

ADAPTIVE CALIBRATION OF RISK-TAKING:
THE ROLE OF PARENTING STYLES, EMOTION
REGULATION, AND STRESS REACTIVITY

By

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Bachelor of Science in Psychology
Oklahoma State University
Stillwater, Oklahoma
2016

Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
MASTER OF SCIENCE
May, 2019

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Title of Study: ADAPTIVE CALIBRATION OF RISK TAKING: THE ROLE OF
PARENTING STYLES, EMOTION REGULATION, AND STRESS
REACTIVITY

Major Field: PSYCHOLOGY

Abstract: The present study was designed to understand whether experienced parenting style in childhood and emotion regulation strategies can predict the relationship between stress reactivity and risk-taking behaviors. Based on the *Adaptive Calibration Model* it was predicted that the relationship between cortisol and risk-taking behavior would be influenced by early life experiences with parents and emotion regulation strategies. To test the hypotheses, 156 college age students were recruited to participate in an experimental study in which half of the participants experienced a stressful social situation before engaging in a computerized risk-taking task. The results of the present study suggested that cortisol reactivity marginally increased risk-taking propensity. However, the present study did not find evidence that differences in risk-taking were influenced by interactions between parenting style and emotion regulation with cortisol reactivity. Results of the present study have implications in future research design of studies using computerized measures of risk-taking, and areas of future research in the field based on trends in the current data.

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CHAPTER I

INTRODUCTION

Risk-taking is a universal behavior observed in both humans and non-human animals (Laviola, Macri, Morley-Fletcher, & Adriani, 2003; Weber, Shafir, & Blas, 2004). Consequences of risk-taking can involve either rewards or punishments. Many individuals engage in risk-taking behaviors in hope of potential rewards, but others will often engage in risk-averse behaviors if the potential costs are too high (Anderson & Galinsky, 2006; Platt & Huettel, 2008; Zaleskiewicz, 2001). Risk-taking is of interest to researchers, educators, and policy makers because consequences of risk-taking can lead to unintentional injuries which are one of the leading causes of death and disability in the United States, and are often avoidable (Center for Disease Control and Prevention [CDC]; 2014). In the United States, unintentional injury is the leading cause of death for individuals under the age of 44 (CDC, 2014). Furthermore, while risk-taking can have physical consequences on the victim, risk-taking behaviors can also create emotional and financial consequences for the victim's family and community (National Safety

Council [NSC]; 2015).

Risk-taking behaviors begin in childhood, peak in adolescence, and continue into adulthood (see Boyer, 2006, for review). While negative consequences of risk-taking are often undesirable, risk-taking is an integral part of learning and development. In early childhood, engaging in risk-taking behaviors can assist with the understanding of the world as children learn how to modify or avoid situations in which negative consequences can cause serious harm (e.g., climbing trees, running in traffic; Granie, 2009; Little, 2006). In adolescence and adulthood, risk-taking can help gain peers, social status, or both short- and long-term mates (Peake, Dishion, Stormshak, Moor, & Pfeifer, 2013; Pearson & Michell, 2009; Sylwester & Pawlowski, 2011; Steinberg 2007). Furthermore, individual differences in sensitivity to potential positive and negative consequences of risk-taking can influence the likelihood that an individual is to engage in risk-taking behaviors. The aim of the present research was to understand the relationship between biological sensitivity to stressors, parenting style in childhood, and emotion regulation, to predict risk-taking in young adults.

The *Adaption Calibration Model* (ACM) posits that individuals who are able to attune their biological responses to match the structure and support of their environments will have optimal outcomes (Del Giudice, Ellis, & Shirtcliff, 2011). Optimal developmental outcomes occur for individuals who are highly responsive to their environment when there are no chronic stressors, or who lack response to their environment when there are chronic stressors in their environment (Boyce et al., 1995; Del Giudice et al., 2011). Deleterious outcomes occur when there is an environmental mismatch between biological attunement and environmental stressors (Nederhof & Schmidt, 2012). Stress can affect

the ability of an individual to accurately weigh the potential consequences of the behavior and can therefore lead to risk-taking behaviors (Starcke, Wolf, Markowitsch, & Brand, 2008).

Because stress is expressed through activation of the neuroendocrine system, individual differences in stress sensitivity can be predicted by biological sex (Preston, Buchanan, Stansfield & Bechara, 2007; Van den Box, Harteveld, & Stoop, 2009). When using cortisol as a proxy measure of HPA-axis activation and stress response, males are seen to have greater sensitivity to stressors than females, and overall sensitivity is determined by experience with the stressor (Denson, Creswell, Gransville-Smith, 2011; Dickerson & Kemeny, 2004; Rimmele et al., 2007). Males are also more likely to engage in risk-taking behaviors when having heightened reactions to stressful events while females are more likely to engage in risk-adverse behaviors (Byrnes, Miller, Schafer, 1999; Pawlowski, Atwal, & Dunbar, 2008). While biological sex differences can lead to differences in stress reactivity and risk-taking, life-history factors can calibrate biological sensitivity stressors leaving some individuals more sensitive to stressors and risk-taking than others.

How individuals respond to stress and the extent to which they engage in risk-taking have been shown to be related to early childhood factors, such as the parenting style one has experienced (Lovallo, 2013; Obradovic, Bush, Stamperdahl, Adler, & Boyce, 2010). Across the lifespan, risk-taking behaviors are seen to be higher in individuals reared by parents who adopt more negative parenting strategies (Kapungu, Holmbeck, and Paikoff, 2006). These effects are seen to be magnified in individuals with compounding harsh ecological factors, including: low SES, parental absence, and neighborhood violence

(Belsky, Steinberg, Houts, & Halpern-Felsher, 2010; Brumbach, Figueredo, & Ellis, 2009; Furr-Holden, Milam, Reynolds, MacPherson, & Lejuez, 2012). However, although the literature has provided evidence that parenting style can influence stress-reactivity and related processes, there has been very little laboratory evidence of this effect. The current study seeks to inform this gap in the literature by understanding how past relationship with parents and emotion regulation strategies can predict the relationship between stress reactivity and risk-taking behaviors.

CHAPTER II

REVIEW OF LITERATURE

The Adaptive Calibration of Biological and Social Responses to Environment

Humans are born with the ability to adapt to their social and ecological environments to promote survival and reproduction (Bateson et al., 2004; Bateson & Gluckman, 2012). Early childhood is a pivotal time for our bodies to understand the consistency of our social relationships, and how to best adapt to differences to promote optimal outcomes (Ellis & Boyce, 2008; Hochberg et al., 2011). Optimal outcomes occur for individuals who are able to adapt their biological responses to stressors in ways that match their environment, but worst outcomes occur for individuals who experience a mismatch between their environment and their biological responses to stressors (Boyce et al., 1995; Del Giudice et al., 2011). Most notably, if an individual is in a chronically stressful environment, but has high reactivity to stressors this can put an individual at higher risk for physical and mental disease (Baum, Garofalo, Yali; 1999; Shapero et al., 2013; Steptoe & Feldman, 2001). Further, high sensitivity in chronically stressful

environments can lead to other maladaptive coping behaviors such as alcohol and substance abuse, over/under eating, and gambling (Schreiber, Grant, & Odlaug, 2012). Therefore, if the environment is harsh or unpredictable in early childhood (i.e., child abuse, neighborhood violence, parental absence) it is best for these individuals to develop systems that make them less sensitive to their environment to reduce the likelihood that the individual will be able to adapt to their environment (Lovallo, 2013).

One of the ways that our bodies adapt to our social environments is through the development of the hypothalamic-pituitary-adrenal (HPA) axis (Maniam, Antoniadis, & Morris, 2014). The HPA axis begins to develop in childhood, and therefore the calibration of its processes are highly dependent on environmental factors (Tarullo & Gunnar, 2006). The HPA axis is activated when an individual is exposed to stressors to prepare the body to engage in either a fight or flight response (Kudielka & Kirschbaum, 2005). The activity level of the HPA axis can be indirectly measured through the secretion of the hormone cortisol (Clow, Hucklebridge, Stalder, Evans & Thorn, 2010; Sexbe, 2008). For most individuals, experiencing acute stress is associated with an increased level of cortisol secretion (Dickerson & Kemeny, 2004; Ditzen et al., 2007; Ditzen, Schmidt, Strauss, Nater, Ehlert, & Heinrichs, 2008; Lucas-Thompson & Granger, 2014; Simonovic, Stupple, Gale, & Sheffield, 2016). Because activation of the HPA axis is an indicator to our body that there is a potential threat to our health and safety, cortisol reactivity to stressors is seen to lead to behavioral changes in individuals to promote survival (Dickerson & Kemeny, 2004). Levels of circulating cortisol influences risk-taking propensity by modifying the emotional state of an individual when the opportunity to engage in a risky behavior is presented (Clark, Iversen. & Goodwin, 2001). However,

these behavioral differences are seen to be dimorphic due to stress and sensitivity to environmental contexts.

Cortisol Reactivity to Stressors and Risk-Taking

Research has shown that there are broad associations between cortisol reactivity and risk-taking behaviors. In the case of anticipatory stress and acute psychological stress, males are seen to have increased cortisol reactivity to stressors and increased risk-taking behaviors (Daughters, Gorka, Matusiewicz, & Anderson, 2013; Lighthall, Mather, & Gorlick, 2009; Preston et al, 2007; van den Bos, Harteveld, & Stoop, 2009). Females experiencing high cortisol reactivity to anticipatory or acute stressors are seen to have decreased risk-taking behaviors (Lighthall et al., 2009). The disparities in risk-taking behaviors in response to stressors can not only be used to explain why males are more likely to engage in risk-taking behaviors, but may also explain why males are 2-3 times more likely to die or seek emergency treatment for unintentional injuries when compared to females (CDC, 2014; Harris, Jenkins, & Glasser, 2006; Owens, 2002).

Diverging risk-taking behaviors for males and females have also been found to be related to our evolutionary pasts. When exposed to stressors, both males and females experience high cortisol reactivity, but the appraisal of the stressors is different. For males, stressors may be seen as a form of competition, but for females, stressors are more likely to be seen as a threat (Preston et al., 2007). Due to the evolutionary differences in mating and parenting strategies between males and females, it would be more adaptive for females to engage in more avoidance behaviors when stressed than males to ensure future reproductive and parenting success (Geary & Flinn, 2002; Wang, Kruger, & Wilke, 2009).

Parenting influences on emotion regulation and risk-taking behaviors.

Parents are known to have a large influence on the behavioral and emotional development of their children across the lifespan. In terms of the stress response, parents are thought to influence the adaptation of biological systems and social behavior by potentially acting as buffers to environmental stressors, and modeling behaviors that can be used to combat stressors (Hazel et al., 2014). Depending on parenting style and parent-child relationship quality, parents can either promote resiliency or place their child at greater risk for problems later in development (Masten, Best, & Garmezy, 1990).

Traditionally, researchers have categorized parenting types as: authoritative, authoritarian, and permissive (Baumrind, 1967), and these parenting types are often characterized according to levels of parental warmth and demandingness (Power, 2013). Authoritative parents and authoritarian parents are both seen to be high in demandingness, but authoritative parents are also seen to be high in warmth, whereas authoritarian parents are seen to be low in warmth (Bornstein & Bornstein, 2007; Fan & Zhang, 2014). Permissive parents are low in demandingness and can have varying levels of warmth (Bornstein & Bornstein, 2007). Although traditional research has examined how these three parenting types influence child outcomes, more recent approaches have examined parenting styles by using dimensional models. One of the more commonly used measures of parenting style is Skinner and colleagues' (2005) Six Dimensions of Parenting Style questionnaire. The dimensional model created by Skinner and colleagues (Skinner et al., 2005; see also Robinson et al., 1995) assesses parenting style on three positive aspects (e.g., warmth, structure, and autonomy) and three negative aspects (e.g., rejection, coercion, and chaos). Assessing positive and negative parenting behaviors is

advantageous to strict categorization of parenting style because children are likely to experience some combination of positive and negative interactions with parents, and allowing for positive and negative styles allows for more intraparental variation (Byrd-Craven, Auer, Granger, & Massey, 2012; Kennison & Byrd-Craven, 2015; in press; Kennison et al., 2016).

Across the lifespan, children of parents who engage in more positive parenting strategies have been shown to engage in fewer risk-taking behaviors than children of parents who engage in more negative parenting strategies (Braza et al., 2015; Coolahan, & Nelson, 2002; Rodriguez, Donovan, & Crowley, 2009; Schwebel & Gaines, 2007; Wood & Kennison, 2017). In early childhood, children of parents who engage in more negative parenting behaviors are more likely to engage in physical risk taking behaviors (Wood & Kennison, 2017; Wood & Kennison, 2019). In adolescence, children of parents who engage in more negative parenting engage more frequently in riskier sexual behaviors (e.g., unprotected sex, multiple partners; Raffaelli & Crockett, 2003). These behaviors often continue into early adulthood, as these children are more likely to also drink alcohol in excess, especially in the presence of peers (Borsari, Murphy, & Barnett, 2007; Patock-Peckham & Morgan-Lopez, 2006). Further, the relationship between parenting style and risk-taking behavior may also be associated with financial or social consequences in early adulthood. Specifically, for males, financial risk-taking behaviors in male college students were predicted by negative mother relationships, and ethical risk-taking was predicted by negative father relationships. Ethical risk-taking in females was predicted by both negative father relationships and positive mother relationships (Kennison, Wood, Byrd-Craven, & Downing, 2016).

Because previous research has indicated that risk-taking behaviors can vary based on biological responses to stress, parents can further influence risk-taking behaviors by assisting with the attunement of biological responses to the environment. In infancy, maternal sensitivity is seen to promote biological and hormonal attunement which can assist with the calibration of biological responses (e.g., cortisol) in later life (Byrd-Craven & Clauss, 2019; Hibel, Granger, Blair, Finegood, & Family Life Project Key Investigators, 2015). In later development, individuals who are raised by parents who engage in more negative parenting styles are found to have blunted diurnal rhythms and higher cortisol responses to stressors than individuals from families that adopt more positive parenting styles (Pendry & Adam, 2007; Sheikh et al., 2014; Zalewski, Lengua, Kiff, & Fishedr, 2012).

Importantly, previous research suggests that parenting style needs to be consistent across large periods of development for children to show any differences in cortisol response to stressors. Young infants raised by mothers experiencing postpartum depression have higher cortisol response to stressors while their mothers are experiencing depression symptoms; however, five years later, children of mothers who are no longer experiencing symptoms of depression do not have significantly different cortisol responses than children of non-depressed mothers (Murray, Halligan, Goodyer, & Herbert, 2010). Thus, for individuals who experience negative parenting styles for a long period of development, there may be biological and physiological changes that become entrenched and contribute to long term risk. For example, research has shown that individuals who experience consistently negative parenting styles across childhood have decreased white matter density in the brain, particularly in the areas associated with

emotion regulation and stress response—potentially placing these individuals at risk for emotional and behavioral dysregulation (Sheikh et al., 2014)

Emotion Regulation and Stress Sensitivity

Similar to how parenting style is shown to influence cortisol response to stressors by providing information about the stability and nature of our environment, research has found that sensitivity to stressful situations might also be dependent on individual emotion regulation strategies. Emotion regulation strategies are used to regulate both positive and negative emotions to facilitate health and well-being (Lam, Dickerson, Zoccola, & Zaldivar, 2009; Richardson, Rice & Devine, 2014). By being able to regulate our emotions when we are presented with a stressor we might actually be able to influence our cortisol reactivity to stressors and our risk-taking propensity (Betts, Gullone, & Allen, 2009; Magar, Phillips, & Hosie, 2008).

Although there are an array of different emotion regulation strategies that an individual might engage in to regulate positive and negative emotions, two of the more prevalent emotion regulation strategies discussed in the literature are cognitive reappraisal and expressive suppression (Gross & John, 2003). Cognitive reappraisal techniques are commonly used to inhibit negative emotions by shifting attention to the more positive aspects of the situation (Bradley et al., 2011; Gross & John, 2003; Morris, Silk, & Steinberg., 2007; Robinson, Morris, Heller, Scheeringa, Boris, & Smyke, 2009). Cognitive reappraisal occurs when an individual discusses negative emotions with others to shift their attention to the more positive aspects of the issues causing their negative affect (Kim et al., 2011). For individuals facing stressors, cognitive reappraisal techniques can help an individual modify their behaviors to reduce their psychological or

physiological feelings of stress (Carlson, Dikecligil, Greenberg, & Mujlica-Parodi, 2012). In experimental studies, participants utilizing cognitive reappraisal techniques when facing an acute stressor have been shown to have decreased physiological response (e.g., heart rate, respiration, cortisol), and report higher levels of happiness (Carlson et al., 2012). Further, reappraisal techniques significantly decrease cortisol response to anticipated stressors (i.e., public speaking tasks) than distraction techniques in experimental studies (Priem & Solomon, 2009).

Contrariwise, expressive suppression is used to regulate negative emotions by decreasing the behaviors and affect associated with negative situations by consciously subduing the emotion (Goldin, McRae, Ramel, & Gross, 2008; Gross & John, 2003). Individuals who engage in expressive suppression are at higher risk for developing internalizing and externalizing disorders, such as depression and conduct disorder, respectively (Balan, Dobrea, Roman, & Balazsi, 2017). Due to the biological and emotional dysregulation associated with expressive suppression, individuals who report higher rates of expressive suppression emotion regulation have been seen to engage in higher levels of risk-taking behaviors as a form of maladaptive coping (Magar et al., 2008, 2008; Schreiber et al., 2012). Research has posited that expressive suppressive emotion regulation strategies may require more cognitive effort than cognitive reappraisal, which may lead to increased risk-taking behavior (Betts et al., 2009).

However, it should be noted that the effectiveness of emotion regulation strategies depends on the individual's culture and environment. Whereas expressive suppression is seen to lead to more negative outcomes for individuals in western populations, the outcomes for individuals using expressive suppression and cognitive reappraisal emotion

regulation strategies is reversed in at-risk populations and eastern cultures (Kim et al., 2011). Therefore, it is expected that parents and community members can influence the emotional development of their children as they socialize their children to use emotion regulation strategies that are both culturally and environmentally appropriate.

Current Study

The present study used an experimental approach to understand whether individuals' relationships with parents, emotion regulation strategies, and stress reactivity determine risk-taking behaviors. Previous literature has shown that cortisol reactivity to stressors can predict differences in risk-taking behaviors between males and females (Lighthall et al., 2009). There is also evidence that parents can assist with the development of emotion regulation strategies that can ultimately influence cortisol reactivity to stressors and risk-taking behaviors (Baumrind, 1991; Kim et al., 2011; Magar et al., 2008; Tarullo & Gunnar, 2006). However, although research has shown that both parenting style and emotion regulation can separately influence stress reactivity and risk-taking behaviors, there has been no laboratory experiment to examine how parenting and emotion regulation factors can interact with cortisol reactivity to predict risk-taking behaviors.

To address the gap in the literature, participants in the present study were randomly assigned to experience either an acute stressor or a non-stressor task prior to engaging in a computerized risk-taking task. Biological reactivity to stressors was measured via salivary cortisol which was taken prior to the stressor, twenty minutes after the stressor, and forty minutes after the stressor. Based on prior literature, the experiment was designed to address three central hypotheses:

First, participants in the stress condition were expected to have significant differences in risk-taking when compared to participants in the control condition. Further, it was expected that there would be a significant interaction for group and sex effects on risk-taking behaviors such that females in the experimental condition would have decreased risk-taking than females in the control condition, and males in the experimental condition would have increased risk-taking than males in the control condition.

Second, for the experimental group, it was expected that differences in risk-taking would be explained by variance in cortisol reactivity such that it was expected that male participants in the stress condition who had increased cortisol reactivity would have increased risk-taking behaviors, and female participants in the stress condition who had increased cortisol reactivity would have decreased risk-taking behaviors (Byrnes et al., 1999; Lighthall et al., 2009; Pawlowski et al., 2008).

Third, individual differences in risk-taking due to stress and cortisol reactivity were expected to be related to early childhood experiences with parents and emotion regulation strategies. It was expected that higher levels of parental warmth during childhood and cognitive reappraisal would interact with cortisol reactivity to decrease risk-taking behaviors. Similarly, it was expected that lower levels of parental warmth during childhood and higher levels of expressive suppression would interact with cortisol to increase risk-taking behaviors (Belsky et al., 2010; Magar et al., 2008).

CHAPTER III

METHODOLOGY

Participants

One-hundred fifty-six healthy young adults (79 male, Mage = 19.59 years, age range:18-32 years) were recruited to participate in this study. Those who participated were compensated with course credit and were placed in a drawing to win \$150. The ethnicity of the participants was 72.4% Caucasian; 4.5% African American, 7.4% Hispanic; 2.6% Asian; 3.2% Native American; 14.7% belonged to more than one ethnicity; 0.6% reported being from a non-reported ethnicity. Participants also reported coming from a variety of SES backgrounds with 10.4% reporting coming from a family that earned less than \$24,999; 9.6% came from families that earned between \$25,000 and \$49,999; 18.6% came from families that earned between \$50,000 and \$74,999; 11.5% came from families that earned between \$75,000 and \$99,999; 43.6% came from families earning more than \$100,000 annually.

Material

Multiple measures were used in the study to assess the following variables: parenting style experienced in childhood, emotion regulation strategies, risk-taking behaviors, and self-reported changes in stress.

Parenting Style Experienced in Childhood. Participants completed the Six Dimensions of Parenting Style Questionnaire (SDPS) created by Skinner, Johnson, & Snyder (2005) to assess how parenting style influenced emotion regulation, stress reactivity. The questionnaire was modified for participants to respond to questions for both relationships with their mother and with their father on a 7-point Likert-scale in which “1” indicates “not at all true” and “7” indicates “very true.” The questionnaire is composed of six separate dimensions of: warmth (“My mother/father knew a lot of what was going on for me”), rejection (“My mother/father didn’t understand me very well”), structure (“My mother/father made it clear to me what he expected from me”), chaos (“My mother/father let me get away with thing she really shouldn’t have allowed”), autonomy support (“My mother/father encouraged me to express my feelings even when they were hard to hear”), and coercion (“My mother/father felt like he had to push me to do things”).

In prior research the three individual facets (comprised of 23 items) of warmth, structure, and autonomy were collapsed to create a “positive” relationship category (Byrd-Craven et al., 2012; Kennison & Byrd-Craven, 2015; Kennison et al., 2016). The three individual facets (comprised of 24 items) of rejection, chaos, and coercion were collapsed to create a “negative” relationship category. A single composite score reflecting overall parenting style was created from the positive and negative parenting scores by

subtracting the average scores of the negative style category from the average scores of the positive style category. Positive scores indicate more positive parenting styles and negative scores indicate more negative parenting styles. The two factors created were seen to have high internal reliability, *mother* $\alpha = .973$, *father* $\alpha = .975$.

Emotion Regulation Strategies. The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) was used to assess the participants self-reported level of emotion regulation. The ERQ is a 10 item questionnaire on a 5-point Likert-scale in which a “1” indicated that the individual “strongly disagreed” with the statement and a “5” indicated that they “strongly agreed” with the statement. The ERQ includes questions pertaining to cognitive reappraisal (“When I’m faced with a stressful situation, I make myself think about it in a way that helps me stay calm.”) and expressive suppression (“When I am feeling negative emotions, I make sure not to express them”). The *cognitive reappraisal* and *expressive suppressive* factors each created by averaging ($\alpha = .797$ and $.808$, respectively) the items in each group.

Risk-Taking Behaviors. To measure observed risk-taking behaviors, computerized version of the automatic Balloon Analogue Risk-Task (BART; Pleskac, Wallsten, Wnag, & Lejuez, 2008) using Millisecond Software. This task is a modified version of the manual BART task designed by Lejuez et al., 2002. For the automatic BART, the participant is provided 30 trials in which they are given the option to pump the balloon for a reward of \$.05 or collect their winnings. For each pump in which the balloon does not pop, the participant will earn \$.05, but if the balloon pops, the participant will lose any collective winnings for the popped balloon but will retain the money earned from un-popped balloons. The automatic BART differs from the manual

BART because in the automatic BART participants are required to provide their desired number of pumps in a text box prior to the balloon inflating; in the manual BART participants must make individual clicks on the “pump” button and stop inflating either by the balloon exploding or by hitting “collect earnings” if they wish to earn the payout prior to the balloon popping.

For scoring the BART, the higher average number of pumps indicated higher levels of risk-taking and impulsivity. In the traditional manual-BART it is suggested for researchers to use the adjusted average number of pumps so that the number of desired pumps is no longer constrained by explosion points (Bornovalova et al., 2009; Lighthall et al., 2009; Tull et al., 2009). However, for the automatic BART, participants provide their desired number of pumps prior to any pumps being added to the balloon and therefore the desired number of pumps on a given balloon is not influenced by explosion points and therefore the total or average unadjusted number of pumps can be used for analyses (Daughters et al., 2013; Euser, Evans, Greaves-Lord, Huizink, & Franken, 2013; Euser, van Meel, Snelleman, & Franken, 2011; Pelskac et al., 2008; Vaca et al., 2013; Young & McCoy, 2018). For the present study, all reports are based on the average number of pumps provided across all thirty balloons.

Self-Reported Stress Reactivity (Manipulation Check). To assess the effectiveness of the experimental manipulation participants were provided the State-Trait Anxiety Inventory-Short (STAIS; Marteau & Bekker, 1992) and State-Trait Anger Inventory (STAXI; Spielberger, 1988). The STAIS was a 6 item scale and the STAXI was a 14 item scale, and both were rated on a 7-point Likert-Scale in which higher scores indicated higher anxiety. Responses were collapsed across questionnaires to create one factor and

participants completed this questionnaire prior to ($\alpha = .815$) and after the experimental manipulation ($\alpha = .818$).

Procedure

Participants were recruited through a University SONA system at a large university in the Southwestern United States. Exclusion criteria for the study included consumption of alcohol, medication, illicit substances, smoking, and exercising one hour prior to participation. These exclusion criteria were included to ensure that there was no contamination to the salivary samples. No participants were excluded based on these criteria.

Participants arrived at the lab between 1230h-1500h to ensure that the salivary samples are not influenced by diurnal cortisol rhythms. When participant first arrived to the lab they completed the demographic, SDPS, and ERQ questionnaires to allow them time to habituate to the laboratory environment. Questionnaires took approximately 30 minutes. After the habituation period, participants provided salivary samples to be used to establish baseline salivary cortisol levels.

Next, participants were randomly assigned to the experimental stress or control no-stress group. Using modified protocols from the Trier Social Stress Task (TSST; Kirschbaum, Pirke, Hellhammer, 1993) both conditions were provided ten minutes to prepare a five-minute speech indicating why they should be hired for a job. Participants in the experimental group were told that the speech would be recorded using a web-camera on the laboratory computer and would be reviewed by a panel of judges. Participants in the experimental group were required to use the whole five minutes for their presentation; if a participant stopped, they were told to continue after a 20 second

period of silence. Next, participants in the experimental group engaged in a five-minute subtraction task where they were asked to subtract from 1,022 to 0 in increments of 13 as quickly as possible, having to re-start when they messed up. Control group participants were only required to prepare a speech, but they were instructed they would not present the speech, and they did not engage in the subtraction task.

After completing a manipulation check, participants were then to wait for twenty minutes to allow for the activity in the HPA axis to peak and allow for larger secretion of cortisol (Hankin, Badanes, Abela, & Watamura, 2010). After providing a secondary salivary sample, participants engaged with the computerized BART task. Twenty minutes after the second salivary sample, participants provided a third salivary sample to examine the recovery rate of participant's stress response. Once the samples had been collected and all surveys have been completed, participants were debriefed about the manipulation and released from the study.

Salivary Cortisol Collection, Assay and Analysis. Three 1.8 ml saliva samples per participant were collected, stored, and assayed at Oklahoma State University. Saliva samples were collected in cyrovial tubes using a "passive drool" technique. Samples were stored at -20C prior to assay. Assays were conducted using commercially available salivary cortisol enzyme immunoassay kits from a national research supply company (Salimetrics, State College, PA). Assays were conducted on 96-well plates using double control settings. After assay, one participant was removed from analyses because results were inconclusive. All salivary assays remaining in analyses had CV values under 12.75% and the final average CV value of assayed samples was 3.19%.

During analysis, raw cortisol data was found to be positively skewed for all three time-points. Raw values were then transformed using the square root transformation method, and can be found in *Table 1*. Raw values were used to calculate Area Under the Curve (AUC) in respect to ground (AUC_G) and in respect to increase (AUC_I) using the formulas provided by Prussner et al. (2003). Area under the curve with respect to ground was found to have a significant positive skew and was later transformed.

Data Analysis

All statistical analyses were conducted using SPSS 24.0 software. Analyses conducted to review the results of this experiment include two-way ANOVA, two-way ANCOVA, and linear multiple and hierarchical regression. All results were conducted while controlling for wake-time and medication usage.

Manipulation Check

Participants in the stressor group reported feeling more stress after the experimental manipulation than participants in the control group, $F(1, 62) = 34.742, p < .001$. (See *Figure 1*). Females in the experimental group reported feeling more stress after the experimental manipulation than males $F(1, 62) = 13.806, p < .001$.

CHAPTER IV

RESULTS

Data for six participants who did not complete the entire study were excluded from subsequent analyses, and one participant was excluded from the analysis due to inconclusive results of cortisol assay reports. Further, to determine differences based on the manipulation, control participants who had unexplained increases in cortisol response without exposure to a stressor (i.e., greater than a 10% increase in cortisol secretion), and experimental participants who did not show a cortisol response to the stressor (i.e., less than 10% increase in cortisol secretion), and experimental participants who did not show a cortisol response to the stressor (i.e., less than 10% increase in cortisol secretion) were excluded from statistical analyses (Glienke & Piefke, 2017; Stock & Merz, 2018). The final sample included 47 males (26 experimental condition; $M_{age} = 19.85$, $SD = 2.25$) and 37 females (17 experimental condition; $M_{age} = 19.41$, $SD = 1.98$). Listwise deletion was used in the case of missing data for any of the self-report measures. A summary of all descriptive statistics for the variables examined are included in *Table 2*, and a

summary of all Pearson correlational data for all variables examined in the present study are included in *Table 3*.

Effect of Stress Condition on Risk-Taking

To test the first hypothesis to understand the relationship between sex and experimental stress condition on risk-taking behaviors, a two-way ANOVA was conducted. The second ANOVA analysis assessed risk-taking differences by experimental group and sex using the average wanted balloons in the BART, but yielded no significant main effects or interaction effects. In summary, results of this analysis did not support the first hypothesis because there were no differences in risk-taking between men and women, or between the experimental or control group (*Table 4*).

Effect of Cortisol Reactivity on Risk-Taking

To test the second hypothesis, a second set of analyses were conducted to determine if cortisol reactivity could influence risk-taking behaviors. To test this hypothesis, an ANOVA analyses was first conducted to establish that the experimental manipulation led to differences in cortisol activity. There was a significant main effect for group for both AUCI ($F[1, 77] = 30.719, p < .001, \text{partial } \eta^2 = .285$) and AUCG ($F[1, 77] = 4.417, p = .039, \text{partial } \eta^2 = .054$), confirming that participants in the experimental condition had higher cortisol reactivity than participants in the control condition. No sex differences in cortisol reactivity were found (*Table 5*).

Next, a linear multiple regression analysis was conducted within the experimental group to determine if variation in cortisol reactivity could influence risk-taking behaviors for males and females. For the model, the BART score was the dependent variable. The independent variables included participant sex, cortisol reactivity, and an interaction

between sex and cortisol reactivity. Results were significant in the model using AUCG (Model 1; $F[5, 36] = 2.514, p = .047$) and were marginally significant in the model using AUCI (Model 1; $F[5, 36] = 2.247 p < .070$). Results from Model 1 indicate that there was a marginally significant effect for AUCG on risk-taking behaviors ($\beta = -.820; p = .091$); however, there was no evidence of an interaction between cortisol and sex, suggesting that the relationship between cortisol and risk taking did not vary according sex (*Table 6*). In summary, the second hypothesis was partially supported because although the significant linear multiple regression model suggests that cortisol response can have small influences on risk-taking propensity following exposure to a stressor, the analysis only yielded marginal results for AUCG. Thus, the following analyses testing the third hypothesis cannot be definitively supported and should be considered exploratory to determine whether parenting style or emotion regulation strategy may play a role in risk taking behaviors.

Effect of Parenting Style, Emotional Regulation Strategy, and Cortisol Response on Risk-Taking Behaviors

To test the third hypothesis, a series of linear regressions were conducted within the experimental group to examine if the marginally significant differences in risk-taking due to AUCG could be exacerbated by early childhood experiences with parents and emotion regulation strategies. The first regression model was conducted to determine whether parenting styles interacted with cortisol reactivity to stressor to predict risk-taking behaviors. In the model, risk-taking was the dependent variable and the independent variables included participant sex, cortisol reactivity, mother style, father style, and two interactions between the two parenting styles and cortisol reactivity. The

present model accounted for 7.8% of the variance in risk-taking scores, and the model was not significant and there were no individual significant predictors in the model, $F(8,34) = 1.443$, $p = .215$. These results indicate that parenting style experienced in childhood was not significant a predictor of risk-taking behavior, and parenting style did not interact with cortisol reactivity to influence risk-taking propensity for participants in the experimental group after exposure to a stressor (*Table 7*).

The second regression model analysis was conducted to determine whether emotion regulation strategies interacted with cortisol reactivity to stressor to predict risk-taking behaviors. In the model, risk-taking was the dependent variable and the independent variables included participant sex, cortisol reactivity, cognitive reappraisal, expressive suppressive, and two interactions between the two emotion regulation strategies and cortisol reactivity. The present model accounted for 8.5% of the variance in risk-taking scores, and the model was not significant and there were no individual significant predictors in the model, $F(8,33) = 1.474$, $p = .204$. These results indicate that emotion regulation strategies were not significant predictors of risk-taking behavior, and emotion regulation strategy did not interact with cortisol reactivity to influence risk-taking propensity for participants in the experimental group after experiencing a stressor (*Table 8*). In summary, the present study cannot find support for the second study because neither of the two models in the analysis were significant indicating that individual differences in risk-taking behavior were not further explained by either parenting style or emotion regulation strategy.

CHAPTER V

DISCUSSION

The present study was designed to understand how past relationship with parents and emotion regulation strategies can predict the relationship between stress reactivity and risk-taking behaviors. Based on the previous literature, the present study was designed to test three main hypotheses: 1) participants who experienced a stressor would have changes in risk-taking behavior and these differences would be influenced by cortisol reactivity and sex, 2) that cortisol reactivity would predict differences in risk-taking for males and females in the experimental group and 3) the relationship between cortisol reactivity and risk-taking would be attenuated by parenting style and emotion regulation strategies. Results of the present study found trends that cortisol response to stressor can predict decreased risk-taking behavior, but these effects were not found to be different based on sex. However, the results did not support the second hypothesis that cortisol reactivity interacted with parenting style or emotion regulation to influence risk-taking behaviors.

The marginal differences in risk-taking as a function of cortisol increase could indicate support for prior research that has found that risk-taking behaviors change after experiencing a stressor (Daughters et al., 2013; Lighthall et al., 2009; Preston et al, 2007; van den Bos et al., 2009). However, because there were no interactions found between sex and cortisol in predicting risk-taking behavior, the present study does not support the previous literature that has found that increased cortisol reactivity increases risk-taking in males, but decreases risk-taking in females (Lighthall et al., 2009). Because previous research examining the role of stress and cortisol on risk-taking behaviors has found significant effects for stress and sex using pain-related stressors (i.e., cold-pressor task), it may be the case that only certain stressors can elicit changes in risk-taking behaviors, which is suggestive of both limitations and future directions for the present study.

Limitations

There are several limitations for the present study. First, the present study is limited by a small sample size due to the number of participants excluded from the study for being either unexplained nonresponders (i.e., participants who did not have a stress response to the experimental stressor) or unexplained responders (i.e., participants who had a stress response to the control condition). By having a small sample size, any potential significant effects of parents and emotion regulation might have gone undetected because the sample was underpowered. Further, the issues seen regarding the unexplained responses for individuals in the experimental and control groups might have been due to the choice in experimental manipulation.

Second, in the present study participants engaged in a modified Trier Social Stress Task (TSST). Use of the TSST could potentially be a limitation for the present study

because all data was collected in the department of psychology at a large university in which many labs use modified versions of the TSST. There is a possibility that participants were aware of the nature of the stressor prior to participating in the study. Secondly, there is also evidence that for college students who are high achieving, use of an experimental manipulation such as the TSST might not be an effecting way to activate a stress response as the manipulation does not provide greater stress than a task they are asked to do on a daily basis (i.e., class presentations; Richardson et al., 2014).

Further, because the present study relied on a convenience sample of college students, variation in parenting styles and emotion regulation strategies might have been constrained with little variance within the sample. In the present study, the majority of college students reported having both mothers and fathers who engaged in more positive parenting styles. Therefore, the present study might be missing significant effects for parenting style and other factors because the lack of variation in reported parenting styles between participants. Potentially, if the sample had been more diverse the present study would not only have been able to find significant effects for parenting, but the results would also be able to generalize to more of the general public.

Lastly, the decision to use the balloon analogue risk-taking task (BART) could have influenced the ability to find differences in risk-taking behaviors based on biological and social factors. Inability to find differences in BART performance could be due to the fact that research has found that large sample sized are needed to find small effects for the BART (Lauriola, Panno, Levin, & Lejuez, 2013). This could indicate that the present study might have been underpowered to find a significant result in the BART task. By using the BART, the present study might have been limited due to framing effects based

on the instructions and financial compensation for participants, which have been seen to influence BART task engagement (Benjamin & Robbins, 2007). The present study used the standard instructions for the BART, however, because participants were students from a university research pool receiving course credit instead of financial compensation this might have decreased the saliency of the task because all earnings were hypothetical. By not having true, financial incentive, participants might not have been actively motivated to activate the reward related processes associated with risk-taking and the BART task. Because of these limitations with the BART task, future research should examine if the risk-taking differences can be found across levels of cortisol reactivity, parenting style, and emotion regulation strategy with larger samples sizes and financial compensation. In addition, it might be of benefit to assess observational risk-taking with another empirical measure of risk-taking to determine if risk-taking and reward sensitivity are context dependent.

Implications and Future Directions

The present study found trends that an individual's risk-taking propensity may be modified by cortisol reactivity (AUC_G) after experiencing a stressor. This could have potential implications for research using computerized measures of risk-taking behaviors by indicating that some individual variation might be due to increases in cortisol—even if the researcher is not intending to induce a stress response. Therefore, future studies using tasks like the BART should be careful to avoid creating stressful environments (i.e., novel environments) that could confound the results of future risk-taking research.

However, because the present study did not find that the relationship between cortisol and risk-taking was influenced by parenting styles or emotion regulation

strategies when using a college sample, future research should utilize community samples to understand how greater variation in familial and community processes can influence risk-taking behaviors. By using a community sample, future research would have access to a larger sample sizes, and a community sample might not be as desensitized to experimental stress manipulations as members of a college age sample. Use of a community sample would also allow for greater understanding of how community and cultural dynamics shape the influence of biological stress response systems and subsequent behaviors, and increase external validity of results.

Lastly, because the present study did not find and support that parenting style or emotion regulation interacted with cortisol reactivity to predict differences in risk-taking behavior, future research should examine other factors that might interact with cortisol reactivity to predict risk-taking behaviors. Previous literature has indicated that an individual's sensitivity to stressors might be influenced by genetic variation of the oxytocin receptor gene OXTR rs53576 (Auer et al., 2015; Tost et al.2010). Research has largely found that individuals who are G-homozygous allele carriers have significantly lower levels of self-reported and physiological stress reactivity than A-allele carriers (Auer et al., 2015; Rodrigues et al., 2009). Because the present study found marginal differences in risk-taking behavior based on AUC_G, future research examining cortisol interactions with OXTR rs53576 might be able to provide a more detailed picture of the biological predictors of risk-taking propensity. However, because the relationship between stress sensitivity and OXTR rs53576 has been found to be reversed for individuals from eastern cultures or at-risk populations, future research examining

cortisol and OXTR rs53576 could potentially extend implications for the *Adaptive Calibration Model* (Kim et al., 2011).

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APPENDICES

Table 1. Means of Raw and Transformed Cortisol Over Time in Males and Females in Stress and No-Stress(NS) Groups

	Time 1		Time 2		Time 3	
	R	T	R	T	R	T
<i>Females – Stress</i>	.114	.329	.185	.403	.157	.380
<i>Females-NS</i>	.175	.404	.150	.370	.187	.384
<i>Men – Stress</i>	.096	.302	.210	.441	.168	.364
<i>Male – NS</i>	.143	.370	.117	.334	.122	.341

Table 2. Means and Significant Differences for Variables of Interest

	Females				Males				N	Between Sex		Between Stress-Group		Total	
	No-Stress		Stress		No-Stress		Stress			F	p	F	p	F	p
	M	Std. D	M	Std. D	M	Std. D	M	Std. D							
Mother Style	3.87	1.39	4.17	2.07	4.19	1.43	3.52	1.46	84	.219	.641	.282	.597	1.952	.166
Father Style	3.66	1.50	3.26	2.41	4.00	1.45	3.37	1.57	84	.341	.561	1.857	.177	.084	.773
Cog. Reap.	3.51	.60	3.67	.82	3.61	.86	3.51	.67	83	.024	.878	.034	.854	.612	.436
Exp. Supp.	2.63	1.16	2.49	1.02	2.85	1.11	3.14	.83	83	3.780	.055	.119	.731	.932	.337
BART	56.88	10.62	53.90	18.09	58.94	12.74	65.44	9.89	84	5.798	.018*	.387	.536	2.827	.097
AUC _G	2.72	.93	2.91	.95	2.46	.52	3.05	.77	84	.112	.738	4.87	.030	1.365	.246
AUC _I	-.51	2.61	2.53	4.61	-.86	1.25	4.13	3.52	84	.795	.375	32.647	<.001* **	1.93	.169
Self-Reported Stress	.04	.55	1.57	1.23	-.07	.50	.41	.52	66	13.806	<.001* **	34.74	<.001* **	9.33	.003

Note: * $p < .05$, *** $p < .001$; Wake-up time and medication use were not controlled for in comparative analyses between groups.

Table 3: *Correlational Values for All Variables of Interest*

	1.	2.	3.	4.	5.	6.	7.	8.
1. BART	--	-.340	-.132	-.187	-.155	.098	.115	-.046
2. Self-reported Stress	.053	--	0.122	0.282	-.184	-.483**	-0.051	0.056
3. AUC _G	.284	.352*	--	.784***	.205	.005	-0.029	-0.019
4. AUC _I	.121	0.265	.702***	--	.203	.025	-0.019	-0.039
5. Mother Style	-.026	.076	-.027	-.374**	--	.711***	.038	.184
6. Father Style	.013	.074	.068	-.224	.835***	---	.356*	0.096
7. Cognitive Reappraisal	.030	0.106	-0.068	-0.280	.225	.379**	--	0.041
8. Expressive suppression	.824	0.001	-0.109	0.148	-.15	-.293*	-.0126	--

Note: Values at the top of the matrix are female responses, values at the bottom of the matrix are male responses; *** $p < .001$, ** $p < .01$, * $p < .05$

Table 4. *Summary of ANOVA results for Risk-Taking as a function of Sex and Experimental Condition*

Dependent	Source	Mean Sq.	F	Sig	eta
Average Wanted Pumps	Group	41.910	.262	.610	.003
	Sex	133.169	.834	.346	.011
	Group*Sex	401.031	2.511	.117	.032

Note: Female = 0

Table 5. Summary of ANOVA results for Cortisol Change as a function of Sex and Experimental Condition

Dependent	Source	Mean Sq.	F	Sig	eta
AUC _G	Group	2.926	4.417	.039*	.054
	Sex	.049	.073	.787	.001
	Group*Sex	.894	1.349	.249	.017
AUC _I	Group	322.231	30.719	>.001***	.285
	Sex	1.976	.188	.666	.002
	Group*Sex	17.268	1.646	.203	.021

Note: *p < .05; *** p < .001

Table 6: Multiple regressions for risk-taking change as a function of cortisol reactivity and sex for individuals in the experimental condition.

Predictors	Model 1				Model 2			
	B	SE	β	Sig.	B	SE	β	Sig.
Cortisol	-14.396	8.288	-.820	.091 ⁺	-2.321	1.748	-.635	.192
Sex	-14.745	17.842	-.497	.414	5.240	7.591	.117	.494
Sex*Cortisol	7.732	5.247	1.086	.149	1.153	1.111	.531	.306
ΔR^2	.153*				.129 ⁺			
F	2.514* ($p = .047$)				2.247 ⁺ ($p = .070$)			

Note: In Model 1 AUC_g was used as the measure for cortisol change, In Model 2 AUC_I was used as the measure for cortisol change; For sex, female = 0; ⁺ $p < .10$; * $p < .05$

Table 7: Multiple regressions for risk-taking change as a function of cortisol reactivity, parenting style, and sex for individuals in the experimental condition.

Predictors	B	SE	B	t	Sig
Sex	8.576	6.614	.289	1.297	.203
Cortisol	.798	8.862	.045	.090	.929
Mother Style	2.365	7.160	.280	.330	.743
Father Style	1.443	9.177	.189	.157	.876
Cortisol*Mother Style	-.986	2.203	-.430	-.430	.657
Cortisol*Father Style	.093	3.189	.039	.039	.977
ΔR^2			.078		
F			1.443	($p = .215$)	

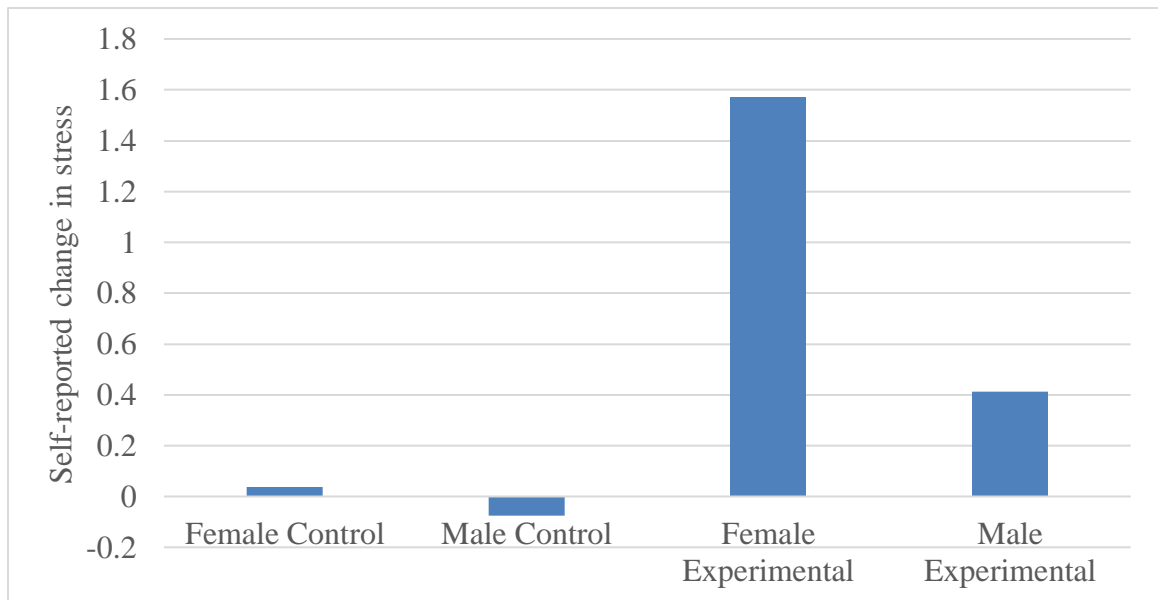
Note: AUC_g was used as the measure for cortisol change; For sex, female = 0

Table 8: *Multiple regressions for risk-taking change as a function of cortisol reactivity, emotion regulation, and sex for individuals in the experimental condition.*

Predictors	B	SE	β	t	Sig
Sex	13.045	6.788	.436	1.922	.063 ⁺
Cortisol	-24.152	19.073	-1.372	-1.266	.214
Cog. Reap.	-1.803	14.490	-.088	-.124	.902
Exp. Supp.	-17.109	12.002	-1.104	-1.425	.163
Cortisol*CR	.629	4.782	.125	.132	.896
Cortisol*ES	5.742	4.108	1.620	1.398	.171
ΔR^2			.085		
F				1.474 ($p = .204$)	

Note: Cog. Reap = Cognitive Reappraisal; Exp. Supp. = Expressive Suppression; AUC_g was used as the measure for cortisol change; For sex, female = 0; ⁺ $p < .10$

Figure 1: *Self-reported changes in stress after experimental manipulation*



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