Assistant and Undergraduate Honors Thesis
Mentor: Michelle Hebl, Ph.D.

UNIVERSITY OF TEXAS, AUSTIN ***AUSTIN, TX***

2013 Adolescent Development Lab— Research Assistant
Summer Undergraduate Research Experience
Mentor: David Yeager, Ph.D.

STATISTICAL AND METHODOLOGICAL TRAINING

2019 Longitudinal Data Analysis (Taught by Paul Allison)
2019 Latent Class and Latent Transition Analysis (Taught by Karen Nylund-Gibson)
2018 Time-Varying Effects Modeling (Self-Taught)
Longitudinal Structural Equation Modeling (Taught by Candice Odgers and Sandra Simpkins)
Treatment Effects Analysis (Steven Vaisey)
2017 Salivary Analyte and Immunoassay Basics (Taught by Douglas Granger)
Structural Equation Modeling (Taught by John Hipp)
2016 Applied Longitudinal Data Analysis (Taught by JoAnn Prause)
2015 Advanced Quantitative Methods (Taught by JoAnn Prause)

MENTORSHIP

2014-2020 UCI Undergraduate Research Opportunities Program (UROP)— Mentor
-Oversee team of undergraduate researchers in developing an empirical research question. Aid in the team's securing of funds, as well as completing applications to and presentations at professional conferences.

2016, 2017, 2018 UCI Competitive Edge Summer Research Program— Graduate Peer Mentor
-Advised incoming graduate students one-on-one to develop and execute an independent research project.

2017, 2018 UCI Department of Psychology and Social Behavior Post-Baccalaureate Program— Graduate Student Summer Mentor
-Advised two post-baccalaureate students one-on-one to develop their writing and draft applications for graduate programs.

2017-2018 UCI Undergraduate Campuswide Honors Program— Thesis Supervisor
Mentee: Elizabeth Duran

Title: The Association Between Immigration Status, Academics, and Delinquency among Justice-Involved Latino Youth

2016-2018 UCI Department of Psychology and Social Behavior— Graduate Peer Mentor
-Provided mentorship and advice for first-year students.

MENTORED STUDENTS' CONFERENCE PRESENTATIONS

Ochoa, A., Farias, L., Martin, J., Shickler, S., Brown, C., **Simmons, C.,** & Cauffman, E. (2020, August). *Callous-Unemotional Traits and Gun Use: Examining the mediating role of impulsivity* [Poster]. American Psychological Association Conference, Washington, D.C., United States.

Martin, J., Farias, L., Ochoa, A., Shickler, S., Brown, C., **Simmons, C.,** & Cauffman, E. (2020, May). *Examining Substance Use Trajectories Among Callous-Unemotional Variants* [Poster]. Association for Psychological Science Annual Convention, Chicago, IL, United States.

Meneses, B., Garcia, J., Gonzalez, E., Alanis, S., **Simmons, C.,** & Cauffman, E. (2019, May). *An Examination of the Influence of Sibling Criminality on Juvenile Delinquency* [Poster]. Association for Psychological Science Annual Convention, Washington, D.C., United States.

Iniguez, C., Sadri, A., Shelton, K., Hasal, S., **Simmons, C.,** & Cauffman, E. (2018, May). *The Influence of Justice System Involvement on the Offending Behavior of Callous-Unemotional Youth* [Poster]. Association for Psychological Science Annual Convention, San Francisco, CA, United States.

Sadri, A., Inguez, C., Shelton, K., Hasal, S., **Simmons, C.,** & Cauffman, E. (2018, August). *Examining the Individual and Contextual Differences Between Successful and Unsuccessful CU Youth* [PowerPoint presentation]. Young Investigator Symposium, American Psychological Association Conference, San Francisco, CA, United States.

Duran, E., **Simmons, C**., & Cauffman, E., (2018, July). *The Relationship Between Immigration Status, Academics, and Delinquency among Justice-Involved Latino Youth* [Poster]. American Psychological Association Conference, San Francisco, CA, United States.

Tom, K., Hruza, S., Galystan, E., **Simmons, C.**, & Cauffman, E. (2017). *Social relationships of callous-unemotional adolescents.* Presentation at the UCI Undergraduate Research Symposium in Irvine, CA, United States.

Galystan, E., Tom, K., Hruza, S., **Simmons, C.**, & Cauffman, E. (2017). *The Quantity and Quality of Peer Relationships among Callous-Unemotional Youth* [Poster]. Western Psychological Association Convention, Sacramento, CA, United States.

Hruza, S., Tom, K., Galystan, E., **Simmons, C.**, & Cauffman, E. (2017*). Social Relationships of Callous-Unemotional Youth: The Effects of Relationship Quality on Offending Behavior.* [Poster]. American Psychological Society Convention, Boston, MA, United States.

Clyde, A., Conod, P., Hernandez, B., Williams, L., **Simmons, C.**, & Cauffman, E. (2016). *The influence of probation officers on substance use and delinquency among youth from single-parent households* [PowerPoint presentation]. UCI Undergraduate Research Symposium, Irvine, CA, United States.

Clyde, A., Williams, L., Conod, P., Hernandez, B., **Simmons, C**, & Cauffman, E. (2016). *Substance Use, Juvenile Offending, and the Moderating Effect of Parent Structure.* [Poster]. American Psychological Society Convention, Denver, CO, United States.

Williams, L., Clyde, A., Conod, P., Hernandez, B., **Simmons, C**., & Cauffman, E. (2016). *Substance use, parents, and delinquency among juvenile offenders* [Poster]. Western Psychological Association Convention, Long Beach, CA, United States.

Serrano, V., Rodriguez, A., Castro, A., Hernandez, B., O'Bara, C., **Simmons, C**., Donley, S., &

Cauffman, E. (2015). *Mean boys: Relational aggression and resistance to peer influence among male juvenile offenders* [PowerPoint presentation]. UCI Undergraduate Research Symposium, Irvine, CA, United States.

Hernandez, B., Rodriguez, A., O'Bara, C., Castro, A., Serrano, V., **Simmons, C.**, Donley, S., & Cauffman, E. (2015*). Relational aggression and resistance to peer influence in male offenders* [Poster]. American Psychological Science Convention, New York City, NY, United States.

MENTORED STUDENTS' GRANTS AND AWARDS

2020	UCI Undergraduate Research Opportunities Program (UROP) Grant— $600
2019	UCI Undergraduate Research Opportunities Program (UROP) Grant— $600
2018	UCI Undergraduate Research Opportunities Program (UROP) Grant— $400
2018	UCI Undergraduate Research Opportunities Program (UROP) Grant— $400
2018	Association for Psychological Science Student Caucus— Travel Award
2017	UCI Undergraduate Excellence in Research Award—Belinda Hernandez
2017	UCI Undergraduate Research Opportunities Program (UROP) Grant— $500
2016	UCI Undergraduate Research Opportunities Program (UROP) Grant— $400
2015	UCI Undergraduate Research Opportunities Program (UROP) Grant— $700

TEACHING EXPERIENCE

COURSE DEVELOPMENT

2018 UC Irvine Online Master of Legal and Forensic Psychology Program
-Invited to develop the content and materials for a course entitled "Race and the Law" for L. Song Richardson, Dean and Chancellor's Professor of Law at UC Irvine

GUEST LECTURES

2019	*Understanding Independent Samples t-tests and ANOVAs*, Statistical Analysis in Social Ecology, Undergraduate
2018	*Latent Class and Latent Profile Analyses*, Graduate Seminar
2018	*School and the Purpose of Secondary Education*, Adolescent Development, Undergraduate
2017	*Adolescence in Time and Space*, Adolescent Development, Undergraduate
2016	*Substance Use*, Adolescent Development, Undergraduate

TEACHING ASSISTANTSHIPS

2019-2020	UC Irvine Online Master of Legal and Forensic Psychology Program Statistical Analysis in Social Ecology, Undergraduate
2016	Naturalistic Field Research, Undergraduate Childhood, Undergraduate Adolescent Development, Undergraduate
2015	Forensic Psychology, Undergraduate Human Sexuality, Undergraduate
2014	Abnormal Psychology, Undergraduate

SERVICE AND LEADERSHIP

2019-	Experimental Liaison—American Psychology-Law Society (AP-LS) Student Committee
2018-2019	Experimental Liaison— American Psychology-Law Society (AP-LS) Student Committee

2018	Planning Committee— Summer Institute for the UC Consortium on the Developmental Science of Adolescence
2018-	Ad Hoc Reviewer— *Journal of Research on Adolescence*

UNIVERSITY OF CALIFORNIA, IRVINE

2019	Coordinator— DECADE Professional Development Series for Enhancing Access and Quality of Research with Diverse Populations
2018-	Peer Grant Reviewer— School of Social Ecology
2018	Peer Grant Reviewer— Competitive Edge Summer Research Program
2016-2018	Student Representative— School of Social Ecology Climate Committee
2017-	Grant and Funding Information Coordinator— Department of Psychology and Social Behavior
2017	Guest Speaker— Department of Psychology and Social Behavior Funding Workshop
2017	Guest Speaker— Graduate Division Funding Workshop
2016	Guest Speaker— Department of Psychology and Social Behavior Funding Workshop
2016	Colloquium Coordinator— Psychology and Social Behavior Developmental Psychology
2015	Guest Speaker— ACCESS Social Ecology Program

PROFESSIONAL MEMBERSHIPS AND AFFILIATIONS

2018-	Society for Personality and Social Psychology
2017-	American Psychological Association (APA)
2016-	Society for Research in Child Development (SRCD)
2015-	American Psychology-Law Society (AP-LS)
2014-	Center for Psychology and Law, UC Irvine— Distinguished Student Fellow
2014-	Society for Research on Adolescence (SRA)— Student Member

ABSTRACT OF THE DISSERTATION

Examining the Psychobiology of Callous-Unemotional Traits

By

Cortney Simmons

Doctor of Philosophy in Psychological Science

University of California, Irvine, 2020

Professor Elizabeth Cauffman, Chair

Callous-unemotional (CU) traits (e.g., lack of empathy, deficient guilt/remorse, and shallow affect) are a risk factor for delinquent behavior. Youth high in CU traits exhibit a range of cognitive and emotional deficits, such as fearlessness, insensitivity to punishment, and reward dependency, that predispose them to antisocial behavior and justice system involvement. This two-part dissertation study sought to better understand how CU traits predispose youth to delinquent behavior by examining the hormones thought to underlie these emotional and cognitive deficits. Employing a sample of 55 justice-involved male youth recruited from an ongoing longitudinal study of first-time juvenile offenders, Study One examined the association between CU traits, adverse experiences (prior exposure to violence and hostile parent-child relationships), and cortisol and alpha-amylase reactivity. The results indicate that individuals with elevated levels of CU traits exhibit lower cortisol reactivity than their counterparts. However, they exhibited the same pattern of alpha-amylase reactivity. In addition, prior adversity did not moderate the association between CU traits and either indicator of stress reactivity. Study Two tested whether cortisol and testosterone mediated the association between CU traits, reward and punishment sensitivity, and risk taking. Unfortunately, the study was

underpowered and could not determine whether there was a direct or indirect association between CU traits, risk taking, or reward/punishment sensitivity. However, the study provided evidence that individuals with high CU traits exhibited the same pattern of testosterone reactivity as their counterparts. Taken together, the results of the two studies suggest youth with elevated levels of CU traits exhibit a pattern of stress reactivity that may contribute to their behavior. Future research should continue to explore the relation between stress reactivity, CU traits, and other aspects of antisocial behavior, such as aggression and retaliation.

INTRODUCTION

Callous-unemotional (CU) traits (e.g., lack of empathy, deficient guilt/remorse, and shallow affect) are a robust and well-established risk factor for delinquent behavior (Frick, Ray, Thornton, & Kahn, 2014). Youth high in CU traits exhibit a range of cognitive and emotional deficits, such as impaired recognition of and consideration for the emotions of others, fearlessness, insensitivity to punishment, and reward dependency, that predispose them to delinquent behavior (Blair, 2005; Herpers, Scheepers, Bons, Buitelaar, & Rommelse, 2014; van Goozen, Fairchild, Snoek, & Harold, 2007). Indeed, compared to other antisocial youth, youth with elevated CU traits are more likely to engage in violent, aggressive, and severe antisocial behavior. Studies demonstrate that even after accounting for other known risk factors and protective factors, such as IQ, peer deviancy, impulse control, and psychosocial maturity (Hampton, Drabick, & Steinberg, 2013; Kahn, Byrd, & Pardini, 2013; Muñoz, Frick, Kimonis, & Aucoin, 2008; Simmons et al., 2020), CU traits are a strong predictor of delinquency. Alarmingly, youth high in CU traits exhibit a more stable pattern of delinquency such that their criminal behavior is more likely to persist into adulthood compared to other antisocial youth (McMahon, Witkiewitz, & Kotler, 2010). Considering the extensive societal and monetary costs associated with criminal behavior and justice-system involvement, preventing and reducing delinquent behavior among youth with high levels of CU traits is critical.

To determine how CU traits predispose youth to delinquent behavior, researchers have sought to identify the biological mechanisms underlying the emotional and cognitive deficits exhibited by youth high in CU traits (for review see Glenn & Raine, 2014; Mouls et al., 2018). Neurological research on these deficits suggests that CU and psychopathic traits are associated with atypical function in the brain regions underpinning emotional, social, and reward-

processing (Cardinale et al., 2018; Raschle et al., 2018; Sethi et al., 2018; Seara-Cardoso & Viding, 2015). Hormones are one of the primary mechanisms through which brain functioning affects behavior—in response to certain stimuli, they are released into the body to influence how individuals perceive, process, and physiologically respond to the stimuli (Gunnar & Quevedo, 2007). Importantly, hormone levels can be influenced by an individual's environment and behavior and affect how the brain functions. Hormones produced by the hypothalamus-pituitary-adrenal (HPA) axis, sympathetic nervous system (SNS), and hypothalamus-pituitary-gonadal (HPG) axis can influence the brain regions implicated in CU traits (Blair, 2005; Terburg, Morgan, & van Honk, 2009). As such, hormones may be prime targets in behavioral treatments for youth with CU traits as they may be able to address the aspects of cognitive and emotional functioning that contribute to delinquency.

Existing knowledge of the biological correlates of CU traits is largely drawn from neurological and physiological research on a related adult disorder: psychopathy. Although often equated and used interchangeably, psychopathy and CU traits have distinct definitions and developmental considerations. Adults with psychopathic traits exhibit an arrogant and deceitful interpersonal style, an impulsive and irresponsible behavioral style, and deficiencies in affect. CU traits correspond with the affective dimension of psychopathy, with youth high in CU traits exhibiting a lack of guilt and empathy, show callous use of others for one's own gain, and exhibit deficient emotionality (Frick et al., 2014). Many of the behavioral features of psychopathy are normative during adolescence (e.g. impulsivity and irresponsbility), causing researchers to question the downward extension of adult psychopathy to youth (Skeem & Cauffman, 2003). Consequently, it is unclear whether the findings of the psychobiology of psychopathy should apply to CU traits.

A better understanding of CU traits is crucial for developing effective early delinquency prevention and treatment efforts. To contribute to this important line of research, this dissertation focused on the relation between CU traits and hormones released by three biological systems: cortisol from the hypothalamic-pituitary-adrenal (HPA) axis, the alpha-amylase from the sympathetic nervous system (SNS), and testosterone from hypothalamic -pituitary-gonadal (HPG) axis. Employing a sample of justice-involved male youth recruited from an ongoing longitudinal study of first-time juvenile offenders, Study One examined the association between CU traits, adverse experiences (prior exposure to violence and hostile parent-child relationships), and cortisol and alpha-amylase reactivity. Study Two tested whether cortisol and testosterone mediated the association between CU traits, reward and punishment sensitivity, and risk taking. By examining the association between CU traits and these hormones, this dissertation provides some insight into the malleable factors underlying the cognitive and emotional deficits exhibited by individuals with elevated CU traits exhibit.

STUDY ONE

Research Rationale

Youth high in CU traits exhibit a range of cognitive and emotional deficits, such as insensitivity to punishment and fearlessness, that suggest there are distinct causal factors underlying their behavior. Many researchers believe the stress response system and its effects on the amygdala contribute to these deficits (Blair, 2005; Herpers et al., 2014; van Goozen et al., 2007). When the amygdala is functioning normally, increased activity in this brain region is associated with increased fear and withdrawal behavior. Accordingly, dysfunction in the amygdala may explain why youth high in CU traits are less responsive to the threat of punishment and more likely to engage in antisocial behavior (Frick, Ray, Thornton, & Kahn, 2014). Research suggests that impairments in amygdala functioning are likely caused by abnormal levels of hormones released by two components of the stress response system: the sympathetic nervous system (SNS) and the hypothalamus-pituitary-adrenal (HPA) axis. Cortisol—the end-product of the HPA axis— and norepinephrine—a product of the SNS—typically surge in response to threats (Dickerson & Kemeny, 2004; Gunnar, Talge, & Herrera, 2009; Quas et al., 2014; Stroud et al., 2009) and act on the amygdala. Increased cortisol levels potentiate a state of fear and withdrawal behaviors, while increased norepinephrine is associated with activation of neural circuits in the amygdala and enhances ones' ability to recognize and learn from threatening stimuli (Gunnar & Quevedo, 2007). Importantly, individual and environmental factors may reduce how much of these hormones are released by the stress response system (Bauer, Quas, & Boyce, 2002), subsequently disrupting amygdala functioning and increasing one's risk for behavioral problems.

Callous-Unemotional Traits, Adversity, and Stress Reactivity

Callous-unemotional and psychopathic individuals exhibit patterns of cognitive and emotional responses associated with reduced activity in the stress system— youth with high levels of CU traits exhibit fearlessness, insensitivity to punishment, and impaired emotional processing (Gao, Baker, Raine, Wu, & Bezdjian, 2009; Jones, Happe, Gilbert, Burnett, & Viding, 2010; van Honk & Schutter, 2006). These findings lead many researchers to believe that individuals with high CU traits have a stress response system that reduces responsivity in the amygdala. Several studies provide evidence of a relation between elevated CU traits and reduced HPA activity (for review see Glenn & Raine, 2014; Moul et al., 2018). For example, Loney and colleagues (2006) examined the relation between CU traits and basal cortisol in a sample of 108 adolescents ages 12 to 18 years old. Among the male youth, CU traits were associated with low basal cortisol levels. Subsequent studies on cortisol reactivity among college undergraduate students suggest that males high in psychopathic traits also exhibit a blunted cortisol response to a social evaluative threat (O'Leary, Loney, & Eckel, 2007; O'Leary, Taylor, & Eckel, 2010). A slightly different pattern was observed in a study of incarcerated young adults who either responded (i.e., showed an increase in cortisol levels) or did not respond to a stressor (M. M. Johnson, Mikolajewski, Shirtcliff, Eckel, & Taylor, 2015). Among the responders, the researchers found individuals with higher levels of affective psychopathic traits (e.g., callousness) did not exhibit a lower cortisol response. Among the non-responders, individuals with high affective psychopathic traits exhibited a significant *decrease* in cortisol following a stressor.

It is unclear whether youth high in CU traits also exhibit reduced SNS reactivity in response to stressors. Deficits in norepinephrine, a product of the SNS that influences amygdala functioning, may contribute to the cognitive and emotional deficits observed among youth with

high levels of CU traits. For example, an experimental study found that when the effect of norepinephrine was blocked (accomplished with the betablocker propranolol), amygdala activation to negative emotional images decreased (van Stegeren et al., 2005). This finding suggests norepinephrine is needed to generate an appropriate response to emotional stimuli. Despite its importance, norepinephrine has not been examined in psychobiological research on CU traits. Studies on psychopathy and other indicators of SNS activity, such as electrodermal arousal and heart rate, are mixed—some studies indicate psychopathic and callous-unemotional individuals are less reactive to aversive stimuli, while others find no association between psychopathy and SNS activity (Anastassiou-Hadjicharalambous & Warden, 2008; de Wied, van Boxtel, Matthys, & Meeus, 2012; Fung et al., 2005; Lorber, 2004). Researchers can potentially address this gap in the literature by examining the relation between CU traits and alpha-amylase. Alpha-amylase is an enzyme found in saliva that is highly correlated with changes in norepinephrine and SNS activity (Granger, Kivlighan, el-Sheikh, Gordis, & Stroud, 2007; Rohleder, Nater, Wolf, Ehlert, & Kirschbaum, 2004; Thoma, Kirschbaum, Wolf, & Rohleder, 2012; van Stegeren, Rohleder, Everaerd, & Wolf, 2006). SNS activity triggers increases in norepinephrine, which in turn stimulates the production of alpha-amylase by the salivary glands (Bosch, de Geus, Veerman, Hoogstraten, & Nieuw Amerongen, 2003). As such, alpha-amylase can be used as an indicator for the release of norepinephrine and the SNS response to stress.

There is substantial evidence that adversity during childhood and adolescence is also associated with dysfunction in the stress response system (Koss & Gunnar, 2018). In regard to the HPA axis, exposure to prolonged and chronic stress may alter HPA activity and produce patterns of hypocortisolism, or suppression of cortisol (for review, see Koss & Gunnar, 2018). Low cortisol levels attributed to adversity are observable even in response to acute threats. For

example, Lovallo and colleagues (2012) found that adults who were exposed to high levels of lifetime adversity exhibited blunted cortisol reactivity in response to a laboratory stressor. This pattern is seen among younger individuals as well. Compared to those with low levels of exposure to violence, male adolescents with high exposure to violence exhibited lower cortisol reactivity in response to a laboratory stressor (Peckins, Dockray, Eckenrode, Heaton, & Susman, 2012). Similarly, previous research suggests early adversity can have lasting effects on SNS reactivity; however, the direction of the effect is inconsistent. Several studies indicate adversity is associated heightened and prolonged SNS responses (Lovallo et al., 2012; McLaughlin et al., 2015). Employing a sample of 41 young adults, one study examined the association between childhood adversity and alpha-amylase reactivity in response to a social evaluative threat (Kuras et al., 2018). Individuals with a history of adversity exhibited a greater stress response, indicated by higher levels of alpha-amylase. In contrast, other studies have found that individuals exposed to a greater number of adverse experiences showed a smaller change in heart rate in response to a social threat (Lovallo et al., 2012; Voellmin et al., 2015). Regardless of the direction, it is apparent that adversity has the potential to disrupt both SNS and HPA activity.

Although research has connected both CU traits and adversity to altered stress reactivity, the simultaneous effect of CU traits and adversity on stress reactivity has only been examined in two studies on cortisol. In the first study, researchers examined the association between psychopathy, traumatic childhood experiences, and diurnal cortisol within a sample of incarcerated male adults (Cima, Smeets, & Jelicic, 2008). They found that diurnal cortisol levels were lower among adults with high levels of psychopathic traits. However, within the group scoring high in psychopathy, those who experienced child abuse exhibited significantly higher cortisol levels. In the second study, researchers examined the association between CU traits, life

stress exposure, and two indicators of HPA activity—diurnal rhythm and cortisol awakening response—in a sample of incarcerated male youth (Gostisha et al., 2014). Consistent with previous findings (Cima et al., 2008), the results indicated that both greater life stress exposure and elevated CU traits were related to low levels of cortisol. However, life stress moderated the association between CU traits and cortisol, such that the lowest waking cortisol levels were observed among youth with high CU and high stress exposure. Given the discrepant findings, measurements of cortisol, and different developmental periods examined in these studies, additional research is needed to clarify how CU traits and adversity independently and jointly affect stress reactivity functioning in youth.

Asymmetry in HPA Axis and SNS Activity

Although there is evidence that the HPA axis and SNS are interrelated, most studies on individuals with CU or psychopathic traits focus on only one component of the stress response system. Research on the HPA axis and SNS suggests that these components each generate a physiological response to stressors (Chrousos, 2009; Sapolsky, Romero, & Munck, 2000). SNS activation is responsible for the fast-acting defensive "flight or fight" response (e.g., enhanced respiratory rate and blood flow to muscles), while HPA activation is slow-acting and mobilizes energy resources. Bauer, Quas, and Boyce (2002) theorized that concurrently low or asymmetric HPA and SNS reactivity may place youth at risk for adjustment or behavioral problems. The results of studies testing this hypothesis have been mixed. The first study examined whether alpha-amylase and cortisol reactivity were related to adolescent aggression (Gordis, Granger, Susman, & Trickett, 2006). Aggression was highest among individuals with low cortisol and alpha-amylase, and lowest among those with high cortisol and low alpha-amylase. The finding was replicated in samples of community (Susman et al., 2010) and justice-involved male

adolescents (de Vries-Bouw et al., 2012). In contrast to the studies on antisocial behavior, research on maltreated youth have observed asymmetry in HPA and SNS activity (Ali & Pruessner, 2012; Vigil, Geary, Granger, & Flinn, 2010). For example, Gordis and colleagues (2008) found that while cortisol and alpha-amylase were positively associated among non-maltreated youth, there were no significant association among maltreated youth. As of now, only one study has examined how the HPA axis and SNS operate together among youth with elevated levels of CU traits. Glenn and colleagues (2015) examined the relation between alpha-amylase reactivity, cortisol reactivity, and psychopathic traits. Surprisingly, psychopathy was unrelated to both cortisol and alpha-amylase reactivity. The researchers also reported there were no significant increases in either cortisol or alpha-amylase, which was likely a result of reactivity being assessed with a single sample collected twelve minutes post-stressor. Given alpha-amylase and cortisol are expected to peak five and 20 minutes after the stressor (Granger et al., 2007), they may have failed to capture the increase. Considering the limitations of this study, additional research on CU traits and both cortisol and alpha-amylase is needed.

Aims and Hypotheses

This study examined the association between CU traits, adversity, and stress reactivity. The first aim was to determine whether CU traits and adversity (exposure to violence and parental hostility) were independently associated with cortisol and alpha-amylase reactivity. I hypothesized that CU traits and both types of adversity would predict cortisol and alpha-amylase stress reactivity. Specifically, higher levels of CU traits and greater adversity would be associated with lower levels of cortisol and alpha-amylase reactivity. The second aim was to determine whether the association between CU traits and stress responsivity was moderated by adversity. I hypothesized that the CU/stress reactivity association would be moderated by

adversity, such that youth high in both CU traits and adversity would exhibit lower cortisol and alpha-amylase reactivity than individuals with high CU traits and low levels of adversity. The final aim was to examine whether CU traits were associated with asymmetric stress responses. I hypothesized that youth high in CU traits would exhibit greater asymmetry in cortisol and alpha-amylase levels than youth with low CU traits. Further, youth high in both CU traits and adversity would exhibit the greatest asymmetry.

Method

Participants

Participants were recruited from the Crossroads Study, a longitudinal study that prospectively examines the effects of juvenile justice system contact on the development of 1,216 male first-time offenders. Youth were ages 13-17 when they were recruited into the study after being arrested for a range of low-level (misdemeanor) offenses. Crossroads youth were recruited from three sites— Philadelphia, Pennsylvania (n = 533); Jefferson Parish, Louisiana (n = 151); and Orange County, California (n =532). Crossroads Study participants were eligible for this dissertation if they had completed at least five Crossroads interviews (80% retention rate). Recruiting Crossroads participants who were not missing data for four or more interviews allowed for more reliable estimates of their adverse experiences. Additionally, due to location and transportation limitations, only Crossroads participants from the Orange County site were eligible for the study.

A total number of 465 Crossroads participants met the inclusion criterion and were eligible for the current study. Of the 465 eligible participants, 55 youth were successfully recruited and completed the study procedures. The final sample was aged 21 to 25 years (*M*= 22.84, SD= 1.15), with 80.00% having at least one parent who had a high school degree or more.

In regard to race/ethnicity, 78.18% of the sample self-identified as Latino, 16.36% White, 1.82% Black, and 3.64% Other (which is reflective of the Orange County sample; see Table 1.1).

The remaining 410 eligible youth did not participate because contact was unsuccessful during the recruitment period (n= 148), they refused (n= 55), the youth scheduled a session and did not show (n= 35), they had moved out of Orange County (n= 25), youth were in custody (n= 10), the youth had recently withdrawn from the Crossroads Study (n= 2), or contact was not attempted before the recruitment period ended (n= 134). One participant was a UCI student and was not contacted in order to preserve the integrity of the Crossroads Study. To determine whether the final sample was characteristically different than the eligible youth who did not participate, we tested differences in demographics, CU traits, adverse experiences, and lifetime offending between the final sample, non-participating eligible Crossroads youth, refusals, and no-shows (Table 1.1.). The results indicate youth in the final sample exhibited significantly higher IQs than non-participating eligible youth (d= -0.47) and no-show youth (d= -0.44). Compared to the final sample, a smaller proportion of the non-participating eligible youth (φ= -0.13) and refusals (φ= -0.22) reported that their parent had obtained a HS degree or more. The final sample reported higher parental hostility (d= -0.30) than the non-participating youth. The final sample youth had lower levels of CU traits than the no-show youth (d= -.44).

Table 1.1.
Differences between Dissertation Sample, full Crossroads Study sample, and No-Shows

	Dissertation Sample (N=55)	Eligible Crossroads Sample (N= 410)		Refusals (N= 55)		No-Shows (N= 35)	
	M / %	*M* / %	*d* / φ	*M* / %	*d* / φ	*M* / %	*d* / φ
Baseline Age	15.35	15.49	0.12	15.60	0.21	15.31	-0.03
Black[a]	1.82	0.73	-0.04	0.00	-0.10	5.71	0.11
Latino[b]	78.18	78.05	0.00	81.82	0.05	82.86	0.06
White[c]	16.36	17.56	0.01	12.73	-0.05	8.57	-0.11
Parent Education[d]	80.00	60.98	**-0.13****	60.00	**-0.22***	80.00	0.00
IQ	94.87	89.65	**-0.47*****	91.47	0.30	89.77	**-0.44***
CU Traits	19.56	20.63	0.13	19.79	0.03	23.03	**0.43***
Parental Hostility	1.49	1.42	**-0.30***	1.42	-0.29	1.50	0.07
Exposure to Violence	3.24	3.35	0.04	3.04	-0.07	4.37	0.38
Lifetime Offending	4.95	4.89	-0.01	4.07	-0.24	6.31	0.32

[a]Black= 1, Non-Black= 0, [b]Latino= 1, Non-Latino= 0, [c]White= 1, Non-White= 0, [d]HS degree or more= 1 , Less than HS degree= 0
**p< .05, **p< .01, ***p< .001*

Procedures

Crossroads Study. The Institutional Review Board (IRB) at all three institutions approved the Crossroads study procedures. Signed parental consent and youth assent were obtained for all participants before interviews were conducted. Based on the sensitive nature of the sample, a Privacy Certificate was obtained from the Department of Justice, which protects participants' privacy by exempting both their identity and responses from subpoenas, court orders, and other types of involuntary disclosures. Youth completed a baseline interview after the disposition hearing for their first arrest. Following their baseline interview, youth were re-interviewed ten times: every six months for three years, once a year for two years, and once every two years. The face-to-face interviews with the youth ranged from 2–4 hours and were documented using a secure, computer-assisted program.

Dissertation Study. Trained research assistants contacted eligible participants using information (i.e. phone numbers, email addresses) collected during their most recent Crossroads Study interview. During initial contact, research assistants described the study and determined whether the youth was interested in participating. If the youth agreed to participate, then a session was scheduled for an afternoon that was convenient for the youth. All sessions were conducted in the afternoon or early evening to control for diurnal changes in cortisol as research on the diurnal pattern of cortisol production indicates that cortisol levels are highest in the morning and decline throughout the day (Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). Previous research also indicates that food or drink residue in the mouth may influence the saliva samples by altering salivary pH or composition, or by interacting with the chemicals in the immunoassays (Granger, Johnson, Szanton, Out, & Schumann, 2012). Therefore, the participants were asked to refrain from eating for at least two hours before the scheduled interview time. Since many licit and illicit substances can reduce salivary flow (Rees, 1992), participants were also instructed not to consume any alcohol, drugs, or stimulants the day of the session.

At the beginning of the session, a trained research assistant reviewed the consent form and answered any questions about the study procedures. Participants were then asked to complete self-report measures of various domains (e.g., behavior, mood, health behaviors, personality) and a stressor task. After the stressor task, participants completed a gambling game that measures sensitivity to reward and punishment, and a driving task designed to assess risk taking. Participants were also asked to provide six saliva samples. The self-report measures, gambling game, and driving task were completed on the computer (e.g., Qualtrics for self-report measures, Inquisit for gambling and driving). The total duration of the session ranged between

1.75 and 2.5 hours. Initially, participants were compensated $50 for their time. However, several eligible youth refused to participate in this study due to relatively low amount of compensation they would receive (youth receive more than $100 for Crossroads interviews). Compensation was increased to $75 to help improve the recruitment success rate.

Stressor. To acquire information on cortisol and alpha-amylase reactivity, participants completed the Trier Social Stress Test (TSST). The TSST is an effective method of inducing stress (Dickerson & Kemeny, 2004). Participants were asked to deliver a 5-minute speech about themselves (a mini autobiography) and complete a 5-minute math task (N-back task) in front of two confederate evaluators. Previous research suggests that cortisol increases only occur when the evaluators are of opposite gender (Duchesne, Tessera, Dedovic, Engert, & Pruessner, 2012). Therefore, in each session at least one of the evaluators was female to increase likelihood of observing a cortisol response to the TSST. The confederate evaluators were trained to provide no feedback throughout the task (ex. keeping a neutral facial expression and refraining from gestures such as head nodding).

Salivary Collection. The salivary sampling scheme in the current study was based on recommended sampling design for salivary cortisol and alpha-amylase reactivity. This involved a pre-pre-[task]- post-post-post-post sampling scheme with samples collected on arrival to the lab (after consent), immediately before the task (after a period of relaxation), then again immediately after , 15, 30 and 50 minutes after the start of the stressor (Granger et al., 2012; Stroud et al., 2009; Gordis et al., 2006). This sampling scheme aligns with findings that suggest cortisol is slow-responding and takes approximately 20 minutes to peak after the TSST, and that cortisol samples must be collected at least 40 minutes after the TSST in order to observe recovery

(Dickerson & Kemeny, 2004). Alpha-amylase is more "fast-responding" and peaks around 5 minutes following a social stressor (Granger et al., 2007).

Once participants arrived on campus, they were given a bottle of water and instructed to rinse their mouths—this act served as an additional safeguard against contamination of saliva samples. Participants provided their first samples after their informed consent was obtained. Since rinsing may dilute salivary analytes (Granger et al., 2007), the first saliva sample was collected at least 10 minutes after participants rinsed their mouths to allow salivary analytes to return to undiluted levels. Participants were not allowed to consume any food or drink during the remainder of the study.

The saliva samples were collected using the passive drool technique (Granger et al., 2012). In this technique, participants were asked to use a short straw to gently force saliva into a storage vial. For each saliva sample, participants were instructed to try to fill the vial within a 5-minute period. If they filled the vial before the 5-minute period ended, participants were instructed to stop and move on to the next task. The participants were also instructed to stop if the 5-minute period ended and they had not filled the vial. Trained research assistants tracked the volume of saliva each adolescent provided for each saliva sample as well as the time the participant spent providing the sample (always less than or equal to 5 minutes). From these markers of volume and time, participants' salivary flow rates were calculated (saliva sample volume in millimeters / saliva sample collection duration in minutes). Research indicates alpha-amylase concentrations in saliva are influenced by flow rate (Beltzer et al., 2010), therefore this information was considered during the hormone data analysis. It is important to note that salivary cortisol levels are not affected by flow rate (Vining & McGinley, 1987). A research assistant also noted the exact time of day at which each saliva sample is collected and when the stressor began.

The start time of the stressor is included as a control in each regression model. At the end of the TSST, samples were frozen at-20 degrees Celsius (or lower). This process helped reduce the bacterial growth that can significantly influence levels of salivary analytes (Whembolua, Granger, Singer, Kivlighan, & Marguin, 2006). Samples were kept frozen until they are hand-delivered to the Institute for Interdisciplinary Salivary Bioscience Research at UCI for assaying.

Measures

Data for the current study was drawn from two sources: the Crossroads Study and the on-campus session. A summary of the measures is available in Table 1.2.

Table 1.2.
Summary of measures administered pre and post the Trier Social Stress Test.

Measures	Crossroads Study	On-Campus Session	
		Pre-Stressor	Post-Stressor
Dependent Variable			
Cortisol		X	X
Alpha Amylase		X	X
Independent Variables			
CU Traits	X		
Exposure to Violence	X		
Parental Hostility	X		
Covariates			
Demographics	X	X	
Stressor Start Time		X	

Salivary Cortisol and Alpha-Amylase. Salivary cortisol and alpha-amylase were used as markers for HPA- and SNS-activity in response to the Trier Social Stress Test (TSST). Participants were asked to provide six saliva samples during the session. All samples were assayed in duplicate for alpha-amylase, and in both singlet (10%) and duplicate (90%) for cortisol. Descriptive statistics are provided in Table 1.3.

Callous-unemotional (CU) Traits. At baseline and each follow-up interview of the Crossroads Study, CU traits have been assessed using the 24-item Inventory of Callous-

Unemotional traits (Kimonis et al., 2008). Participants self-rated items (e.g., "I feel bad or guilty when I do something wrong" or "I try not to hurt others' feelings") on a 4-point Likert scale from 0 (not at all true) to 3 (definitely true). The ICU demonstrated adequate internal validity at each interview (α = .77-.81). Scores on all 24 items are summed to create an additive index, with higher scores indicating greater levels of CU traits. The ICU total score has been supported in factor analyses conducted with both detained (Kimonis et al., 2008) and community (Essau, Sasagawa, & Frick, 2006) adolescent samples, and is positively correlated with antisocial behavior and negatively correlated with pro-social behavior in samples of community and detained adolescents (Essau et al., 2006; Kimonis et al., 2008). Descriptive statistics are provided in Table 1.3.

Exposure to Violence. At baseline and each follow-up interview of the Crossroads Study, individuals' exposure to violence and victimization was assessed using the 17-item Exposure to Violence Inventory (Selner-O'Hagan, Kindlon, Buka, Raudenbush, & Earls, 1998). Six dichotomous items assess youths' exposure to violence as a victim (e.g., "Have you ever been beaten up, mugged, or seriously threatened by another person?"). Seven dichotomous items assess youths' history of witnessing violence ("Have you ever seen someone else get beaten up, mugged, or seriously threatened by another person?"). Four items assess exposure to death (e.g. "Has anyone close to you tried to kill him/herself?"). An ETV total score was computed by counting the items the participant had experienced as a victim or a witness during the past seven years, with higher scores indicating a greater exposure to violence. Descriptive statistics are provided in Table 1.3.

Parental Hostility. At baseline and each follow-up interview of the Crossroads Study, the affective tone of the relationship with each parent was assessed using the Quality of Parental

Relationships Inventory (Conger, Ge, Elder, Lorenz, & Simons, 1994). Twelve items assessed parent hostility (e.g. "How often does your father/mother get angry at you?"). The participants responded to each question on a 4- point Likert scale ranging from "Always" to "Never". The scale demonstrated adequate internal validity at each interview (α = .79-.81). A parent hostility total score was computed by averaging the maternal and paternal hostility scores from the past seven years, with higher scores indicating a greater overall parental hostility. Descriptive statistics are provided in Table 1.3.

Demographics. At baseline and each follow-up interview of the Crossroads Study, participants provided information on their race/ethnicity and the highest level of education that either of their parents had received. During the on-campus sessions, participants reported their current age. Parent education was used as a proxy for socioeconomic status (Galobardes, Lynch, & Smith, 2007). Prior research supports the validity of this measure for use with adolescent samples (Lien, Friestad, & Klepp, 2001). Previous research has found an association between cortisol levels increased with age and race, such that older youth and racial minorities exhibit either lower basal cortisol or diminished cortisol reactivity compared to younger individuals and non-Hispanic Whites (Hostinar, McQuillan, Mirous, Grant, & Adam, 2014). Further, adolescents of lower socio-economic status may exhibit higher cortisol reactivity in responses to the TSST (Harkness, Stewart, & Wynne-Edwards, 2011). Consequently, age, race, and parent education were used as covariates in all analyses. Descriptive statistics are provided in Table 1.3.

Table 1.3.
Sample Descriptives

	M / %	*SD*	Min	Max
Cortisol (AUC_I)	1.10	1.03	0.00	3.10
Alpha-Amylase (AUC_I)	4.57	3.34	0.00	8.24
Callous-Unemotional Traits	19.56	7.67	6.00	36.00
Exposure to Violence	3.24	2.67	0.00	10.00
Parental Hostility	1.49	0.26	1.06	2.13
Age	22.84	1.15	2.00	25.00
Start Time	16.67	1.39	14.75	19.03
Parent Education	80.00			
Race				
White	16.36			
Black	1.82			
Hispanic	78.18			
Other	3.64			

Plan of Analysis

Cortisol and Alpha-Amylase Analyses

Saliva samples were assayed for cortisol and alpha-amylase using commercially available immunoassay kits without modification to the manufacturers (Salimetrics) recommended protocols. A competitive immunoassay kit was used to assay for cortisol. Cortisol in standards and samples compete with Cortisol conjugated to horseradish peroxidase for the antibody binding sites on a microtitre plate. After incubation, unbound components are washed away. Bound Cortisol Enzyme Conjugate can be spectrophotometrically measured (optical density) at 405 nm using a standard laboratory plate reader. A kinetic reaction immunoassay kit was used to assay for alpha-amylase. The assay employs a chromagenic substrate, 2-chloro-p-nitrophenol, linked to maltotriose. The enzymatic action of alpha-amylase on this substrate yields 2-chloro-p-nitrophenol, which can be spectrophotometrically measured at 405 nm using a standard laboratory plate reader. The amount of alpha-amylase activity present in the sample is directly proportional to the optical density increase (over a 2 min period) in absorbance at 405 nm.

Two indicators of data quality were calculated for each analyte: inter-assay precision and intra-assay precision. Inter-assay precision is a measure of the reliability of assays across microtiter plates. Intra-assay precision is a measure of the reliability of the assay for individual samples by comparing duplicate samples (each participant saliva sample were assayed more than one time). The inter-assay CVs for cortisol and alpha-amylase were 1.89% and 6.6%, respectively. For cortisol, the intra-assay CV for low controls was 3.43%, and the inter-assay CV for high controls was 0.34%. For alpha-amylase, the intra-assay CV for low controls was 7.2%, and the inter-assay CV for high controls was 5.9%. All CVs met recommended criteria (Chard, 1981).

For cortisol, standard curves were created by plotting the optical density versus the concentrations of a series of wells containing known amounts of cortisol. Comparing the standard curve to participants' saliva samples allows for a measure of how many standard units of cortisol is in participants' saliva samples. To determine if the standard curve is valid, the standard curve R^2 was assessed. The acceptance criteria were met for each plate. Additionally, the range of the standard curves were examined to ensure they included the highest and lowest values of cortisol in the saliva samples. The range of standards was large enough to detect cortisol levels for all assays.

Statistical Analyses

Preliminary Analyses.

Prior to hypothesis testing, the distribution of the raw salivary assay data was examined for non-detects, zero values, insufficient quantity, and values beyond the upper and lower limits of detection (ULOD/LLOD). The data was also examined for outliers, defined as values more than three standard deviations from the mean score (Gordis, et al., 2006), which were dropped

from the data. Cortisol and alpha-amylase reactivity were measured by calculating the area under the curve with respect to increase (AUC_i) for the five samples obtained during the session (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). The formula for AUC_i is:

$$\left(\sum_{i=1}^{n-1}[t_i(m_i + m_{i+1})/2]\right) - (n-1)\cdot m_1$$

where t_i is the precise interval between sample i and sample i+1, m_1 is the first sample, m_i is the level of the cortisol for sample i, and n is the total number of samples. This formula results in one number representing a general index of cortisol reactivity for each subject. The same formula was used to generate one number representing alpha-amylase reactivity. It is possible that some participants showed a stronger decrease than increase over time, resulting in negative values. In cases with negative values, the AUC_i was set to 0, avoiding negative areas and denoting the fact that no increase was seen in the particular subject. The AUCi values were used as the dependent variables in the subsequent statistical analyses.

Because salivary assay data is often positively skewed with a disproportionate number of low-value cases (Granger et al., 2007), tests to determine whether Box-cox, log, square-root, or inverse square-root transformation are most appropriate were conducted. Individual cortisol and alpha-amylase values were transformed using the Box-Cox transformation (Miller & Plessow, 2013). The AUCi values for cortisol and alpha-amylase were log-transformed. Pairwise correlations were calculated to examine the associations between the independent, dependent, and covariates.

Table 1.4.

Cortisol and Alpha-Amylase (Untransformed)

	Cortisol (µg/dL)						Alpha-Amylase (U/mL)					
	N	*M (SD)*		Min, Max		Skew	*n*	*M (SD)*		Min, Max		Skew
Time												
Pre-2 Minutes	54	0.19	0.15	0.06	0.99	**3.36**	53	103.30	70.39	15.74	343.74	**1.44**
Post-0 Minutes	54	0.22	0.16	0.07	1.03	**2.95**	52	130.33	76.22	21.16	348.34	**0.82**
Post-5 Minutes	54	0.26	0.16	0.05	0.88	**1.47**	53	103.34	59.58	9.84	318.49	**1.24**
Post-20 Minutes	54	0.28	0.20	0.05	0.93	**1.64**	54	111.71	63.32	10.00	308.65	**0.88**
Post-40 Minutes	54	0.20	0.14	0.05	0.78	**2.17**	53	117.72	71.00	20.50	321.28	**0.91**
AUC_I	54	3.98	5.14	0.00	21.24	**1.65**	52	967.58	1120.93	0.00	3773.23	**1.02**

Table 1.5.

Cortisol and Alpha-Amylase (Transformed)

	Cortisol					Alpha-Amylase				
	N	*M (SD)*	Min, Max		Skew	*n*	*M (SD)*	Min, Max		Skew
Sample										
Pre-2 Minutes	54	0.64	0.10	0.49	1.00	**1.19**	53	3.07	0.50	1.99	4.31	0.36
Post-0 Minutes	54	0.66	0.10	0.51	1.01	**1.09**	52	3.27	0.51	2.14	4.32	-0.08
Post-5 Minutes	54	0.69	0.10	0.48	0.97	0.22	53	3.09	0.47	1.77	4.22	-0.22
Post-20 Minutes	54	0.70	0.12	0.46	0.98	0.35	54	3.14	0.50	1.78	4.19	-0.45
Post-40 Minutes	54	0.65	0.10	0.47	0.94	**0.70**	53	3.18	0.50	2.13	4.23	-0.05
AUC_I	54	1.10	1.03	0.00	3.10	1.65	52	4.57	3.34	0.00	8.24	-0.51

Note. Bolded values indicate significance of $p < .05$.

Hypothesis Testing.

Research Aim 1.1. The first research aim was to determine whether CU traits and adversity (exposure to violence and hostile parent-child relationships) were independently associated with cortisol and alpha-amylase reactivity. It was hypothesized that CU traits and adversity would each significantly predict cortisol and alpha-amylase stress reactivity. Higher levels of CU traits and greater adversity would be associated with lower levels of cortisol and alpha-amylase reactivity.

Multiple regression models were used to examine the association between CU traits, adversity, stress reactivity. In the first model, cortisol AUC_i was regressed on CU traits, parental hostility, and exposure to violence. In the second model, alpha-amylase AUC_i was regressed on CU traits, parental hostility, and exposure to violence. Consistent with previous research (Glenn, Raine, Schug, Gao, & Granger, 2011; Glenn et al., 2015; Gostisha et al., 2014), the start time of the stressor, age, race/ethnicity (Latino=1, Non-Latino=0), and parent education (HS degree or more=1, Less than HS degree= 0) were included as covariates.

Research Aim 1.2. The second research aim was to determine whether the association between CU traits and stress reactivity was moderated by adversity (exposure to violence and hostile parent-child relationships). It was hypothesized that CU/stress association would be moderated by adversity, such that youth high in both CU traits and adversity would exhibit lower cortisol and alpha-amylase reactivity than individuals high in CU traits exposed to low levels of adversity. Youth high in CU traits and adversity would also exhibit lower levels of reactivity than low CU individuals.

Multiple regression models with interaction terms for CU traits and adversity (CU traits x parental hostility, CU traits x exposure to violence) were used to address this aim. Separate

models were conducted for cortisol and alpha-amylase. Consistent with previous research (Glenn et al., 2011; Glenn et al., 2015; Gostisha et al., 2014), the start time of the stressor, age, race/ethnicity (Latino=1, Non-Latino=0), and parent education (HS degree or more=1, Less than HS degree= 0) were included as covariates.

Research Aim 1.3. The third research aim was to examine whether CU traits were associated with asymmetric stress responses. It was hypothesized that youth high in CU traits would exhibit greater asymmetry in cortisol and alpha-amylase levels than youth with low CU traits.

Three multiple regression models were used to address this aim. First, alpha-amylase was regressed on CU traits and cortisol. An interaction term with CU traits and cortisol was added into the second model to test for moderation. In the third model, three-way interaction terms with CU traits, cortisol, and each adversity variables were added into the model. Consistent with previous research (Glenn et al., 2011; Glenn et al., 2015; Gostisha et al., 2014), the start time of the stressor, age, race/ethnicity (Latino=1, Non-Latino=0), and parent education (HS degree or more=1, Less than HS degree= 0) were included as covariates.

Alternative Analyses. Due to concerns about the small sample size and limited power, fixed effect models were estimated as an alternative method to test the hypotheses. Fixed effects regressions are perhaps more ideal for the current study because they focus exclusively on understanding within-individual variability in stress responses. Each individual is treated as his own "control variable," which means that all time-invariant factors about the individual and his environment (e.g., race/ethnicity, SES, health behaviors) are automatically controlled (Allison, 2009). Although coefficients cannot be estimated for time-invariant predictors, interactions between time-varying predictors (i.e. time before and after TSST) and time-invariant predictors

(CU traits, parental hostility, exposure to violence) can be estimated. This allows me to determine whether hormone levels before and after the stressor are different for those with varying levels of CU traits, parental hostility, and exposure to violence (time-invariant predictors). The first set of fixed effect models examined how cortisol and alpha-amylase varied by the timing of each sample. Interactions between time and CU traits were added in the second set of models. The models were repeated with time by parental hostility and exposure to violence interactions. The third set of models tested three-way interactions between time, CU traits, and parental hostility. The interaction between time, CU traits, and exposure to violence was also tested.

Fixed effect models were also estimated to test whether individuals with increased CU traits exhibited asymmetric stress response. The first model examined the association between cortisol and alpha-amylase. An interaction term with cortisol and CU traits was added to the model to determine whether the association between cortisol and alpha-amylase was different among individuals with high, low, and average levels of CU traits.

Results

Preliminary Results

Inter-assay and intra-assay CVs were calculated as indicators of data quality. The inter-assay CVs for cortisol and alpha-amylase were 1.89% and 6.6%, respectively. For cortisol, the intra-assay CV for low controls was 3.43%, and the inter-assay CV for high controls was 0.34%. For alpha-amylase, the intra-assay CV for low controls was 7.2%, and the inter-assay CV for high controls was 5.9%. All CVs met recommended criteria (Chard, 1981). The raw salivary assay data was examined for non-detects, zero values, insufficient quantity, and values beyond the ULOD and LLOD. For cortisol, two samples were concluded to have insufficient quantity for

testing (coded as missing values), four samples were at the ULOD and were retested at a 1:10 dilution, and zero samples were at the LLOD. There were no non-detects or zero values for cortisol. For alpha-amylase, zero samples were concluded to have insufficient quantity for testing, zero samples were at the LLOD, and there were no non-detects or zero values. Thirteen samples were at the ULOD—seven were retested at a 1:10 dilution, six retested at a 1:800 dilution. Outliers more than 3 SD from the mean were also excluded from analyses. Six cortisol and eleven alpha-amylase values were dropped. Descriptive statistics for untransformed cortisol and alpha-amylase are presented in Table 1.4.

Pairwise correlations were calculated to determine the association between the main study variables (Table 1.6). Results indicate that alpha-amylase and cortisol were significantly correlated (r= 0.31), such that increases in alpha-amylase were associated with increases in cortisol.

Table 1.6.
Pairwise correlations between study variables

		1	2	3	4	5	6	7	8	9
1	Cortisol (AUCi)	-								
2	Alpha-Amylase (AUCi)	**0.31**	-							
3	Callous-Unemotional Traits	-0.26	-0.12	-						
4	Exposure to Violence	-0.11	-0.19	0.21	-					
5	Parental Hostility	-0.19	-0.04	0.15	0.08	-				
6	Age	-0.07	-0.10	-0.09	0.15	-0.01	-			
7	Parent Education	0.06	0.11	-0.09	-0.09	-0.14	0.13	-		
8	Race	-0.05	0.09	0.05	0.11	0.20	-0.19	**-0.26**	-	
9	Start Time	-0.07	0.16	0.01	-0.06	-0.20	**-0.29**	0.05	-0.09	-

[A] HS degree or more= 1 , Less than HS degree= 0, [B] Latino= 1, Non-Latino= 0
Note. Bolded values indicate significance of p< .05.

Research Aim 1.1.

Multiple regression models were used to examine the association between stress reactivity, CU traits, parental hostility, and exposure to violence (Table 1.7). For cortisol reactivity, the results

indicate there were no significant associations between cortisol reactivity and CU traits (X), parental hostility, or exposure to violence. Similar results were observed for alpha-amylase—alpha-amylase reactivity was not associated with CU traits or either indicator of adversity.

Research Aim 1.2.

Interactions terms for CU traits and adversity (CU traits x parental hostility, CU traits x exposure to violence) added to the multiple regression models to determine whether the association between CU traits and stress reactivity was moderated by adversity. The results indicate the association between CU traits and cortisol reactivity was not significantly moderated by either parental hostility or exposure to violence (Table 1.8). The pattern of results was the same for alpha-amylase, such that neither type of adversity moderated the association between CU traits and alpha-amylase reactivity.

Research Aim 1.3.

Multiple regression models were used to determine whether individuals with higher levels of CU traits exhibited asymmetric stress responses (Table 1.9). Results from the first model indicated there was a significant positive association between cortisol and alpha-amylase. An interaction term with cortisol and CU traits was added to the model. The interaction term was not significantly associated with alpha-amylase, indicating CU traits did not moderate the association between cortisol and alpha-amylase. Finally, the three-way interaction terms with cortisol reactivity, CU traits, and adversity were added to the model (Table 1.10). Neither interaction term was significantly associated with alpha-amylase.

Table 1.7.

Multiple regression model with callous-unemotional traits, exposure to violence, and parental hostility predicting stress reactivity

	Cortisol (AUCi)					Alpha Amylase (AUCi)				
	B	SE	*p*	95% CI		*B*	SE	*p*	95% CI	
CU Traits	-0.03	0.02	0.11	-0.07	0.01	-0.03	0.07	0.61	-0.17	0.10
Exposure to Violence	-0.01	0.06	0.86	-0.12	0.10	-0.21	0.19	0.27	-0.60	0.17
Parental Hostility	-0.71	0.59	0.23	-1.89	0.46	0.07	1.99	0.97	-3.94	4.09
Age	-0.13	0.14	0.35	-0.40	0.14	-0.08	0.46	0.87	-1.00	0.85
Parent Education[a]	0.07	0.37	0.85	-0.67	0.82	1.07	1.25	0.40	-1.44	3.59
Race[b]	-0.08	0.37	0.83	-0.82	0.66	1.20	1.28	0.36	-1.39	3.79
Start Time	0.00	0.00	0.32	0.00	0.00	0.00	0.00	0.35	0.00	0.00
R^2			*F*(7, 46)= 0.91					*F*(7, 44)= 0.66		
F			R-squared= 0.12					R-squared= 0.10		

Table 1.8.

Multiple regression models with interactions between callous-unemotional traits, parental hostility, and exposure to violence predicting stress reactivity

	Cortisol (AUCi)					Alpha Amylase (AUCi)				
	B	SE	*p*	95% CI		*B*	SE	*p*	95% CI	
CU Traits	-0.03	0.02	0.14	-0.07	0.01	-0.04	0.07	0.59	-0.18	0.10
Exposure to Violence	-0.01	0.06	0.82	-0.13	0.10	-0.20	0.20	0.32	-0.60	0.20
Parental Hostility	-0.74	0.63	0.25	-2.01	0.53	0.13	2.17	0.95	-4.25	4.51
Age	-0.13	0.15	0.40	-0.43	0.18	-0.09	0.51	0.87	-1.12	0.94
Parent Education[a]	0.06	0.41	0.88	-0.77	0.90	1.07	1.42	0.45	-1.79	3.93
Race[b]	-0.05	0.39	0.90	-0.83	0.74	1.13	1.36	0.41	-1.61	3.87
Start Time	0.00	0.00	0.41	0.00	0.00	0.00	0.00	0.44	0.00	0.00
CU Traits x Exposure to Violence	0.00	0.01	0.80	-0.02	0.02	0.00	0.03	0.97	-0.06	0.06
CU Traits x Parental Hostility	0.00	0.10	0.96	-0.21	0.20	-0.03	0.35	0.93	-0.73	0.67
			F(9, 43)= 0.68					*F*(9, 41)= 0.43		
			R-squared= 0.13					R-squared= 0.09		

Table 1.9.

Multiple regression models with interaction between cortisol reactivity and callous-unemotional traits predicting alpha-amylase reactivity

	Model 1					Model 2				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
Cortisol (AUCi)	**1.04**	**0.21**	**0.00**	**0.63**	**1.45**	**1.00**	**0.21**	**0.00**	**0.59**	**1.41**
Callous-Unemotional Traits	-0.01	0.03	0.62	-0.07	0.04	0.04	0.04	0.34	-0.04	0.11
Age	-0.02	0.19	0.91	-0.39	0.34	0.02	0.19	0.91	-0.35	0.39
Parent Education[a]	0.97	0.51	0.06	-0.03	1.97	**1.02**	**0.51**	**0.04**	**0.03**	**2.02**
Race[b]	**1.28**	**0.52**	**0.01**	**0.26**	**2.31**	**1.13**	**0.52**	**0.03**	**0.09**	**2.16**
Start Time	**0.00**	**0.00**	**0.01**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
Cortisol x CU Traits						-0.05	0.03	0.07	-0.10	0.00

[A] HS degree or more=1 , Less than HS degree= 0, [B] Latino=1, Non-Latino=0

Table 1.10.

Multiple regression models with interactions between cortisol reactivity, callous-unemotional traits, parental hostility, and exposure to violence predicting alpha-amylase reactivity

	Model 3				
	B	*SE*	*p*	95% CI	
Cortisol	**1.13**	**0.55**	**0.05**	**0.00**	**2.25**
Callous-Unemotional Traits	-0.03	0.09	0.72	-0.21	0.15
Age	0.23	0.52	0.66	-0.83	1.29
Parent Education[a]	0.37	1.54	0.81	-2.77	3.50
Race[b]	0.50	1.42	0.73	-2.38	3.38
Start Time	0.00	0.00	0.14	0.00	0.00
Parental Hostility	1.36	2.43	0.58	-3.58	6.30
Exposure to Violence	-0.15	0.22	0.50	-0.61	0.30
Cortisol x Callous-Unemotional Traits	-0.06	0.09	0.47	-0.24	0.11
Cortisol x Parental Hostility	0.85	2.31	0.72	-3.84	5.53
Cortisol x Exposure to Violence	-0.33	0.21	0.14	-0.76	0.11
CU Traits x Parental Hostility	-0.03	0.42	0.95	-0.88	0.83
CU Traits x Exposure to Violence	0.00	0.04	0.92	-0.08	0.07
Cortisol x CU Traits x Parental Hostility	-0.26	0.44	0.55	-1.16	0.63
Cortisol x CU Traits x Exposure to Violence	0.01	0.04	0.80	-0.07	0.09

[A] HS degree or more=1 , Less than HS degree= 0, [B] Latino=1, Non-Latino=0

Alternative Analyses

To address concerns regarding the small sample size and limited power, fixed effect models were used to examine within-person variability in cortisol and alpha-amylase levels over time. The results from the first models indicate that participants exhibited increased cortisol and alpha-amylase levels following the stressor (Table 1.11). Specifically, cortisol levels 5-minutes and 20-minutes after the TSST were significantly higher than pre-TSST levels (Figure 1.1). Alpha-amylase levels immediately after the TSST (0-minutes) were also significantly higher than pre-TSST levels (Figure 1.2).

Table 1.11.
Fixed Effects Regressions for Time predicting Cortisol and Alpha-Amylase Reactivity

	Cortisol					Alpha Amylase				
	B	SE	*p*	95% CI		*B*	SE	*p*	95% CI	
Time										
Post-0 Minutes	**0.03**	**0.01**	**0.01**	**0.01**	**0.05**	**0.22**	**0.04**	**0.00**	**0.14**	**0.30**
Post-5 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**	0.02	0.04	0.61	-0.06	0.10
Post-20 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**	0.06	0.04	0.14	-0.02	0.14
Post-40 Minutes	0.01	0.01	0.32	-0.01	0.03	**0.12**	**0.04**	**0.01**	**0.04**	**0.20**

Figure 1.1.
Cortisol Response to the Trier Social Stress Test

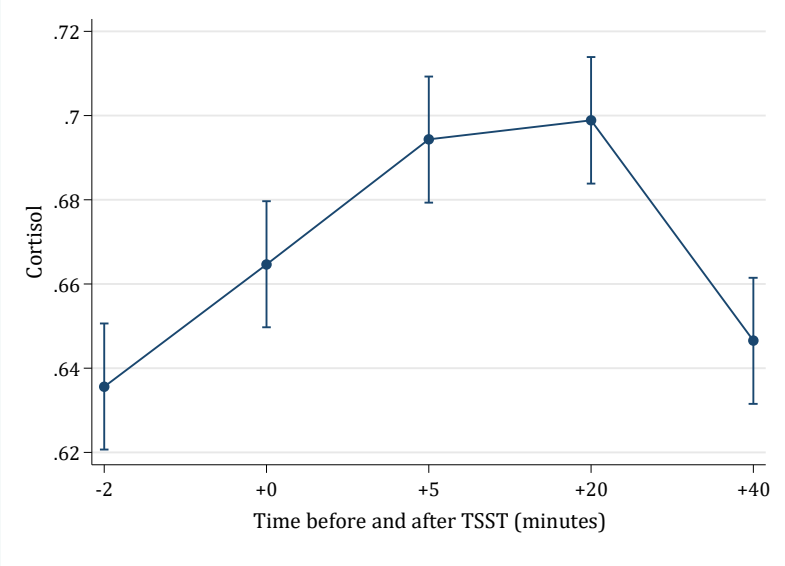
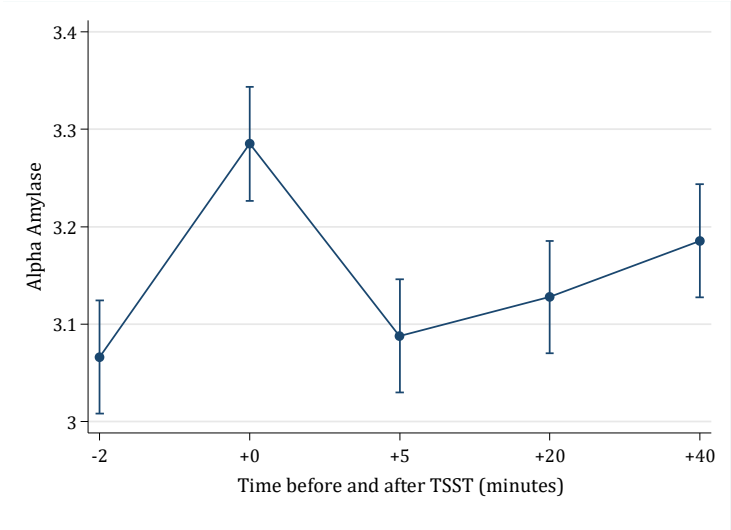


Figure 1.2.
Alpha-Amylase Response to the Trier Social Stress Test



Two-way interaction terms for time with CU traits, parental hostility, and exposure to violence were added to the fixed effect models. The results indicated the interaction between time and CU traits was significant (Table 1.12). Participants with higher levels of CU traits had significantly lower levels of cortisol 20- and 40-minutes after the TSST (Table 1.13, Figure 1.3). The three-way interaction terms did not significantly predict cortisol or alpha-amylase (Table 1.12), indicating that the effect of CU traits was not moderated by either parental hostility or exposure to violence.

Table 1.12.
Wald Test for the Interactions in Fixed Effects Regressions predicting Cortisol and Alpha-Amylase Reactivity

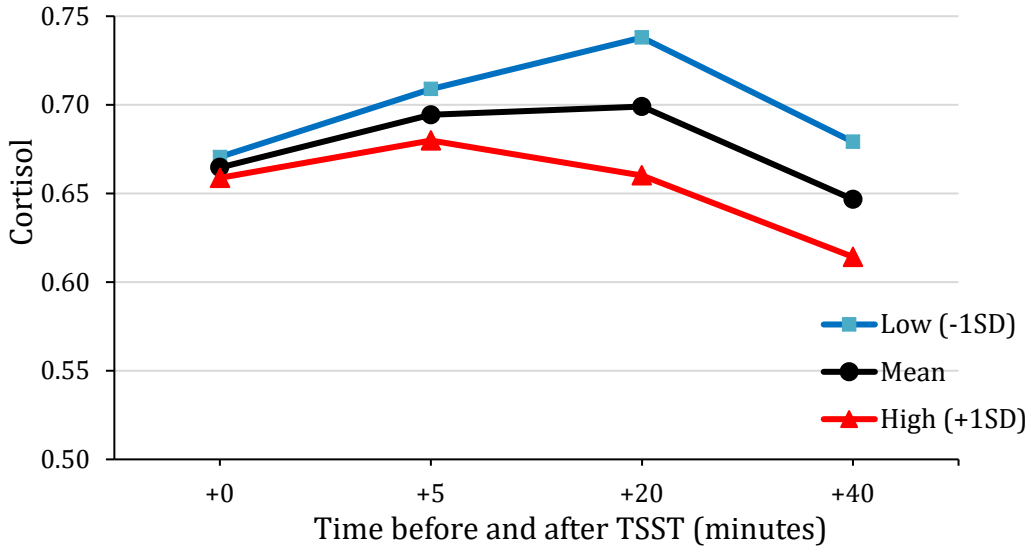
	Cortisol	Alpha-Amylase
	F	*F*
Time x CU Traits	**5.28*** **	0.81
Time x Exposure to Violence	0.91	0.90
Time x Parent Hostility	1.53	2.18
Time x CU Traits x Exposure to Violence	0.46	1.28
Time x CU Traits x Parental Hostility	0.69	0.30

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

Table 1.13.
Interactions between Time and Callous-Unemotional Traits in Fixed Effects Regressions predicting Cortisol Reactivity

	B	SE	*p*	95% CI	
Time					
Post-0 Minutes	**0.03**	**0.01**	**0.01**	**0.01**	**0.05**
Post-5 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**
Post-20 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**
Post-40 Minutes	0.01	0.01	0.29	-0.01	0.03
Time x CU Traits					
Post-0 Minutes	0.00	0.00	0.57	0.00	0.00
Post-5 Minutes	0.00	0.00	0.16	0.00	0.00
Post-20 Minutes	**-0.01**	**0.00**	**0.00**	**-0.01**	**0.00**
Post-40 Minutes	**0.00**	**0.00**	**0.00**	**-0.01**	**0.00**

Figure 1.3.
Cortisol Response by Saliva Sample and Callous-Unemotional Traits



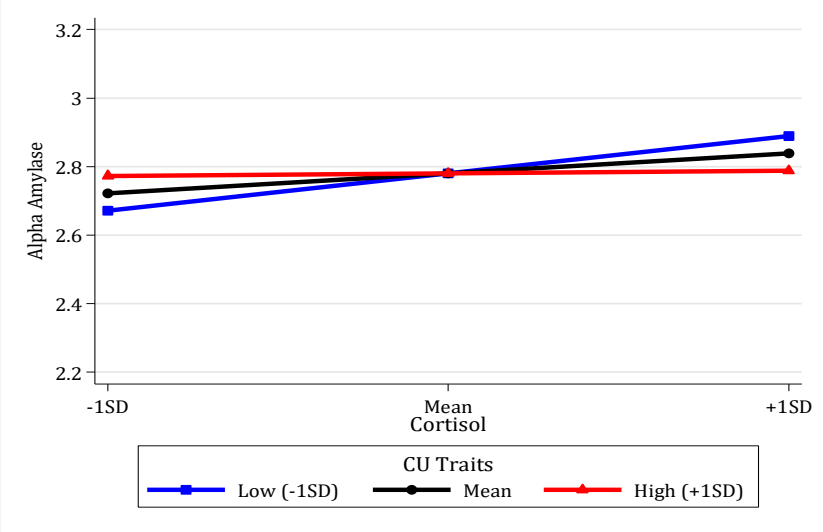
Separate fixed effect models were estimated to test whether individuals with increased CU traits exhibited symmetric or asymmetric stress responses (Table 1.14). The first model examined the association between cortisol and alpha-amylase. Cortisol was significantly associated with alpha-amylase, such that increases in cortisol were associated with increases in alpha-amylase. An interaction term with cortisol and CU traits was added to the model, and the

results indicated that the interaction was significantly associated with alpha-amylase (Figure 1.4). Post-hoc analysis revealed that cortisol and alpha-amylase were positively associated among individuals with low (-1SD) (*dydx*= 1.00, SE= 0.32, *p*= 0.002) and average levels of CU traits (*dydx*= 0.54, SE= 0.25, p= 0.03). Interestingly, there was no significant association between cortisol and alpha-amylase among individuals with high CU traits (*dydx*= 0.08, SE= 0.37, *p*= 0.84). These result suggest that individuals with higher CU traits exhibited asymmetric stress reactivity.

Table 1.14.
Fixed Effects Regressions with Cortisol Reactivity and CU Traits predicting Alpha-Amylase Reactivity

	Model 1					Model 2				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
Time										
Post-0 Minutes	**0.20**	**0.04**	**0.00**	**0.11**	**0.28**	**0.20**	**0.04**	**0.00**	**0.11**	**0.28**
Post-5 Minutes	-0.02	0.05	0.63	-0.11	0.07	-0.04	0.05	0.44	-0.13	0.05
Post-20 Minutes	0.02	0.05	0.61	-0.07	0.11	0.01	0.05	0.84	-0.08	0.10
Post-40 Minutes	**0.11**	**0.04**	**0.01**	**0.03**	**0.20**	**0.10**	**0.04**	**0.02**	**0.01**	**0.18**
Cortisol	**0.61**	**0.25**	**0.02**	**0.11**	**1.11**	**0.54**	**0.25**	**0.04**	**0.04**	**1.04**
Cortisol x CU Traits						**-0.06**	**0.03**	**0.05**	**-0.12**	**0.00**

Figure 1.4.
Association between Alpha-Amylase and Cortisol by Callous-Unemotional Traits



Discussion

Individuals with elevated levels of CU traits engage in more serious and persistent levels delinquent behavior (Frick et al., 2014). This behavioral pattern is attributable to the various socioemotional deficits (e.g., fearlessness, insensitivity to punishment) that increase the likelihood of engaging in antisocial behavior when the opportunity arises. Identifying and understanding the mechanisms underlying these deficits may help us more effectively treat CU traits and the accompanying antisocial behaviors. This study was designed to clarify the relation between HPA and SNS activity and CU traits by parsing out confounding effects of prior adversity. Surprisingly, the initial results indicated that CU traits did not predict either cortisol or alpha-amylase reactivity. There was also no relation between parental hostility, exposure to violence, and either indicator of stress reactivity.

The lack of an association between any of the key variables conflicted with the majority psychobiological research on CU and psychopathic traits (Glenn & Raine, 2014; Moul et al., 2018) and prior work on the consequences of adversity (Koss & Gunnar, 2017). The unexpected findings led me to question whether the limited sample size or omitted confounding variables may have influenced the results. Indeed, a post-hoc power analysis conducted in G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) indicated there was not enough statistical power to detect a small effect for cortisol and alpha-amylase (power= 0.17) or a medium effect for alpha-amylase (power= 0.78) (Cohen, 1965). Adding important covariates, like nicotine use (de Vries-Bouw et al., 2012) or anxiety (Spear, 2009), would only further reduce statistical power. To address these concerns, alternative models were used to test the hypotheses. Specifically, fixed effect models were estimated to examine the within-person changes in cortisol and alpha-amylase levels, and how these changes differed by the individual's CU traits and adversity. Importantly, the fixed

effect models account for all measured and unmeasured time-invariant factors that may lead to differences between individuals (i.e., prior nicotine use, psychopathology, genetics).

In contrast to the original models, the fixed effect models revealed that individuals with elevated CU traits did exhibit lower cortisol reactivity than their low CU counterparts. During the time points where cortisol was expected to peak (20 minutes) and decline (40 minutes), participants with high levels of CU traits exhibited lower cortisol than participants with average or low levels of CU traits. Surprisingly, alpha-amylase levels were similar among individuals with high, average, and low levels of CU traits. In sum, the results of fixed effect models indicate that individuals with elevated CU traits exhibited a typical alpha-amylase response and a blunted cortisol response. These findings are particularly interesting considering the prevailing psychobiological theories on psychopathy and CU traits propose their socioemotional deficits are a result of low arousal in both the HPA axis and SNS (Blair, 2005; van Goozen et al., 2007; Herpers et al., 2014). Much of the empirical support for those theories is drawn from studies on antisocial outcomes (i.e., psychopathy, CU traits, delinquency) that primarily examine HPA and SNS functioning separately. As suggested in Bauer et al. (2002), dysfunctional HPA and SNS activity, which may increase an individual's susceptibility to their environment and the likelihood of maladaptive behavior, can be characterized by either concurrently low reactivity or asymmetrical reactivity. The observed pattern may depend on the characteristics of the sample (e.g., unassessed differences in psychopathology, age) or the stressor (e.g., physical versus social threat, controllability) (Bauer et al., 2002; Dickerson & Kemeny, 2004). Given the mixed results regarding the stress profiles of individuals with CU traits, it is clear that researchers must continue to examine functioning in both systems under different conditions

The asymmetrical response observed in this study may provide insight into why individuals with CU traits are more likely to engage in aggressive behaviors. Youth with high levels of CU traits in this study exhibited the same increase in alpha-amylase levels as their counterparts with low or average levels of CU traits, yet their cortisol levels were blunted in comparison. Researchers have posited that this response pattern is exhibited when stressors that are perceived as requiring increased effort, active coping, and "defense" reactions (Henry, 1992). Consequently, individuals high in CU traits may approach and engage threats instead of removing themselves from the situation. Paired with a lack of empathy or sensitivity to punishment, this response may increase one's susceptibility to aggression- or anger-inducing stimuli. Future research should determine whether an asymmetric stress response is a mechanism through which CU traits are related to increased aggressive behavior.

There are several important limitations that must be noted. The foremost limitation is the small sample size which likely contributed to the null findings from the original multiple regression models. With this small sample, there was inadequate statistical power to detect a small and medium effects (Cohen, 1965). Further, additional relevant moderators (e.g., antisocial behavior) and covariates (nicotine use, anxiety) were not included since it would further decrease power. Although the alternative fixed effect models addressed these concerns, additional research with larger sample sizes is needed. An additional concern is the considerable number of individuals who exhibited a decreased in cortisol (n= 19) and alpha-amylase (n= 17) following the stressor. Since the primary focus of this study was to examine how stress levels increased following a stressor, negative AUCi values were coded as zero for the analyses (Pruessner et al., 2003). Decreased HPA activity (i.e., a drop in cortisol levels instead of a blunted response) could be a correlate of CU traits. In a study of incarcerated young adults whose cortisol levels either

increased or decreased following a stressor (Johnson, et al., 2015), the researchers found cortisol levels among individuals with higher levels of affective psychopathic traits decreased at a faster rate than others. Future research should further investigate the association between CU traits and negative HPA responses. Another limitation is that the results of this study may not be generalizable to females, in general, and females involved in the justice system, in particular. There is research that suggests females with elevated levels of CU traits may exhibit different stress responses than males (O'Leary et al., 2007; O'Leary et al., 2010). As such, it is important that future work on the relation between CU traits and stress reactivity includes females.

Finally, although the results of the fixed effect models indicated that the association between CU traits and cortisol was not attenuated by adversity, this does not entirely clarify whether the CU traits themselves are the reason why individuals with high CU traits exhibit blunted stress responses. Researchers attribute the reduced stress reactivity of youth with elevated levels of CU traits to the same adverse experiences (e.g., maltreatment, neglectful and harsh parents) that led to the development of CU traits (Glenn & Raine, 2014). Even though this study benefitted from the extensive longitudinal data on the participants' exposure to violence and parental hostility during adolescence, it was also limited by the lack of information about their childhood experiences (e.g., age during the event, type and frequency of trauma). This limitation may not be a fatal flaw. A recent study suggests the HPA axis recalibrate during adolescence (Gunnar, DePasquale, Reid, Donzella, & Miller, 2019). If this is also the case for the SNS, then the finding that CU traits predict stress reactivity above and beyond adolescent adversity is valid. Regardless, longitudinal data from early childhood into young adulthood is needed to better understand the causal relationship between adversity, CU traits, and blunted cortisol reactivity.

The current study has several strengths. As previously discussed, the central strength of this study is the assessment of both HPA and SNS activity in relation to CU traits. Despite evidence that these systems work together to produce a stress response and modify brain functioning (Bauer et al., 2002; Chrousos, 2009), the coordination between these systems has been largely overlooked in previous work on CU traits. This study addresses the gap in the literature and provides important insights on the stress response systems of individuals with high CU traits. A second strength is the sample of justice-involved youth. Given overarching goal of better understanding CU traits in order to prevent and reduce antisocial behavior, it is important to conduct this research with relevant samples who exhibit variability in the key variables. This study also greatly benefitted from the use of longitudinal data on parental hostility and exposure to violence. Prior work primarily utilizes retrospective reports of adversity (Cima et al., 2008; Gostisha et al., 2014), which is subject to memory biases. Access to extensive data on parental hostility and exposure to violence allowed me to assess the effects of chronic and acute stressors. Acute and chronic stressors exhibit different effects on stress response systems (Dickerson & Kemeny, 2004), therefore it was important to consider both types of adversity in the analyses. Finally, the fixed effect models allowed me to focus on within-person changes in cortisol and alpha-amylase and isolate the effect of CU traits. Although they may include important covariates, studies that utilize traditional between-individual statistical models cannot rule out the possibility that any associations could be attributable to selection effects or unmeasured confounding factors. The fixed effect models in this study allow me to make stronger claims regarding the association between CU traits and stress reactivity.

Countless resources are dedicated to the rehabilitation of delinquent youth and young adults. Despite the effort of parents, teachers, and justice officials, interventions for delinquent

youth and youth high in CU traits are typically ineffective because they fail to address the specific needs of their participants (Hawes, Price, & Dadds, 2014; Lipsey & Cullen, 2007). To better promote positive development and outcomes, researchers and practitioners should take into account the biological factors that contribute to delinquent behavior. Developmental researchers widely agree that behavior is the product of both nature and nurture, and there is evidence that up to 68% of the variation in CU traits and 65% of the variation in antisocial behavior has a genetic basis (Burt, 2009; Frick et al., 2014). Hormones, like those released by the HPA axis and SNS, act as a mechanism through which genetic *and* environmental factors affect brain functioning. Utilizing psychosocial interventions known to produce changes in SNS or HPA activity could improve brain functioning and subsequently reduce CU traits and antisocial behaviors. Caldwell, Skeem, Salekin, and Van Rybroek (2016) found promising results in test of an intensive treatment that utilized reward-oriented approaches, targeted the self-interests of the adolescent, and taught empathy skills. Their results showed that adolescent offenders with high levels of psychopathic traits who were enrolled in the treatment were less likely to recidivate than their counterparts who were enrolled in a standard treatment program. Johnson, Vitacco, and Shirtcliff (2018) examined whether the same treatment could alter hormone levels among incarcerated adolescents. They found that youth with high levels of CU and life adversity were the most responsive to treatment— after participating in the treatment for four months, their cortisol levels increased to more normative levels. Given these encouraging findings, research should continue to explore the relation between stress reactivity, CU traits, and delinquency, as well as the value of using stress reactivity to identify individuals less amenable to treatment and pair them with appropriate interventions. By understanding both CU traits and their biological correlates, interventions may be more effective in reducing the risk of antisocial behavior.

STUDY TWO

Research Rationale

Callous-unemotional (CU) traits (e.g., lack of empathy, deficient guilt/remorse, and shallow affect) are a robust and well-established risk factor for delinquent behavior (Frick et al., 2014). Compared to other antisocial youth, youth with elevated CU traits are more likely to engage in violent, aggressive, and severe antisocial behavior, which in turn increases their likelihood of justice system involvement. Studies demonstrate that callous-unemotional traits account for a significant portion of delinquency even after accounting for other known risk factors and protective factors, such as IQ, peer deviancy, and impulse control (Hampton, Drabick, & Steinberg, 2014; Kahn, Byrd, & Pardini, 2013; Munoz, Frick, Kimonis, & Aucoin, 2008). Alarmingly, youth high in CU traits exhibit a more stable pattern of delinquency such that their criminal behavior is more likely to persist into adulthood compared to other antisocial youth (McMahon, Witkiewitz, & Kotler, 2010).

This susceptibility to delinquent behavior has been attributed to a motivational imbalance of increased reward dependency and decreased sensitivity to punishment (Herpers, Scheepers, Bons, Buitelaar, & Rommelse, 2014; van Honk & Schutter, 2006). Specifically, individuals with elevated levels of CU traits are less responsive to punishment than rewards (Mitchell, Colledge, Leonard, & Blair, 2002) and are less likely to deprioritize rewards despite the possible negative consequences (Baskin-Sommers, Wallace, MacCoon, Curtin, & Newman, 2010; Finger et al., 2011). Some researchers believe this imbalance may be the result of the joint effects of the hypothalamus -pituitary-adrenal (HPA) and hypothalamus-pituitary-gonadal (HPG) axes on the amygdala (Montoya, Terburg, Bos, & van Honk, 2012). Cortisol acts on the amygdala and increases fear reactivity, sensitivity to punishment, and withdrawal-related behavior (Gunnar &

Quevedo, 2007). In contrast, testosterone increases reward sensitivity, aggression, and approach-related behaviors (Terburg et al., 2009; van Honk & Schutter, 2006). Typically, the two hormones counteract each other to produce an appropriate response to stressful or rewarding stimuli— cortisol suppresses the activity in the HPG axis and inhibits approach-related behavior, while testosterone suppresses activity of the HPA axis and inhibits withdrawal-related behavior. Disproportionate levels of these hormones, specifically higher levels of testosterone than cortisol, may result in an increased reward seeking and lower punishment sensitivity (van Honk, Schutter, Hermans, & Putman, 2003).

There are several studies linking the imbalance of cortisol and testosterone to risk taking and antisocial behavior (Dekkers et al., 2019; Knight, Sarkar, Prasad, & Mehta, 2020). For example, in a community sample of adults, Mehta and colleagues (2017) examined whether cortisol moderated the association between testosterone and risk taking. Results indicated that individuals with high basal testosterone and low basal cortisol exhibited heightened risk taking in both self-reports and laboratory tasks, while there was no association between testosterone and risk taking among individuals with high cortisol. Higher levels of testosterone relative to cortisol have also been observed in perpetrators of intimate partner violence (Romero-Martinez, Gonzalez-Bono, Lila, & Moya-Albiol, 2013) and linked to physical aggression in justice-involved samples. In a sample of male youth participating in a delinquency diversion program, researchers tested whether cortisol moderated the association between testosterone and self-reported aggression (Popma et al., 2007). Individuals with high testosterone and low cortisol levels reported the highest levels of overt aggression (e.g., hitting, making threats). Together, these findings highlight the importance of understanding how these hormones operate together among youth high in CU traits and contribute to delinquent behavior.

The results of empirical tests on the association between CU traits, cortisol, and testosterone are not entirely consistent. Low cortisol levels are associated with elevated psychopathic traits among samples of community and incarcerated adults (for review, see Glenn & Raine, 2014). For example, O'Leary, Loney, and Eckel (2007) examined resting cortisol levels and cortisol reactivity among a sample of college undergraduate students who varied in psychopathic traits. Results showed that there were no differences in resting cortisol between adults who scored high and low in psychopathy. However, adults who scored high in psychopathy did exhibit a blunted cortisol response to a social stressor. In contrast, Glenn and colleagues (2011) found no significant direct association between psychopathy and basal cortisol or cortisol reactivity in an adult sample. The findings regarding the testosterone are even more inconsistent (Dekkers et al., 2019; Glenn & Raine, 2014; Welker, Lozoya, Campbell, Neumann, & Carre, 2014). In one of the few studies on the association between CU traits and testosterone in adolescents, the researchers found no difference in resting testosterone levels between youth high and low in CU traits (Loney et al., 2006).

The lack of consistent findings on the relation between cortisol, testosterone, and CU traits may be a result of researchers' focus on the individual levels of each hormone. In an extension of prior work, Terburg and colleagues put forth the Dual-Hormone Serotonergic Hypothesis (2009) which posits that high levels of testosterone combined with low levels of cortisol would promote sensitivity to reward over fearfulness and approach-related behaviors over withdrawal-related behaviors. The results of a study on the association between psychopathy, cortisol, and testosterone levels in adults supports this idea (Glenn et al., 2011). Results indicated that there were no associations between psychopathy, baseline testosterone, baseline cortisol, or cortisol reactivity. However, an association was found when the hormones

were considered together— individuals with elevated psychopathy scores had an increased ratio of testosterone to cortisol reactivity. It is important that a different pattern of results was observed in a later study (Welker et al., 2014)— psychopathy was highest among individuals with high testosterone and high cortisol levels. The findings from both studies have not been replicated, however there is some evidence of cortisol-testosterone divergence among male adolescents high in CU traits. Johnson and colleagues (2014) examined daily fluctuations in cortisol, DHEA, and testosterone in a sample of incarcerated male youth. Results indicated that individuals high in CU traits had low cortisol levels during moments when their testosterone levels were high. Similar results were found in a recent study on the relation between DHEA, cortisol, maltreatment, and psychopathology in male juvenile offenders (Kimonis, Fleming, Wilbur, Groer, & Granger, 2019). Accounting for maltreatment, higher levels of CU traits were associated higher concentrations of DHEA and lower cortisol/DHEA ratios. Given the mixed findings and different assessments of cortisol and testosterone (i.e., basal vs. diurnal vs. reactivity), additional research is needed to clarify the relation between CU traits, cortisol, and testosterone.

Aims and Hypotheses

The overarching goal of the second study was to determine whether CU traits were directly or indirectly associated with risk taking and sensitivity to reward/punishment through cortisol and testosterone. The first research aim was to determine whether youth high and low in CU traits exhibit differences in cortisol reactivity, testosterone, or the ratio of testosterone to cortisol reactivity. I hypothesized that elevated levels of CU traits would be associated with lower cortisol reactivity, higher testosterone reactivity, and a higher testosterone/cortisol reactivity ratio. The second research aim was to determine whether CU traits predicted risk

taking, sensitivity to reward, and sensitivity to punishment. It was hypothesized that individuals with elevated levels of CU traits would display riskier behavior, greater sensitivity to rewards, and lower sensitivity to punishment. The third and final aim was to test whether testosterone and cortisol mediate the association between CU traits and risk taking and sensitivity to reward/punishment. It was hypothesized that only the testosterone/cortisol ratio would partially mediate the association between CU traits, risk taking, and sensitivity to reward and punishment. Specifically, youth with higher levels of CU traits would exhibit a larger testosterone/cortisol ratio, which in turn predicts greater sensitivity to reward, lower sensitivity to punishment, and higher levels of risk taking.

Method

Participants and Procedures

The details regarding sampling and procedures were previously described in Study 1. Youth in the Crossroads study who met the eligibility criteria were contacted and asked to participate in an on-campus session. Of the 465 eligible youth, 55 individuals were successfully recruited into the study. During the on-campus session, participants completed self-report measures of various domains (e.g., behavior, mood, health behaviors, personality), a stressor task, a gambling game that measures sensitivity to reward and punishment, and a driving task designed to assess risk taking. Participants were also asked to provide six saliva samples to be analyzed for cortisol and testosterone. Please refer to the Procedure section in Study One (pg. 12) for additional details on the sample and procedures.

Measures

Data for Study 2 was drawn from two sources: the Crossroads Study and the on-campus session. Callous-unemotional traits, demographics, and cortisol reactivity were assessed using

the same measures from Study 1 (please refer to pages 19-23 for measure details and descriptive information). A summary of Study Two measures is provided in Table 2.1.

Table 2.1.
Summary of measures administered pre- and post-TSST

Measures	Crossroads Study	On-Campus Session	
		Pre-Stressor	Post-Stressor
Dependent Variables			
Risk-Taking— Stoplight Runs			X
Risk-Taking— Stoplight Crashes			X
Sensitivity to Reward—Iowa Gambling Task Advantageous Plays			X
Sensitivity to Punishment—Iowa Gambling Task Disadvantageous Plays			X
Independent Variables			
CU Traits	X		
Mediating Variables			
Cortisol		X	X
Testosterone		X	X
Covariates			
Demographics	X	X	
Stressor Start Time		X	

Salivary Cortisol and Testosterone. Salivary cortisol and testosterone were used as markers for HPA- and HPG-activity in response to the Trier Social Stress Test (TSST). Participants were asked to provide six saliva samples during the session. All samples were assayed in duplicate for testosterone, and in both singlet (10%) and duplicate (90%) for cortisol. Descriptive statistics are provided in Table 2.2.

Sensitivity to Reward and Punishment. During the on-campus session, a modified version of the Iowa Gambling Task (Cauffman et al., 2010) was used to measure reward sensitivity and punishment. The Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994) was designed to approximate real-life decision making under conditions of uncertainty. The modified version of the IGT allows for the separate assessment of decisions in response to positive versus negative feedback— participants receive information on the net gain

or loss associated with a card, rather than information on both a gain and the loss separately (Bechara et al., 1994). Additionally, the modified task is more sensitive to individual differences in performance caused by one's ability to determine the independent effects of gains and losses on subsequent card selection (Peters & Slovic, 2000). By forcing participants to make decisions about each deck in a pseudorandom order, the task eliminates the possibility that individuals employed different search strategies across the decks.

In the modified IGT, participants are presented with four decks of cards and a key they can press to select a card from any of the decks. Each deck contains cards that reward or punish the participants by adding or subtracting points or amounts of money from his account. Two of the decks lead to net increases throughout the task (the gain decks) while the other two lead to net losses (the loss decks). The gain decks are equally advantageous in the long term but vary in the frequency and magnitude of punishment. Similarly, the two loss decks are equally disadvantageous in the long term but vary in the frequency and magnitude of punishment. A running total of the participants' "earnings" appeared on the screen. Participants were instructed to maximize winnings, which requires determining which decks lead to long-term gains and which to long-term losses.

Sensitivity to reward was operationalized as the percentage good plays, which is calculated by dividing the number of times a participant played from advantageous decks during a given task block by the total number of times they were presented with advantageous decks during that block. The quotient was then multiplied by 100 to yield a percentage, with higher percentage indicating a greater sensitivity to reward. Sensitivity to punishment, or the percentage bad plays, was calculated in the same way as sensitivity to reward. Descriptive statistics are provided in Table 2.2.

Risky Decision-Making. During the on-campus session, participants were asked to participate in a computerized, simulated driving task, the Stoplight Driving Task (Chein, Albert, O'Brien, Uckert, & Steinberg, 2011). During the task, participants were instructed to "drive" a car to a distant location where a party is taking place. Participants were told that most people are able to reach the destination in under 2 min. In order to reach the destination, the participants must pass through 20 intersections that are randomly placed throughout the course and marked by a traffic signal. They were informed that when they are approaching an intersection, the traffic signal may turn yellow, and that if this happens, they must decide whether to stop the car and either wait for the light to cycle from yellow to red to green (STOP decision) or attempt to cross through the intersection (GO decision). Participants were also told that if the car is driven through the intersection and the light turns red, there is a chance that it may crash into another vehicle. For the purposes of the current study, the outcome variables were the percentage of risky decisions (GO decisions) and the number of crashes (Chein et al., 2011). Descriptive statistics are provided in Table 2.2.

Demographics. At baseline and each follow-up interview of the Crossroads Study, participants provided information on their race/ethnicity and the highest level of education that either of their parents had received. During the on-campus sessions, participants reported their current age. Parent education was used as a proxy for socioeconomic status (Galobardes et al., 2007). Prior research supports the validity of this measure for use with adolescent samples (Lien et al., 2001). Previous research has found an association between cortisol levels increased with age and race, such that older youth and racial minorities exhibit either lower basal cortisol or diminished cortisol reactivity compared to younger individuals and non-Hispanic Whites (Hostinar et al., 2014). Further, adolescents of lower socio-economic status may exhibit higher

cortisol reactivity in responses to the TSST (Harkness et al., 2011). Consequently, age, race, and parent education were used as covariates in all analyses. Descriptive statistics are provided in Table 2.2.

Table 2.2.
Sample Descriptives

	M / %	SD	Min	Max
Stoplight- Number of Lights Run	6.19	2.64	2.00	16.00
Stoplight- Number of Crashes	2.69	1.60	1.00	8.00
IGT- Advantageous Plays	63.88	22.37	1.67	100.00
IGT- Disadvantageous Plays	70.18	17.86	21.67	98.33
Cortisol (AUC_I)	1.10	1.03	0.00	3.10
Testosterone (AUC_I)	3.22	3.23	0.00	7.44
Callous-Unemotional Traits	19.56	7.67	6.00	36.00
Age	22.84	1.15	2.00	25.00
IQ	94.87	12.26	73.00	128.00
Parent Education	80.00			
Race				
White	16.36			
Black	1.82			
Hispanic	78.18			
Other	3.64			

Plan of Analysis

Cortisol and Testosterone Data Analysis.

Saliva samples were assayed for cortisol and testosterone using the same process outlined for cortisol in Study One (pg. 18). Two indicators of data quality were also calculated for testosterone: standard curve, inter-assay precision, and intra-assay precision. It is important to note that the acceptance criteria for the standard curve, inter-assay precision, and intra-assay precision for testosterone are the same criteria for cortisol (for further details, please refer to pg. 19).

Two indicators of data quality were calculated for each analyte: inter-assay precision and intra-assay precision. Inter-assay precision is a measure of the reliability of assays across microtiter plates. Intra-assay precision is a measure of the reliability of the assay for individual samples by comparing duplicate samples (each participant's saliva sample was assayed more than one time). The inter-assay CVs for cortisol and testosterone were 1.89% and 10.9%, respectively. The intra-assay CVs were 3.69% for cortisol and 3.38% for testosterone. All CVs met recommended criteria (Chard, 1981).

Statistical Analyses.

Preliminary Analyses. Prior to hypothesis testing, the distribution of the raw salivary assay data was examined for non-detects, zero values, insufficient quantity, and values beyond the upper and lower limits of detection (ULOD/LLOD). The data was also examined for outliers, defined as values more than three standard deviations from the mean score (Gordis, et al., 2006), which were dropped from the data. Cortisol and testosterone reactivity were measured by calculating the area under the curve with respect to increase (AUC_i) for the five samples obtained during the session (Pruessner et al., 2003). The formula for AUC_i is:

$$\left(\sum_{i=1}^{n-1}[t_i(m_i + m_{i+1})/2]\right) - (n-1)\cdot m_1$$

where t_i is the precise interval between sample i and sample i+1, m_1 is the first sample, m_i is the level of the cortisol for sample i, and n is the total number of samples. This formula results in one number representing a general index of cortisol reactivity for each subject. The same formula was used to generate one number representing testosterone reactivity. It is possible that some participants showed a stronger decrease than increase over time, resulting in negative values. In cases with negative values, the AUC_i was set to 0, avoiding negative areas and denoting the fact that no increase was seen in the particular subject. The AUCi values were used

as the independent variables in the subsequent statistical analyses. Because salivary assay data is often positively skewed with a disproportionate number of low-value cases (Granger et al., 2007), tests to determine whether Box-cox, log, square-root, or inverse square-root transformation are most appropriate were conducted. The individual testosterone values were not skewed (Table 2.3.). Individual cortisol values were transformed using the Box-Cox transformation (Miller & Plessow, 2013). The AUCi values for cortisol and testosterone were log-transformed.

A testosterone/cortisol ratio was also calculated in two steps (Glenn et al., 2011). The distributions for testosterone and cortisol reactivity (AUC_i) was standardized to t scores (mean 50; SD 10). The t-scores were used to calculate a testosterone/cortisol reactivity ratio. Pairwise correlations were calculated to examine the associations between the independent, dependent, and covariates.

Hypothesis Testing.

Research Aim 2.1. The first research aim was to determine whether CU traits predicted cortisol reactivity, testosterone reactivity, or the ratio of testosterone to cortisol. It was hypothesized that those with elevated levels of CU traits would exhibit lower cortisol reactivity, higher testosterone levels, and a larger testosterone/cortisol reactivity ratio than those with low levels of CU traits.

Multiple regression models were used to examine the association between CU traits and cortisol, testosterone, and the testosterone/cortisol ratio. In separate models, cortisol AUCi, testosterone AUCi, and the ratio were regressed on CU traits. Consistent with previous research (Cauffman et al., 2010; Glenn et al., 2011; Gostisha et al., 2014), the start time of the stressor, age, IQ, race/ethnicity (Latino=1, Non-Latino=0), and parent education (HS degree or more=1,

Less than HS degree= 0) were included as covariates. A total of three regression models were estimated (one per analyte).

Research Aim 2.2. The second research aim was to determine whether CU traits predicted risk taking, sensitivity to reward, and sensitivity to punishment. It was hypothesized that individuals with elevated levels of CU traits would display riskier behavior, greater sensitivity to rewards, and lower sensitivity to punishment.

Multiple regression models were used to examine the association between CU traits, sensitivity to rewards, sensitivity to punishment, and risk taking. In separate models, Stoplight light runs (risk taking), Stoplight crashes (risk taking), advantageous IGT plays (sensitivity to reward), and disadvantageous IGT plays (sensitivity to punishment) were regressed on CU traits. Consistent with previous research (Cauffman et al., 2010; Glenn et al., 2011; Gostisha et al., 2014), the start time of the stressor, age, IQ, race/ethnicity (Latino=1, Non-Latino=0), and parent education (HS degree or more=1, Less than HS degree= 0) were included as covariates. A total of four regression models were estimated (one per risk-taking and sensitivity outcome).

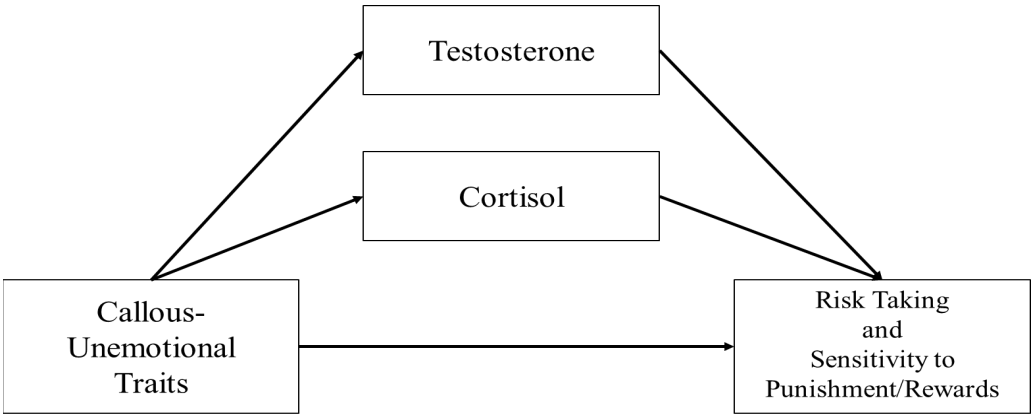
Research Aim 2.3. The third research aim was to determine whether testosterone and cortisol mediate the association between CU traits and each outcome. It was hypothesized that only the testosterone/cortisol ratio would partially mediate the association between CU traits, risk taking, and sensitivity to reward and punishment. Specifically, youth with higher levels of CU traits would exhibit a larger testosterone/cortisol ratio, which in turn predicts greater sensitivity to reward, lower sensitivity to punishment, and higher levels of risk taking.

Mediation models were estimated in Mplus v8 to examine the extent that CU traits influenced risk taking and sensitivity to reward and punishment through cortisol and testosterone levels. For each outcome, the maximum likelihood parameter estimates with standard errors

(MLR) estimator was used. To assess the significance of the indirect effects, percentile bootstrapping was conducted by taking 10,000 samples to construct 95% bias-corrected confidence intervals (Hayes & Scharkow, 2013; Preacher & Hayes, 2008).

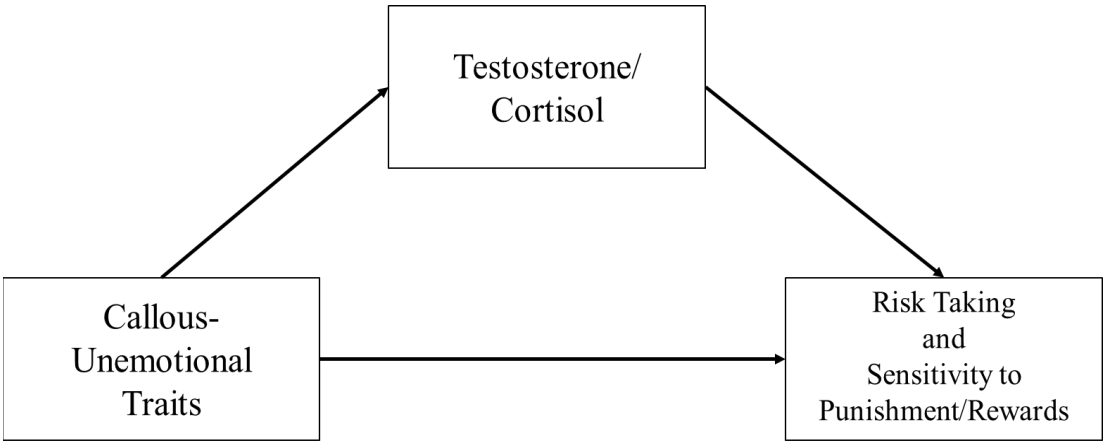
Multiple mediation models were first conducted to determine whether testosterone and cortisol reactivity mediated the association between CU traits and each outcome (Figure 2.1.).

Figure 2.1.
Hypothesized indirect effect of CU traits on risk taking and sensitivity to punishment/rewards through testosterone and cortisol.



Next, simple mediation models were conducted to determine whether the testosterone/cortisol reactivity ratio mediated the association between CU traits and each outcome (Figure 2.2). Consistent with previous research (Glenn et al., 2011; Johnson et al., 2014; Cauffman et al., 2010), the start time of the stressor session, age, race/ethnicity, IQ, and parent education were used as controls in both sets of mediation models. A total of four multiple mediation models and four simple mediation models were estimated.

Figure 2.2.
Hypothesized indirect effect of CU traits on risk taking and sensitivity to punishment/rewards through the testosterone/cortisol ratio.



Supplemental Analyses. Due to concerns about the small sample size and limited power to detect significant but small effects, fixed effect regression models were used to further examine the relation between the main study variables. The benefits of using fixed effect models were described in Study One (pg. 24). First, a set of fixed effect models were estimated to examine how within-individual changes in testosterone and cortisol were related to CU traits. The first models examined how cortisol and testosterone changed in response to the stressor. Interactions between time and CU traits were added to the models to determine whether individuals with different levels of CU traits responded differently to the stressor.

The second set of supplemental analyses included fixed effect models that were used to test whether individuals with increased CU traits exhibited decoupling between cortisol and testosterone reactivity. The first model examined the association between cortisol and testosterone. An interaction term with cortisol and CU traits was added to the model to determine whether the association between cortisol and testosterone was different among individuals with high, low, and average levels of CU traits.

Finally, fixed effect models were used to examine how changes in decision-making during the Iowa Gambling task was related to CU traits, cortisol, and testosterone. The first models examined the change in the number of advantageous and disadvantageous plays across

each block of the task. Interactions between block and CU traits, cortisol, and testosterone were tested in separate models to determine whether individuals with varying levels of these factors performed differently.

Results

Preliminary Results

The raw salivary assay data was examined for non-detects, zero values, insufficient quantity, and values beyond the ULOD and LLOD. For cortisol, two samples were concluded to have insufficient quantity for testing (coded as missing values), four samples were at the ULOD and were retested at a 1:10 dilution, and zero samples were at the LLOD. There were no non-detects or zero values for cortisol. For testosterone, two samples were concluded to have insufficient quantity for testing, zero samples were at the LLOD or ULOD, and there were no non-detects or zero values. Outliers more than 3 SD from the mean were also excluded from analyses. Six cortisol values were dropped. Descriptive statistics for untransformed cortisol and testosterone are presented in Table 2.3. Pairwise correlations were calculated to determine the association between the main study variables (Table 2.5). Results indicate that cortisol and testosterone were significantly correlated (r= 0.52), such that increases in cortisol were associated with increases in testosterone. IQ and the number of crashes in the Stoplight task were also positively correlated (r= .34).

Table 2.3.
Cortisol and Testosterone (Untransformed)

	Cortisol (µg/dL)						Testosterone (pg/mL)					
	n	*M (SD)*		Min, Max		Skew	*n*	*M (SD)*		Min, Max		Skew
Time												
Pre-2 Minutes	54	0.19	0.15	0.06	0.99	**3.36**	55	177.13	55.04	75.87	316.34	0.23
Post-0 Minutes	54	0.22	0.16	0.07	1.03	**2.95**	55	184.47	55.33	72.74	365.08	0.41
Post-5 Minutes	54	0.26	0.16	0.05	0.88	**1.47**	55	168.57	52.64	68.64	319.60	0.37
Post-20 Minutes	54	0.28	0.20	0.05	0.93	**1.64**	55	178.85	58.71	54.23	333.31	0.23
Post-40 Minutes	54	0.20	0.14	0.05	0.78	**2.17**	55	180.60	59.49	57.61	346.71	0.16
AUC_I	54	3.98	5.14	0.00	21.24	**1.65**	55	349.61	456.13	0.00	1693.54	**1.26**

Note. Bolded values indicate significance of $p < .05$.

Table 2.4.
Cortisol and Testosterone (Transformed)

	Cortisol						Testosterone					
	n	*M (SD)*		Min, Max		Skew	*n*	*M (SD)*		Min, Max		Skew
Time												
Pre-2 Minutes	54	0.64	0.10	0.49	1.00	**1.19**	55	177.13	55.04	75.87	316.34	0.23
Post-0 Minutes	54	0.66	0.10	0.51	1.01	**1.09**	55	184.47	55.33	72.74	365.08	0.41
Post-5 Minutes	54	0.69	0.10	0.48	0.97	0.22	55	168.57	52.64	68.64	319.60	0.37
Post-20 Minutes	54	0.70	0.12	0.46	0.98	0.35	55	178.85	58.71	54.23	333.31	0.23
Post-40 Minutes	54	0.65	0.10	0.47	0.94	**0.70**	55	180.60	59.49	57.61	346.71	0.16
AUC_I	54	1.10	1.03	0.00	3.10	1.65	55	3.22	3.23	0.00	7.44	0.03

Note. Bolded values indicate significance of $p < .05$.

Table 2.5

Pairwise correlations between study variables

		1	2	3	4	5	6	7	8	9	10	11	12
1	Stoplight- Number of Lights Run	-											
2	Stoplight- Number of Crashes	**0.84**	-										
3	IGT- Advantageous Plays	-0.10	-0.04	-									
4	IGT- Disadvantageous Plays	-0.10	-0.19	**0.71**	-								
5	Cortisol (AUC_I)	-0.15	-0.21	-0.05	0.06	-							
6	Testosterone (AUC_I)	0.06	0.06	-0.16	-0.13	**0.52**	-						
7	Callous-Unemotional Traits	-0.06	0.00	0.09	0.07	-0.26	-0.13	-					
8	IQ	**0.34**	0.20	-0.21	-0.07	0.02	0.03	-0.15	-				
9	Age	0.04	0.03	0.06	0.05	-0.07	0.11	-0.09	0.01	-			
10	Parent Education[A]	-0.04	0.02	0.05	0.06	0.06	0.15	-0.09	0.05	0.13	-		
11	Race[B]	-0.02	-0.05	0.00	0.10	-0.05	0.02	0.05	**-0.32**	-0.19	**-0.26**	-	
12	Start Time	0.02	0.15	0.10	0.10	-0.07	-0.07	0.01	-0.14	**-0.29**	0.05	-0.09	-

[A] HS degree or more= 1 , Less than HS degree= 0, [B] Latino= 1, Non-Latino= 0. *Note.* Bolded values indicate significance of $p < .05$.

Table 2.6.

Multiple regression model with callous-unemotional traits predicting cortisol reactivity, testosterone reactivity, and testosterone/cortisol ratio

	Cortisol (AUCi)					Testosterone (AUCi)					Testosterone/Cortisol				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
CU Traits	-0.04	0.02	0.06	-0.07	0.00	-0.05	0.06	0.45	-0.17	0.08	0.00	0.00	1.00	-0.01	0.01
Age	-0.14	0.13	0.32	-0.41	0.13	0.24	0.43	0.58	-0.62	1.10	0.05	0.03	0.07	0.00	0.10
IQ	-0.01	0.01	0.65	-0.03	0.02	0.01	0.04	0.85	-0.07	0.09	0.00	0.00	0.92	0.00	0.01
Parent Education[a]	0.11	0.37	0.76	-0.63	0.85	1.26	1.18	0.29	-1.10	3.63	0.00	0.07	0.97	-0.15	0.15
Race[b]	-0.21	0.39	0.59	-0.99	0.57	0.72	1.23	0.56	-1.76	3.20	0.09	0.08	0.27	-0.07	0.24
Start Time	0.00	0.00	0.39	0.00	0.00	0.00	0.00	0.79	0.00	0.00	0.00	0.00	0.48	0.00	0.00

Research Aim 2.1.

Multiple regression models were used to examine the association between CU traits and cortisol, testosterone, and the testosterone/cortisol ratio (Table 2.6). The results indicated there were no significant associations between cortisol reactivity and CU traits. Similar results were observed for testosterone and the testosterone/cortisol ratio— CU traits were not significantly associated with either outcome.

Research Aim 2.2.

Multiple regression models were used to examine the association between CU traits, risk taking, sensitivity to rewards, and sensitivity to punishment. In separate models, Stoplight lights runs (risk taking), Stoplight crashes (risk taking), advantageous plays (sensitivity to reward), and disadvantageous plays (sensitivity to punishment) were regressed on CU traits. The results indicated there was no significant association between CU traits and the number of lights run or the number of crashes made in the Stoplight task (Table 2.7).

Table 2.7.
Multiple regression models with callous-unemotional traits predicting runs and crashes in the Stoplight Driving Task

	Stoplight- Lights Run					Stoplight- Crashes				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
CU Traits	0.00	0.01	0.80	-0.01	0.02	0.00	0.01	0.89	-0.02	0.02
Age	0.01	0.05	0.76	-0.08	0.11	0.03	0.07	0.66	-0.11	0.18
IQ	0.01	0.00	0.13	0.00	0.02	0.02	0.01	0.01	0.00	0.03
Parent Education[a]	0.01	0.14	0.93	-0.27	0.29	-0.07	0.21	0.75	-0.48	0.35
Race[b]	0.03	0.14	0.84	-0.25	0.31	0.13	0.22	0.56	-0.30	0.56

[A] HS degree or more=1 , Less than HS degree= 0, [B] Latino=1, Non-Latino=0

Similar results were observed for performance in the Iowa Gambling task (Table 2.8). Specifically, CU traits were not significantly associated with the number of advantageous plays

or the number of disadvantageous plays. Overall, these results indicate that CU traits did not predict risk taking, sensitivity to rewards, or sensitivity to punishment.

Table 2.8.
Multiple regression models with callous-unemotional traits predicting advantageous and disadvantageous plays in the Iowa Gambling Task

	IGT- Advantageous Plays					IGT- Disadvantageous Plays				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
CU Traits	0.21	0.41	0.61	-0.61	1.04	0.16	0.33	0.62	-0.51	0.84
Age	0.95	2.77	0.73	-4.61	6.50	1.00	2.25	0.66	-3.51	5.51
IQ	-0.41	0.27	0.14	-0.95	0.14	-0.04	0.22	0.84	-0.49	0.40
Parent Education[a]	2.45	8.02	0.76	-13.67	18.56	4.10	6.51	0.53	-8.98	17.19
Race[b]	-3.08	8.26	0.71	-19.69	13.52	5.17	6.71	0.45	-8.32	18.65

[A] HS degree or more=1 , Less than HS degree= 0, [B] Latino=1, Non-Latino=0

Research Aim 2.3.

Multiple mediation analyses were conducted to examine to what extent CU traits influences risk taking, sensitivity to reward, and sensitivity to punishment through cortisol and testosterone levels. The first models examined whether CU traits predicted the number of lights run and crashes made in the Stoplight task (Table 2.9). The results indicated that the total, direct, and indirect effect of CU traits on both risk-taking outcomes were not significant. The results were similar for performance in the Iowa Gambling task (Table 2.10). CU traits were neither directly nor indirectly related to the percentage of advantageous or disadvantageous plays.

Table 2.9.

Mediation models with callous-unemotional traits, cortisol and testosterone predicting runs and crashes in the Stoplight Driving Task

	Stoplight- Lights Run			Stoplight- Crashes		
	B	*SE*	*p*	*B*	*SE*	*p*
CU Traits	-0.01	0.01	0.84	-0.01	0.01	0.64
Cortisol	**-0.14**	**0.06**	**0.02**	-0.15	0.10	0.13
Testosterone	0.03	0.02	0.15	0.03	0.03	0.34
Age	-0.01	0.04	0.89	0.01	0.07	0.89
IQ	0.01	0.00	0.10	**0.02**	**0.01**	**0.00**
Parent Education[a]	-0.01	0.13	0.94	-0.09	0.17	0.62
Race[b]	0.01	0.12	0.97	0.11	0.19	0.58
Effects from X to Y						
Total	0.00	0.01	0.80	0.00	0.01	0.91
Indirect	0.00	0.00	0.29	0.00	0.00	0.36
Specific Indirect						
Lights Run ← Cortisol ← CU Traits	0.01	0.00	0.16	0.01	0.00	0.25
Lights Run ← Testosterone ← CU Traits	0.00	0.00	0.59	0.00	0.00	0.62

Table 2.10.

Mediation models with callous-unemotional traits, cortisol and testosterone predicting advantageous and disadvantageous plays in the Iowa Gambling Task

	IGT- Advantageous Plays			IGT- Disadvantageous Plays		
	B	*SE*	*p*	*B*	*SE*	*p*
CU Traits	0.21	0.48	0.66	0.24	0.44	0.59
Cortisol	1.74	3.11	0.58	4.03	0.29	0.16
Testosterone	-1.41	0.94	0.13	-1.52	0.95	0.11
Age	1.51	2.64	0.57	1.81	1.86	0.33
IQ	-0.09	0.26	0.14	-0.01	0.23	0.96
Parent Education[a]	4.02	9.52	0.67	5.56	7.51	0.46
Race[b]	-1.74	7.88	0.83	6.90	8.49	0.42
Effects from X to Y						
Total	0.23	0.43	0.60	0.19	0.42	0.66
Indirect	0.02	0.18	0.92	-0.06	0.17	0.73
Specific Indirect						
Lights Run<-- Cortisol <-- CU Traits	-0.06	0.16	0.70	-0.14	0.17	0.41
Lights Run<-- Testosterone <-- CU Traits	0.08	0.12	0.52	0.08	0.14	0.55

Simple mediation analyses were conducted to determine whether the testosterone/cortisol ratio mediated the association between CU traits and risk taking, sensitivity to reward, and sensitivity to punishment. Again, the results indicated that the total, direct, and indirect effect of CU traits on risk taking in the Stoplight task (Table 2.11) and plays in the Iowa Gambling Task (Table 2.12) were not significant. Altogether, these results indicate that CU traits are not directly associated with risk taking and sensitivity to punishment/reward, nor do they operate indirectly through cortisol or testosterone.

Table 2.11.
Mediation models with callous-unemotional trait and testosterone/cortisol predicting runs and crashes in the Stoplight Driving Task

	Stoplight- Lights Run			Stoplight- Crashes		
	B	*SE*	*p*	*B*	*SE*	*p*
CU Traits	0.00	0.01	0.75	0.00	0.01	0.93
Testosterone/Cortisol	**0.55**	**0.28**	**0.05**	0.49	0.32	0.13
Age	-0.01	0.04	0.79	0.01	0.07	0.93
IQ	0.01	0.00	0.07	0.02	0.01	0.00
Parent Education[a]	0.01	0.14	0.93	-0.07	0.18	0.71
Race[b]	-0.01	0.11	0.92	0.10	0.19	0.60
Effects from X to Y						
Total	0.00	0.01	0.78	0.00	0.01	0.91
Indirect	0.00	0.00	0.92	0.00	0.00	0.93

[A] HS degree or more=1 , Less than HS degree= 0, [B] Latino=1, Non-Latino=0

Table 2.12.
Mediation models with callous-unemotional traits, cortisol and testosterone predicting advantageous and disadvantageous plays in the Iowa Gambling Task

	IGT- Advantageous Plays			IGT- Disadvantageous Plays		
	B	*SE*	*p*	*B*	*SE*	*p*
CU Traits	0.21	0.45	0.64	0.16	0.43	0.71
Testosterone/Cortisol	-13.89	13.81	0.31	-26.71	14.59	0.07
Age	1.55	2.66	0.56	2.16	0.19	0.27
IQ	-0.41	0.25	0.11	-0.05	0.22	0.83
Parent Education[a]	2.52	9.60	0.79	4.25	6.83	0.53
Race[b]	-0.20	7.83	0.80	7.19	7.62	0.35
Effects from X to Y						
Total	0.218	0.429	0.611	0.175	0.411	0.67
Indirect	0.007	0.083	0.934	0.014	0.135	0.92

[A] HS degree or more=1 , Less than HS degree= 0, [B] Latino=1, Non-Latino=0

Supplemental Analyses

To address concerns regarding the small sample size and limited power, fixed effect models were estimated to examine how within-individual changes in testosterone and cortisol were related to CU traits. The results from the first models indicate that participants exhibited increased cortisol and testosterone levels following the stressor (Table 2.13). Specifically, cortisol levels 5-minutes and 20-minutes after the TSST were significantly higher than pre-TSST levels (Figure 2.3). Testosterone levels immediately after the TSST (0-minutes) were also significantly higher than pre-TSST levels (Figure 2.4). Interestingly, testosterone levels 5-minutes after the TSST dropped and were significantly lower that pre-TSST levels.

Table 2.13.

Fixed Effects Regressions for time predicting cortisol and testosterone reactivity

	Cortisol					Testosterone				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
Time										
Post-0 Minutes	**0.03**	**0.01**	**0.01**	**0.01**	**0.05**	**7.34**	**2.87**	**0.01**	**1.69**	**12.99**
Post-5 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**	**-8.56**	**2.87**	**0.00**	**-14.21**	**-2.90**
Post-20 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**	1.72	2.87	0.55	-3.93	7.38
Post-40 Minutes	0.01	0.01	0.32	-0.01	0.03	3.47	2.87	0.23	-2.18	9.13

Figure 2.3.

Cortisol Response to the Trier Social Stress Test

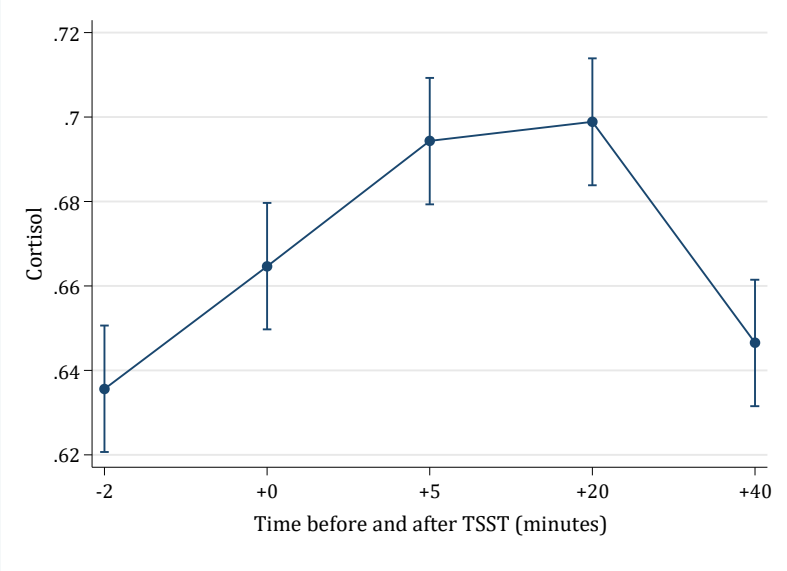
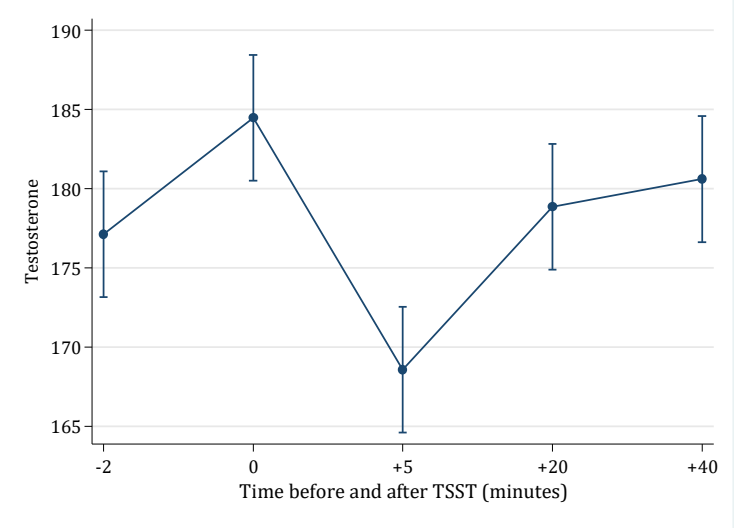


Figure 2.4

Testosterone Response to the Trier Social Stress Test

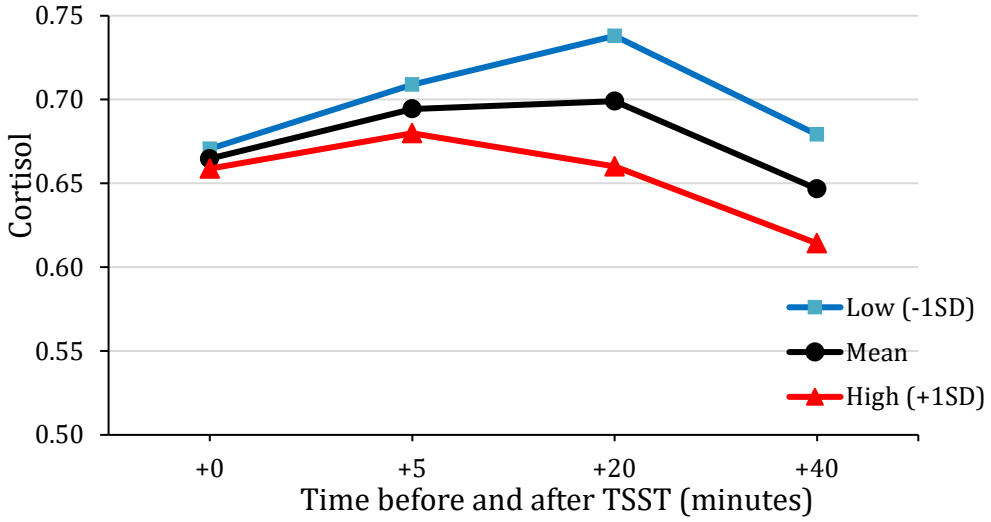


To determine whether individuals with different levels of CU traits responded differently to the stressor, an interaction term with time and CU traits was added to the models. Testosterone reactivity did not differ by levels of CU traits (F(4,212) = 0.69, p= .60). For cortisol, the results indicated the interaction between time and CU traits was significant (F(4, 208)= 5.28, p< .001). Participants with higher levels of CU traits had significantly lower levels of cortisol 20- and 40-minutes after the TSST (Table 2.14, Figure 2.5).

Table 2.14.
Interactions between Time and Callous-Unemotional Traits in Fixed Effects Regressions predicting Cortisol Reactivity

	B	SE	*p*	95% CI	
Time					
Post-0 Minutes	**0.03**	**0.01**	**0.01**	**0.01**	**0.05**
Post-5 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**
Post-20 Minutes	**0.06**	**0.01**	**0.00**	**0.04**	**0.08**
Post-40 Minutes	0.01	0.01	0.29	-0.01	0.03
Time x CU Traits					
Post-0 Minutes	0.00	0.00	0.57	0.00	0.00
Post-5 Minutes	0.00	0.00	0.16	0.00	0.00
Post-20 Minutes	**-0.01**	**0.00**	**0.00**	**-0.01**	**0.00**
Post-40 Minutes	**0.00**	**0.00**	**0.00**	**-0.01**	**0.00**

Figure 2.5.
Cortisol Response by Saliva Sample and Callous-Unemotional Traits



The second set of supplemental analyses included fixed effect models that were used to test whether individuals with increased CU traits exhibited decoupling between cortisol and testosterone. The first model examined the association between cortisol and testosterone. Cortisol was significantly associated with testosterone, such that increases in cortisol were associated with increases in testosterone. An interaction term with cortisol and CU traits was added to the model, and the results indicated that the interaction was not significantly associated with testosterone (Table 2.15). This result suggests that individuals with higher levels of CU traits did not exhibit decoupling between cortisol and testosterone.

Table 2.15.
Fixed Effects Regressions with Cortisol Reactivity and CU Traits predicting Testosterone Reactivity

	Model 1					Model 2				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
Time										
Post-0 Minutes	3.97	2.69	0.14	-1.34	9.29	3.88	2.68	0.15	-1.41	9.16
Post-5 Minutes	**-15.27**	**2.83**	**0.00**	**-20.84**	**-9.70**	**-15.55**	**2.81**	**0.00**	**-21.10**	**-10.00**
Post-20 Minutes	-5.48	2.85	0.06	-11.11	0.15	**-6.48**	**2.89**	**0.03**	**-12.18**	**-0.79**
Post-40 Minutes	1.77	2.66	0.51	-3.46	7.01	0.82	2.69	0.76	-4.48	6.12
Cortisol	**112.29**	**16.82**	**0.00**	**79.14**	**145.44**	**109.65**	**16.78**	**0.00**	**76.57**	**142.73**
Cortisol x CU Traits						-3.93	2.11	0.06	-8.08	0.23

Lastly, fixed effect models were used to examine how performance in the Iowa Gambling task was related to CU traits, cortisol reactivity, and testosterone reactivity. These models tested how the number of advantageous and disadvantageous plays changed throughout the task, and whether performance differed by the individuals' levels of CU traits, cortisol reactivity, and testosterone reactivity (Tables 2.16 and 2.17). The results indicated that the rate of advantageous and disadvantageous plays did not change during the task, suggesting participants did not become increasingly sensitive to gains or losses as they completed the task. CU traits and

testosterone reactivity did not moderate the association between block and task performance, suggesting individuals with high levels of CU traits or testosterone reactivity performed similarly to their low CU and low testosterone counterparts.

Table 2.16.

Fixed Effects Regressions with CU traits, Cortisol Reactivity and Testosterone Reactivity predicting advantageous plays in the Iowa Gambling Task

	Model 1					Model 2					Model 3					Model 4				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
Block	0.86	0.61	0.16	-0.34	2.06	0.86	0.61	0.16	-0.33	2.06	0.72	0.62	0.24	-0.49	1.93	0.86	0.61	0.16	-0.33	2.06
Block x CU Traits						0.14	0.08	0.07	-0.01	0.30										
Block x Cortisol											-0.02	0.12	0.90	-0.25	0.22					
Block x Testosterone																0.00	0.00	0.13	0.00	0.00

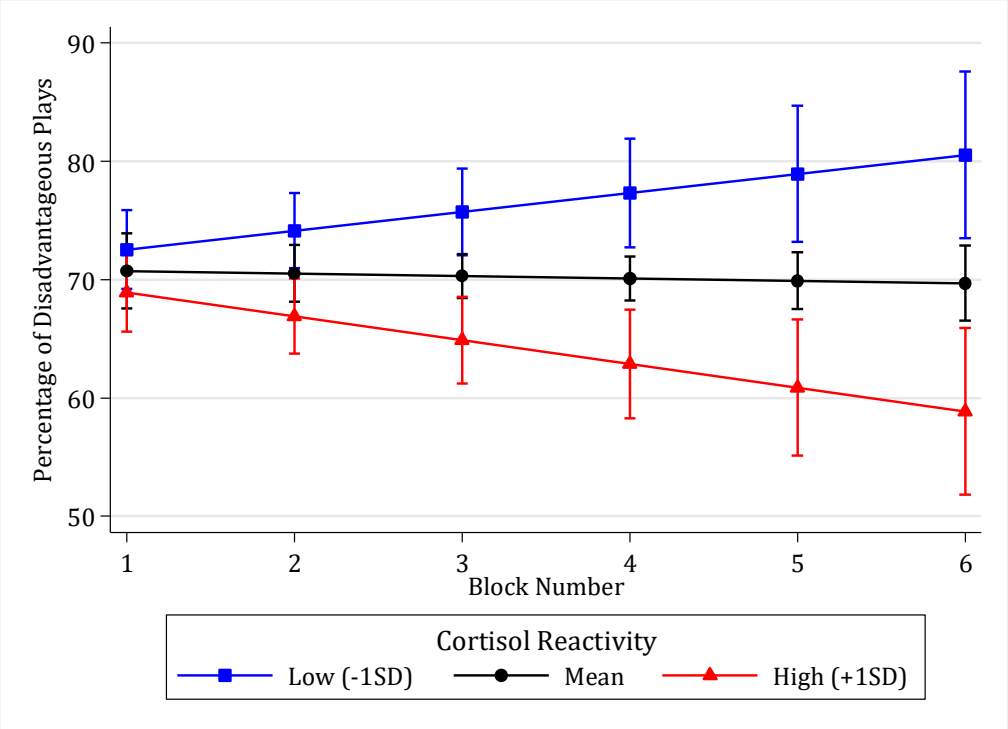
Table 2.17.

Fixed Effects Regressions with CU traits, Cortisol Reactivity and Testosterone Reactivity predicting disadvantageous plays in the Iowa Gambling Task

	Model 1					Model 2					Model 3					Model 4				
	B	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI		*B*	*SE*	*p*	95% CI	
Block	-0.04	0.54	0.94	-1.11	1.02	-0.04	0.54	0.94	-1.11	1.02	-0.21	0.53	0.70	-1.26	0.85	-0.04	0.54	0.94	-1.10	1.02
Block x CU Traits						0.06	0.07	0.37	-0.08	0.20										
Block x Cortisol											**-0.35**	**0.11**	**0.00**	**-0.56**	**-0.15**					
Block x Testosterone																0.00	0.00	0.10	0.00	0.00

The interaction between block and cortisol reactivity was significant (Figure 2.6). Post-hoc analysis revealed that individuals with low (-1SD) cortisol reactivity increasingly made disadvantageous plays (*dydx*= 1.60, SE= 0.76, *p*= 0.03), while individuals with average levels of cortisol reactivity did not exhibit any changes (*dydx*= -.01, SE= 0.54, *p*= 0.70). Interestingly, the number of disadvantageous plays decreased among individuals with high (+1SD) cortisol reactivity (*dydx*= -2.01, SE= 0.76, *p*= 0.01). In sum, these results suggest that individuals with greater cortisol response exhibited increased sensitivity to punishment, while individuals with lower cortisol response exhibited decreased sensitivity to punishment.

Figure 2.6.
Percentage of Disadvantageous Plays by Block Number and Cortisol Reactivity



Discussion

Despite evidence that CU traits, cortisol, and testosterone each contribute to antisocial and delinquent behavior, how these factors operate in conjunction to confer risk is not well

understood. By examining these factors in a sample of justice-involved youth, the current study sought to provide much-needed information on the biological mechanisms through which CU traits place youth at risk for delinquent behavior. Contrary to my hypotheses, the initial results of this study suggested CU traits did not predict either cortisol or testosterone reactivity. Surprisingly, CU traits did not directly or indirectly predict performance in either the Stoplight task or the Iowa Gambling task. These findings suggest that individuals with elevated levels of CU traits do not engage in riskier behavior or differ in sensitivity to punishment/rewards than their low CU counterparts. The discrepancy between these initial results and prior work on CU traits may be due to the small sample size and limited statistical power. Indeed, post-hoc power analyses conducted in G*Power (Faul et al., 2007) and Monte Carlo power analysis simulations conducted in MPlus v8 (Muthén & Muthén, 2002; Thoemmes, Mackinnon, & Reiser, 2010) the study did not have enough power to detect any direct or indirect effects of CU traits.

To circumvent these limitations, fixed effect analyses were used to examine the relation between CU traits and within-person changes in cortisol and testosterone. Similar to the initial results, these models indicate there were no significant differences in testosterone among participants with high, average, and low levels of CU traits. Regardless of CU traits, testosterone levels peaked immediately after the stressor, decreased significantly the following five minutes, then returned to pre-stressor levels. Considering the mixed evidence regarding the relation between CU traits and testosterone (Dekkers et al., 2019; Roy, Cook, Carre, & Welker, 2019), this finding was relatively unsurprising. The fixed effect models also indicated that individuals with elevated CU traits exhibited low cortisol reactivity. Compared to participants with average or low levels of CU traits, participants with high levels of CU traits exhibited a smaller increase

in cortisol levels following the stressor. This finding aligns with majority of the literature on CU traits and HPA activity (for review see Glenn & Raine, 2014; Moul et al., 2018).

Although these results conflict with the prevailing theories about the association between CU traits, cortisol, and testosterone, the pattern of reactivity observed among individuals with elevated CU traits still provides insight into their behavior. During a stressful event, the HPA axis acts to inhibit activity in the HPG axis, and vice versa (E. Johnson, Kamilaris, Chrousos, & Gold, 1992). If the inhibitory effect of cortisol is disrupted or weakened in individuals with elevated CU traits, then testosterone may have an unchecked influence on behavior. Increases in testosterone following stressors or competition have been linked with increased aggression (Zilioli & Bird, 2017). This is particularly concerning considering evidence that personality or temperamental factors may exacerbate the association between testosterone and aggression. For example, Welker and colleagues (2019) found that increased testosterone levels following a competition was associated with higher levels of aggression, but only among individuals who reported feeling generally less connected to others. Unfortunately, a measure of aggression was not included in the current study, therefore I was unable to test this potential mechanism. Future research should determine whether this response contributes to the higher rates of aggressive behavior observed among youth high in CU traits.

Several theories and studies proposed that the ratio of testosterone to cortisol would be more strongly related to CU traits than the individual levels of each hormone (Herpers et al., 2014; Montoya et al., 2012). Specifically, individuals with elevated levels of CU traits would have higher levels of testosterone relative to cortisol. The results of this study did not support this postulation— CU traits were not associated with the testosterone/cortisol ratio. An important differentiating factor between the current study and previous work is how testosterone was

assessed. The few studies on CU traits and the testosterone/cortisol ratio measured resting, basal, or diurnal testosterone (Glenn et al., 2011; Johnson et al., 2014; Loney et al., 2006; Welker et al., 2014), which taps more into total production rather than the change in hormones levels (Khoury et al., 2015). Researchers have yet to delineate whether the overall amount or the fluctuation in testosterone is more relevant to understanding how CU traits predispose youth to antisocial behavior. Given the types of situations in which antisocial behavior occurs (e.g, threats of physical pain, status loss, or legal punishment), I propose reactivity provides a closer estimate of how an individual would respond behaviorally. Additional work is needed to test this assumption.

While this study provided no evidence that CU traits predicted performance in either of the behavioral tasks, it did provide an interesting finding regarding the relation between cortisol reactivity and performance on the Iowa Gambling task. Results from the fixed effect models indicated that individuals with low cortisol reactivity increasingly made disadvantageous plays throughout the task. One possible interpretation of this finding is that individuals with low cortisol reactivity are less sensitive to punishment than individuals with average or high levels of cortisol reactivity. Alternatively, repeatedly making disadvantageous plays may reflect a reduced propensity for avoidant behavior or an inability to process punishment when seeking rewards. These same characteristics have been observed among individuals with psychopathic traits (Baskin-Sommers et al., 2010; Finger et al., 2011; Mitchell et al., 2002). Due to the limited sample size, I was unable to fully test whether CU traits were indirectly associated with punishment sensitivity through cortisol reactivity. Given decreased punishment sensitivity and avoidant behavior are thought to contribute to the antisocial behavior of youth high in CU traits, researchers should test this association in a larger sample.

Despite the unique features of this study, there were several important limitations to consider. The most significant limitation is the small sample size which likely contributed to the null findings from the original multiple regression models. Post-hoc power analyses indicated there was inadequate statistical power to detect small direct effects in the multiple regression models or significant indirect effects in the mediation models. Additional research with larger sample sizes is needed. An additional limitation is the lack of a control group that did not participate in the TSST. It is possible that any effect that CU traits had on risk taking and sensitivity to punishment/reward was masked by the effect of the TSST. In experimental studies, youth assigned to the stress conditions tend to make more risky decisions than youth assigned to non-stressed conditions (S. B. Johnson, Dariotis, & Wang, 2012; Reynolds et al., 2013). Individual characteristics, like impulsivity and social anxiety, can attenuate or exacerbate the effect of stress. It is imperative that future studies are conducted with proper comparison groups in order to determine if there is truly no association between CU traits and performance in the Iowa Gambling Task and Stoplight task. A final limitation is that the sample consisted of only males. Consequently, the results of this study may not be generalizable to females involved in the justice system. There are gender differences in HPG activity and testosterone levels, with females typically having lower levels than males. There is also evidence that females with elevated levels of CU traits exhibit different patterns of cortisol and testosterone than their male counterparts (O'Leary et al., 2007; O'Leary et al., 2010). As such, it is important that future work examines the association between CU traits and HPA/HPG activity in female participants.

Despite these limitations, there were also notable strengths. A key strength of this study is the assessment of both HPA and HPA reactivity in relation to CU traits. Although adolescence is a period in which the HPA and HPG axes are more likely to work together to address stress

(Shirtcliff et al., 2015), cortisol and testosterone have been examined together in only a handful of studies (Glenn et al., 2011; Johnson et al., 2014; Loney et al., 2006; Welker et al., 2014). An additional strength was the inclusion of supplemental fixed effect models that enabled me to isolate the effects of CU traits and examine within-person changes in cortisol, testosterone. Traditional between-individual statistical models cannot rule out the possibility that any associations could be attributable to selection effects or unmeasured confounding factors. The fixed effect models permit stronger claims to be made about the associations between the main study variables. The final notable strength of the study is that the sample consisted of justice-involved youth. Given the overarching goal of better understanding CU traits in order to prevent and reduce antisocial behavior, it is important to conduct this research with samples who have a history of antisocial behavior.

Altogether, the results of the current study help refine our understanding of CU traits and their relation to HPA and HPG reactivity. Individuals with elevated levels of CU traits exhibited a pattern of cortisol and testosterone reactivity that suggests they have an increased propensity to engage in aggressive behavior. Additionally, individuals with low cortisol reactivity displayed decreased sensitivity to punishment and reduced avoidant behavior. Although though this study did not provide evidence that cortisol and testosterone mediate the association between CU traits and sensitivity to reward/punishment or risk taking, this information may still be useful in the context of delinquency prevention and rehabilitation. There is growing evidence hormones can be used to determine which interventions would effectively address behavioral problems and potentially cognitive or emotional deficits. The results of this study suggest youth with elevated CU traits may benefit from programs that could address low HPA reactivity or reduce HPG activity. One promising treatment option is the Fast Track intervention program (CPPRG, 1992)

(CPPRG, 1999), which was designed to address family- and school-based risk factors, build socioemotional competency, and strengthen protective factors. Carre and colleagues (2014) sought to determine if the program reduced reactive aggression following provocation, and whether certain biological factors could explain the effects of Fast Track program. Their results indicated that participation in the program during elementary and middle school was associated with significantly lower levels of reactive aggression at age 26. Further, testosterone reactivity mediated the association between intervention participation and aggression. Participation resulted in significantly lower testosterone reactivity in adulthood, which explained a significant portion of the programs effect on aggression. This study provides compelling evidence that comprehensive psychosocial interventions have the potential to prevent aggressive behavior by influencing the biological profiles of their participants.

Antisocial youth with CU traits are a particularly difficult group to treat, in part because interventions fail to address the specific factors that contribute to their behavior (Hawes et al., 2014; Lipsey & Cullen, 2007). Despite evidence that up to 68% of the variation in CU traits has a genetic basis (Frick et al., 2014), researchers and practitioners typically disregard or avoid addressing biological risk factors. As a result, we squander the opportunity to understand and target malleable risk factors, and fail to fully address the needs of youth with CU traits. Given the results of the current study and prior intervention research, the biological underpinnings of CU traits and the effectiveness of programs that take biology into consideration must be further explored.

CONCLUSION

This dissertation was designed to examine the hormonal correlates of CU traits with the hope of better understanding why youth with CU traits are more likely to engage in antisocial and delinquent behavior. Study One examined how CU traits predicted cortisol and alpha-amylase reactivity after accounting for prior exposure to violence and hostile parent--child relationships. Study Two examined whether cortisol and testosterone were potential mechanisms through which CU traits contributed to sensitivity to rewards/punishment and risk taking. Although several hypotheses were not supported, the findings from both studies provide important insights about the hormonal correlates of CU traits. Specifically, participants with elevated CU traits exhibited lower cortisol reactivity than their counterparts. However, they also exhibited similar increases in testosterone and alpha-amylase shortly after the stressor ended. This pattern of reactivity may explain why individuals with elevated CU traits are more likely to engage in aggressive behavior. When cortisol levels are low, increased testosterone promotes aggression and approach-related behaviors, while increased alpha-amylase promotes defensive responses. When provoked or threatened, individuals high in CU traits may be more likely to approach and initiate a verbal or physical conflict. Paired with other CU-related impairments (e.g., a lack of empathy and concern about the long-term consequences of their actions), this reaction may increase their responsiveness to minor aggression-inducing stimuli and the frequency of aggressive acts.

To better promote positive development and outcomes among individuals with high levels of CU traits, more research is needed to understand the biological factors underlying CU traits. Based on the existing evidence, researchers have already proposed assessing cortisol, alpha-amylase, testosterone, and other hormone levels to determine whether youth high in CU

traits are responsive to certain treatments (Glenn & McCauley, 2018). They posit that by establishing whether a youth would benefit from the treatment prior to enrollment, we would avoid losing time, money, and other resources on ineffective treatments. While this is a worthy goal to pursue, it would be unwise for practitioners to start making treatment decisions based on our current understanding of CU traits. First, research on the hormonal correlates of CU traits is still fairly limited. The results from existing studies are inconsistent due to considerable variation in how hormones are measured. Additional research is needed to determine clinically meaningful thresholds for each hormone in order to ensure assessments are objective and standardized. Second, depending on the type of measurement, hormone levels can vary considerably from day to day (Kuhlman et al., 2019). Consequently, using a single measurement may inappropriately categorize youth as responsive or unresponsive to treatment. A better understanding of how environmental and individual factors contribute to the variation in hormone levels is needed. Finally, improperly interpreting and using assessments of an individual's responsiveness could inadvertently stigmatize youth with high levels of CU traits. CU traits are often measured in juvenile justice settings as a means of assessing an individual's risk of recidivism (Viljoen, McLachlan, & Vincent, 2010). Without a detailed explanation that hormones are malleable, any results indicating youth high in CU traits are less unamenable to treatment at a biological level could cause undue harm in the form of harsher sentences and prolonged stays in juvenile facilities. Guidelines for the use of these assessments must be put in place before hormones can be widely used to determine treatment responsiveness.

Although additional work needed to understand how biological factors could be used to determine treatment responsiveness, practitioners can still use the information gathered in this study and prior research to help individuals with high levels of CU traits. This dissertation found

that the way individuals with high CU traits physiologically respond to threats may increase the likelihood that they act aggressively. Employing interventions known to influence HPA, HPG, and SNS activity, such as the Fast Track intervention program (CPPRG, 1999) or the comprehensive treatment utilized by Caldwell et al. (2016), could help reduce aggression among youth high in CU traits. If these types of treatment options are inaccessible or impractical given the available resources, practitioners can focus on other socioemotional deficits associated with CU traits. For example, the negative effect of this pattern of stress reactivity could be attenuated by enhancing youths' ability to recognize and process the emotional cues in their environment (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012; Hunnikin & van Goozen, 2019; Moul, Hawes, & Dadds, 2018). Negative emotional cues, such as facial expressions of fear and sadness, are thought to prevent antisocial behavior by eliciting a negative physiological response (e.g. increased cortisol), which in turn contributes to empathy and concern about how one's actions caused those feelings in the other person. Impairments in the ability to recognize and respond to emotion are widely observed among individuals with high levels of CU traits (Dadds, Kimonis, Schollar-Root, Moul, & Hawes, 2018). Not only are they more likely to misidentify sadness and fear, but they are also more likely to interpret ambiguous facial expression as angry or threatening. Fortunately, there is evidence that addressing these impairments may help individuals process emotional information more accurately and reduce aggressive behavior (Penton-Voak et al., 2013). A promising treatment option for adolescents, originally developed by Neumann, Babbage, Zupan, & Willer (2015), consists of a two-hour computerized intervention that requires participants to identify facial expressions of happiness, sadness, fear, and anger, describe an event that made them feel those emotions, then mimic the emotions in a mirror. Among a sample of male juvenile offenders, Hubble and colleagues (2015) found that

youth who received the intervention had improved emotion recognition abilities and showed a reduction in the severity of crimes committed during the following six months. Computerized interventions that target the cognitive and emotional deficits associated with CU traits can also be effective among adults. Employing a six-week program consisting of once a week, hour-long computerized trainings designed to specifically address deficiencies in attention to context, Baskin-Sommers, Curtin, and Newman (2015) were able to increase the responsiveness to affective and nonaffective information among individuals with psychopathic traits.

Altogether, the results of intervention research and the current dissertation underscore the importance of understanding the biological factors associated with CU traits. In response to stressful or threatening stimuli, individuals with elevated CU traits exhibited the same increase in alpha-amylase and testosterone levels as their counterparts with low or average levels of CU traits, yet their cortisol levels were significantly lower in comparison. This response may explain, in part, how CU traits predispose youth to aggressive and violent behavior. In order to mitigate this risk factor, practitioners should use existing treatments that enhance the socioemotional capabilities of youth high in CU traits. Considering the extensive costs associated with such behavior, continued efforts to understand and effectively target this underlying biological mechanism are critical.

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