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A Multi-Systemic Analysis of Infant Stress Reactivity

Maria Lauer
Lehigh University, marialauer2@gmail.com

A Multi-Systemic Analysis of Infant Stress Reactivity

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Maria Lauer

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_____
Date

_____
Susan S. Woodhouse, Ph.D.
Associate Professor
Counseling Psychology

_____
Accepted Date

Committee Members:

_____
Grace I. L. Caskie, Ph.D.
Associate Professor
Counseling Psychology

_____
Patricia Manz, Ph.D.
Associate Professor
School Psychology

_____
Deborah Laible, Ph.D.
Professor
Psychology

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Abstract

Understanding the human stress response is important because individual differences in stress response have been linked with psychological outcomes (Allwood et al., 2011). Theorists have suggested that simultaneously examining the two main components (the locus-coeruleus, norepinephrine/sympathetic nervous system (LC-NE/SNS) and the hypothalamic pituitary adrenal (HPA) axis) of the stress response system would be the most appropriate approach to further our understanding (Bauer et al., 2002). Yet most research has focused on only one component of the stress response. Further, although behavioral reactivity has been conceptualized as an important component of the overall stress response, few investigations include markers of behavioral reactivity. The purpose of the present study was to 1) identify homogenous patterns of stress reactivity across both the HPA axis and the LC-NE/SNS, 2) identify homogenous patterns when including a behavioral marker, and 3) examine whether patterns of reactivity are linked with later socio-emotional outcomes. Participants included 219 infants and their mothers who participated in a larger study of maternal caregiving. When the infants were approximately 6 months old, provided saliva samples shortly after arriving in the lab and then again, following a stressor task. Saliva samples were assayed for Cortisol (HPA) and Alpha Amylase (sAA). Infants were 37.4% Black/African American, 24.2% as biracial/multi-racial, 16.4% as White/European American, 14.6% as Latino/Hispanic, 1.4% as Asian/Asian American. Fit statistics of a Latent Profile Analysis supported a three-class solution for the LE-NE/SNS and HPA only model. Most salient was the identification of two classes with reactivity in only one system. A large heterogeneous class, with reactivity in both systems, was also identified. An additional, exploratory LPA was conducted on the infants identified as having reactivity on both systems. Again, three classes emerged, two of which were again characterized

by asymmetrical reactivity. The addition of a behavioral marker for stress reactivity did not result in significant changes to infant classification. No statistically significant class differences on socio-emotional outcomes emerged, although class differences in externalizing behaviors approached significance.

Chapter I

Introduction

Understanding the human stress response is important because accumulating evidence shows that individual differences in stress response are associated with outcomes such as psychological wellbeing and physical health (Allwood, Handwerger, Kivlighan, Granger, & Stroud, 2011; El-Sheikh, Arsiwalla, Hinnant, & Erath, 2011; El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008; Keller & El-Sheikh, 2009; Nederhof, Marceau, Shirtcliff, Hastings, & Oldehinkel, 2015). The physiological, neuroendocrine, and behavioral changes that occur in order to respond to a stressor (i.e., the *stress response*) are facilitated by activation of the stress response system (Chrousos, 2009; Chrousos & Gold, 1992).

The stress response system is composed of two main components, the locus-coeruleus, norepinephrine/sympathetic nervous system (LC-NE/SNS) and the hypothalamic pituitary adrenal (HPA) axis (Chrousos, 2009; Chrousos & Gold, 1992). Although a significant number of research studies have examined the two components of the stress response system separately, theorists have suggested that simultaneously examining both components of the stress response system would be the most appropriate approach to further our understanding of this system (Bauer, Quas, & Boyce, 2002). In fact, researchers have found links between particular patterns of activity across both components of the stress response system and emotion regulation, as well as social functioning (Granger, Kivlighan, El-Sheikh, Gordis, & Stroud, 2007; Kirschbaum & Hellhammer, 1994). Still, the body of research examining both components of the stress response simultaneously remains scant. Moreover, much of the existing research has focused on childhood and adolescence, with very little attention to the stress response system in infancy. Finally, no studies to date have examined links between the stress response system, including

both SNS and HPA, and externalizing and internalizing behaviors in infancy, despite identification of such links in research with children and adolescents (e.g., El Sheikh et al., 2008) and the importance of understanding the stress response system across development. The purpose of the present study is to examine links between the activity of the stress response system following a stressful frustration task with 6 month old infants and mother-rated internalizing and externalizing behaviors at 12 months. This study addresses limitations in previous research by analyzing response patterns across both components of the stress response system via a person-centered approach, by focusing on infants, and by examining links to externalizing and internalizing behaviors as outcomes.

The Two Components of the Stress Reactivity System

A stimulus in the environment that is perceived as posing a threat to survival or wellbeing triggers a set of complex and interrelated responses within the brain and the endocrine system, the sum of which can be considered an organism's stress response (Boyce & Ellis, 2005). The HPA axis and the LC-NE/SNS (also known as the Sympathetic-Adrenal-Medullary system or SAM) are responsible for creating physiological and behavioral change in order to deal with the perceived threat (Chrousos & Gold, 1992).

LC-NE/SNS. The LC-NE/SNS is considered the first line of response to distress and, when it becomes activated, acts upon various organs on the body to elicit changes and respond to the stressor. This activation redirects the individual's attention towards the threatening stimulus and increases cognitive (e.g., alertness, cognitive sharpness) and physical (e.g., respiration, heart rate) resources to deal with the threat. The result of this activation is a behavioral response, often conceptualized as *fight or flight* (Chrousos & Gold, 1992).

The locus coeruleus (LC) system begins in the brainstem and extends through a dense system of neurons to the amygdala, hippocampus, the mesolimbic dopamine system, and the

medial prefrontal cortex (Boyce & Ellis, 2005). Activation of the LC results in the release of norepinephrine (NE) throughout the brain. NE, then, acts on hypothalamic centers, which in turn activate and regulate the sympathetic nervous system (Boyce, & Ellis, 2005). Activation of the LC-NE/SNS can be measured via a non-invasive salivary enzyme, salivary alpha amylase (sAA, Granger et al., 2006).

HPA Axis. Activation of the stress response system may also produce activity in the HPA axis. The HPA axis aids in survival when environmental conditions are extreme, that is, when the necessary resources to deal with the threat exceed available resources (Gunnar & Cheatham, 2003). HPA reactivity ensures short-term survival by redirecting resources to deal with the immediate threat (e.g., creating more readily available energy by metabolizing fats and protein). Energy, then, is redirected away from long-term survival processes (such as reproduction, digestion, and immune functioning; Gunnar & Cheatham, 2003). Additionally, the HPA axis reacts more slowly to the perceived stress due to the need to produce glucocorticoids on demand, making it a second line of defense (Axelrod & Reisine, 1984). Its activity is considered a passive response to distress, which is often characterized by behavioral avoidance, emotional distress, and a sense of lost control (Henry, 1992). The end product of HPA axis activation is cortisol, which can be measured non-invasively via saliva and has long been studied in relation to stress reactivity (see Gunnar & Cheatham, 2003).

Research has shown that both the LC-NE.SNS and the HPA axis systems are each linked to mental health. For example, research on SNS has shown that hyperactivity of the brain's norepinephrine producing centers is linked with panic disorder (Southwick, Bremer, Rassmusson, Morgan, Arnsten, & Charney, 1999) and that low resting heart rate is linked with anti-social behaviors in children and adolescents (Keller & El-Sheikh, 2009). Similarly, research

on the HPA axis indicates that individual differences in the activity of this system are linked with disruptive behaviors (Burke, Davis, Otte, & Mohr, 2002), externalizing behaviors (Shirtcliff, Granger, Booth, & Johnson, 2005), and anxiety (Essex, Klein, Cho, & Kalin, 2002). Although both components of the stress reactivity are independently linked to mental health outcomes, examining both systems concurrently would offer a more complete picture of the human stress response, as both systems are simultaneously involved in responding to threat (Chrousos, 2009). Independent examination of each component ignores the unique contributions of the other component as well as potential interactions between the two components, obscuring the full stress response and diminishing researchers' ability to understand links between physiological arousal and psychopathology (Bauer et al., 2002).

Investigating the Stress Response System via Multi-system Analysis

It makes sense to examine the two components together because evidence suggests that although the two stress response subsystems are anatomically different, they are functionally linked (Chrousos & Gold, 1992). Neurons that express corticotropin-releasing hormone (CRH, a hormone involved in the activity of the HPA axis) from the amygdala project into the locus-coeruleus directly, increasing the rate of firing of NE releasing neurons. Similarly, firing of neurons that release NE may increase CRH production (Habib, Gold, & Chrousos, 2001). The neural and functional relations between the LC-NE/SNS and the HPA axis suggest that activation in one system might activate, de-activate, or in some way influence the activity of the other system (although the exact ways in which these two systems influence one another remains poorly understood; Bauer et al., 2002). Thus, analysis of the stress response system should therefore include both systems in order to obtain a clear picture of coordination across the whole system. Due to the functional connections between the LC-NE/SNS and the HPA axis, theorists

have attempted to describe possible models of concurrent activation (Bauer et al., 2002; Chrousos & Gold, 1992; Munck, Guyre, & Holbrook, 1984). Theorists agree that an inverted U shape best characterizes the relation between arousal (in general) and wellbeing, so that insufficient or extreme arousal is detrimental to functioning and wellbeing (Bauer et al., 2002; Chrousos and Gold, 1992). Bauer and colleagues (2002) suggest analyzing patterns of stress reactivity by taking into account activity in each component (the LC-NE/SNS and the HPA axis) of the system, in order to identify adaptive patterns of activation**.** In addition, Bauer et al. proposed two potential models of stress reactivity: a) a pattern in which the activity of the LC-NE/SNS and the HPA is independent or additive; and b) a pattern in which coordinated activation of the stress response system (the HPA and the LC-NE/SNS) is assumed to be interactive. The additive model suggests that, given that an optimal level of arousal is moderate arousal, the total sum of sAA and cortisol change from baseline should be moderate (neither too low or too high). Thus, according to this model either moderate arousal in both systems or asymmetrical arousal (e.g. one system displays high arousal and the other low arousal) would be most adaptive because the total amount of arousal would be moderate. According to the additive model, symmetrical activation would constitute risk (i.e., high HPA – high SNS activity, or low HPA – low SNS activity).

In contrast, the interactive model suggests that activity of the HPA axis suppresses the activity of the SNS, thereby interacting with the SNS to manage the stress response. Once again, keeping in mind that moderate overall arousal is optimal, adaptive activation of this system would constitute concurrent moderate activation of the two systems. Symmetrical activation (e.g., moderate HPA-moderate SNS; low HPA-low SNS; and high HPA-high SNS) would indicate coordinated responses and low risk. In the case of the interactive model, risk would be

related with asymmetrical activation of the two systems (i.e., high HPA activity-low SNS activity or low HPA activity-high SNS activity), which would suggest the two systems have poor coordination in managing the stress response. Several studies have since attempted to examine the stress reactivity system using the models proposed by Bauer et al. (2002). However, results have been mixed and have not consistently supported one model above the other. Some studies have found support for the additive model (i.e., concurrently high or concurrently low arousal is linked to poor outcomes; Bae et al., 2015; Berry, Blair, Willoughby, Granger, & Family Life Project Key Investigators, 2012; El-Sheikh, et al., 2008; Chen, Raine, Soyfer, Granger, 2015; Gordis, Granger, Susman, & Trickett, 2006). For example, El-Sheikh and colleagues (2008) found that higher cortisol in the context of concurrently higher sAA was linked with more parent rated internalizing and externalizing behaviors. Some studies, however, have found support for the interactive model. Such studies have found that poor outcomes are predicted when high activity in one system occurs with low activity in the other system (Allwood et al., 2011; Chen, Raine, Rudo-Hutt, Glenn, Soyfer, & Granger, 2015; Reeves, Fisher, Newman, & Granger, 2016). For example, a study by Allwood and colleagues (2011) found that cortisol reactivity was linked with total number of parent rated behavior problems only in the context of low sAA reactivity, for a sample of 7 - 16 year olds.

Taken together, results have demonstrated that patterns of activation in the stress reactivity system are linked with important outcomes, such as internalizing and externalizing behaviors in children (Allwood et al., 2011; El-Sheikh et al., 2008; Chen, Raine, Rudo-Hutt, et al., 2015; Chen, Raine, Soyfer, et al., 2015). Yet, results have been mixed and support has not accumulated for one model of stress reactivity over another (i.e., additive model vs. interactive model; Bae et al., 2015; Berry et al., 2012; El-Sheikh, et al., 2008; Chen, Raine, Soyfer, et al.,

2015; Gordis et al., 2006). To date, most empirical efforts have approached the study of the stress reactivity system from a variable centered approach (i.e., examining how variables relate to one another across all individuals). It is possible that approaching the examination of the stress response system from a person-centered approach (that is, examining patterns of reactivity that are similar across subgroups of the population) may contribute to our understanding of how activity of the stress response system is related to important outcomes. In fact, there are several advantages to utilizing a person-centered approach as the next step in studying the stress response. First, a person-centered approach assumes that a population or sample is heterogeneous concerning how key variables interact with one another, thus allowing for the existence of various models of adaptive functioning and of risk (Laursen & Hoff, 2006). Additionally, a person-centered approach can be coupled with a variable-centered approach, thereby allowing for the initial identification of homogenous subgroups which can be followed up with variable-centered approaches (i.e., examining group differences in how predictors and outcomes associate; Laursen & Hoff, 2006). Thus, the present study seeks to fill this gap in knowledge by examining activity in the stress response system from a person-centered approach.

Behavioral Reactivity as Part of the Stress Response System

Infant behavioral reactivity has been proposed as one variable that may be related to individual differences in physiological stress reactivity (Rothbart, 1989; Rothbart & Derryberry, 1981), yet research on models of how the two stress response symptoms work together has neglected behavioral responsivity during stress. Behavioral reactivity encompasses the infants' observable response to a given stimulus, so that infants may cry to painful physical stimulus (e.g., inoculation; Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995), may approach a novel toy

(Putnam & Stifter, 2002), or may avert their gaze from an overwhelming stimulus (Swingler, Perry, Calkins, & Bell, 2014).

Theorists suggest that successfully adapting to stress may involve both physiological and behavioral responses (Eisenberg, Fabes, & Guthrie, 1997; Stansbury & Gunnar, 1994). Infant behavioral reactivity (frequently measured as time spent displaying facial and vocal distress signals; Sherman, Stupica, Dykas, Rams-Marcuse, & Cassidy, 2013) to stress has been empirically linked to physiological markers of stress reactivity, such as cortisol, with studies finding correlations between increases in cortisol and negative reactivity behaviors (e.g., fussing and crying; Towe-Goodman, Stifter, Mills-Koonce, Granger, & Family Life Project Key Investigators, 2012; Ursache, Blair, Granger, Stifter, Voegtline, & Family Life Project Key Investigators, 2014).

Although more is known about links between cortisol and behavioral reactivity, a similar pattern can be observed between SNS activation and behavioral reactivity in infants. For example, links have been established between behavioral reactivity levels and heart rate (a measure of autonomic nervous system activity, which includes SNS activity; Haley & Stansbury, 2003). Nevertheless, few studies have examined behavioral reactivity and physiological reactivity in both the HPA and SNS concurrently. A study Spinrad and colleagues (2009), however, stands as a notable exception because they examined such links in preschoolers. Spinrad et al. investigated links between SNS and HPA axis activation and behavioral responses to a frustration task in a sample of 84 preschoolers. The authors found that sAA reactivity was linked with dispositional anger (although only for girls). No links between HPA and behavioral responses to a frustration task emerged.

Nevertheless, little is known about whether including infant behavioral reactivity, in *addition to* physiological activity of the two components of the stress system, will add to our ability to explain individual differences in internalizing and externalizing symptoms at 12 months. For example, it might be that infants who display a moderate arousal in both the SNS and the HPA to a frustration task, who also display moderate levels of negative reactivity would have an optimal level of overall arousal and therefore be rated as having lower levels of internalizing and externalizing behaviors by their mothers at 12 months.

On the other hand, it may be that infants who do not appear behaviorally distressed (i.e., who show low negative reactivity on a behavioral level), yet show signs of being highly physiologically aroused, might be over controlling their behavior and in turn may be more likely to be rated as displaying greater internalizing behaviors by their mothers later on at 12 months. Given the importance of understanding adaptive patterns of stress response in the context of individual differences, the present study seeks to examine patterns of behavioral and physiological stress reactivity to a frustration/anger stressor in 6-month-old infants and explore whether these patterns of infant physiological and behavioral response are linked with later socio-emotional functioning at 12 months.

The Present Study

First, the present study will use Latent Profile Analysis (LPA) to examine whether patterns of reactivity in the HPA and the LC-NE/SNS systems emerge in response to an infant frustration/anger task with infants 6 months of age. By taking a person-centered approach, the present study might augment our understanding of the stress response system as a whole. Further, although the present study does not seek to empirically test the two opposing patterns proposed by Bauer and colleagues (2002), either, both, or neither of these patterns may emerge through the person-centered approach chosen for this study.

Second, the present study seeks to examine whether patterns of stress reactivity are differentially linked with later socio-emotional functioning (mother rated internalizing and externalizing behaviors) when the infants are 12 months of age.

Then, a second LPA analysis will be conducted adding a behavioral reactivity variable in order to examine the stress response in a more complete manner. Thus, the second LPA analysis will include both the physiological markers (cortisol and salivary alpha amylase) and a behavioral reactivity measure (i.e., average negative reactivity, both facial and vocal, across the paradigm). Including a measure of behavioral reactivity into the examination of the stress response might more clearly define subgroups of individuals for whom stress response variables interact in the same manner. Further, given the expectation that behavioral changes will follow stress reactivity, including negative behavioral reactivity into this analysis will help us identify individuals who display maladaptive patterns at the behavioral level (although they may not display maladaptive patterns at the physiological level).

Finally, during this second stage, relations between new patterns of stress reactivity (which include physiological and behavioral markers) and 12 month socio-emotional functioning (internalizing and externalizing) will be examined.

Chapter II

Literature Review

Responding to stress is necessary for survival, and humans, like other species, have developed systems to respond to threats in the environment, namely, the stress response. The stress response in humans is mediated by two principal components: the locus coeruleus/norepinephrine-sympathetic nervous system (LC-NE/SNS) and the hypothalamic pituitary adrenal (HPA) axis (Chrousos & Gold, 1992). These two systems will be reviewed in depth in the pages to come. First, however, it is important to understand the purpose of the stress response, which is dual. The stress response exists both to cope with a threat and to return the body to its natural state of balance.

Homeostasis, Stress, and the Biology of the Stress Response

The stress response is important to survival as it is responsible for preserving a complex and dynamic balance within and between all systems in the entity (Chrousos & Gold, 1992). This balance is called homeostasis (Chrousos & Gold, 1992). Maintaining homeostasis is vital for an organism's well-being and functioning (Chrousos, 2009). Yet, an organism's homeostasis is constantly threatened by both internal (i.e., emotional) and external (i.e., physical) disturbances. These disturbances, due to their adverse nature, are considered stressors (Chrousos, 2009). Stressors, then, are any stimulus that is perceived as a threat to an organism's equilibrium. The noxious effect of stressors on the organism has been conceptualized as stress (Selye, 1936).

The term stress comes from the indo-european root of *str*, which translates to "exertion of pressure" (p. 375, Chrousos, 2009). The word stress was first used by Hans Selye, a Hungarian-Canadian experimentalist in 1956. Selye was the first to describe the prototypical physiological response to nocuous agents that is now considered the body's stress response (Selye, 1936).

Stress was originally defined as the "state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biological system" (Selye, 1956, p. 423). That is, stress was the response produced by the body regardless of the stressing stimulus. More recently, stress has been conceptualized as the body's recognition of a stimulus that threatens homeostasis and the resulting adaptive responses deployed to re-establish homeostasis (Chrousos, Torpy, & Gold, 1998). More specifically, stress has been conceptualized as any situation in which neurochemicals known to be involved in the brain's stress response, such as the glucocortidoids associated with the HPA axis, become elevated following an experience (McEwen, 2000).

Given the importance of homeostasis for the organism's well-being, a premium is placed in maintaining this equilibrium. When a stimulus is perceived in the environment and it is interpreted as posing a challenge to the body's homeostasis, a response to the stressor may be mounted via activation of the stress response system (Chrousos, 2009; Chrousos & Gold, 1992). Activation of the response system results in a "cascade of hormonal and biochemical events" which ultimately result in behavioral and physical changes that promote survival and homeostasis (pp. 197; Gunnar & Cheatham, 2003). An adaptive response to stress may include increased arousal and alertness, focused cognition and attention, euphoria, analgesia, and higher core temperature in order to allow the person to actively resolve the stressful situation, survive, and ultimately restore homeostasis (Charmandari, Tsigos, & Chrousos, 2005). In order for these physiological changes to occur and to assure prompt response to the stressor, energy is redirected from various body parts towards these functions. In fact, activation of the stress response increases the creation of lipids and glucose, both of which increase the available energy resources (Chrousos & Gold, 1992). Similarly, vegetative functions, such as digestion, mating,

and sleep are slowed or stopped, to preserve energy for the functions directly involved in responding to the stressor (Chrousos & Gold, 1992).

Activation of the stress response system is initiated by rousing of the neural systems that activate the functions necessary to respond to the stressor. Two systems have been identified as the primary components of the stress response system, namely the Locus Coerelous-Norepinephrine/Sympathetic Nervous System (LC-NE/SNS) and the Hypothalamic Pituitary Adrenal (HPA) axis (Charmandari et al., 2005; Chrousos & Gold, 1992).

The Components of the Stress Reactivity System

The stress response is mediated by two systems, the Locus Coeruleus - Norepinephrine/Sympathetic Nervous System (LC-NE/SNS) and the Hypothalamic Pituitary Adrenal (HPA) axis (Charmandari et al., 2005; Chrousos & Gold., 1992). Although each of these components has long been implicated in the human stress response, they have been primarily investigated independently from each other (Bauer et al., 2002). Yet, modern conceptualizations suggest that to best understand the stress response these two systems should be investigated in tandem (Bauer et al., 2002). Further, there are good theoretical and empirical reasons to believe activation of these two systems influence one another (i.e., they are not independent; Munck et al., 1984). Before delving into the overlaps between the two systems, it is important to have an understanding of how each system acts in response to stress. The next sections will detail what is known about the LC-NE/SNS and the HPA axis as it relates to their stress response activity.

The LC-NE/SNS: Part of the Autonomic Nervous System

The autonomic nervous system (ANS) is composed of two functionally and anatomically different systems, the sympathetic (SNS) and parasympathetic nervous systems (PNS). The SNS, alongside the PNS, is responsible for regulating cardiovascular, gastrointestinal, respiratory, renal, and endocrine systems (Charmandari et al., 2005). The sympathetic nervous system is

activated in times of emergency or crisis and it is responsible for the *fight or flight* response (McCorry, 2007). Fight or flight is the term used to characterize the physiological and behavioral (i.e., fighting the threat or fleeing) changes that follow activation of the SNS due to threat (Cannon, 1932). When the SNS activates the fight or flight response, it acts on the whole body in order to facilitate dealing with the environmental threat. For example: heart rate increases to pump more blood per minute; vasoconstriction, the constriction of blood vessels, causes redirection of blood away from inactive tissues (e.g., gastrointestinal systems) towards muscles; maximization of oxygen uptake and carbon dioxide elimination occurs through bronchodilation, or the enlargement of air passages; and increases of glucose molecules on the blood are achieved via increased glycogenolysis and gluconeogenesis (creation of new glucose from sources other than carbohydrates).This widespread response is coordinated, so that nutrientrich, oxygenated blood is delivered effectively to the muscles (McCorry, 2007).

Neurobiology of the LC-NE/SNS. The locus coeruleus (LC) is the brainstem nucleus which begins in the pons with a concentration of noradrenergic neurons that are bluish in color (locus coeruleus means “blue spot” in Latin; Aston-Jones & Cohen, 2005). The LC receives inputs from a restricted number of central nervous system (CNS) structures, including the nucleus paragigantocellularis (PGI) and the nucleus prepositus hypogleuss (PH). SNS-relevant stimuli activate neurons found in the PGI. The PH neurons are linked to control of eye movement (Berridge & Waterhouse, 2003). LC neurons emanate, in an extensive network, from the nucleus to various parts of the brain (Aston-Jones & Cohen, 2005). LC projections are particularly dense in areas linked with attentional processing and motor activity. Within the visual system, NE fibers activate regions related to spatial perception and visual-motor activity (Berridge & Waterhouse, 2003). Additionally, recent findings show that activity of the LC-NE

during stress inhibits the activity of the pre-frontal cortex in adults, favoring a more instinctual and rapid response to the threat situation (Arnsten, 2000).

The LC is one of the brain's main norepinephrine producing centers and it is the primary, and in some cases only, source of norepinephrine to various brain cortices (cerebral, cerebellar, and hippocampal; Ashton-Jones & Cohen, 2005; Berridge & Waterhouse, 2003). Neurons in the LC nucleus contain dopamine beta-hydroxylase, an enzyme involved in the conversion of dopamine to norepinephrine. Norepinephrine, released via activation of the LC-NE, travels to the sympathetic nervous system. Norepinephrine is, in fact, the main neurotransmitter used by the SNS. The SNS uses its efferent pre-ganglionic fibers to communicate with noradrenergic post-ganglionic fibers that connect directly to the organs under influence of the SNS (Charmandari et al., 2005). The SNS, then, is the last step in the stress reactivity chain as its efferent neurons travel from the brain down through the spinal cord to connect with various organs and parts of the body (Aston-Jones, Rajkowski, Kubiak, Valentino, & Shipley, 1996).

The LC-NE/SNS, then, is one of two primary components of the stress response. It is quick acting and its activation creates a chain of events that result in a coordinated response to the threat (attending to the threat, or choosing to run, for example). Yet, individual differences in the activity of this system exist. In fact, individual differences in activity of the LC-NE/SNS have been linked with differences in behavioral responses. For example, Fabes and colleagues (1993) found that increased SNS activity, as measured by skin conductance, was linked with increased visual aversion from a distressing stimulus, for a group of 3rd and 6th graders. Moreover, empirical findings show that individual differences in the activity of the LC-NE/SNS are also linked with social and emotional problems in childhood and adolescence. For example, ,low resting heart rate, a measure of ANS activity has been linked with anti-social behavioral in

children and adolescents (Keller & El-Sheikh, 2009; Lorber, 2004), and hyperactivity of the brain's noradrenergic (norepinephrine producing) systems has been linked with panic disorder and post-traumatic stress disorder (Southwick et al., 1999). Given the wide-ranging influence of the LC-NE/SNS system throughout the body and the links between its activity and wellbeing, it is important to understand adaptive functioning of the LC-NE/SNS in stress reactivity, particularly in conjunction with the second component of the stress reactivity system, the HPA axis. Understanding these two systems, LC-NE/SNS and HPA, in relation to one another will be a primary focus of this study.

Finally, it is important to note that norepinephrine (NE) is the main chemical resulting from the activation of the LC-NE/SNS (Berridge & Waterhouse, 2003). NE is released into the blood stream and acts on organs. Yet, measuring NE through blood samples is a costly and invasive method (Granger et al., 2007). Recently researchers have used a novel method to assess LC-NE/SNS activity non-invasively via saliva. Specifically, scientists have begun using salivary alpha amylase (sAA) as a marker of LC-NE/SNS activity. This new, non-invasive methodology allows us to study the activity of the LC-NE/SNS in individuals for whom more invasive methodology would have been prohibitive, such as infants. The present study will utilize this advanced methodology to further our understanding of the stress response with a sample of 6-month-old infants.

Physiological markers of LC-NE/SNS activity. In addition to skin conductance (El-Sheikh et al., 2008) and resting heart rate (Fabes et al., 1993), a number of physiological indicators of LC-NE/SNS activity have been identified. For example, blood norepinephrine concentration (Rohleder, Nater, Wolf, Ehlert, & Kirschbaum, 2004), and pre-ejection period (Bosch, de Gaus, Veerman, Hoogstraten, & Nieuw Amerongen, 2003) have been found to index

LC-NE/SNS activity. However, these testing for these indicators of LC-NE/SNS activity can be invasive as is the case for blood-based analyses, or subject to error due to movement or other artifact as is the case with the skin conductance and heart rate based measures. In recent years, the advent of sophisticated methods for assaying salivary alpha amylase have presented researchers with a new method for examining sympathetic nervous system activity. The use of salivary assays provide some benefit over previously used measures, as salivary collection is minimally invasive, requires fairly inexpensive equipment, and can be collected outside of the laboratory setting (Granger & Kivlighan, 2003). Further, it is likely that examining SNS activity via sAA will provide unique information in addition to other measures of SNS as dissociation among physiological measures is often found (Granger & Kivlighan, 2003).

Salivary alpha amylase, a digestive enzyme produced in the mouth that digests carbohydrates, has received increased attention in recent years as a non-invasive marker of the sympathetic nervous system activation (SNS; Granger et al., 2006; Nater, & Rohleder, 2009). Alpha amylase is an enzyme that is produced in the mouth by salivary glands and can be assayed from samples of whole saliva (Nater & Rohleder, 2009). This enzyme is responsible for the breakdown of starch in the mouth to initiate digestion. sAA originates in ocinar cells, which are activated by the autonomic nervous system via neurotransmitters. In fact, neurotransmitters emanating from the sympathetic nervous system, such as norepinephrine bind to the adrenergic receptors in ocinar cells to communicate with internal, secondary messengers that activate the secretory process (Nater & Rohleder, 2009). In response to perceived threat, SNS activation can be seen in sAA concentration as quickly as 5 – 10 minutes after the stressor has taken place (Granger & Kivlighan, 2003), and amounts of sAA should return to normal in a window that is shorter than for cortisol (Takai, Yamaguchi, Aragaki, Eto, Uchihashi, & Nishikawa, 2004).

Early studies demonstrated a link between concentration of sAA and sympathetic nervous system activity (see Granger et al., 2006). A series of studies examining links between salivary alpha amylase and activation of the LC-NE/SNS demonstrated that amounts of norepinephrine blood concentration could be estimated by concentration of alpha amylase in whole saliva (Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996). Salivary alpha amylase has also been linked with other well-established measures of ANS activation, such as cardiovascular parameters like pre-ejection period (Bosch et al., 2003), and skin conductance (El Sheikh et al., 2008).

Studies to date have shown that sAA increases in reaction to stimuli throughout the lifespan, and that it is sensitivity to both physical (e.g., exercise) and psychological stressors (e.g., taking an exam; Granger et al., 2006; Granger et al, 2007; Gordis et al., 2006). Baseline sAA levels are present and measurable beginning early in infancy, as young as 2 months old (Davis & Granger, 2009) and changes in amount of sAA in response to stressors have also been identified during infancy. Davis and Granger (2009) found that sAA increased following inoculation for 6 months old infants. Eliciting a response in infant sAA through methods other than inoculation has resulted in mixed outcomes. Laurent, Ablow, and Measelle, (2012), found that 18-month-old infants displayed sAA reactivity to a series of emotion focused lab tasks (fear, frustration, and joy tasks). Granger and colleagues (2006) utilized the arm restraint task to elicit stress in a sample of 86 infants and found no statistically significant reactivity in sAA levels for 6-month-old infants 20 minutes post peak distress. Kivlighan et al (2005) also failed to demonstrate infant sAA reactivity to the arm restraint task in 6-10-month-old infants when assessing sAA 20 minutes post task (as reported in Granger et al., 2007). One possible problem for previous researchers in detecting a change in sAA following a stressor is the timing of the sample collection. The LC-NE/SNS response is thought to be fast acting, resulting in increases in

sAA as quickly as 5-10 minutes following the stressor. Therefore, collecting saliva samples within the expected window of reactivity becomes a key issue in being able to identify changes in sAA.

Salivary alpha amylase and links to socio-emotional outcomes. Given the relatively new ability to measure LC-NE/SNS activity noninvasively via salivary alpha amylase, the body of knowledge about sAA and socio-emotional outcomes remains modest. Some studies that have examined this relationship have found that sAA activity appears to be linked with socio-emotional outcomes, but results have been mixed. Although one study of 87 two-year-olds found that higher amounts of sAA were linked with positive affect and approach behaviors in in a series of emotion-eliciting lab tasks (Fortunato et al., 2009). Other studies have found high sAA levels were linked to poor socio-emotional outcomes. For example, Allwood et al. (2011) found that basal levels of sAA were linked with self-reported rating of anxiety in a sample of 56 children aged 7 to 16 years, and van Veen and colleagues (2008) found higher basal sAA amounts in individuals with generalized social anxiety disorder versus healthy controls. Tanaka et al. (2012) found higher amounts of basal and reactive sAA in patients with major depressive disorder when compared to healthy controls (see Schumacher, Kirschbaum, Fydrich, & Ströhle, 2013 for a review). In contrast, other studies have found no links between sAA and psychopathology (obsessive compulsive disorder, Lord, Hall, Soares, & Steiner, 2011; generalized anxiety disorder, Fisher, Granger, & Newman, 2010).

Of particular interest to the current study are studies that focused on the link between sAA and socio-emotional outcomes in infancy. As previously, mentioned, Fortunato and colleagues (2008) found that baseline sAA amounts were positively linked with approach behavior and positive affect in a sample of toddlers following a series of challenging tasks. Hill-

Soderlund and colleagues (2015) found links between basal sAA at 12 months and internalizing behaviors at 36 months. Results also showed that basal sAA at 12 months and total increase of basal sAA over time (between 12 and 36 months) were linked with externalizing behaviors at 36 months. Little is known, however, about links between sAA in infants younger than 12 months and later internalizing and externalizing behavior problems at 12 months.

The Hypothalamic Pituitary Adrenal Axis

The hypothalamic pituitary adrenal axis is the second and final component of the stress reactivity system. The HPA axis has long received empirical attention, and it has been examined throughout the lifespan and in relation to a variety of outcomes such as psychopathology (Burke et al., 2005), physical health such as inflammation and disease (Franchimont, Kino, Galon, Meduri, & Chrousos, 2003) and in trauma correlates such as post-traumatic stress disorder (Steudte-Schmiedgen et al., 2015). The following pages will focus on the structure and function of the HPA axis and its links to psychopathology and functioning in infancy and childhood.

Neurobiology of the hypothalamic pituitary adrenal axis. The HPA axis begins in the hypothalamus where the brain and body send signals to the paraventricular nucleus (PVN) the first step in HPA axis activation (Gunnar, & Cheatham, 2003). The PVN secretes corticotropin-releasing hormone and arginine vasopressin, two chemicals involved in the activity of the HPA axis (AVP; Gunnar & Vazquez, 2006). The corticotropin-releasing hormone (CRH) travels to the anterior pituitary gland where it begins the synthesis of the adrenocorticotropic hormone (ACTH; Gunnar, & Cheatham, 2003; Gunnar & Vazquez, 2006). ACTH then travels to the adrenal glands, which produce glucocorticoids (GCs; Gunnar & Vazquez, 2006). Glucocorticoids (GCs; e.g., cortisol, corticosterone) are powerful steroids that act on organs to facilitate homeostasis. More specifically, they influence DNA in organs and tissues of the body in order to activate or

deactivate gene transcription (Sapolsky, Romero, & Munck, 2000). GCs are also involved in the brain, where they influence motivation, reward, and mood (de Kloet, 1991). After GCs are produced, they are then released into the blood stream (Gunnar, & Cheatham, 2003).

Cortisol is the principal human GC and it is involved in regulating blood pressure, immune system preparedness, and glucose metabolism (Gold, Goodwin, & Chrousos, 1988). The extra cortisol that is produced in response to stress (additional to basal flowing cortisol) binds to corticotropin binding globulin (CBG) rendering it inactive (Gunnar, & Cheatham, 2003). However, both in basal conditions and during elevated cortisol times such as in response to stress, only a certain percentage of the blood flowing cortisol binds to CBG (Gunnar, & Cheatham, 2003). The left-over cortisol remains active and able to have effects on the body and brain (Gunnar, & Cheatham, 2003). When cortisol binds to receptor cells, it activates or deactivates processes to help the organism maintain or return to balance. For example, maintaining steady electrical currents in neurons to aid communication and facilitating glucose utilization by the brain (Gunnar & Vazquez, 2006). Further, the HPA axis is self-regulating, so that through a complex negative feedback loop, activation of the PVN slows down when too much cortisol is detected in the system (i.e., when too many receptors have bound cortisol, Gunnar & Cheatham, 2003). The process of HPA activation resulting in cortisol in the bloodstream takes approximately 20 minutes (Gunnar, & Cheatham, 2003).

Stressors and the HPA response. The HPA axis is primarily responsible for responding to significant stressors that require additional resources than those that are readily available in the organism (i.e., available energy that is redirected for dealing with the stressor by the SNS, Gunnar, & Cheatham, 2003). HPA activation creates additional resources by redirecting energies that are normally used for long-term survival, such as reproduction, immune function, digestion,

and growth, towards the resolution of the immediate threat (Gunnar, & Cheatham, 2003; Sapolsky et al., 2000). Energy is directed towards the synthesis of protein and fats to aid in mobilization for the purpose of survival (Gunnar, & Cheatham, 2003).

While short-term activation of the HPA axis results in GC levels that promote health maintaining behaviors (Gunnar & Vazquez, 2006). Long-term suspension of long-term survival functions, however, has been conceptualized as detrimental to prolonged wellbeing and healthy life-span development (Gunnar, & Cheatham, 2003). Studies have shown that prolonged activation of the HPA axis leading to increased CRH and GC levels inhibits developments and shapes brain systems (Gunnar & Vazquez, 2006). Changes in the brain related to chronic HPA activation have been linked to the development of psychopathology (Heim, Owens, Plotsky, & Nemeroff, 1997).

The HPA axis may be activated by *systemic* or *processive* stressors (Herman & Cullinan, 1997). Systemic stressors are independent of context and of a physical nature (i.e., infection or hypotension). *Processive* stressors rely on conscious appraisal by the individual. As such the individual must assign the stressor an emotional meaning that in turn activates the HPA axis (Gunnar & Vazquez, 2006). Types of *processive* stressors that have been identified include "novelty, uncertainty, unpredictability, uncontrollability and potential for harm" (Kirschbaum & Hellhammer, 1992, as cited in Gunnar and Vazquez, 2006). Activity of the HPA axis in humans can be observed since birth (Gunnar, 1989). Cortisol is present in response to small stressors and painful stressors in newborns (Gunnar & Cheatham, 2003). Newborns display cortisol elevations to stressful experiences such as physical exams and heelstick blood draws (Gunnar 1989; Gunnar 1992). Interestingly, the magnitude of the response is moderated by repeated exposure, with a decrease in response following physical exams and an increase in cortisol response following

heelstick procedures (Gunnar 1989; Gunnar 1992). A cortisol response to physical exams disappears by 10-11 weeks of age (see Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010 for a review), but inoculations, given between 2 and 6 months of age results in an increase in cortisol (see Jansen et al., 2010 for a review). To sum, adrenocortical activity can be seen since infancy and throughout the first year of age.

Salivary cortisol has long been used as a non-invasive marker of HPA axis activity (Gunnar & Vazquez, 2006). Initial measures of cortisol were obtained via urine and blood, although both of these methods of collection presented significant challenges for research with children and infants (Gunnar, 1986). Development of salivary assays that could measure free flowing cortisol levels in small amounts of saliva appeared in the early 1980s and have been used widely since then, due to the non-invasive nature of saliva collection (Gunnar, 1986). Salivary cortisol has been shown to be highly correlated with plasma cortisol (Kirschbaum & Hellhammer, 1994) and cortisol levels in saliva are unaffected by salivary flow rate due to their transmission to saliva occurring via passive diffusion (Kirschbaum & Hellhammer, 1994).

The HPA axis also has a 24-hour circadian rhythm that influences the amounts of basal flowing cortisol throughout the day (Gunnar, & Cheatham, 2003). In adults, cortisol is at its highest shortly after waking (20-30 minutes post rousing) and decreases during the daytime hours (Gunnar, & Cheatham, 2003). In infants, the typical morning peak of the circadian rhythm seen is adults emerges around 3 months of age (see Gunnar & Donzella, 2002, for a review). In order to control for basal cortisol changes studies examining the activity of the HPA axis should collect cortisol samples outside of the peak and nadir points of the circadian rhythm of the HPA axis (Gunnar & Vazquez, 2006).

HPA axis activity and links to socio-emotional outcomes. A now large body of literature exists documenting links between activity of the HPA axis and socio-emotional outcomes through infancy, childhood, and adolescence. Both basal and post-stressor amounts of cortisol have been linked with a variety of social and emotional issues. Low basal cortisol amoutns have been linked with disruptive behaviors. Children diagnosed with Oppositional Defiant Disorder (ODD) or who are at risk for developing the disorder display blunted basal cortisol levels and no change in cortisol amounts following stressors (see Burke, Loeber, & Birmaher, 2002). In a study of adolescents (N = 724, mean age 13.5 years) boys with low trait cortisol levels displayed greater concurrent externalizing behaviors (Shirtcliff et al., 2005). Another study found heightened basal glucocorticoids in children with anxiety disorders (Carrion et al., 2002). Finally, disruptions in the diurnal activity and the stress response of cortisol in adults with major depressive disorder have been consistently identified in the literature (see review, Pariante, 2003). Although links between cortisol and psychopathology are well established, much of the existing literature has not investigated the HPA axis in the context of the stress reactivity. Yet, as previously stated, it is important to examine both components of the stress response system in order to fully understand links between stress reactivity and outcomes.

Interactions between the HPA and the LC-NE/SNS Systems

Our understanding of the stress response has evolved in recent decades (Boyce & Ellis, 2005). For some time, the conventional understanding was that the stress response developed in a prehistoric environment to deal effectively with threat, and that it is no longer adaptive given the intensity and stressors of modern human life (Boyce & Ellis, 2005). Nesse and Young (2000) argued that although we experience stressors, those of our ancestors were undoubtedly more intense and lethal and that given that the stress response was calibrated during ancestral times, it is likely to produce net losses when activated by our modern day stressors. Yet, other theorists

and researchers have challenged this understanding. Munck and colleagues (1984) were the first to propose that the activity of the HPA axis suppresses the body's normal reactions (i.e., the LC-NE/SNS reaction) to stress, thereby protecting the body from a damaging stress response. They theorized that HPA axis activity did not amplify the stress response, but rather served a protective purpose by down regulating the stress response once the threatening stimulus had been resolved and returning the system to homeostasis. This regulatory function then protects the body from possible overshooting of the normal stress response which would then, in itself, become a stressor. In fact, the nature of the relationship between the two components of the stress response remains a focus of research efforts.

Current understandings of the structural and functional outlines of the HPA axis and the LC-NE/SNS suggest that these two systems are integrated and cross-regulatory (Boyce & Ellis, 2005). If CRH is injected into the LC, the firing rate of the norepinephrine releasing hormones in the LC increases, and norepinephrine in turn stimulates release of CRH (Chrousos & Gold, 1992). CRH producing neurons from the amygdala nucleus extend into the LC and their release increases the release of NE (Meany, 2001). NE secretion in the LC has been linked to increases in CRH production in the PVN (Habib et al., 2001). This understanding is in line with several recent empirical findings that suggest lack of coordination between the two systems is a better indicator of pathology than large responses in either system alone. Bauer (2002) found that children who had asymmetrical activation of the HPA axis and the LC-NE/SNS showed the greatest number of behavioral problems. In adults, a review showed that asymmetry between adrenocortical and sympathetic responses following an acutely stressful event were most predictive of later post-traumatic stress disorder symptomatology (Yehuda, McFarlane, & Shalev, 1998).

Because interactions between the SNS and HPA have been found, modern approaches to the study of stress psychophysiology emphasize a multisystemic approach (Bauer et al., 2002). Yet, the nature of the interaction between the HPA axis and the LC-NE/SNS remains poorly understood. Recent findings show that glucocorticoids both suppress and amplify the effects of sympathetic activation on metabolism, immune function, and cardiovascular activity (see Sapolsky et al., 2000 for a review), suggesting that the activity of the HPA axis following SNS activity may at times aid the efforts of the SNS but at other times it may work to down-regulate this response. In sum, there is reason to believe that in order to understand the human stress response scientists should examine both stress responding systems concurrently, as they likely influence one another.

Theoretical Models of the Stress Response System

Bauer et al. (2002) proposed a model of the possible relations between the HPA axis and the LC-NE/SNS. Bauer and colleagues' (2002) model of stress reactivity builds on an earlier model of optimal arousal by Chrousos and Gold (1992). In this model they propose that a non-linear relationship exist. They suggest that an inverted U shape may best characterize the links between overall (i.e., both HPA and SNS) arousal and well-being. This model, borrowed from classic arousal-performance empirical research (e.g., memory, attention) indicates that there is an optimal level of arousal that leads to the most adaptive response, and that insufficient or extreme overall arousal impairs the organism's ability to respond. This conceptualization has in fact been empirically supported in child development literature by examinations of Kagan's theoretical model of correlates between physiological arousal and behavior (Kagan, 1997, Kagan, Reznick, & Snidman,1987). In his model, Kagan suggests that some children who experience hyperarousal of both the SNS and the HPA axis display inhibited behaviors that he contends are

used to manage the experience of a highly reactive physiological system. Conversely, children who experience low arousal in both the SNS and the HPA axis display aggressive behaviors and inappropriate fear responses, which Kagan argues are deployed due to a lack of appropriate stress-related physiological arousal (Kagan, 1997).

Bauer et al (2002) built on the idea of optimal arousal by proposing two alternative models. Both models rest on the idea of *optimal arousal* that is, the idea that an adaptive response entails moderate arousal (inadequate or excessive arousal are not adaptive; Chrousos & Gold, 1992). The first model suggested by Bauer et al (2002) in one in which the sum of the activity between the two systems best characterizes overall arousal. This model implies that the HPA axis activity augments the initial stress response mounted by the LC-NE/SNS; in other words, this model suggests an *additive* relation between the two systems. In this case, a moderate level of overall arousal would come about by moderate activity in both systems or high arousal in one system that is paired with low arousal in the other system (e.g., high HPA activity and low LC-NE/SNS activity). In this model, if both the LC-NE/SNS and the HPA axis display low arousal, the overall arousal is inadequate. If both the LC-NE/SNS and the HPA axis display high arousal, the overall level of arousal is high, and therefore excessive. Such symmetrical responses (i.e., hypo- or hyper-reactivity) may indicate susceptibility to future disease.

The second model put forth by Bauer et al. (2002) is an *interactive* model that assumes that the HPA axis does not augment the activity of the LC-NE/SNS but in fact suppresses the activity of the LC-NE/SNS system (as initially suggested by Munck et al., 1984). This model regards the two systems as being interactive or coordinated, and as such, an appropriate response is one that shows concurrent moderate activation of the two systems. Maintaining that an optimum level of arousal is neither inadequate nor excessive (Chrousos & Gold, 1992), a

maladaptive response in this model is one in which the two systems display lack of coordination such as low LC-NE/SNS arousal paired with high HPA axis arousal.

In sum, theorists suggest that there may be an optimal relation between the two components of the stress system and that this relation can be seen via their activity in response to stressors. Further, theory suggests that deviation from the optimal response may be related to poor functioning and illness. Understanding this optimal response may be an important step in understanding the interaction between the psychobiological stress response and socio-emotional outcomes

Concurrent LC-NE-SNS and HPA axis activity and links to outcomes. Research is starting to emerge in which both components of the stress response system are examined concurrently. Many of the studies that have examined the LC-NE/SNS and the HPA axis concurrently have focused on children and adolescents, and findings regarding the interactions between cortisol and salivary alpha amylase and their influence on socio-emotional outcomes are mixed. These studies have found that concurrently high or concurrently low amounts of cortisol and sAA are linked with increased behavioral issues. For example, in a sample of 64 children, 9 and 10 years old, El-Sheikh and colleagues (2008) found that interactions between sAA and cortisol averaged across a laboratory visit were linked with number of parent rated behavioral problems. Specifically, they found that in the context of higher sAA, higher cortisol amounts were linked with both externalizing and internalizing behaviors. Similarly, a study of 429, 11-12 years olds, found that externalizing and internalizing behaviors were predicted by the interaction between trait (i.e., a composite between basal and reactivity levels) cortisol and trait sAA. Specifically they found that concurrently low trait HPA and sAA levels were linked with higher externalizing and internalizing behaviors (as rated by parents; Chen, Raine, Soyfer, et al., 2015).

Bae, et al., (2015) found that children with externalizing disorders displayed a lower cortisol response than healthy controls at low sAA levels. A study with 10-14 year olds (N = 67) found a significant interaction between cortisol and sAA area under the curve predicting parent reports of child aggression. Specifically, they found that the inverse link between cortisol and parent reported aggression was significant when sAA was low (1 *sd* below the mean) but not when high (Gordis et al., 2006). In sum, these studies support the *additive* model of stress reactivity, which suggests that symmetrical low or symmetrical high activation of the SNS and the HPA axis is linked with poor outcomes.

Other studies have found contrasting evidence, suggesting that concurrently opposing levels of cortisol and sAA are linked with poor socio-emotional outcomes. A study of 57 children, 7-16 years of age, examined sAA and cortisol activity pre and post a series of laboratory stressors. The results of this study showed that greater reactivity cortisol levels were linked with total number of problem behaviors (as rated by a parent) in the context of low sAA reactivity (Allwood et al., 2011). Another study with a sample (*N* = 425) of primarily African American children, aged 11-12 year olds found that the relationship between harsh parenting and externalizing/internalizing problems was mediated by the activity of the HPA axis and LC-NE/SNS. More specifically, they found that a link between harsh parenting and externalizing/internalizing behaviors only when cortisol and sAA showed asymmetrical basal levels (although only for boys; Chen, Raine, Ruddo-Hutt, et al., 2015). A recent study of 108 adolescents and young adults (mean age 19.16, *SD* = 1.53) with generalized anxiety disorder (GAD) showed that individuals with GAD were more likely to experience high sAA changes following a stressor task paired with low cortisol changes when compared with healthy controls (Reeves et al., 2016). In sum, these studies found support for the interactive model of stress

reactivity, which suggests that asymmetrical activity between the SNS and the HPA is linked to poor outcomes.

What is known about the stress reactivity system and socio-emotional outcomes in infancy and toddlerhood is more limited. In a longitudinal study of infants at 7, 15, 24, and 36 months of age and executive function (EF) and academic achievement, baseline levels of sAA and cortisol were linked with EF at 36 months. Patterns of concurrently high or concurrently low sAA and cortisol were linked with lower EF abilities. Results for academic achievement show a similar pattern (concurrently high or concurrently low sAA/cortisol linked with lower achievement; Berry et al., 2012). Although more is known about links between the individual components of the stress reactivity system and outcomes, understanding the stress response necessitates multisystem investigation.

Shifting to a Person-Centered Approach

Thus far, these two models have provided a useful heuristic guiding research, but the results have been mixed. One potential reason for the mixed results might be the methodological approach taken to this point. To date, the stress response system has been studied from a variable-centered approach. That is, examinations have focused primarily on how variables are associated with one another, attempting to identify a process that works similarly across all members of the sample (Laursen & Hoff, 2006). Variable-centered approaches are predicated on the assumption that a particular phenomenon works in a similar way in a largely homogenous sample (Laursen & Hoff, 2006). A different way to approach our understanding of the stress response system is through a person-centered approach.

A person-centered approach does not assume that a particular phenomenon operates the same across individuals, but rather, seeks to describe differences in how variables interact for

groups of individuals. Put another way, a person-centered approach seeks to identify and describe differences in subgroups of individuals for which key variables interact in a similar way (Laursen & Hoff, 2006)

Thus, the present study will first, seek to identify whether different patterns of stress reactivity exist in a sample of 6-month-old infants who experience a laboratory stressor. Approaching this question from a person-centered approach will allow for identification of subgroups of people for whom the stress response system reacts in a similar way. This approach may provide further insight into the models of reactivity proposed by Bauer et al., (2002) as either, both, or none of the patterns proposed may emerge in this population. Then, the present study seeks to identify whether identified patterns of reactivity differentially predict mother rated internalizing and externalizing disorders at age 12 months.

Behavioral Concomitants of Stress Reactivity

Activation of the stress response system results in physiological and behavioral changes (Chrousos & Gold, 1992). Given that changes in behavior are an expected end product of the stress response, including measures of behavior in examinations of the stress system might provide a clearer picture of the human stress response. Behavioral reactivity, or an individual's observable response to stimulus (Braungart-Rieker & Stifter, 1996) has been conceptualized as one dimension of temperament that might be related to the stress response. Yet, few studies have examined behavioral reactivity along with both components of the stress system. The following section introduces behavioral reactivity and examines the known links between this construct and outcomes of interest.

Negative Behavioral Reactivity

Negative Behavioral reactivity refers to motor and affective behaviors that accompany a specific stimulus. For example, children may approach when an interesting toy is presented

(Putnam & Stifter, 2002). They may also avert their gaze when a stimuli proves overwhelming (Swingler et al., 2014). Finally, infants may vocalize, cry, and contort their faces in ways that demonstrate negative emotionality when confronted with stimulus that is painful, scary, or frustrating (for example crying after a heelstick blood draw, Gunnar et al., 1995). These behaviors can be conceptualized as the infant's response to environmental stimulation.

Theorists have suggested that negative behavioral reactivity, such as fussing and crying, is adaptive (Stenberg, Campos, & Emde, 1983). Crying is an evolutionarily adaptive behavior that helps infants to survive by manipulating adults into increasing caretaking, among other purposes (Lummaa, Vuorisalo, Barr, & Lehtonen, 1998). By displaying negative affect infants are effectively communicating distress to others in order to achieve a change in their environment and or motivating others to act on their behalves to achieve a desired goal (Braungart-Rieker, Stifter, 1996). Frustration and anger, two related affective responses, are common experiences that may result from daily activities such as waiting for food, having their clothes changed, or being in a restrictive environment (Braungart-Rieker & Stifter, 1996). Loss of control and having a desired goal blocked often result in behavioral displays of anger and frustration (Lewis, Alessandri, & Sullivan, 1990).

Negative behavioral reactivity exists since early in development and can be measured in infancy (Rothbart, 2007). In fact, anger and frustration are apparent as early as 2-3 months (Rothbarth, 2007). Although frustration and anger responses are normative, individual differences in reactivity and self-regulation of these affective dimensions have been linked with various developmental outcomes. Both positive and negative developmental outcomes have been linked with frustration/anger reactivity during infancy. Fox (1989) found links between negative reactivity to a frustration task at 5 months and positive approach behaviors at 14 months. Fox

interpreted findings to suggest negative reactivity to frustration was a marker of a social child who wanted to explore his/her environment without limitations (i.e., not an irritable child). In a study using parent reported temperament on 149 infants, Derryberry and Rothbart (1984) found that frustration at 3 months was positively related to activity levels at 6, 9, and 12 months. Anger is linked with later aggression and increased frustration (Rothbart, 2007). Rothbart, Derryberry, & Hershey, 2010 found that frustration at 6 and 10 months was linked with later frustration/anger, high discomfort, low soothability, and high guilt/shame. They also found that 6 and 10 month frustration/anger was related to seven year impulsivity, aggression, and high intensity pleasure. Authors argue that infant frustration is linked with greater approach, externalizing, and internalizing tendencies.

Links between Behavioral Reactivity and Physiological Reactivity

Behavioral reactivity has been theoretically and empirically linked with physiological markers such as heart rate, and cortisol (Haley & Stansbury, 2003; Towe-Goodman et al., 2012). For example, highly fearful children have been found to have higher and more stable resting heart rates (Kagan et al., 1998). Similarly, in a longitudinal study, childhood negative emotionality at ages 9-10 predicted later cortisol reactivity to a laboratory stressor task during adulthood (Hagan, Luecken, Modecki, Sandler, & Wolchik, 2016). In terms of behavioral reactivity and the endocrine system, a recent study found links between cortisol, sAA, and behavioral reactivity in a sample of 2-year-old children. In this study, cortisol was related with negative affect and withdrawal behaviors, whereas sAA was linked with positive affect and approach behavior (Fortunato, et al., 2008). Thus, successfully adapting to stress may involve both physiological and behavioral responses (Eisenberg et al., 1997; Stansbury & Gunnar, 1994). Yet, although some research exists linking behavioral reactivity with the activity of the stress

response system, much is still unknown about this relationship. Further, few studies have examined both components of the stress response alongside behavioral reactivity and self-regulation.

Although both behavioral reactivity and elevations in physiological stress markers have often been used to assess distress in infancy (Gunnar, 1992), the exact patterns of relationship between these two factors remain poorly understood (Gunnar, Isensee, & Fust, 1987). In fact, discrepancies between activity in physiological markers of distress and distress related behaviors abound in the literature. Some findings indicate a linear relationship between cortisol reactivity and behavioral reactivity. For example, Ursache and colleagues (2012) found significant relations between cortisol activity and behavioral reactivity to a frustration task in a sample of 7-month-old infants. Other findings, however, have indicated no coordination between cortisol reactivity and behavioral reactivity. In a sample of 62 six-month infants, cortisol reactivity and recovery was not related to concurrent behavior during inoculation (Ramsay & Lewis, 2003). Another study found that cortisol reactivity to a physical exam decreased over time, but behavioral reactivity remained the same in a cross-sectional study of infants 7 to 15 weeks of age (Larson, White, Cochran, Donzella, & Gunnar, 1998).

Recent studies have begun to shift their approach to studying the links between physiological and behavioral markers of stress reactivity towards examinations of *patterns* of reactivity (Towe-Goodman et al.,2012). Towe-Goodman and colleagues (2012) used growth mixture models to identify patterns of behavioral reactivity and cortisol in response to 3 stressors. Results show various patterns of activity,with 55% of the sample displaying low adrenocortical and low behavioral reactivity; 34% displaying high cortisol reactivity and moderate negative behavior, and two smaller subgroups (6% - showed high negative reactivity

and average cortisol responses; 6% - high baseline cortisol, low cortisol reactivity). These infant patterns were linked to levels of inter-parental violence, suggesting that different regulatory strategies may develop in different contexts.

The approach taken by the research cited above highlights that both the behavioral and the endocrine systems serve stress related functions and should be seen as important components of the stress reactivity system (Quas, Hong, Alkon, & Boyce, 2000). Yet, to date, few empirical studies have focused on examining both behavioral and physiological components of the stress response. The present study seeks to fill this gap.

The Present Study

The present study seeks to extend our understanding of the stress response system by taking a person-centered approach to the study of the stress response and by examining both physiological and behavioral concomitants of responding to stress. There will be two sets of analyses, and within each set analysis will occur in two steps.

The first set of analyses will focus on examining patterns of change in salivary cortisol and sAA following the stressor manipulation. The first step in this set of analyses will be a Latent Profile Analysis (LPA) to identify homogenous subgroups of infants who display similar patterns of physiological (HPA and LC-NE/SNS) reactivity following a frustration/anger task. The models proposed by Bauer at al (2002), describe four potential patterns of change in the two salivary analytes of interest. Thus, the following hypotheses follow these expected patterns.

Hypotheses

I hypothesize that there will be four unique patterns of change in cortisol and sAA that will emerge in the LPA: a) a pattern of relatively large increases from baseline to post stressor in both cortisol and sAA; b) a pattern involving a relatively large increase in one analyte, combined with a pattern of relatively small or no increase in the other analyte; c) a pattern of small or no

change in both of the analytes; and d) a pattern of relatively moderate levels of change in both analytes.

After identification of homogenous subgroups, in the second step of the analytic plan, a MANOVA will be conducted to analyze whether there are group differences in 12-month-old infants' caregiver-rated externalizing and internalizing behaviors. Previous research has identified several variables that influence the activity of sAA and salivary cortisol. Infant age, for example, has been linked with cortisol activity (Gunnar & Donezella, 2002). Thus, situational and demographic factors found to be associated with levels of change in cortisol and sAA will be included in the analysis.

Hypotheses

Based on the extant theory (Bauer, Quas, & Boyce, 2002) and research (e.g., Kagan, 1986) the following hypotheses are proposed:

1) Individuals who experience relatively large increases in both cortisol and sAA post-stressor will display greater internalizing behaviors at 12 months. This hypothesis follows the additive model that suggests that over- or under-reactivity of the stress response is linked with poor outcomes. Further, I hypothesize links to internalizing behaviors following research by Kagan and colleagues (1987; 1988) that has established links between elevated cortisol levels and behavioral inhibition in children following exposure to a novel environment. Behavioral inhibition is associated with childhood anxiety disorders (Biederman, Rosenbaum, Bolduc-Murphy, Faraone, Chaloff, Hirshfeld, & Kagan, 1993).

2) Individuals who experience relatively small increases in both cortisol and sAA post-stressor will display greater externalizing symptoms at 12 months. This hypothesis follows the additive model that suggests that over- or under-reactivity of the stress response is linked with

poor outcomes. In addition, this pattern of reactivity might be linked with externalizing behaviors specifically given research that shows that low cortisol amounts following stressors are linked with externalizing problems such as oppositional defiant disorder (Burke et al., 2002).

3) Individuals who experience moderate increases in both cortisol and sAA post-stressor will display the lowest amount of both externalizing and internalizing symptoms. This prediction is consistent with the underlying theoretical assumption that moderate reactivity across the stress system is the most adaptive response pattern (Bauer et al., 2002). This pattern is congruent with both the additive and the interactive models.

4) Individuals who experience large increases in one analyte, paired with low levels of change in another analyte, will display both internalizing and externalizing symptoms at 12 months. This hypothesis is in line with Bauer et al.,'s (2002) interactive model, which suggests asymmetry between the HPA axis and the SNS response to stress is linked with childhood behavior problems. In fact, asymmetry between the HPA axis and the SNS has been linked with behavioral issues in toddlers (Fortunato et al., 2008) and youth (Allwood et al., 2011).

The proposed patterns of reactivity and their links to respective outcomes encapsulate the two models proposed by Bauer et al (2002). The following table provides a schematic of how the potential results map would map onto Bauer and colleagues proposed models. As can be seen on the table, if patterns of large change in cortisol and sAA, and/or patterns of small change in cortisol and sAA are linked with behavioral symptoms, this would provide support for the additive model. Conversely, if asymmetrical (high-low or low-high) patterns of reactivity in cortisol and sAA are linked with symptoms, this result would provide support for the interactive model.

Table 1

Comparison of Additive and Interactive Models by Expected Classes

	Additive Model	Interactive Model
Large Change Cort + Large Change in sAA linked with symptoms	Support	
Small Change Cort + Small Change in sAA linked with symptoms	Support	
Moderate Change in Cort + Moderate Change in sAA NOT linked with symptoms	Support	Support
Large Change in Cort + Small Change in sAA/ OR Small Change in Cort + Large Change in sAA linked with symptoms		Support

The second set of analyses will replicate and extend the first set of analyses by adding infant behavioral reactivity to the LPA analysis in order to examine subgroups of infant stress responding (including corstisol, sAA, and behavioral reactivity). Homogenous subgroups that emerge will then be compared with each other to examine any significant differences in levels of externalizing and internalizing symptoms at 12 months as rated by the infants' mother/caregiver. Given the complexity associated with consideration of this additional variable and the lack of previous studies to guide development of hypotheses, the second set of analyses will be considered exploratory analyses.

Chapter III

Method

Participants

A total of 219 infants and their mothers participated in the larger study. Infants were 45% (99) male, 53% (116) female (gender was not available for 4 of the infants or 1.8%). In terms of race and ethnicity, mother-reported infant race and ethnicity was as follows: 37.4% (82) Black/African American, 24.2% (53) as biracial/multi-racial, 16.4% (36) as White/European American, 14.6% (32) as Latino/Hispanic, 1.4% (3) as Asian/Asian American. Information about race and ethnicity was missing for 5.9% (13) of the infants either due to data collection error or mothers declining to answer. Four mothers reported "other" as their infants' race/ethnicity but did not provide further clarification. Regarding family income, 49.8% (109) of mothers in this sample reported a household income of $10,000 or less per year, 23.7% (52) indicated earning between $10,000 and $30,000, 9.1% (20) reported earning between $30,000 and $50,000, and 8.7% (19) reported earning over $50,000 per year. Data on household income was not known for 8.7% (19) of mothers. Mothers also reported on their highest educational attainment with 13.7% (30) not finishing high school, 36.5% (80) reported completing high school or vocational school, 18.7% (41) completing some college but not finishing a degree, 5% (11) of mothers had completed an associate's degree, 11% (24) completing a bachelor's degree, and 6.4% (14) reporting attending some graduate school or completing a graduate degree. No information regarding educational attainment was available for 8.7% (19) mothers.

Table 2

Infant Characteristics

Variable Name	Frequency	Percent
Gender (*N* = 219)	Male = 99	Male = 45.2%
	Female = 116	Female = 53%
	Missing = 4	Missing = 1.8%
Premature Status (*N* = 219)	Yes = 19	Yes = 8.7%
	No = 184	No = 84%
	Unknown = 3	Unknown = 1%
	Missing = 13	Missing = 5.9%
Infant Race/Ethnicity (*N* = 192)	Asian/Asian American = 3	1.4%
	Black/African American = 82	37.4%
	White = 36	16.4%
	Latino/a = 32	14.6%
	Bi-racial/Multiracial = 53	24.2%
	Missing = 13	5.9%

Table 3

Maternal Income and Education

Variable Name	Frequency	Percent
Household Income (*N* =200)	$10,000 or less = 109	49.8%
	$10,000 – 30,000 = 52	23.7%
	$30,001 – 50,000 = 20	9.1%
	$50,001 or more = 19	8.7%
	Missing = 19	8.7%
Educational Achievement (*N* = 200)	Some High School = 30	13.7%
	High School Diploma/GED = 80	36.5%
	Some College = 41	18.7%
	Associate's Degree = 11	5%
	Bachelor's Degree = 24	11%
	Some Graduate/ Graduate Degree = 14	6.4
	Missing = 19	8.7%

Measures

Demographics. Demographic characteristics were collected via maternal self-report when infants were six months old. The demographics questionnaire requested the following information: infant date of birth, infant gender, infant weight and length at birth, infant and

mother race and ethnicity, household income, household composition, maternal employment status, maternal employment title, maternal highest level of education, infant health status, number of siblings, full-term vs. premature birth, maternal first language, language spoken at home and other languages spoken by mother, and mother's current romantic status.

All questionnaire data were collected via data collection software, Questionnaire Development System™ (V. 3.0, NOVA Research Company, Silver Spring, MD.). This software automatically reads the questionnaire questions and multiple-choice answers aloud to allow participants who may have reading difficulties to complete the questionnaire portion of the study. Participants were given the option to switch to a silent mode if they preferred to read the questions themselves.

Infant gender was reported by the infant's mother or caregiver. Infant race/ethnicity was also reported by the infant's mother or caregiver. Caregivers were able to select more than one race/ethnicity variable if applicable. Thus, if a caregiver selected more than one race/ethnicity for their infant, the variable was recoded into a new variable, multi-racial/multi-ethnic. Infant weight as birth was entered as pounds and ounces. This variable was recoded into total ounces. Mothers or caregivers were able to report on prematurity status of the infant, with additional space for more information (e.g., weeks of gestation). Caregivers reported on the household income by selecting from a predetermined range of values (as reported above). Finally, caregiver educational status was self-reported via open text response to the prompt "please tell us about your highest level of education completed". Individual responses were then grouped by educational achievement as detailed above (Table 3).

Stressor task. To elicit infant frustration and anger, the arm restraint procedure (Goldsmith & Rothbart, 1999) was administered when the infant was six months of age. In this

procedure, mothers were asked to place their infants in a car seat, secure the straps for safety, and position themselves behind the infants' field of view. A research assistant proceeded to gently restrain the infant by holding the infant's arms down to the sides. After two minutes of arm restraint, or 30 seconds of hard crying (i.e., when the infant's cry is at peak distress and the infant cannot increase the distress level any further), the research assistant released the infant's arms. The infant was then left in the car seat for 60 seconds to allow for the use of self-regulatory behaviors such as self-soothing (e.g., sucking thumb). Finally, the mother was allowed to parent her infant in whatever way she thought appropriate. The whole procedure was video-taped for later coding.

Salivary Analytes

Infant saliva samples collected at six-months were assayed for salivary cortisol and salivary alpha amylase using a commercially-available assay (Salimetrics LLC, Carlsbad, CA). Samples were assayed in duplicate for cortisol and the average of the duplicates was used in all analyses. Samples were assayed in singlet for alpha-amylase.

Salivary alpha amylase. In order to best understand sympathetic activity from sAA, it is important to take into account the confounding influence of salivary flow rate (which is primarily governed by the parasympathetic nervous system; Bosch, Veerman, de Geus, & Proctor, 2011). Salivary flow rate is the amount of saliva produced over time. In order to correct for salivary flow rate, the measured level of sAA (U/mL) was multiplied by the saliva flow rate (mL/min). For the present samples, saliva collection procedures were standardized as 90 seconds for each sample. Thus, salivary flow rate was calculated as milligrams of saliva over 1.5 minutes. This conversion results in sAA output (U/min; Beltzer, Fortunato, Guaderrama, Peckins, Garramone, & Granger, 2010). This output measure was used in all statistical analyses. Next, in order to

calculate a sAA reactivity score, each individual's alpha amylase scores across the four post-stressor time points (10, 20, and 30 minutes) was examined, in order to identify peak reactivity (i.e., highest concentration of alpha amylase). Previous studies have shown that sAA concentration can peak as quickly as 10 minutes after a stressor has occurred (Granger & Kivlighan, 2003). Thus, identifying the largest concentration of sAA within the first 30 minutes of stressor task likely captures an individual's peak. In order to account for individual variation in starting (i.e., baseline) levels of the salivary analyte, a reactivity score was calculated in the following manner: individual's peak reactivity minus individual's baseline score. This sAA reactivity score was used in all analyses (e.g., Engert, Smallwood, & Singer, 2014). See Table 4 for frequency counts.

Salivary cortisol. All cortisol samples were assayed in duplicate and averaged across the two assays for a final cortisol concentration score per time point. As with sAA, each individual's cortisol scores across the four post-stressor time points (10, 20, 30, and 40 minutes) were examined, in order to identify peak reactivity (e.g., highest concentration of cortisol). For cortisol, HPA activation can result in peak cortisol in the bloodstream as soon as 20 minutes post-stressor (Gunnar & Cheatham, 2003). Thus, peaks within 40 minutes post stressor were considered in order to identify an individual's cortisol peak. In order to account for individual variation in starting (e.g., baseline) levels of cortisol, reactivity score was calculated in the following manner: individual's peak reactivity minus individual's baseline score. This cortisol reactivity score was used in all analyses. See Table 4 for frequency counts.

Table 4

Cortisol and Salivary Alpha Amylase Peak Times

	Cortisol		Salivary Alpha Amylase	
Variable Name	Frequency	Percent	Frequency	Percent
10 Minutes	76	34.7%	87	39.7%
20 Minutes	58	26.5%	79	36.1%
30 Minutes	42	19.2%	51	23.3%
40 Minutes	39	17.8%	-	-
Missing	4	1.8%	2	.9%
	N = 219		*N* = 219	

Infant socio-emotional functioning. Infants' social and emotional functioning at 12 months was assessed via maternal self-report on the internalizing and externalizing behavior scales of the Infant-Toddler Social and Emotional Assessment (ITSEA; Carter & Briggs-Gowan, 1993, 2000). This parent-report instrument was developed to assess social and emotional problems and competencies in infants and children ages 0 – 3. The complete scale includes 166 items, which are rated on a three-point scale (0 = *not true/rarely*, 1 = *somewhat true/sometimes*, and 2 = *very true/often*). Additionally, each item allows parents to respond "*no opportunity*" if they have not witnessed a particular behavior (such as their child interacting with peers). ITSEA raw scores can be transformed into T-scores in order to assess severity and frequency of behaviors across the four domains. On average, this assessment takes approximately 30 minutes to complete (Carter, Briggs-Gowan, Jones, & Little, 2003).

The ITSEA assesses behaviors in four broad domains (internalizing, externalizing, dysregulation, and competencies) and includes indices to assess more severe problems (maladaptive, atypical behavior, and social relatedness). The internalizing domain contains 30 items and is composed of four subscales: General Anxiety, Depression/Withdrawal, Separation Distress, and Inhibition to Novelty. The externalizing domain contains 24 items, and it is composed of Activity/Impulsivity, Aggression/Defiance, and Peer Aggression subscales.

Dysregulation is composed of Sleep, Eating and Sensory Sensitivity, and Negative Emotionality. Competencies include Attention, Limitation/Play, Mastery Motivation, Compliance, and Prosocial Peer Relations scales. The Maladaptive index includes items that assess for toileting issues, sexualized behavior, pica, Tourette's syndrome, and post-traumatic stress disorder. The Social Relatedness and the Atypical Behavior indices assess for Pervasive Developmental Delay and Autism. Only the internalizing and externalizing scales are examined in the present study.

The ITSEA internalizing and externalizing scales have shown good construct validity. The internalizing scale of the ITSEA is highly and negatively correlated with the Colorado Child Temperament Inventory, sociability scale (Buss & Plomin, 1975) and with the CBCL internalizing subscale (Carter et al., 2003). Similarly, the ITSEA externalizing scale has high concordance with the externalizing scale of the Child Behavior Check List 1-1.5 (CBCL; Achenbach 1991; Carter et al., 2003). Previous studies utilizing the ITSEA have shown that these scales have good internal consistency (Cronbach's alpha of 0.87 and 0.80 for externalizing and internalizing, respectively; Carter et al., 2003). Internal consistency for the present study was 0.84 for externalizing and 0.67 for internalizing, suggesting adequate internalizing consistency. Test-retest reliability ranged from 0.82 - 0.90 for the four main domains, and from 0.69 -0.85 for the subscales (tested 44 days apart; Carter et al., 2003).

Externalizing and internalizing global scores were derived according to the standard procedures detailed in the user's manual (Carter & Briggs-Gowan, 2005). First, items that needed reserving for the purpose of scoring were identified and processed. Then ITSEA subscales (activity/impulsivity, aggression/defiance, peer aggression, depression/withdrawal, general anxiety, separation distress, and inhibition to novelty) were scored according to scoring directions (average across items). Rules for number of missing items allowed were followed for

each subscale according to the ITSEA manual (e.g., generally no more than two items per subscale; Carter & Briggs-Gowan, 2005). Internalizing and externalizing domain scores were derived from averaging the appropriate subscales. The internalizing domain score is composed of the depression/withdrawal, general anxiety, separation distress, and inhibition-to-novelty subscales. The externalizing domain score is composed of activity/impulsivity, aggression/defiance, peer-aggression subscales.

Infant negative behavioral reactivity. Infant negative behavioral reactivity was coded from videotapes of the arm restraint task (prior to the reunion with the mother) by coding length and intensity of infant behavioral reactivity (vocal and facial behaviors; Braunger-Rieker & Stifter, 1996; Sherman et al., 2013). First, all infant vocalizations and facial expressions were coded for quality of distress every five-seconds on a four-point scale (0 = *no vocalizations, no negative facial affect*, 1= *mild vocal and/or facial negative reactivity*; 2 = *moderate vocal and/or facial negative reactivity*, and 3 = *high vocal and/or facial negative reactivity*). Mild negative reactivity is negative reactivity of low intensity, so that the infant shows a fussy facial expression, a fussy vocalization, or both. A fuss with a flat expression may also be counted here. Moderate negative reactivity is characterized as moderate in intensity, including crying, an open squared mouth, and open or partially opened eyes. Finally, high negative reactivity refers to high-intensity cues, such as screams, closed or partially closed eyes, and wide or open mouth. Breath holding, tears, silent screams, and color changes in the face would also indicate high intensity reactivity. Average negative reactivity was calculated by averaging negative reactivity segment ratings across five-second epochs to obtain general affective display.

Behavioral coding of infant videotaped behaviors began immediately following the start of the task (i.e., when the research assistant places her hands on the infant's arms to restraint

them). Coding ended one minute after the research assistant has released the infant's arms, or as soon as the mother comes to the baby or the research assistant turns the car seat away from the camera. Each infant's negative behavioral reactivity was coded from videos of the arm restraint task by trained coders. Five percent of cases (i.e., 10 cases) were used as training cases. In this process, randomly matched raters independently coded assigned cases. These independent codes were used to calculate coder reliability during training. Coders then met with their partners to review their ratings and discuss discrepancies. A final rating was assigned via consensus. Once raters achieved ICC scores above .90, they were allowed to code as independent coders. Reliability was checked throughout the coding process to monitor for coder drift. In the final dataset, 15% (i.e., 31 cases) were double coded to calculate overall rater reliability. Intra-class correlation (ICC) estimates and 95% confidence intervals were calculated based on a single-rater, absolute agreement, two-way random effects model. ICC values less than 0.5 indicate poor reliability, between 0.5 and 0.75 moderate reliability, between 0.75 and 0.9 good reliability and, values above 0.9 indicate excellent reliability (Koo & Li, 2016). For the present study, ICC estimates were .98 (CI = .96 - .99), indicating excellent inter-rater reliability.

Previous research has validated the use of distress vocalizations as a measure of negative affect and arousal (Calkins, Fox, & Marshall, 1996; Stifter & Braungart, 1995). Specifically, previous empirical work with infants has shown that the quality of distress vocalizations (e.g., intensity of behavioral reactivity) is linked to other markers of distress such as duration of crying and escape attempts (i.e., from an arm restraint procedure; Porter et al., 2009). Latency to distress, duration of negative vocalizations, and intensity of distress measures at six months via laboratory observation predicted seven-year frustration and anger (Rothbart et al., 2000).

Procedures

The study procedures consisted of two laboratory visits that occurred approximately six months apart, the first one taking place when the infant was six-months old. Mothers who were interested in participating in the study after receiving information from flyers, posters, and/or speaking with a research staff member were screened for eligibility over the phone. If mothers were eligible to participate, they were invited to come into the laboratory to take part in the first phase of the study.

The first laboratory visit, which occurred when the infant was six-months old, lasted approximately two hours. Shortly after arriving to the lab, mothers were asked to provide informed consent for themselves and consent for their infant to participate in the study. If a mother of an infant was not yet 18 years old, her parents were asked to provide informed consent for the mother, who was then able to assent for herself and consent for her infant's participation in the study.

Although mothers and their infants completed various tasks and activities during their first visit to the laboratory, only those tasks relevant to the current study are described here. Soon after providing informed consent, but before any other laboratory tasks began, baseline saliva samples were collected from both mother and infant. Approximately 30 minutes into their lab visit infants were administered a frustration/anger task (i.e., arm restraint procedure). The remainder saliva samples were collected in 10-minute increments beginning 10 minutes after the arm restraint ends (at 10, 20, 30, and 40 minutes after the research assistant releases the infant's arms from her grasp). At this time, mothers were asked to complete a series of questionnaires about herself and her child, including a health questionnaire to provide information about the infant's current health status. When the infant is approximately 12-months old, the dyads were

asked to return to the laboratory in order to complete follow-up measures. At that visit, mothers completed a socio-emotional measure about their infants (ITSEA).

Each part of the study was accompanied by a monetary incentive. Specifically, mothers received $50 for participating in the two-hour, six-month laboratory visit and $60 for participating in three 30-minute home visits (not included in the current study). Mothers received $70 for their 1.5 hour laboratory visit when their child is 12-months old, and finally, they received $80 for two 1.5 hour home visits (not included in the current study) following the second laboratory visit. In total, research participants could be compensated up to $260 for their time over the four parts of the study.

Saliva Collection. Saliva samples were collected at five time intervals. The first sample was collected shortly after the dyad arrived in the lab and served as a baseline measure of salivary alpha amylase and cortisol. The final four samples were collected in 10-minute intervals beginning 10 minutes post stress task (i.e., 10 minutes after the experimenter releases the infant's arms during the arm restraint task). The four sample times are as follows: 10 minutes post arm restraint, 20 minutes post arm restraint, 30 minutes post arm restraint, and finally, 40 minutes post arm restraint. Infants mouthed polymer swabs (125 mm in length), which were designed to collect saliva from the interior of the mouth. The swabs were immediately deposited into swab storage tubes and frozen to -20° C at the laboratory in Harrisburg, PA. All data collection occurred in the afternoon to control for the diurnal pattern of the salivary analytes. Frozen samples were transported on dry ice to a laboratory to be assayed (Salimetrics, Carlsbad, CA).

Design

For this study, I used classification and multivariate analysis of variance designs. Data were analyzed using M*plus* for Windows, version 7.4 (Muthén & Muthén, 2015). Preliminary

analyses were conducted to identify any potential effects of situational (e.g., family income) and demographic (e.g., infant gender) variables on the salivary analytes. Bivariate correlations and t-tests were conducted between potential covariates and the change from baseline to post-distress in sAA and cortisol. Based on published recommendations and previous studies (e.g., Granger et al., 2007; Gunnar & Cheatham, 2003), the following variables were considered as potential covariates: infant gender, infant race/ethnicity, infant age, family income, and infant weight at birth. Any variables found to explain additional variance in changes in sAA and cortisol were included in the main analyses.

The first stage of data analysis was to conduct a Latent Profile Analysis (LPA; Williams & Kibowsky, 2016) to identify homogenous subgroups of participants based on their scores in the following variables: change in salivary alpha amylase post-stressor and change in salivary cortisol post-stressor. LPA is an empirically-driven approach to identifying latent categories or groups of people who share common characteristics (in this case, similar changes in sAA and cortisol in response to a stressor). Groups are defined via maximum likelihood estimation (Little & Rubin, 1989). Key fit statistics have been selected *a priori* based on available recommendations.

LPA uses an iterative approach whereby classes are added to each model in order to determine which model fits the data best (Williams & Bikowsky, 2016). In order to determine the ideal number of groups for this sample, each model was evaluated using the Lo-Mendell-Rubin Adjusted Likelihood Ratio Rest (LMRT; Lo, Mendell, & Rubin, 2001) and the Bootstrapped Likelihood Ratio Test (BLRT; McLachlan & Peel, 2000). These two fit statistics compare the fit of a model of interest (e.g. a three group model) with a comparison model that is composed of one less group (e.g., a two group model). The LMRT and the BLRT produce a *p*-

value that indicates which model fits better ($p < .05$ suggest the model with more classes is a better fit). Additionally, the Akaike information criteria (AIC; Akaike, 1974) and the sample size-adjusted Bayesian information criteria (sBIC; Schwarz, 1978), two descriptive fit indices, were considered to understand model fit. For both AIC and sBIC, smaller values suggest better fit. LPA was conducted via M*plus* 7.4 (Muthén & Muthén, 2015).

After selection of best fitting model, a factorial multivariate analysis of variance (MANOVA) test was used to examine links between group membership and the outcome measures (externalizing and internalizing behaviors). A MANOVA is a statistical test used to assess differences in dependent variables by groups. To run a MANOVA, one must first check that statistical assumptions are met. In order to assess multivariate normality, I checked the skewness and kurtosis of each dependent variable (which should fall between -2 and +2; Lomax, 2001). I also examined the probability plots of each dependent variable and a scatterplot to assess bivariate normality (Stevens, 2009). Finally, I tested homogeneity of covariance matrices by requesting Box's test in SPSS (Stevens, 2009). Then, I conducted a factorial MANOVA test with the externalizing and internalizing scores as dependent variables and class membership and income as independent variables.

Exploratory Analyses

In order to explore the role of behavioral variables in predicting internalizing and externalizing, a second LPA was conducted with the addition of the behavioral reactivity variable, average behavioral distress. Model fit was assessed one more time following the same steps and criteria that were used in the first LPA. Then, a second factorial MANOVA was conducted with the new classes as independent variables and the same outcome variables as the first test (i.e., internalizing and externalizing total scores).

Chapter IV

Results

Data Preparation

To ensure high data quality, data were entered into a database in duplicate by different research assistants and compared via CompareIt software (Version 3.0, Grig Software, 2006) a software program that identifies incongruent data fields making data entry errors easier to identify. Discrepancies were resolved by checking the original or raw data and entering the correct value.

Missing Data

The quantity and nature of missing data per variable was examined. Missing data per variable can be found on Table 5.

Table 5

Missing Data per Variable

Study Variable	Missing	Percent	Variable Total
Baseline Alpha Amylase	1	0.5%	218
Baseline Cortisol	3	1.4%	216
Reactivity Alpha Amylase	2	0.9%	217
Reactivity Cortisol	4	1.8%	215
Negative Behavioral Reactivity	22	10.0%	197
Internalizing	87	39.7%	132
Externalizing	87	39.7%	132
Covariates			
Infant Weight	32	14.6%	187
Household Income	19	8.7%	200
Infant Race/Ethnicity	13	5.9%	219

Note: Highest variable $N = 228$.

Missing data in latent profile analyses. Latent Profile Analysis allows for missing data on the observed variables so long as the data are missing at random (Roesch, Villodas, & Villodas, 2010). Exploratory analyses were conducted in order to qualify the missing data in the

observed variables. Rhoads (2012) highlights the lack of empirical tests that ascertain whether variables are missing at random (MAR) and explains that analysis examining whether variables are missing completely at random (such as Little's MCAR test) rest on a priori MAR assumption. Following this assumption, I conducted a Little's Missing Completely at Random (MCAR) analysis on cortisol reactivity, alpha amylase reactivity, and negative behavioral reactivity. Little's MCAR analysis tests the hypothesis that any missing data cannot be predicted by another variable (Little, 1988, as cited by Garson, 2015). If this test results in a non-significant finding, data can be deleted listwise without affecting analyses. For the variables of interest, cortisol and salivary alpha amylase reactivity, and negative behavioral reactivity, Little's MCAR was not significant, suggesting that data cannot be predicted by another variable in the study (Little's MCAR test: $\chi^2 = 6.98$, $df = 7$, $p = .43$). A decision to proceed with LPAs was made based on the available evidence. LPAs use full information maximum likelihood estimation to deal with missing data.

Missing data in analysis of variance. Although the overall number of participants in this study was 228, due to both the longitudinal nature of the design and other factors such as mothers declining to return for 12 month lab visits and difficulties in contacting the mothers in this highly transient group, some cases were lost on follow up. Overall, 87 cases were missing for the dependent variables of interest, internalizing and externalizing behaviors. Of those 87 missing cases, six cases were intentionally dropped from the final sample due to the project ending prior to the infant becoming eligible to complete a 12 month lab visit. The final sample size is reduced to $n = 141$ for the multivariate analyses of variance.

Data Normality Check, Descriptive Statistics, and Correlation

Descriptive statistics are reported in Table 6. Normality of continuous data was examined via descriptive statistics. Acceptable ranges for skewness and kurtosis were decided *a priori* as -2 to +2 (Weston & Gore, 2006). Acceptable ranges of skewness and kurtosis were observed for the outcome variables, internalizing and externalizing behaviors. The outcome variables were transformed into T scores and examined for scores falling into the cut off range "of concern" which, in the ITSEA, is scores above 1.5 standard deviations (T scores greater than 65). Results show that 14.2% of infants had externalizing T scores greater than 65 and 6.8% had internalizing T scores greater than 65. The dependent variable, negative behavioral reactivity also demonstrated skewness and kurtosis within acceptable ranges. Thus, these variables are likely normally distributed.

As is common in the extant literature (e.g., Gordis et al., 2008; Miller & Plessow, 2013), the salivary alpha amylase and the cortisol variables demonstrate skewness and kurtosis levels that fell outside acceptable ranges, suggesting these variables were not normally distributed. As can be seen from Table 6, the alpha amylase baseline and reactivity variables show slight kurtosis, whereas the cortisol baseline and reactivity variables show large kurtosis. The salivary variables were transformed in order to achieve normal distribution. The descriptive statistics for the log transformed salivary analyte variables can be found in Table 7. Logarithmic transformation achieved skewness and kurtosis within normal limits. Additionally, and following procedures identified in the literature (Fortunato et al., 2008) outliers < or > 3 SD were identified and recoded to the next value in the respective distribution in order to reduce undue influence of outliers while also retaining cases.

Table 6

Descriptive Statistics for Dependent and Independent Variables

	n	Min	Max	*M*	*SD*	Skewness	Std. Error	Kurtosis	Std. Error
Baseline sAA	218	.70	210.00	54.88	44.78	1.41	.16	2.05	.33
Baseline Cortisol	216	.03	2.70	0.32	0.42	3.90	.17	17.00	.33
Reactivity sAA	217	.001	226.00	40.70	53.73	1.73	.16	2.40	.33
Reactivity Cortisol	215	.001	2.88	0.33	0.54	3.01	.17	10.07	.33
Negative Behavioral Reactivity	197	.00	3.00	1.37	0.89	0.15	.17	-1.16	.34
Internalizing Global	132	.04	1.08	0.54	0.21	0.07	.21	-0.46	.42
Externalizing Global	132	.00	1.42	0.56	0.28	0.71	.211	0.50	.42

Table 7

Descriptive Statistics for Log Transformed Variables

	n	Min	Max	*M*	*SD*	Skewness	Std. Error	Kurtosis	Std. Error
Log Transformed Baseline sAA	218	-0.15	2.32	1.56	0.45	-1.04	.16	1.34	.33
Log Transformed Baseline Cortisol	216	-1.49	0.43	-0.66	0.34	0.87	.17	1.23	.33
Log Transformed Reactivity sAA	217	-6.91	5.42	1.05	4.37	-1.12	.16	-0.41	.31
Log Transformed Reactivity Cortisol	215	-6.91	1.06	-2.70	2.44	-0.74	.17	-0.70	.33

A bivariate correlation matrix for log transformed baseline cortisol, log transformed baseline sAA, negative behavioral reactivity, and outcome variables was examined in order to explore relationships between study variables. Cohen and Cohen (1983) suggest small (1%), medium (9%), and large (25%) as benchmarks for interpreting effect sizes. As can be seen in Table 8, internalizing and externalizing were significantly and positively correlated (r^2 = .15, a medium effect size). Internalizing behavior problems were correlated with negative behavioral reactivity (r^2 = .03, a small effect size), although the correlation approached but did not attain conventional levels of significance. Externalizing behavior problems were not significantly correlated with negative behavioral reactivity. Log transformed baseline cortisol and internalizing were significantly correlated in a positive direction (r^2 = .04, a small effect size). Log transformed baseline cortisol and externalizing were not significantly correlated. Log transformed baseline sAA was not correlated with the two outcome variables.

Table 8

Bivariate Correlations Baselines, Behavior, and Outcome Variables

		Baseline sAA	Baseline Cortisol	Behavioral Reactivity	Intern	Extern
Baseline sAA	Correlation	1	.03	-.14	.09	.05
	p		.60	.05	.30	.60
	n		215	196	132	132
Baseline Cortisol	Correlation		1	.11	.19*	.10
	p			.12	.03	.26
	n			196	132	132
Behavioral Reactivity	Correlation			1	.16^	.09
	p				.08	.33
	n				124	124
Internalizing	Correlation				1	.39**
	p					.00
	n					132

Externalizing	Correlation	1
	p	
	n	

Note. Log transformed salivary variables were used in these correlations.
^$p < .10$. *$p < .05$. **$p < .001$.

As detailed in the method section, salivary reactivity scores were calculated by subtracting the baseline value from the highest post stressor value. This resulted in a difference score between pre and post stressor. Given that some infants had elevated levels of cortisol or sAA at baseline (e.g., due to finding the laboratory visit stressful, or experiencing a stressor prior to arriving in the lab) or that some infants may not have experienced later tasks a particularly stressful (and cortisol tends to fall over the course of the day), some infant reactivity scores resulted in a negative value (i.e., baseline scores were higher than any post stressor scores). As one of the primary aims of this study was to better understand the stress reactivity pattern negative reactivity scores were converted to near zero (e.g., .001 as a mark for no reactivity following the stressor). This allowed for salivary analyte scores to be transformed in order to address non-normality, a step that is common in working with salivary analytes and that was necessary for the present study. This procedure, however, prevented examining a correlation between reactivity scores and other variables, as the range of values now included a subsample of infants with a constant value, effectively forming two groups. In order to explore how the physiological reactivity variables related to the rest of the study variables, a correlation was conducted with the raw pre and post task difference scores (e.g., those containing negative values). Descriptive statistics for these two variables are found on Table 9.

Table 9

Descriptive Statistics for Difference Scores

	n	Min	Max	*M*	*SD*	Skewness	Std. Error	Kurtosis	Std. Error
Cortisol Reactivity Difference Score	215	-0.72	2.85	0.29	0.57	2.59	.17	8.36	.33
sAA Reactivity Difference Score	217	-125	226	33.85	62.07	0.91	.16	1.54	.33

These two variables were then used in a correlation to explore links between baseline and reactivity scores, and between reactivity scores and other study variables. It should be noted that the cortisol variable displays positive kurtosis, even by more relaxed standards (West, Finch, & Curran (1995) have recommended interpreting a substantial departure from normality when kurtosis is greater than absolute value 7). Baseline-reactivity correlations can be found on Table 10. As seen in Table 10, cortisol reactivity is significantly and positively related to cortisol baseline (r^2 = .06, a small effect size) and sAA baseline is negatively related to cortisol reactivity (r^2 = .03, a small effect size). Cortisol baseline and sAA baseline were not significantly relate. Similarly, sAA reactivity and sAA baseline were not statistically significantly related. Finally, cortisol reactivity was not significantly linked with sAA reactivity.

Table 10

Bivariate Correlations for Baseline and Reactivity Variables

		sAA Reactivity	Cortisol Reactivity	sAA Baseline	Cortisol Baseline
sAA Reactivity Difference Score	Correlation	1	.06	.07	.05
	p		.35	.29	.49
	n		213	217	1214
Cortisol Reactivity Difference Score	Correlation		1	-.16*	.25**
	p			.02	.00
	n			214	214
sAA Baseline	Correlation			1	.03

	p	.60
	n	214
Cortisol Baseline	Correlation	1
	p	
	n	

Note. ^$p < .10$. *$p < .05$. **$p < .001$.

Correlations between reactivity and other variables can be found on Table 11. Cortisol reactivity was marginally linked with behavioral reactivity ($r^2 = .02$, a small effect size) and sAA reactivity was marginally and negatively linked with behavioral reactivity ($r^2 = .02$, a small effect size). Cortisol reactivity and sAA reactivity were not significantly related with either internalizing or externalizing.

Table 11

Bivariate Correlations for Reactivity Variables and Outcome Variables

		sAA	Cortisol	Behavioral Reactivity	Intern	Extern
sAA Reactivity Difference Score	Correlation	1	.08	-.14^	.13	.02
	p		.26	.06	.13	.85
	n		213	195	132	132
Cortisol Reactivity Difference Score	Correlation		1	.13^	.08	.12
	p			.08	.36	.18
	n			194	131	131
Behavioral Reactivity	Correlation			1	.15^	.09
	p				.08	.33
	n				124	124
Internalizing	Correlation				1	.39**
	p					.00
	n					132
Externalizing	Correlation					1
	p					
	n					

Note. Variables in their original values were used in these correlations.
^$p < .10$. *$p < .05$. **$p < .001$.

Partial correlations were examined to further explore the relations between negative behavioral reactivity and physiological reactivity. Partialing out the variance explained by cortisol reactivity, sAA reactivity was negatively related to behavioral reactivity (pr^2 = -.14, p = .04, r^2 = .02, a small effect size). Partialing out the variance explained by sAA reactivity, cortisol is positively related to negative behavioral reactivity (pr^2 = .14, p = .05, r^2 = .02, a small effect size).

Covariates and physiological response to challenge for sample. Data on infant body temperature were collected in order to exclude any infants who were sick at the time of the study. However, no febrile infants (i.e., body temperatures above 100.4, American Academy of Pediatrics, 2015) were identified in the present sample. Thus, no infants were excluded due to illness. A bivariate correlation was conducted between infant weight at birth and sAA reactivity, cortisol reactivity, and negative behavioral reactivity. Infant weight at birth was positively correlated with negative behavioral reactivity $r(176) = .16$, $p = .03$, $r^2 = .03$, a small effect size. Student's *t*-tests were conducted to examine potential mean differences in sAA reactivity, cortisol reactivity, and negative behavioral reactivity based on infant gender. No significant gender differences were found on any of these variables.

Racial and ethnic group differences on sAA and cortisol baseline were examined via the Kruskal-Wallis Test, a non-parametric alternative to the one-way ANOVA. A non-parametric test was necessary due to the small number of infants who were identified as being Asian/Asian American (thereby violating the homogeneity of variance assumption needed for an ANOVA test). No significant differences were found in cortisol baseline by infant race or ethnicity. Significant differences in sAA baseline by race/ethnicity were found $\chi^2(4) = 10.96$, $p = .03$. Pairwise comparisons using Dunn's (1964) procedure with Bonferroni correction for multiple

comparisons were performed. The adjusted *p* value for statistical significance was .005 (.05 divided by 10 possible comparisons). Post hoc analysis revealed significant differences only between White (median = 1.84) and Bi-Multiracial (median = 1.54) infants on sAA baseline (p = .004). There were no other significant racial/ethnic group differences found. An additional ANOVA test was conducted in order to examine differences in cortisol and sAA baseline by income group. No significant group differences emerged.

An additional test was conducted between race/ethnicity and cortisol, sAA, and behavioral reactivity. No significant differences were found in sAA reactivity or cortisol reactivity by infant race or ethnicity. Significant differences in negative behavioral reactivity by race/ethnicity were found $\chi^2(4) = 11.89$, $p = .002$. Pairwise comparisons using Dunn's (1964) procedure with Bonferroni correction for multiple comparisons were performed. The adjusted *p* value for statistical significance was .005 (.05 divided by 10 possible comparisons). Post hoc analysis revealed significant differences only between African American (median = 0.92) and White (median = 1.84) infants on ratings of negative behavioral reactivity (p = .004). There were no other significant racial/ethnic group differences found. See Table 12 for a full list of values. An additional Kruskal-Wallis test was conducted in order to examine differences in sAA, cortisol, and negative behavioral reactivity by income group. No significant group differences emerged.

Table 12

Kruskal-Wallis Test of Infant Race/Ethnicity on Target Variables

Variable Name	Chi-Square	*df*	*p*
Cortisol Baseline	6.51	4	.16
sAA Baseline	10.96	4	.03
Cortisol Reactivity	2.56	4	.63
sAA Reactivity	5.53	4	.24
Negative Behavioral Reactivity	11.90	4	.02

	n	Median Behavioral React Score	*n*	Median sAA Baseline
Asian/Asian American	3	1.47	3	1.31
Black/African American	78	.92	82	1.69
White	31	1.84	36	1.53
Latino/a	28	1.65	32	1.68
Bi-racial/Multiracial	46	1.54	53	1.71

In summary, preliminary analyses identified two covariates, infant race/ethnicity and infant weight at birth. Infant gender did not appear to be linked to the variables of interest in this sample. Household income was not identified as a covariate by preliminary examination; however, given the documented links between family income and behavioral and physiological variables (cortisol; Fernald & Gunnar, 2009), income was retained as a covariate. Thus, household income was included as a covariate in the first MANOVA (which examined differences in internalizing and externalizing based solely on classes based on patterns of salivary reactivity that were identified in the first LPA). (Note that infant weight at birth and infant race/ethnicity were planned as covariates in the second MANOVA, as these two variables were linked with negative behavioral reactivity. Because the second LPA analysis, however, showed that the addition of negative behavioral reactivity did not result in any changes to infant membership in the classes identified in the first LPA, a second MANOVA was not conducted).

Manipulation check. Next, in order to examine infant response to the stressor task at the group level, two repeated measures ANOVA with four levels for time (baseline, 10-minutes post, 20-minutes post, 30-minutes post and 40-minutes post) and either sAA or cortisol as the dependent variable were conducted. For cortisol, there was a significant effect for time, Wilks' Lambda=.89, $F(4, 173) = 5.40$, $p < .001$. For sAA, there was a significant effect for time, Wilks' Lambda=.85, $F(4, 173) = 7.43$, $p < .001$.

Table 13

Validity Check for Stressor Task

Variable Name	Wilks' Lambda	*F*	*df*	*p*
sAA (U/ml)	.85	7.43	4	<.001
Cortisol (μg/dl)	.89	5.40	4	<.001

Note. U/ml = units per milliliter. μg/dl = micrograms per deciliter.

Hypothesis Testing Latent Profile Analysis: Salivary Alpha Amylase and Cortisol Stress Response Profiles

An LPA was conducted in order to identify homogenous subgroups of infants based on their physiological response to the stressor task. The goal of utilizing this approach is to identify groups of individuals who are similar to each other in terms of stress reactivity but different from individuals in other groups (Muthén & Muthén, 2007). An LPA is a model-based technique that classifies each individual in a group based on the likelihood of belonging to that group (Orpinas et al, 2014). Although the number of latent groups or classes is not known a priori in exploratory applications of LPA, theory and model fit indices can be used to select the best fitting number of classes (Orpinas et al, 2014).

To determine the ideal number of groups for this sample, fit statistics selected *a priori* were examined. The fit statistics of the latent profiles of a one-class through a four-class solution were requested. First, the Lo-Mendell-Rubin Adjusted Likelihood Ratio Rest (LMRT; Lo, Mendell, & Rubin, 2001) and the Bootstrapped Likelihood Ration Test (BLRT; McLachlan & Peel, 2000), two fit statistics that compare the fit of a model of interest (e.g., a three-class model) with a comparison model that is composed of one less group (e.g., a two-class model) were examined. The LMRT and the BLRT produce a *p*-value that indicates which model fits better ($p < .05$ suggest the model with more classes is a better fit). For the present sample, the LMRT and the BLRT both supported a three-class model.

Additional fit indices were also examined. The Akaike information criteria (AIC, Akaike, 1974) and the sample size-adjusted Bayesian information criteria (sBIC; Schwarz, 1978) suggest better fit in models where these statistics are lower. For this sample, the AIC and the sBIC also supported a 3-class model. Models with greater than 3 classes (e.g., 4-class model and above) resulted in unstable parameters due to classes with too few members (Muthén & Muthén, 2009). These model fit statistics for a 4-class model were not examined because of the unstable parameters. See Table 14 for LPA fit statistics and Table 15 for class means and descriptions.

The ultimate goal of this study was to examine group differences in externalizing and internalizing based on reactivity profile. In order to do this, each individual's posterior probabilities for each class were exported, using the most likely class membership (Nagin, 2005). This can be problematic, as it is introducing error and decreasing precision in forcing each individual to belong to one class, which the LPA handles by modeling the uncertainty (Berlin, Williams, & Parra, 2014). However, if most cases have a high likelihood of belonging to only one class, error related to forced categorization remains minimal (Muthén & Muthén, 2009). Entropy, which is a measure of how well the majority of individuals fit into their forced categories, can be used in order to examine issues with this forced classification. A value close to 1 indicates good classification in that many individuals have probability values close to either 0 or 1 (Muthén & Muthén, 2009). For this LPA, Entropy was .97, suggesting that many individuals have probability values of either 0 or 1 (e.g., high likelihood of belonging to one class). Thus, proceeding with exporting forced class membership to use with the proposed analysis of variance is warranted.

Table 14

Model Selection Fit Statistics for sAA and Cortisol Reactivity for LPA1

Model	AIC	BIC	sBIC	LMRT (*p*)	BLRT (*p*)	Entropy	Class	Membership (%)
1 Class	2255.51	2269.07	2256.39	-	-	1.00	1	219 (100%)
2 Class	1934.46	1958.20	1936.01	<.000	<.000	.99	1	172 (78%)
							2	47 (22%)
3 Class	1873.42	1907.31	1875.62	= .013	<.000	.97	1	47 (21%)
							2	40 (18%)
							3	132 (60%)
4 Class	-	-	-	-	-	-	-	-

Note. AIC = Akaike information criterion; BIC = Bayesian information criterion; sBIC = sample adjusted information criterion; LMRT = Lo-Mendell-Rubin likelihood ratio test. Bootstrap Likelihood Ratio Test. Lower numbers of AIC, BIC, and sBIC signify better fit. A significant LMRT and/or BLRT statistic signifies that the model with more classes is a better fit than a model with one less class.
* A four-class model resulted in unstable parameters due to requesting too many classes.

Table 15

Means, Standard Deviations, and Class Descriptions for LPA1

Variable	Class 1 (*n* = 47) M*(SD)*	Class 2 (*n* = 40) M*(SD)*	Class 3 (*n* = 132) M*(SD)*
Cortisol Reactivity Mean	-3.01(.58)	-6.64(.32)	-1.38(.32)
sAA Reactivity Mean	-6.91(.00)	3.46(.42)	3.18(.36)
Description	No sAA reactivity/ HPA only group	No-to-low Cortisol/Primarily SNS Reactivity Group	Moderate-to-high sAA and moderate-to-high cortisol reactivity

Note. Values are for transformed variables.

Class Descriptions

Class 1: No sAA reactivity, HPA only group (*n* = 47). This class was composed of infants who displayed no salivary alpha amylase reactivity but a varying range of cortisol reactivity to the stressor task. The log transformed alpha amylase reactivity value (class *M* = -6.91) is in the no reactivity range (sample *M* = 1.05, *SD* = 4.37). The log transformed cortisol value (class *M* = -3.01) was in the low-average range for the sample as a whole (sample *M* -2.70, *SD* = 2.44); however, the log transformed class cortisol reactivity values ranged from near zero (-6.91) to high-average (-0.05). Given the wide range of cortisol reactivity, this group is best characterized by the lack of sAA reactivity.

Class 2: No to low Cortisol, primarily SNS Reactivity Group (*n* = 40). This class had infants who had average to high log transformed alpha amylase reactivity to the stressor task, with a class mean of 3.46 (sample *M* = 1.05, *SD* = 4.37). Log transformed class sAA values ranged from a minimum of 0.73 to a maximum of 5.41. This group had no-to-low log transformed cortisol reactivity (class *M* = -6.64, sample *M* = -2.70, *SD* = 2.44). This group is characterized by the lack of cortisol reactivity.

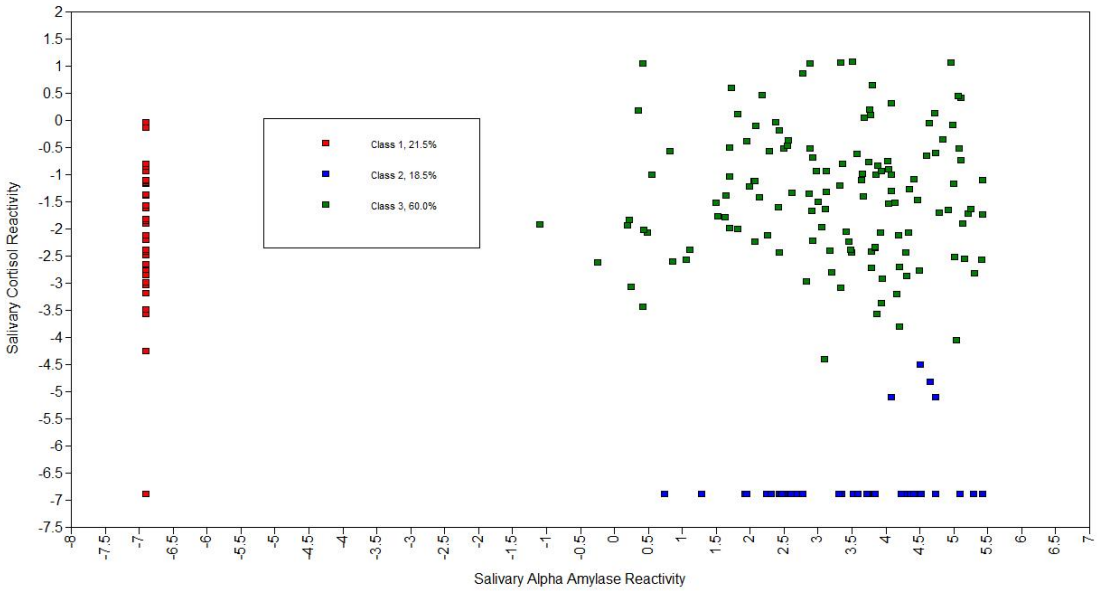
Class 3: Moderate-to-high sAA and moderate-to-high cortisol reactivity (*n* = 132). This class contained the most members and was comprised of infants who displayed moderate to high reactivity on both stress systems in response to the stressor task. The log transformed alpha amylase reactivity mean for this class was 3.18, (sample *M* = 1.05, *SD* = 4.37, range = -1.01 - 5.42). The log transformed cortisol reactivity mean for this class was -1.38 (sample *M* = -2.70, *SD* = 2.44, range = -4.42 - 1.06).

In addition to class means and standard deviations, a scatterplot was requested to examine spread and position of the classes (see Figure 1 for log transformed variables, and Figure 2 for a

scatterplot with original values). As can be seen in the figure, the classes identified by this LPA cluster primarily around the cases that display lack of reactivity from one system or the other, creating two classes from these data (Class 1 and 2). The rest of the data, nearly every case with reactivity on both systems, is then grouped in a third class. Although this 3 class model solution identified three homogenous groups, it provides little information about the infants who did display reactivity across both components of the stress reactivity system. As one of the primary aims of this study was to understand how the two reactivity systems interact, an additional LPA was conducted on the Class 3 cases (those with both cortisol and sAA activity) in order to elucidate whether homogenous groups existed in this subgroup.

Figure 1

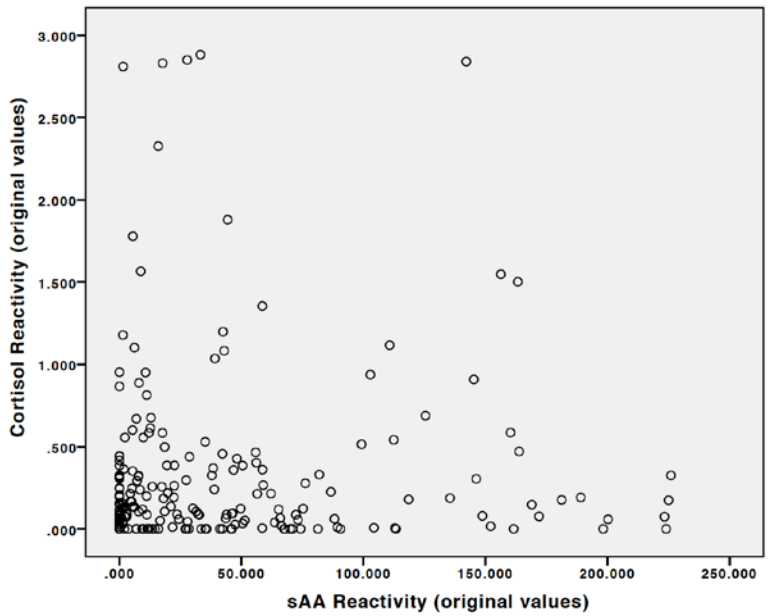
Three Class Reactivity Model Scatterplot



Note.: Values are for transformed variables

Figure 2

Three Class Reactivity Model Scatterplot (Original Values)



Understanding the Classes of LPA1

Classes were examined for significant differences on demographics variables. A one way ANOVA resulted in no significant differences between classes on infant weight at birth. Chi-square tests revealed no significant class differences in family income, race/ethnicity, or infant gender ($p > .05$). In addition, classes were examined on differences in baseline cortisol and sAA. A MANOVA showed significant differences in baseline cortisol and sAA by class, Wilks' Lambda = .92, $F(4, 422) = 4.70$, $p = .001$, partial eta squared = .04, a small effect size. Univariate tests showed significant differences in baseline cortisol by class $F(2, 212) = 4.50$, $p = .012$, partial eta squared = .04, with post hoc Tukey comparisons showing that Class 1 had significantly higher baseline cortisol (log transformed) than Class 2 ($p = .011$) and Class 3 ($p = .045$). Class 2 and 3 were not significantly different. Univariate tests also showed significant differences in baseline sAA by class $F(2, 212) = 4.91$, $p = .008$, partial eta squared = .04, with post hoc comparisons showing that Class 3 was significantly higher than Class 2 ($p = .006$). No differences between Class 1 and 2, or 1 and 3 were found.

Additional LPA (LPA2) with Class 3 identified in LPA1

All of the infants previously classified in Class 3 were identified. Descriptive statistics for this group can be found in Table 16. As can be seen from this table, the skewness and kurtosis for sAA are within acceptable limits. The kurtosis of cortisol remains slightly elevated (West et al., 1996). Thus, for ease of result interpretation, and because skewness and kurtosis for sAA are within acceptable limits, the cortisol and sAA variables used in LPA2 were not log transformed.

The second LPA (LPA2) was conducted on the 132 infants who had previously been categorized in Class 3 (LPA1). The fit statistics for the LPA2 can be found in Table 17. Based

on fit statistics and theory, a three class model was selected as the best fit to the data. Class means, standard deviation, and class descriptions can be found on Table 18.

Table 16

Descriptive Statistics for Class 3 Cortisol and Salivary Alpha Amylase

	n	Min	Max	*M*	*SD*	Skewness	Std. Error	Kurtosis	Std. Error
Reactivity sAA	131	.33	226.00	51.45	56.71	1.45	.21	1.28	.42
Reactivity Cortisol	132	.01	2.88	0.48	0.66	2.41	.21	5.85	.42

Table 17

Model Selection Fit Statistics for sAA and Cortisol Reactivity for LPA2

Model	AIC	BIC	sBIC	LMRT (*p*)	BLRT (*p*)	Entropy	Class Number	Membership (%)
1 Class	1700.50	1712.03	1699.38	-	-	1.00	1	132 (100%)
2 Class	1614.23	1634.41	1612.27	= .024	<.000	.97	1	121 (91%)
							2	10 (8%)
3 Class	1550.12	1578.95	1547.32	= .012	<.000	.96	1	101 (76%)
							2	22 (16%)
							3	9 (6%)
4 Class	1518.06	1555.54	1514.42	= .184	<.000	.96	1	93 (70%)
							2	17 (13%)
							3	16 (12%)
							4	6 (4%)
5 Class	1497.03	1543.15	1492.54	= .235	<.000	.97	1	6 (4%)
							2	16 (12%)

	3	12 (9%)
	4	92 (69%)
	5	6 (4%)

Note. AIC = Akaike information criterion; BIC = Bayesian information criterion; sBIC = sample adjusted information criterion; LMRT = Lo-Mendell-Rubin likelihood ratio test. Bootstrap Likelihood Ratio Test. Lower numbers of AIC, BIC, and sBIC signify better fit. A significant LMRT and/or BLRT statistic signifies that the model with more classes is a better fit than a model with one less class.

Table 18

Means, Standard Deviations, and Class Descriptions for LPA2

Variable	Class 1 (*n* = 101) *M(SD)*	Class 2 (*n* = 22) *M(SD)*	Class 3 (*n* = 9) *M(SD)*
Cortisol Reactivity Mean	0.32(0.18)	0.46(0.32)	2.45(.46)
sAA Reactivity Mean	29.25(1.67)	161.20(3.09)	34.30(3.83)
Description	Low-to-Moderate Cortisol and Low-to-Moderate sAA	Low-to-Moderate Cortisol and High sAA	Moderate-to-high Cortisol and Low-to-Moderate sAA

Note. Means and Standard Deviations are for the original values.

Class Descriptions for LPA2

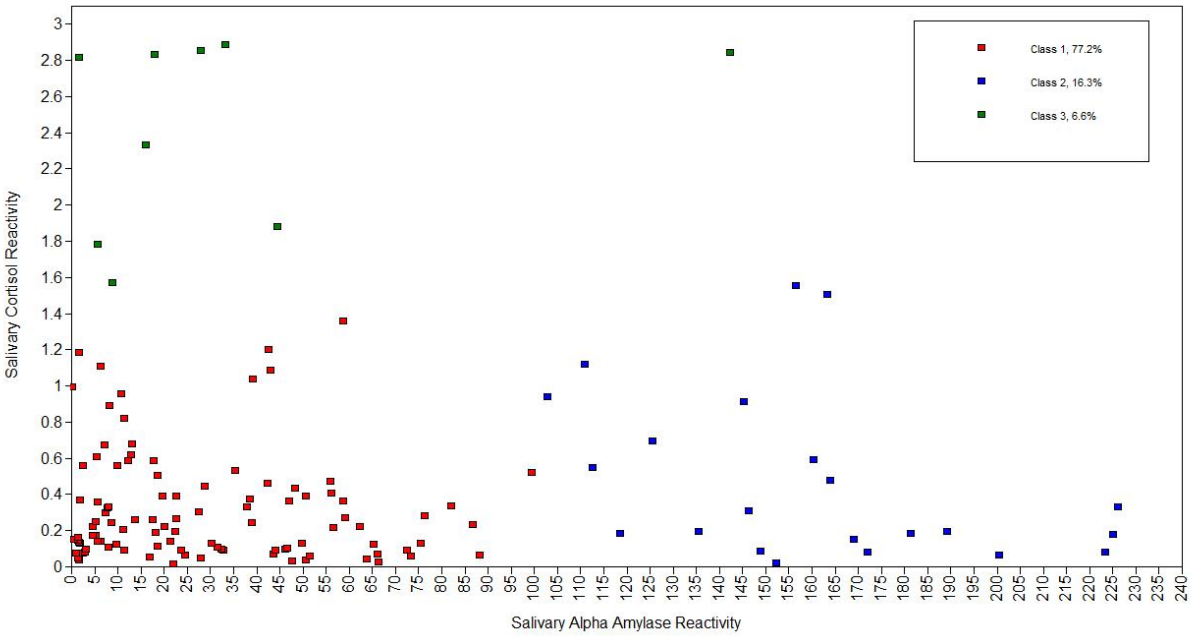
Class 1. Low to moderate cortisol and sAA (*n* = 101). This largest class was composed of infants who displayed a low to low-moderate cortisol reactivity, paired with low-to-moderate sAA reactivity.

Class 2 Low to moderate cortisol and high sAA (*n* = 22). This class was composed of infants who displayed high sAA reactivity paired with low to moderate cortisol reactivity.

Class 3. Moderate to high cortisol and low to moderate sAA (*n* = 9). This smallest class includes infants with low sAA (although one outlier can be seen in the scatterplot Figure 2) paired with moderate to high Cortisol activity.

Figure 3

Three Class Model Scatterplot (LPA2)



Note. These variables are presented here in original values.

Class Mean Differences in Internalizing and Externalizing Behaviors at 12 Months

These analyses examined mean differences in internalizing and externalizing across the three initially identified reactivity classes (in LPA1), as well as the subclasses identified subsequently in LPA2. After identifying the most likely number of classes via the LPA, each individual's class was extracted and used a grouping variable. The mean of each group on the outcome variables of interest (internalizing and externalizing) was then compared via a factorial multivariate analysis of variance (MANOVA). A factorial MANOVA approach was selected (as opposed to a multivariate analysis of covariance) as household income variable violated the homogeneity of regression slopes assumption necessary to use as a covariate in a multivariate analysis of covariance. Household income, which was previously identified as a variable related to infant alpha amylase reactivity scores, was included as an additional categorical dependent

variable. Household income was recoded as a categorical variable (0 = < $10,000; 1 = $10,001-$30,000, 2 = > $30,000) in order to run a MANOVA.

A factorial multivariate analysis of variance was conducted to compare maternal scores on externalizing and internalizing behaviors at 12 months across initial infant stress reactivity and household income groups. The independent variables were internalizing and externalizing and the dependent variables were class membership and household income. Assumptions for MANOVA were checked. The internalizing and externalizing variables produced skewness and kurtosis values that fell within acceptable ranges (-2 to 2; Weston & Gore, 2006), suggesting these variables were normally distributed. Normal probability plots displayed linear distributions, suggesting univariate normality. Bivariate scatter plots appear elliptical in shape, suggesting bivariate normality. Homogeneity of covariance matrices assumption was met (Box's M = 33.48, F = 1.24, p = .19). Results of the factorial MANOVA can be found on Table 19. No significant difference on 12 month externalizing and internalizing scores were found when the interaction between income and stress reactivity class were examined, Wilks' Lambda = *.93*, $F(8, 226)$ = 1.03, p = .41. No significant difference between the three reactivity classes on 12 month externalizing and internalizing scores were found, Wilks' Lambda = *.96*, $F(4, 226)$ = 1.15, p = .33. No significant differences between the three income classes on 12 month externalizing and internalizing scores were found, Wilks' Lambda = *.93*, $F(4, 226)$ = .1.93, p = .11. A Chi-Square test was conducted to examine frequency differences across classes on internalizing and externalizing T scores (i.e., having a T score above the clinical cut off of 65). No significant differences emerged ($p > .05$).

Table 19

Results of the Factorial MANOVA for LPA1 Classes

	Wilks' Lambda	*df*	Error *df*	*F*	*p*	Partial Eta Squared
Class (LPA1)	.96	4	226	1.15	.33	.02
Income	.93	4	226	1.93	.11	.03
Class*Income	.93	8	226	1.03	.41	.03

Note. Dependent variables are internalizing and externalizing scores.

An additional factorial multivariate analysis of variance was conducted to compare maternal scores on externalizing and internalizing behaviors at 12 months across the three secondary classes (identified via LPA2; i.e., those derived uniquely from the moderate-to-large reactivity group from LPA1) of infant stress reactivity and household income groups. Again, the independent variables were internalizing and externalizing and the dependent variables were class membership and household income.

Table 20

Factorial MANOVA Results for LPA2 Classes

	Wilks' Lambda	*df*	Error df	*F*	*p*	Partial Eta Squared
Class(LPA2)	.90	4	126	1.62	.17	.05
Income	.81	4	126	3.47**	.01	.10
Class*Income	.83	4	126	2.04^	.07	.09

Note. Dependent variables are internalizing and externalizing scores.

Significant differences in internalizing and externalizing scores depending on income group emerged, Wilks' Lambda = *.81, F*(4, 126) = 3.47, *p* = .01 (see Table 20). There was a significant main effect for externalizing scores (*F*(2,64) = 7.11, *p* = .002), but not for internalizing scores. This result parallels preliminary results presented earlier, indicating lower income was associated with higher levels of externalizing. An income group by physiological

reactivity class interaction effect that approached, but did not reach, conventional levels of significant emerged in predicting externalizing and internalizing behavior problems, Wilks' Lambda = *.83*, $F(4, 126) = 2.04$, $p = .07$, Partial eta squared = .09, a medium effect size. Univariate tests showed an interaction effect for externalizing scores ($F(3,64) = 2.01$, $p = .10$, Partial eta squared = .09) that approached, but did not attain, conventional levels of significance. No significant interaction effect emerged for internalizing scores.

In order to probe the interaction effect, an interaction variable was constructed by grouping infants by physiological reactivity class and income level. Because some physiological reactivity by income level cells were empty when all three income groups were used, the two income groups over $10,000 per year were combined into one group in order to probe the interaction effect. See Table 21 for frequency counts and cell means on externalizing. A One-way ANOVA examining whether there were significant differences between the cell means on externalizing scores was significant $F(5,66) = 4.55$, $p < .001$, Partial eta squared = .26. Follow up pairwise comparisons showed that the one infant in Class 3/income < $10,000 (cell e) was rated as having more externalizing behaviors than infants in all the others class by income groups (*p* values range between <.001 and .009). In addition, the 25 infants in Class 1/income < $10,000 (cell a) had greater externalizing symptoms than infants in Class 1/income > $10,000 (cell b) ($p = .004$). Additionally, infants in Class 2/income < $10,000 had more externalizing behaviors than infants in Class 1/income > $10,000, but at levels that only approached but did not attain conventional levels of significance, $p = .07$. No significant differences emerged between the rest of the comparisons (see Table 21 for full details). In sum, it is difficult to interpret this interaction effect because examination of the cells suggests that other than a main effect for income (with lower income associated with higher externalizing), there is only one infant in cell

e that has more externalizing behavior that the other infants in every other class. Thus, it is impossible to ascertain whether there is something unique about the interaction of income with levels of physiological reactivity shown in class 3 or whether the one infant in cell 3 reflects a unique anomalous finding.

Table 21

Frequency Counts and Means of Class by Income Cells

	Income < $10,000			Income > $10,000		
	Group	Frequency	*M*	Group	Frequency	*M*
Class 1	(a)	25	$.63_1$	(b)	33	$.43_2$
Class 2	(c)	6	$.64_5$	(d)	4	$.65_5$
Class 3	(e)	1	1.42_4	(f)	3	$.54_5$

Note: Means are for externalizing behaviors. Means that share a subscript do not differ significantly.

Follow up univariate analyses were conducted to explore the main effects identified through the factorial MANOVA. Levene's test of homogeneity revealed lack of homogeneity across groups for this analysis thus, a more stringent alpha level of .025 was used to manage this violation (Field, 2009). Based on the results of the follow up one-way ANOVA, infant externalizing behaviors differed significantly based on income level, $F(2, 69) = 10.62$, $p < .001$, Partial eta squared = .23, a large effect size (Cohen, 1969; Richardson, 2011). Scheffé post hoc comparisons revealed greater externalizing behaviors both for infants whose caregivers reported earning less than $10,000 ($p < .001$) and for infants whose caregivers reported earning between $10,000 and $30,000 ($p = .008$) when compared with infants whose caregivers reported earning $30,000 of more ($p < .001$). There was no significant difference in externalizing behaviors between infants in the under $10,000 group and those in the $10,000 to $30,000 group ($p > .05$).

Of note, although the omnibus test of group differences across reactivity levels (i.e., differences between the classes identified through LPA2) was not significant, an examination of the univariate tests showed a trend for group differences in externalizing behaviors across reactivity groups ($F(2,64)= 2.78$, $p = .07$, partial eta squared = .08, a small-to-medium effect size). Post hoc pairwise comparisons did not identify any significant differences, however. When a multivariate test is not significant but a univariate test is, Tabachnick and Fidell (2001) suggest that there may be insufficient power to detect relationships at the multivariate level and encourage researchers to offer the significant univariate test as a guide for future research.

Finally, a Freeman-Halton extension of the Fisher's Exact test was conducted to examine frequency distributions across classes on internalizing and externalizing T scores (i.e., having a T score above the clinical cut off of 65). No significant differences in probability distribution emerged for internalizing ($p > .05$). The frequency distribution across classes for externalizing T scores above 65 was significant ($p = .05$), suggesting that the number of infants showing externalizing behaviors in the "at risk" category varied by class. A Fisher's Exact Test showed a significant frequency difference between infants "at risk" and those not at risk in Class 1 ($p < .001$). Frequencies in Class 2 and 3 between "at risk" and those not at risk were not different than expected ($p > .05$).

Table 22

Frequency of T Scores above and below 65 across classes

	T < 65	T > 65
Class 1	52	9
Class 2	7	4
Class 3	2	2

Latent Profile Analysis: Behavioral Reactivity, Salivary Alpha Amylase and Cortisol Stress Response Profiles (LPA3)

To test the second set of hypotheses, a third LPA was conducted in which the variable Negative Behavioral Reactivity was added as an indicator. As before, fit statistics selected *a priori* were examined. The fit statistics of the latent profiles of a one-class through a four-class solution were requested. First, the Lo-Mendell-Rubin Adjusted Likelihood Ratio Rest and the Bootstrapped Likelihood Ratio Test were examined. As a reminder, a significant difference between the models compared in the LMRT and the BLRT suggests the model with more classes is a better fit. For this second LPA, the LMRT and the BLRT both supported a 3-class model.

As in the first LPA (LPA1), the AIC and the sBIC for this LPA also support a 3-class model. Entropy for the 3-class model was .97. Models with greater than 3 classes (e.g., 4 class model and above) resulted in unstable parameters due to classes with too few members (Muthén & Muthén, 2009). These model fit statistics were not examined. See Table 23 for LPA3 fit statistics and Table 24 for Class means and descriptions.

Table 213

Model Selection Fit Statistics for sAA, Cortisol, and Negative Behavioral Reactivity

Model	AIC	BIC	sBIC	LMRT (*p*)	BLRT (*p*)	Entropy	Class	Membership (%)
1 Class	2771.44	2791.77	2772.76	-	-	-	1	219(100%)
2 Class	2446.41	2480.30	2448.61	<.001	<.001	.99	1	172(78%)
							2	47(22%)
3 Class	2383.74	2431.19	2386.82	= .014	<.001	.97	1	47(21%)
							2	41(18%)
							3	131(60%)
4 Class	-	-	-	-	-	-	-	-

Note. AIC = Akaike information criterion; BIC = Bayesian information criterion; sBIC = sample adjusted information criterion; LMRT = Lo-Mendell-Rubin likelihood ratio test. Bootstrap Likelihood Ratio Test. Lower numbers of AIC, BIC, and sBIC signify better fit. A significant LMRT and/or BLRT statistic signifies that the model with more classes is a better fit than a model with one less class.
* A four-class model resulted in unstable parameters due to requesting too many classes.

Table 224

Means, Standard Deviations, and Class Descriptions for LPA3 Classes

Variable	Class 1 (*n* = 41) *M*(*SD*)	Class 2 (*n* = 131) *M*(*SD*)	Class 3 (*n* = 47) *M*(*SD*)
Cortisol Reactivity Mean	-6.64(.32)	-1.38(.32)	-3.02(.58)
sAA Reactivity Mean	3.46(.42)	3.18(.36)	-6.91(.00)
Negative Behavioral Reactivity	1.03(.34)	1.37(.29)	1.67(.36)
Description	No-to-low Cortisol/Primarily SNS Reactivity Group – Lower	Moderate to high sAA and moderate to high cortisol reactivity – Moderate	No sAA reactivity/ HPA only group/High behavioral reactivity

	Behavioral Reactivity	Behavioral Reactivity	– High Behavioral Reactivity

Note. Cortisol and salivary alpha amylase values are for log transformed variables. Behavioral reactivity is presented in its original values.

Class Descriptions

Class 1: No-to-low Cortisol, primarily SNS Reactivity, lower behavioral reactivity (*n* = 41). This class had infants who had high log transformed alpha amylase reactivity to the stressor task, with a class mean of 3.46 (sample *M* = 1.05, *SD* = 4.37). This group had no-to-low log transformed cortisol reactivity (class *M* = -6.64, sample *M* = -2.70, *SD* = 2.44). Behaviorally, this group displayed, on average, lower behavioral distress (class *M* = 1.00, sample *M* = 1.37, sample *S.D.* = 0.89).

Class 2: Moderate to high sAA, moderate to high cortisol reactivity, moderate behavioral reactivity (*n* = 131). This class contained the most members and was comprised of infants who, on average, displayed high log transformed reactivity on both stress systems and moderate behavioral reactivity. The log transformed alpha amylase reactivity mean for this class was 3.18 (sample M = 1.05, *SD* = 4.37). The log transformed cortisol reactivity mean for this class was -1.38 (sample M = -2.70, *SD* = 2.44). Behaviorally, this class displayed average amounts of distress (class *M* = 1.37, sample *M* = 1.37, sample *SD* = 0.89).

Class 3: No sAA reactivity, low HPA, high behavioral reactivity (*n* = 47). Physiologically, this class was composed of infants who displayed nearly no log transformed salivary alpha amylase and very low log transformed cortisol reactivity to the stressor task. The alpha amylase reactivity value (class *M* = -6.91) is in the no reactivity range (sample *M* = 1.05, *SD* = 4.37). The cortisol value (class *M* = -3.02) was in the low average range for the sample (sample *M* = -2.70, *SD* = 2.44). Behaviorally, these infants displayed on average the most

negative behavioral reactivity (e.g., crying) of the three classes. In comparison to the sample mean and standard deviation (M = 1.37; SD = 0.89), the mean behavioral reactivity of the infants in this class (M = 1.67) was on the high-average range.

Examining the scatterplot of the three classes (Figure 3), however, reveals that the addition of the behavioral reactivity variable did not change the composition of the classes, meaning that members who belonged to either class 1, 2, or 3 at LPA1 typically remained in that classification in LPA3. Upon further inspection of each infant's classification, only one infant moved from Class 3 at LPA1 (moderate to high sAA and cortisol reactivity) to Class 1 at LPA3 (no-to-low cortisol, high sAA reactivity).

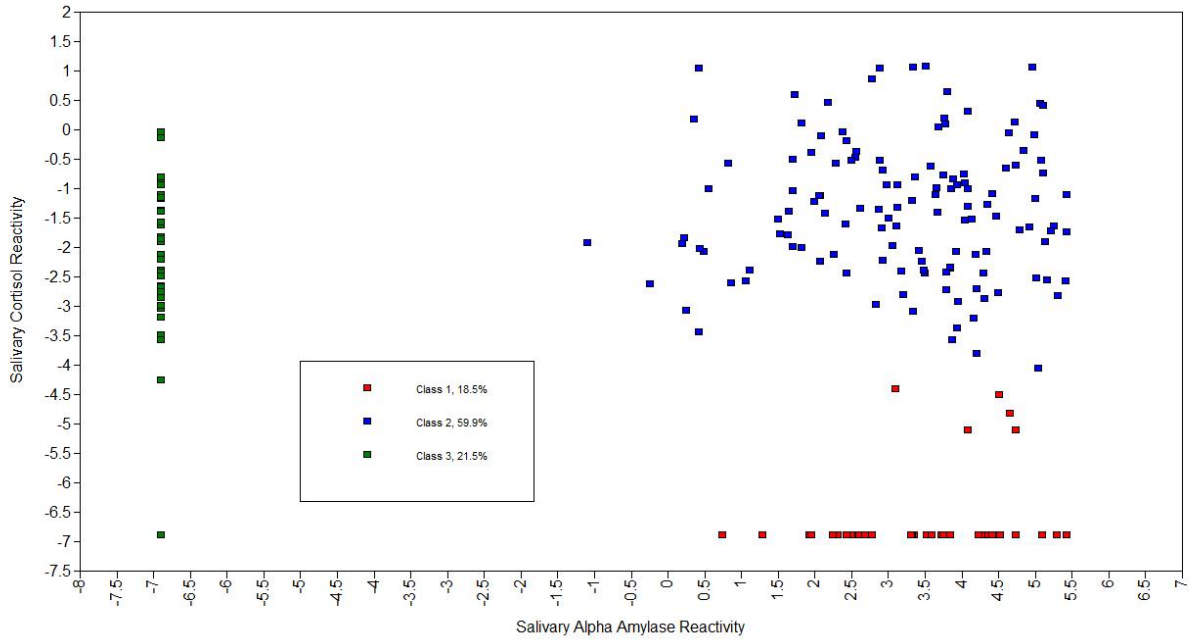
Post hoc test were conducted to further understand how the classes identified primarily by the physiological markers vary with relation to negative behavioral reactivity. A one-way ANOVA was conducted to examine class differences on negative behavioral reactivity. Due to violation of Equality of Variances, a more stringent alpha level of .016 was used for statistical significance. Results of this analysis of variance show that classes vary significantly by negative behavioral reactivity, $F(2, 194) = 3.69$, $p = .009$, partial eta squared = .048, a small effect size. Scheffé post hoc comparisons revealed significantly greater negative behavioral reactivity in Class 3 when compared with Class 1 ($p < .006$). However, as infant race/ethnicity had previously been identified as covariate for negative behavioral reactivity, a factorial ANOVA with infant race/ethnicity was conducted. When race/ethnicity is taken into account, the relationship between class membership and negative behavioral reactivity becomes non-significant $F(2, 151) = 3.38$, $p = .11$.

Although this 3 class model solution identified three homogenous groups, it once again clustered all infants with reactivity in both systems in a large class (Class 2), providing little

information about these infants and their stress reactivity. As was previously stated, one of the primary aims of this study was to understand how the two reactivity systems interact. Given that having classes with no reactivity in one of the two physiological markers acts as an outlier that pulls for a group, an additional LPA (LPA4) was conducted with Class 2 only (131 infants who display moderate to high physiological reactivity) to further classify those infants who showed reactivity in both systems.

Figure 4

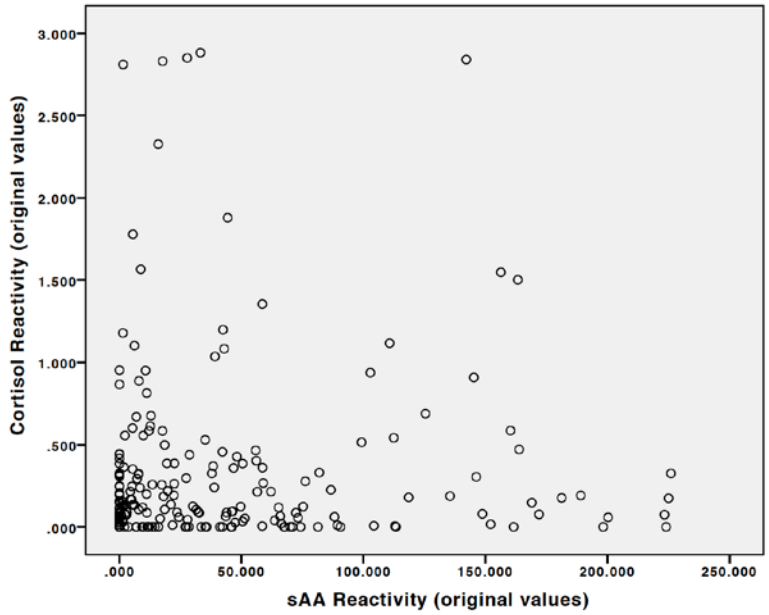
Three Class Reactivity Model Scatterplot (PLA3)



Note. Values are for transformed variables.

Figure 5

Three Class Reactivity Model Scatterplot (Original Values)



Additional LPA with Class 2 (LPA4)

A fourth LPA was conducted on the 131 infants who had previously been categorized in Class 2 in LPA3. Descriptive statistics for this group can be found in Table 23. Based on fit statistics and theory, a three class model was selected as the best fit to the data. The fit statistics for the LPA4 can be found in Table 24. Notably, the 3 classes identified in LPA4 and the 3 classes identified in LPA2 were identical (contained the same infants classified in the same way), suggesting that no additional information was added by inclusion of behavioral reactivity).

Table 235

Descriptive Statistics for Class 2 Cortisol and Salivary Alpha Amylase LPA3

	n	Min	Max	*M*	*SD*	Skewness	Std. Error	Kurtosis	Std. Error
Reactivity sAA	130	.33	226.00	51.67	56.86	1.45	.21	1.24	.42
Reactivity Cortisol	131	.02	2.88	0.49	0.63	2.41	.21	5.81	.42
Negative Behavioral Reactivity	122	.00	3.00	1.37	0.92	.147	.22	-1.23	.43

Table 246

Model Selection Fit Statistics for sAA, Cortisol, and Negative Behavioral Reactivity LPA4

Model	AIC	BIC	sBIC	LMRT (*p*)	BLRT (*p*)	Entropy	Class Number	Membership (%)
1 Class	2008.03	2025.28	2006.30	-	-	-	1	131(100%)
2 Class	1923.62	1952.37	1920.74	= .05	<.001	.97	1	11(8%)
							2	120(91%)
3 Class	1863.40	1903.65	1859.37	= .01	<.001	.96	1	22(16%)
							2	100(76%)
							3	9(6%)
4 Class	1827.70	1879.45	1822.52	=.16	<.001*	.96	1	6(4%)
							2	17(13%)
							3	91(7%)
							4	17(13%)
5 Class	1809.13	1872.38	1802.80	=.24	<.001*	.97	1	91(69%)
							2	16(12%)

	3	12(9%)
	4	6(4%)
	5	6(4%)

Note. AIC = Akaike information criterion; BIC = Bayesian information criterion; sBIC = sample adjusted information criterion; LMRT = Lo-Mendell-Rubin likelihood ratio test. Bootstrap Likelihood Ratio Test. Lower numbers of AIC, BIC, and sBIC signify better fit. A significant LMRT and/or BLRT statistic signifies that the model with more classes is a better fit than a model with one less class.
* = log likelihood value not replicated in all bootstrap draws.

Class Mean Differences in Internalizing and Externalizing Behaviors at 12 Months

Close examination of the new classes revealed that no changes occurred with the addition of the negative behavioral reactivity variable (e.g., all infants remained in the same subclass to which they were assigned at LPA2). Thus, no additional analyses were conducted at this point, as these would have reproduced the analyses previously shown.

Chapter V

Discussion

Research to date shows that individual differences in the human stress response are associated with important outcomes such as psychological wellbeing and physical health (Allwood et al., 2011; El-Sheikh et al., 2011; El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008; Keller & El-Sheikh, 2009; Nederhof et al., 2015). Yet, much of our knowledge about the stress response comes from studies examining only one component of the stress system (i.e., HPA axis or SNS, but not both). Theory, however, suggests that the stress reactivity system should be examined in a multisystemic manner, accounting for activity in both components of the stress system (Bauer et al.,2002). Further limiting our understanding of the stress response is the lack of research focusing on the stress reactivity system in infancy. Thus, the present study sought to identify homogenous patterns of stress reactivity measured by activation of the HPA axis and the LC-NE/SNS exist in a sample of 6-month-old infants and examine links between specific patterns of response and internalizing and externalizing symptoms of the same infants at age 12 months.

In addition to physiological reactivity, this study included markers of behavioral reactivity. Behavioral reactivity has been conceptualized as a component of the stress response, as successfully adapting to stress may require both physiological and behavioral responses (Eisenberg et al., 1997; Stansbury & Gunnar, 1994). However, research examining both physiological and behavioral reactivity in infancy remains limited, and links between physiological and behavioral reactivity are poorly understood. Thus, in order to address the gaps in the literature, the present study examined physiological and behavioral markers for stress reactivity in order to identify homogenous patterns of stress reactivity in 6-month-old infants. A

final and additional objective was to examine links between patterns of stress reactivity (physiological and behavioral) and internalizing and externalizing symptoms in infants at 12 months.

Two sets of analyses were used to examine patterns of stress reactivity and their links to socio-emotional outcomes. First, an LPA was conducted to identify homogenous classes of reactivity across the LC-NE/SNS and the HPA axis. Three groups emerged from LPA1. Due to the identification of a large, heterogeneous class through LPA1, and this study's particular focus on understanding patterns of reactivity across both components of the stress system, a follow up LPA (LPA2) was conducted on this large class. Three additional classes were identified through LPA2. The classes identified through LPA1 and those identified through LPA2 were used as grouping variables to examine differences in socio-emotional outcomes in two separate analyses of variance.

The second set of analyses consisted of adding a behavioral reactivity variable as an additional stress reactivity marker. An LPA (LPA3) with three markers for stress reactivity was conducted. Again, a large heterogeneous class emerged through LPA3 and was followed up by an additional LPA (LPA4). Three classes emerged from LPA4. The addition of a behavioral marker of stress reactivity did not change infants' classifications. Thus, relationships between stress reactivity patterns and socio-emotional outcomes were only conducted on the classes identified through LPA1 and LPA2.

In general, three patterns of reactivity were identified in both sets of examinations (with and without behavioral marker), but no statistically significant relationships between patterns of stress reactivity and internalizing and externalizing behaviors at 12 months emerged. Yet, when differences in externalizing behaviors by reactivity class were examined on the infants with

reactivity in both systems (LPA2), several trends nearing conventional levels of significance emerged. First, an interaction effect of reactivity by income on externalizing behaviors was identified. A link between income and externalizing behaviors was also identified. Finally, a trend emerged between physiological reactivity patterns and externalizing behaviors. In order to maintain a focus on both Type II and Type I error, given the early stage of research examining links between reactivity in two physiological stress systems (SNS and HPA) and later infant behavior problems, the fact that the sample size was small for detecting an interaction (Aiken & West, 1991), and findings of respectable effect sizes, determinations were made to provide tentative interpretations for those links that approached significance. In fact, a number of researchers have argued that less emphasis should be placed on statistical significance testing (Cohen 1994; Thompson & Snyder, 1997, 1998), and that instead interpretations should focus on effect sizes (Wilkinson & APA Task Force on Statistical Inference, 1999). Findings will be discussed with regards to research and methodological implications, implications for stress reactivity theory, directions for future research, and limitations.

Findings and Consistency with Prior Research

Baseline cortisol and salivary alpha amylase: links to behavioral reactivity, internalizing, and externalizing

Baseline cortisol was significantly and positively related to internalizing behaviors at 12 months. Previous evidence shows links between unstimulated cortisol and internalizing disorders in 12 to 36-month-olds (links in particular with waking cortisol, Scher, Hall, Zaidman-Zait, & Weinbeer, 2010) and in preschool aged children (aged 4.5; Essex, Klein, Cho, & Kalin, 2002). In older, clinic referred children, links between unstimulated salivary cortisol and parent rated internalizing behaviors have been identified (7-14 year olds; Scerbo & Kolko, 1994). No

links between baseline sAA and socio-emotional outcomes were identified in the present study.

Baseline sAA was significantly and inversely related to behavioral reactivity. As the incorporation of sAA as a marker of SNS activity remains fairly new (first articles appeared in the literature in the mid-2000s, Granger et al., 2006), knowledge about behavioral concomitants of sAA reactivity remains scarce. In fact, this is the first study to examine sAA and concurrent negative behavioral reactivity. The inverse relationship suggests that higher levels of baseline sAA are linked with less negative behavioral reactivity to the arm restraint task.

Cortisol and salivary alpha amylase reactivity: Links to behavioral reactivity, internalizing, and externalizing

Both cortisol and sAA reactivity were linked with behavioral reactivity, but in opposite ways. sAA was inversely related, so that greater sAA reactivity was linked with lesser negative behavioral reactivity. This implies that as SNS activation increases in response to a specific stressor, infants tend to demonstrate lower levels of negative behavioral reactivity, such as fussing and crying. Cortisol, on the other hand, was positively related to behavioral reactivity, so that as cortisol reactivity increased, so did negative behavioral reactivity. This means that when HPA axis activation occurs in the context of an immediate stressor, infants tend to display greater negative behavioral reactivity, such as fussing and crying.

The present findings are in line with a body of research that finds differential associations between SNS, HPA axis, and behavior. There appears to be an established link between cortisol reactivity and negative emotionality/behavioral reactivity. Ursache and colleagues (2014) found that cortisol reactivity to a stress task was linked with increased negative behavioral reactivity in a sample of 7-month-old infants. In a sample of toddlers, post task cortisol was linked with negative affect and withdrawal behaviors (Fortunato et al., 2008). Thus, the present findings

linking increases in cortisol with increases in negatively behavioral reactivity appear to be congruent with previous literature.

Links between sAA and behavioral reactivity, however, have not been as clearly elucidated. In fact, few studies have examined links between sAA and behavioral reactivity in infancy. Fortunato and colleagues (2008) found that sAA was related to approach behavior and positive affect (and not negative affect) in a sample of toddlers. Some theory suggests that there is a more sensitive threshold for SNS activation than for HPA axis activation, so that SNS reactivity should occur in response to milder challenges than those that would activate the HPA axis (Lovallo & Thomas, 2000). If in fact the SNS reacts when a stressor is perceived as more manageable, an SNS signal might suggest the infant is marshaling resources but that the stressor is manageable and does not necessitate a behavioral response, thus resulting in little or no behavioral activation as the infant does not need to cue behaviorally for help. If the HPA axis activation results due to an unmanageable stressor, then signaling for assistance becomes important. Then, infants displaying high HPA axis activity would also demonstrate high behavioral reactivity. In fact, a negative relationship between sAA reactivity and negative behavioral reactivity emerged even when controlling for cortisol reactivity (i.e., HPA axis activity). When partial out sAA reactivity, cortisol reactivity remained positively linked with negative behavioral reactivity. These inverse findings may potentially suggest that the SNS and the HPA axis serve somewhat different functions in the stress response.

Finally, negative behavioral reactivity was marginally related to internalizing, so that as infants displayed greater negative behavioral reactivity to the stressor task, they were rated as displaying greater numbers of internalizing behaviors by their caregivers later on at 12 months. This finding is consistent with previous findings relating negative behavioral reactivity to later

caregiver rated internalizing behaviors in infants (Garstein, Putnam, & Rothbart, 2012; Miner & Clarke-Stewart, 2008). For example, negative affect in a still face paradigm at 6 months linked with internalizing behaviors at 18 months (Moore, Cohn, & Campbell, 2001). Links between negative behavioral reactivity, such as crying, and later socio-emotional outcomes have been conceptualized within a differential susceptibility model, suggesting that highly reactive infants may be more sensitive to potential environmental and interpersonal moderators (parenting). For example, if infants who display significant negative emotionality to challenges have parents who have difficulty navigating these challenges, an ongoing pattern of dysregulation to stressors in the parent-infant dynamic may develop exacerbating the infant's difficulty to regulate. This places the infant at risk for later behavior problems (Rothbart, 2011).

Patterns of Physiological Reactivity

LPA1. The first set of analyses (LPA1) involved identifying groups of infants who demonstrated homogenous patterns of stress reactivity in both the LC-NE/SNS and the HPA axis systems. Three classes were identified, 1) a no sAA reactivity/HPA only class, 2) a no-to-low cortisol/primarily SNS reactivity class, and 3) a moderate-to-high sAA and cortisol class. The most salient finding of LPA1 was the identification of two classes primarily characterized by reactivity in only one system (i.e., reactivity in the SNS or the HPA axis).

Class 1 and Class 2 each demonstrated asymmetrical reactivity, with one system displaying reactivity and the other displaying low to no reactivity. Class 1 was composed of infants who displayed no sAA reactivity and variable cortisol reactivity. The majority of this class is composed by infants who displayed no reactivity in the LC-NE/SNS system and who varied in their HPA axis reactivity, from lack of reactivity to high reactivity. Class 2 was the exact inverse of Class 1, as it was composed primarily of infants who displayed no cortisol

reactivity, with average-to-high sAA reactivity. This group of infants demonstrated no-to-low HPA axis reactivity and average-to-high LC-NE/SNS reactivity (all but 4 infants in this class displayed no HPA axis reactivity). Together, these two groups of infants comprised approximately 40% of the sample.

These patterns of reactivity could be seen as consistent with the interactive model of reactivity (Bauer et al., 2002). The interactive model posits that the HPA axis and the LC-NE/SNS interact with each other, with the HPA axis actually suppressing the activity of the LC-NE/SNS (as initially suggested by Menck et al., 1984). Although the infants classified in either Class 1 or Class 2 appear to demonstrate patterns of reactivity that are poorly coordinated, with one system responding to the stressor and the other system displaying a low or nonexistent response, the present study findings do not support a conceptualization that these patterns represent poor adaptation. In fact, no clear support for either model (interactive or additive) in the theory emerged from this study, as no statistically significant links between reactivity patterns and socioemotional outcomes emerged.

It is important to consider potential shortcomings related to the data collection process could explain the lack of LC-NE/SNS or HPA axis stress reactivity found in these two groups. It is possible that for this subgroup of infants, sAA reactivity occurred very quickly and in a shorter window than for other infants in the sample. Studies that include sAA in infancy and childhood are few and methods for assessing sAA evolving. Although most researchers appear to collect data between 10 and 20 minutes following the stressor (Granger et al., 2006), there is some evidence, although with older individuals, that sAA can peak within the first 10 minutes after a stressor. Gordis and colleagues (2008) found that in their sample of youth (9-14 years old), on average, sAA peaked immediately following the stressor task and returned to normal within 10

minutes. Thus, if there was sAA reactivity that peaked and returned to near baseline within the first ten minutes following the stressor task, infants would appear to have no reactivity when in fact this reactivity was missed due to the structure of the data collection.

In order to address this possibility, it would be important to identify or develop practical ways to measure the activity of the SNS. There are significant benefits to utilizing salivary measures, as it allows for measurement of the HPA axis and the SNS simultaneously (i.e., both can be measured from the same sample). However, in order to capture peak reactivity as well as returns to baseline levels, several samplings must take place. Complicating the picture is the fact that the SNS and the HPA axis have variant reactivity timelines. Therefore, although an earlier salivary collection point might be ideal, it could be difficult to implement in the laboratory setting, as salivary collections take up a significant amount of time. Measuring the activity of the SNS by other means might provide additional avenues of data collection. For example, recent improvements in technology allowing measurement of skin conductance continuously in an ambulatory setting, during and after stressor, would now allow us to better characterize the activity of the SNS in future research.

In addition, it is important to consider methodological issues that may have affected detection of physiological reactivity. One possibility is that the way the reactivity variables were calculated prohibited finding a true reactivity. Some infants may have found arriving in the lab visit to be a distressing experience and may have responded with a stress response. It is plausible that, if a stress reaction took place towards the baseline salivary collection time, the “baseline” salivary amount might have been elevated. An elevation in baseline may have diminished or completely obscured milder elevations in analyte following the stressor task (the arm restraint). There is some evidence in our data to suggest this might not be the case, however. Classes

identified through LPA1 varied significantly by baseline cortisol and sAA. Thus, infants who had high baseline cortisol were also later classified as having high cortisol reactivity. Therefore, it is likely that the infants who found the laboratory visit stressful are more generally high reactors and responded with a stress response to the laboratory visit but also to the stressor task (in greater magnitude).

The last and largest class identified through the first LPA, Class 3, was composed of infants who had moderate-to-high reactivity in both the LC-NE/SNS and the HPA axis. This is a reactive group, demonstrating reactivity across both systems of the stress response. Due to the theoretical interest in more closely examining those who reacted in both systems and the size and heterogeneity of Class 3, an additional LPA was conducted with just this sample in order to better understand patterns of stress reactivity across both components of the stress reactivity system.

LPA2. The second LPA also resulted in three classes, 1) a low to moderate cortisol and low to moderate sAA class, 2) a low to moderate cortisol and high sAA class, and 3) a moderate to high cortisol and low to moderate sAA. Class 1 in LPA2 was the largest, with 101 infants. This class was composed of infants who on average displayed low to moderate cortisol reactivity and low to moderate sAA reactivity. Inspection of the scatterplot reveals that this class is composed of infants who display several combinations of reactivity, such as low-low, moderate-moderate, and low-moderate across the two systems. Class 2 in LPA2 contained 22 infants, and was comprised of infants who displayed high sAA reactivity. Inspection of the scatterplot reveals that infants in this class displayed various combinations of low to moderate cortisol and moderate to high sAA (e.g., low cortisol and moderate sAA, moderate cortisol and moderate sAA, etc). Overall, this class can be characterized as a high sAA reactivity class. Class 3 in

LPA2 contained 9 infants, and was comprised primarily of infants who displayed high cortisol reactivity and low sAA reactivity. Of note, one outlier classified in this group can be observed as having high cortisol and moderate-high sAA, whereas the rest of the infants are better characterized by low sAA. Overall, this class can be characterized as a high cortisol reactivity class.

An additional, exploratory LPA was conducted on the largest class identified through LPA1, as this was a heterogeneous class, composed of infants with reactivity across both systems. The purpose of LPA2 was to focus on infants who displayed reactivity across both systems of the stress response. Class 1 showed more or less equivalent reactivity across the two systems, whereas the two other classes showed asymmetrical arousal (Class 2 more sAA reactivity and Class 3 more cortisol reactivity). The emergence of this class does show that, across the whole sample (N = 219), the most common physiological response is, in fact, some combination of reactivity across both systems (as opposed to no reactivity in only one system). This is congruent with modern conceptualization of stress reactivity, which assumes some coordination between both systems (Boyce & Ellis, 2005). In addition, across this sample of 6-month-old infants, a low to moderate response across both physiological systems of the stress reactivity system appears common, which is also congruent Chrousos and Gold (1992) model of optimal arousal, which posits that optimal arousal is neither inadequate nor excessive.

Patterns of Physiological Reactivity and Links to Symptomatology

When rates of internalizing and externalizing behaviors were examined between classes across the whole sample (LPA1), no significant differences emerged. When levels of internalizing and externalizing behaviors were examined between classes across the reactive subsample (e.g., those with reactivity in both systems, LPA2), however, some significant

differences did emerge. Class membership identified though the LPA2 was used as a grouping variable in order to compare differences in externalizing and internalizing behavior problems at 12 months. Household income was also used as a grouping variable, as it was identified as a covariate in preliminary analyses.

A link between income and externalizing behaviors did emerge, showing greater externalizing behaviors in infants whose caregivers reported earning less than $10,000 when compared with infants whose caregivers reported earning $30,000 of more. As well as greater externalizing behaviors in infants whose caregivers reported earning between $10,000 and $30,000 when compared with infants whose caregivers reported earning $30,000 of more. No significant differences between infants whose caregivers earned less than $10,000 and those earning between $10,000 and $30,000. This finding is congruent with a large body of research that has documented links between poverty and later externalizing behaviors in infants and toddlers (see Knitzer & Perry, 2009 for a review).

An interaction effect between income and reactivity class emerged at the multivariate level. Although this link did not reach conventional levels of statistical significance, a medium effect size (partial eta squared = .09) was identified. As previously discussed, in order to maintain a focus on both Type II and Type I error, given the early stage of research examining links between reactivity in two physiological stress systems (SNS and HPA) and later infant behavior problems, the fact that the sample size was small for detecting an interaction (Aiken & West, 1991), and findings of respectable effect sizes, interpretations of the post hoc analyses are presented here. These interpretations are to be considered with caution and as guides for future research.

An examination of the interaction effect proved difficult to interpret beyond a main effect for income (with lower income associated with higher externalizing) given that a cell e was composed of one infant with more externalizing behavior that the other infants in every other class. In addition, at the univariate level, a link between physiological reactivity patterns and externalizing behaviors approached significance, and had a small to medium effect size (partial eta squared = .08) . Pairwise comparisons across the three reactivity levels did not results in significant differences, however.

Finally, a significant difference in the frequency of having infants rated as "of concern" on externalizing behaviors in class 1 emerged, with 52 infants rated below the threshold "of concern" and 9 rated above. Class 1 (LPA2), which was composed of infants with low-moderate reactivity in both the HPA axis and the LE-NE/SNS had more infants without significant externalizing behaviors than might be expected by chance. This finding provides initial evidence supporting a model of adaptive stress reactivity, in which both components of the stress system activate in response to a stressor and the activation of each component is not excessive. This is in line with modern conceptualizations of stress reactivity (Bauer et al., 2002; Boyce & Ellis, 2005).

There are several potential ways to interpret the findings between physiological reactivity and outcomes. First, there is a lack of published studies examining both systems of the stress response in infancy. Existing research that has identified links between physiological-behavioral reactivity and internalizing/externalizing problems later in life, however, is primarily composed of older samples (Chen et al., 2015; Gordis et al., 2006; Reeves et al., 2016). For example, El-Sheikh and colleges (2008) found links between patterns of stress response and internalizing and externalizing behaviors in a sample of 9-10 years old. Allwood and colleagues (2011) also found

patterns of stress reactivity to be linked with parent rated aggression in a sample of 7-16 year olds. No published studies to date identify these links in infancy. The lack of statistically significant findings establishing links between stress reactivity in infancy and later outcomes in the present study, as compared to the clear links that have been found in older children, may suggest such relationships potentially develop or strengthen over time, whether due to interactions with the environment or natural development shifts that occur at various developmental stages. Further research will be needed in order to examine trajectories of stress reactivity from infancy into adolescence.

There are several theoretical ways to understand how these links could develop across infancy and childhood. Boyce and Ellis (2005) have suggested an evolutionary-developmental model in which individual differences in stress reactivity develop over the first years of life by interacting with environmental stressors which shape the response over time. They suggest that that genetic predisposition (i.e., individual differences in stress response) interact with external variables (such as maternal caregiving) to shape the reactivity pattern in one or more systems of the stress response (i.e., HPA axis or LC-NE/SNS). In the present case, it is possible that the relationship between physiological stress reactivity and internalizing and externalizing symptoms is moderated by experiencing certain stressors, such as harsh parenting, which effectively "turn on" these links. On the other hand, it is possible that the relationship between physiological stress reactivity and socio-emotional outcomes is moderated by a buffering variable, such as sensitive parenting, thereby preventing a negative association. In fact, the present findings hint at family income providing a buffering effect on links between patterns of reactivity and externalizing behaviors at 12 months. Given the evidence of relations emerging in toddlerhood, childhood, and adolescence, it is likely that environmental moderators are at play. In fact,

evidence shows that environment and interpersonal variables do influence the development of the HPA axis circuity (Tarullo, Quevedo, & Gunnar, 2008). Future studies that examine moderating variables, such as maternal sensitivity, on the relations between stress reactivity and socio-emotional outcomes seem particularly worthwhile.

In addition, physiological changes occur to the stress reactivity system across childhood, and so the stress reactivity system of a 6-month-old is not the same continuous construct as that of a child or adolescent (Tarullo, Quevedo, & Gunnar, 2008). It is possible that the stress reactivity system during infancy is continuing to undergo significant developmental changes, and the way the system responds at this stage of development will not be the same way it will respond later on. Thus, in order to understand how and why links between stress physiology and poor socio-emotional outcomes develop, a careful longitudinal exploration of the development of the stress response across early childhood becomes increasingly important. Careful consideration and measurement of hypothesized moderators would aid in clarifying a moderated relationship between stress physiology and socio-emotional outcomes.

Other factors related to the actual sample may have prohibited finding any significant links with the outcome variables. The infant sample was fairly homogenous in terms of internalizing and externalizing behaviors, showing on average low frequency of these behaviors. Overall, only 14.2% of infants displayed externalizing behaviors that placed them above a T score of 65, which corresponds to 1.5 standard deviations from the mean and is often considered "of concern" (Briggs-Gowan & Carter, 2006). Similarly, only 6.8% of infants displayed internalizing behaviors above a T score of 65 (Briggs-Gowan & Carter, 2006). Of note, other studies have found similar rates of internalizing and externalizing in non-referred samples. In an economically stable, racially and ethnically diverse sample, mothers reported social and

emotional functioning of infants aged between 12-40 months. Nearly ten percent of the sample had high internalizing symptoms and 9.4% of the sample had high externalizing symptoms (Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006). Although the low incidence of clinically significant symptoms of behavioral problems might have prevented the discovery of significant links between physiological/behavioral arousal and later behavioral problems, it is notable to have found small effects (although not significant at conventional levels) given the compression of scores in this community sample of healthy infants.

In addition, the sample size was greatly reduced at the 12 month point due to attrition and other barriers to data collection. The final sample size for analyses containing internalizing and externalizing ratings was 132 infants. It is possible that this sample is not large enough to detect a true relationship between the variables of interest. Of note, a trend approaching conventional levels of significance emerged between physiological reactivity patterns and externalizing behaviors at 12 months (a small effect size). This might suggest that links between these two variables exist early in development, but larger samples are needed for detection. Although the effect size is small, it is quite notable that any trends emerged, given the sample limitations discussed, bolstering the importance of future research re-examining these questions with larger samples. In fact, the American Psychological Association (APA) Task Force on Statistical Inference emphasized the importance of focusing interpretations on effect sizes (Wilkinson & APA Task Force on Statistical Inference, 1999).

Finally, this study's focus was on a historically hard to reach population facing several barriers (low resources, transportation limitations). Further, many of the study's participants belong to a community with a deep-seated mistrust for researchers and research, given long histories of abuse of this population by the research community (for examples see Bussey-Jones,

Garrett, Henderson, Moloney, Blumenthal, & Corbie-Smith, 2010; Dula, 1994). Thus, our final sample size represents strengths in the study's design and execution, resulting in reaching and successfully maintaining relationships with an underrepresented community. In addition, our attrition rates are similar to those reported in the literature by investigators working with low income, racially and ethnically diverse samples (Tohannan, Carlson, Shepherd, & Batsche-McKenzie, 2017).

Behavioral Reactivity as an Additional Marker of Stress Reactivity

The second set of analyses (LPA3) involved identifying groups of infants who demonstrated similar patterns of stress reactivity across physiological (HPA axis and the LC-NE/SNS systems) and behavioral reactivity systems. Following the methodology utilized in LPA1 and LPA2, and additional LPA (LPA4) was conducted with the largest group of infants (*n* = 131). The most salient finding of LPA3 and LPA4 was that the addition of a behavioral reactivity variable did not change the classification of individual infants. Thus, nearly all infants (except for 1) remained in the group to which they were initially classified. Classes remained identified by the physiological reactivity variables, with no semblance of pattern emerging when the behavioral reactivity is examined.

Behavioral reactivity has been conceptualized as a component of the stress response, as successfully adapting to stress may require both physiological and behavioral responses (Eisenberg et al., 1997; Stansbury & Gunnar, 1994). At a basic and evolutionary level, the ultimate purpose of physiological reactivity to an environmental stressor would be to fight or run away from the threat (Chrousos & Gold, 1992). Therefore, there is reasonable expectation that physiological activity would be in some way coordinated with behavioral activity. In the present sample, adding a behavioral measure of stress reactivity resulted in three classes that were

essentially identical to the initial classes in terms of physiological arousal (e.g., the same patterns at the physiological level) with corresponding negative behavioral reactivity. Thus, the classes remained primarily identified by their physiological reactivity.

Several ways of interpreting such findings might be appropriate. First, it is possible that there is a dependent relationship where behavioral reactivity depends on the activity in the physiological system. The fact that adding a behavioral marker to the LPA did not change the composition of the classes might suggest that behavior does not provide additional information, but rather is directly related to the physiological activity. This pattern can be seen at an observational/qualitative level. Preliminary examinations showed links between sAA, cortisol, and behavioral reactivity. Generally, sAA was negatively linked with behavioral reactivity and cortisol was positively linked with behavioral reactivity. Examining class averages in LPA1, a similar pattern could be observed, such that the class with high cortisol reactivity also had more negative behavioral reactivity compared to the other two classes and the class with high sAA had lower negative behavioral reactivity compared to the other two classes. A low to moderate across all three systems class also emerged. Thus, it might appear that physiological reactivity is linked in an expected manner to behavioral reactivity. However, an exploratory group differences test did not reveal group differences on negative behavioral reactivity based on class membership when covariates were also examined, suggesting that additional variables may be moderating or mediating links between physiology and behavior.

Conversely, it might be that each component of the stress reactivity system is activated differentially depending on how a stressor is perceived. For example, it might be that a stressor warrants low-moderate activity across all three components (LPA 3, Class 2), a mild stressor might only warrant enough SNS activity and some behavioral activity to marshal sufficient

resources to manage the stressor (LPA 3, Class 1), and that a significant stressor warrants HPA activation and great behavioral signaling to obtain additional support (e.g., caregiver help, LPA3, Class 3). In this way, the three components might coordinate to produce an adequate overall response, but each serves a specific purpose and is differentially activated. Future research could examine this hypothesis by utilizing within person models and exposing infants to stressors that vary in intensity.

Along those lines, these findings could suggest that there are additional factors that moderate the relationship between the physiological response and the behavioral response. It might be that stressors must be of a certain quality in order to activate both the behavioral and the physiological response at the same time or in a congruent fashion. For example, research regarding the HPA axis suggests that it should be activated by events that are novel, painful, or threatening (Gunnar & Cheatham, 2003). Thus, milder stressors or those with whom the infant has had experience before may result in a response from just the behavioral system (for example, it could be that the infants with only SNS response, no HPA axis, and low behavioral response have some comfort with the arm restraint procedure and thus are able to cope). Or, it might be that temperament must be taken into account to control for individual variations in behavioral reactivity to fully clarify physiology-behavior relationships.

Finally, if all three components are important parts of the stress response, as theory suggests, it is possible that relationships amongst these three markers may change or develop over time. Some research has shown that HPA reactivity becomes attenuated to a particular stressor over time, even though the behavioral response may remain the same (Gunnar et al., 1996). This process is conceptualized as being protective, as the deleterious effects of chronic

activation of the HPA axis prevented, but communication of distress can continue via the behavioral response (Quas, Hong, Alkon, & Boyce, 2000).

In sum, while interesting patterns of physiology-behavior reactivity emerged, further research is needed to fully elucidate 1) whether behavioral system activation results from physiological activation or whether the two systems work independently to mount a stress response, 2) whether the stress system responds differentially to stressors based on intensity or previous experience, and 3) whether links between physiology and behavior are mediated or moderated by additional variables, such as caregiving or temperament.

Limitations

The present study had several limitations. First, due to the non-experimental nature of the study design, conclusions about causality cannot be drawn from the results of this study. Generalizability of the findings should be limited to infants similar to those used in this study, as the sample consists primarily of low income mothers and their infants, and as such, results cannot be generalized to infants in other socio-economic groups. Similarly, given the social stressors that this sample faces (such as poverty and neighborhood violence among others), the physiological activity observed in the stress reactivity system may only be representative of infants who are born into stressful environmental and interpersonal conditions and may not generalize to infants who are not born into similar conditions. Early exposure to various stressors such as poverty, substandard housing, and interpersonal turmoil has been linked to altered physiological markers of stress reactivity, such as cortisol (Evans & Kim, 2007).

Finally, it is important to note that some patterns in attrition did emerge, such that return at 12 months was more likely if the infant was White, and less likely if the infant was Asian/Asian American or Hispanic. In addition, it was less likely that a family would return for

the 12 month visit when they reported earning less than $10,000 yearly. It is possible that this missing data comprises important data that would have provided a clearer picture of stress reactivity. However, it is also likely that these patterns of missingness are related to additional factors, such as comfort with participating in research and chronic relocation due to socioeconomic hardship.

Threats to Validity

Several threats to validity exist in the present study. Internal validity may have been threatened by maturation, as infants undergo intense developmental changes throughout the months between the two testing points. It is possible that natural maturation changes that occurred during the intervening six months may have obscured relationships between the variables of interest and may have prohibited a rejection of the null hypothesis when such a rejection is in fact warranted. Loss of participants to attrition and other barriers also posed a threat to internal validity. It is possible that individuals who dropped out of the study prior to completing all components represented an important subsample of individuals in one or more variables of interest (e.g., infants with the greatest behavioral reactivity may become too distressed by the laboratory tasks and mothers may decide to not return).

Mono-operation bias, reactivity to the experimental situation, and novelty and disruption effects may constitute barriers to construct validity. Mono-operation bias was present in the design of this study, as socio-emotional functioning was only assessed via the ITSEA. It is possible that this measure does not fully capture the essence of the constructs it seeks to measure (e.g., internalizing and externalizing). Although maternal report might not have given a full picture of the internalizing and externalizing variables, there is evidence to support that this

measure does provide significant information about these behaviors in infancy (Carter et al., 2003).

Bringing infants and mothers into a laboratory creates a novel situation for both the mother and the infant. This creates a threat to validity as the infant may become reactive to the changed environment and could respond with a stress reaction while arriving (thus influencing baseline markers) or throughout the entire visit. In this sample, however, LPA 1 classes varied by reactivity, so that infants who had high reactivity in HPA axis post stressor also showed high HPA axis activity at baseline, supporting the validity that the reactivity measures are identifying high reactors.

Future Directions for Research

The present study focused on the stress response in infancy. It is particularly important to understand the stress response in infancy for several reasons. First, it is important to understand the stress response from a developmental perspective, as examining functioning and response patterns in early development would allow for comparisons at various ages and for an understanding of the continuity (or lack thereof) of stress response patterns. Also, given that links between the stress response and internalizing and externalizing behaviors have emerged in childhood and adolescence, it is important to identify when these links first emerge in order to develop effective interventions. Thus, understanding whether these links are already evident in infancy helps us to answer these questions. In sum, a focus on understanding the stress response in infancy is important in order to answer both basic questions about the stress response system and questions about the role of individual differences in stress response and developmental psychopathology.

With this in mind, future research should continue to focus on examinations of the sress response in infancy. One promising approach would be to collect HPA axis, LE-NE/SNS reactivity markers, and socio-emotional functioning levels cross-sectionally at frequent intervals during early development (e.g. 6, 12, 18, 24, months). Such an approach would help to elucidate coordinated development of the stress reactivity systems across early childhood. It would also help to identify any structural changes that may occur in one component and whether these changes are related to the other component of the stress system. In addition, it would provide evidence of emerging links between structural/functional changes in the stress reactivity system and links with socio-emotional function.

Similarly, in order to increase our understanding of the stress response, including additional markers in future LPAs may help clarify the complexities in the system. For example, including baseline markers of reactivity may help to identify groups of infants who vary in their unstimulated levels of activity. Markers for latency to distress (how long until a stress response is initiated) or recovery (how long does the distress response last) might provide additional information about the ways the stress response varies person to person. Further examining behavioral markers, such as including markers of regulatory behaviors (e.g., looking at a caregiver) would provide additional evidence of interactions between stress physiology and behavior. In sum, it would be important to increase the number of stress markers used to identify homogenous groups as this might help us elucidate the larger, multi-component stress response.

Another promising future research approach would be to select a sample of infants-toddlers who are experiencing higher levels of internalizing and externalizing behaviors in order to increase power. The present sample was primarily composed of infants who were functioning well socio-emotionally at 12 months. It is possible that links between stress reactivity and

internalizing/externalizing are most evident when there is significant functional impairment. Thus, increasing the level of pathology in the sample might help to identify any existing links. Infants and young toddlers are less frequently referred for intervention than older toddlers and school age children. One way to increase access to such a population might be collaborating with pediatric primary care offices, many of whom already screen for socio-emotional development in young children (Briggs et al., 2012).

As was proposed throughout the discussion, identification and measurement of possible mediators and/or moderators that may serve to activate links between stress reactivity and socio-emotional function appears warranted. As discussed above, there is theory and empirical evidence suggesting an interaction between individual and environmental characteristics and negative outcomes (Gunnar & Cheatham, 2003; Heims et al., 1997). Although studies concurrently examining both the SNS and the HPA axis remain scant, potential mediators and moderators can be identified based on the cortisol literature. Potential variables that should be considered as mediators/moderators of the stress response include: quality of caregiving behaviors (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995), maternal prenatal psychopathology (Tollenaar et al., 2010), child abuse (Tarullo & Gunnar, 2006), interparental violence (Sturge-Apple, Davies, Cicchetti, & Manning, 2012), mother's emotional unavailability (Sturge-Apple et al., 2012), poverty (Blair et al., 2011) and infant temperament (Dettling, Gunnar, & Donzella, 1999). It is likely that many other variables might exert an influence on links between the stress response and outcomes, especially throughout childhood and adolescence, such as attending daycare and peer relationships. It is thus imperative to understand the stress response in a developmental manner, beginning in infancy.

Finally, further theoretical and empirical work is needed to fully understand how the stress system works. Concurrently collecting various measures of HPA axis and LC-NE/SNS reactivity markers may help to add valuable information to our understanding of the stress response.

Implications for Intervention

The aims of this study addressed both basic and applied questions. This study sought to observe and describe the activity of the stress response system in a sample of 6 month old infants, an important initial step in furthering our understanding of the human stress response. At a basic science level, furthering our knowledge of the human stress response is important. Understanding how the two components of the stress response interact allows us to further understand both the nature of the two individual components, as well as the global stress response. Further, understanding normative patterns of stress reactivity helps us identify abnormal patterns of response. In fact, a main finding of this study was the identification of a large class of infants with a low-moderate response across the SNS and the HPA axis, a pattern of reactivity that is congruent with theorized normative response (e.g., neither extreme nor insufficient). At the applied level, a better understanding of the stress response would allow us to ask vital questions about the nature of individual differences in responding to stress and relations to psychopathology.

Given the early stages of this research area, it is premature to make assertions about targets of intervention. However, continuing this line of research is necessary in order to address several important remaining questions, which would in turn get us closer to identifying targets for intervention. For example, it would be important to identify whether differences in stress response result from inherited risk or are developed through an interaction with other important

variables. If individual differences are in fact inherited, it is possible that variability in one's stress response is one factor involved in the intergenerational risk for psychopathology (that is, may be an explanatory variable in psychopathology with a genetic load). This would potentially encourage the use (and reimbursement) of preventative interventions that modulate physiological arousal and the stress response (e.g., mindfulness based interventions, Brand, Holsboer-Trachsler, Naranjo, & Schmidt, 2012; Matousek, Pruessner, & Dobkin, 2011). Such prevention interventions could be focused on those infants whose stress response profiles would tend to put them at higher risk for later problems.

On the other hand, if links between individual differences in stress response and poor outcomes develop over time, due to interactions with the environment, interventions targeting those mediators or moderators that influence the development of poor outcomes would be important. For example, an established body of research has identified longitudinal effects of child maltreatment on cortisol regulation (see Tarullo & Gunnar, 2006). These findings highlight the need for: a) interventions to prevent child maltreatment and thus prevent any changes to the biological stress response and b) interventions that might modulate the ill effects of child maltreatment and return the activity of the stress response to pre-abuse levels (e.g., relational interventions that result in biological changes such as Attachment and Biobehavioral Catch-Up; Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008). Understanding that child maltreatment changes the activity in the HPA axis, and that changes in the HPA axis are linked with poor outcomes allows scientist to develop targeted interventions that address these biological changes, hopefully preventing the longer term effects. Developing a similar body of research examining the stress response in a multisystemic manner would help us understand what variables affect the

activity of the stress response as a whole, and develop interventions targeting those variables, thereby potentially preventing poor outcomes.

Finally, an additional application to developing this line of research is in its application in intervention process and outcome research. The overall response to stress could be used as a marker of intervention effectiveness if further evidence for normative and maladaptive response patterns were to be identified.

References

Achenbach, T M. (1991). *Manual for the Child Behavior Checklist 4-8 and 1991 profile.* Burlington: University of Vermont, Department of Psychiatry.

Akaike, H. (1974). A new look at the statistical model identification. *IEEE Transactions on Automatic Control*, *19*(6), 716-723.

Allwood, M. A., Handwerger, K., Kivlighan, K. T., Granger, D. A., & Stroud, L. R. (2011). Direct and moderating links of salivary alpha-amylase and cortisol stress-reactivity to youth behavioral and emotional adjustment. *Biological Psychology*, *88*(1), 57-64. doi:10.1016/j.biopsycho.2011.06.008

American Academy of Pediatrics. (2015, November 11). Signs and Symptoms of Fever. Retrieved from https://www.healthychildren.org/English/health-issues/conditions/fever/Pages/Signs-and-Syptoms-of-Fever.aspx

Arnsten, A. F. (2000). Stress impairs prefrontal cortical function in rats and monkeys: role of dopamine D1 and norepinephrine α-1 receptor mechanisms. *Progress in Brain Research*, *126*, 183-192. doi:10.1016/S0079-6123(00)26014-7

Aston-Jones, G., & Cohen, J. D. (2005). An integrative theory of locus coeruleus-norepinephrine function: adaptive gain and optimal performance. *Annual Review of Neuroscience*, *28*, 403-450. doi:10.1146/annurev.neuro.28.061604.135709

Aston-Jones, G., Rajkowski, J., Kubiak, P., Valentino, R. & Shipley, M. T. (1996). Role of the locus ceoruleus in emotional activation. In G. Holstege, R. Bandler, & C. B. Saper (Eds.), *Progress in Brain Research* (pp. 379-402). Amsterdam, NY: Elsevier.

Axelrod, J., & Reisine, T. D. (1984). Stress hormones: their interaction and regulation. *Science*, *224*(4648), 452-459. doi:10.1126/science.6143403

Bae, Y. J., Stadelmann, S., Klein, A. M., Jaeger, S., Hiemisch, A., Kiess, W., ... & Thiery, J. (2015). The hyporeactivity of salivary cortisol at stress test (TSST-C) in children with internalizing or externalizing disorders is contrastively associated with α-amylase. *Journal of Psychiatric Research, 71*(2015), 78-88. doi:10.1016/j.jpsychires.2015.09.013

Bauer, A. M., Quas, J. A., & Boyce, W. T. (2002). Associations between physiological reactivity and children's behavior: Advantages of a multisystem approach. *Journal of Developmental & Behavioral Pediatrics, 23*(2), 102-113. doi:10.1097/00004703-200204000-00007

Beltzer, E. K., Fortunato, C. K., Guaderrama, M. M., Peckins, M. K., Garramone, B. M., & Granger, D. A. (2010). Salivary flow and alpha-amylase: collection technique, duration, and oral fluid type. *Physiology & Behavior*, *101*(2), 289-296. doi: https://doi.org/10.1016/j.physbeh.2010.05.016

Berridge, C. W., & Waterhouse, B. D. (2003). The locus coeruleus–noradrenergic system: modulation of behavioral state and state-dependent cognitive processes. *Brain Research Reviews*, *42*(1), 33-84. doi:10.1016 / S0165-0173(03)00143-7

Berry, D., Blair, C., Willoughby, M., Granger, D. A., & Family Life Project Key Investigators. (2012). Salivary alpha-amylase and cortisol in infancy and toddlerhood: Direct and indirect relations with executive functioning and academic ability in childhood. *Psychoneuroendocrinology*, *37*(10), 1700-1711. doi:10.1016/j.psyneuen.2012.03.002

Biederman, J., Rosenbaum, J. F., Bolduc-Murphy, E. A., Faraone, S. V., Chaloff, J., Hirshfeld, D. R., & Kagan, J. (1993). A 3-year follow-up of children with and without behavioral inhibition. *Journal of the American Academy of Child & Adolescent Psychiatry*, *32*(4), 814-821.

Blair, C., Granger, D. A., Willoughby, M., Mills-Koonce, R., Cox, M., Greenberg, M. T., ... & FLP Investigators. (2011). Salivary cortisol mediates effects of poverty and parenting on executive functions in early childhood. *Child Development, 82*(6), 1970-1984. doi:10.1111/j.1467-8624.2011.01643.x.

Bosch, J. A., de Geus, E. J., Veerman, E. C., Hoogstraten, J., & Amerongen, A. V. N. (2003). Innate secretory immunity in response to laboratory stressors that evoke distinct patterns of cardiac autonomic activity. *Psychosomatic Medicine, 65*(2), 245-258. doi:10.1097/01.PSY.0000058376.50240.2D

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. an evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17*(02), 271-301. doi:10.1017/S0954579405050145

Brand, S., Holsboer-Trachsler, E., Naranjo, J. R., & Schmidt, S. (2012). Influence of mindfulness practice on cortisol and sleep in long-term and short-term meditators. *Neuropsychobiology, 65*(3), 109-118. DOI: 10.1159/000330362

Braungart-Rieker, J. M., & Stifter, C. A. (1996). Infants' responses to frustrating situations: Continuity and change in reactivity and regulation. *Child Development, 67*(4), 1767-1779. doi:10.2307/1131730

Briggs, R. D., Stettler, E. M., Silver, E. J., Schrag, R. D., Nayak, M., Chinitz, S., & Racine, A. D. (2012). Social-emotional screening for infants and toddlers in primary care. *Pediatrics, 129*(2), e377-e384. Doi: 10.1542/peds.2010-2211

Briggs-Gowan, M. J., Carter, A. S., Bosson-Heenan, J., Guyer, A. E., & Horwitz, S. M. (2006). Are infant-toddler social-emotional and behavioral problems transient? *Journal of the*

American Academy of Child & Adolescent Psychiatry, *45*(7), 849-858. Doi: 10.1097/01.chi.0000220849.48650.59

Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: a meta-analysis. *Psychoneuroendocrinology*, *30*(9), 846-856. doi:10.1016/j.psyneuen.2005.02.010

Burke, J. D., Loeber, R., & Birmaher, B. (2002). Oppositional defiant disorder and conduct disorder: a review of the past 10 years, part II. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*(11), 1275-1293. doi:10.1176/foc.2.4.558

Buss, A. H., & Plomin, R. (1975). *A temperament theory of personality development*. Oxford, England: John Wiley & Sons Inc

Bussey-Jones, J., Garrett, J., Henderson, G., Moloney, M., Blumenthal, C., & Corbie-Smith, G. (2010). The role of race and trust in tissue/blood donation for genetic research. *Genetics in Medicine, 12*(2), 116–121. doi: 10.1097/gim.0b013e3181cd6689.

Calkins, S.D., Fox, N.A. & Marshall, T.R. (1996). Behavioral and physiological antecedents of inhibited and uninhibited behavior. *Child Development*, 67, 523-540. 10.2307/1131830

Canon, W. B. (1932). *The wisdom of the body* (2nd ed.). New York, NY: Basic Books

Carrion, V. G., Weems, C. F., Ray, R. D., Glaser, B., Hessl, D., & Reiss, A. L. (2002). Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biological psychiatry*, *51*(7), 575-582. doi:10.1016/S0006-3223(01)01310-5

Carter, A. S., & Briggs-Gowan, M. (1993). *The Infant–Toddler Social and Emotional Assessment* (ITSEA). Unpublished Measure. Yale University Department of Psychology, New Haven, CT. University of Massachusetts Boston Department of Psychology, Boston, MA.

Carter, A.S., & Briggs-Gowan, M. (2000). *The Infant–Toddler Social and Emotional Assessment* (ITSEA). Unpublished Manual. Yale University Department of Psychology, New Haven, CT. University of Massachusetts Boston Department of Psychology, Boston, MA.

Carter, A. S., & Briggs-Gowan, M. J. (2006). ITSEA: Infant-Toddler Social and Emotional Assessment Examiner's Manual. San Antonio, TX: PsychCorp.

Carter, A. S., Briggs-Gowan, M. J., Jones, S. M., & Little, T. D. (2003). The infant–toddler social and emotional assessment (ITSEA): factor structure, reliability, and validity. *Journal of Abnormal Child Psychology*, *31*(5), 495-514.

Charmandari, E., Tsigos, C., & Chrousos, G. (2005). Endocrinology of the stress response. *Annual Review of Physiology*, *67*, 259-284. doi:10.1146/annurev.physiol.67.040403.120816

Chatterton, R. T., Jr., Vogelsong, K. M., Lu, Y. C., Ellman, A. B., & Hudgens, G. A. (1996). Salivary alpha-amylase as a measure of endogenous adrenergic activity. *Clinical Physiology*, 16, 433-448.

Chen, F. R., Raine, A., Soyfer, L., & Granger, D. A. (2015). Interaction of adrenocortical activity and autonomic arousal on children's externalizing and internalizing behavior problems. *Journal of Abnormal Child Psychology*, 43(1), 189-202. doi:10.1007/s10802-014-9900-y

Chen, F. R., Raine, A., Rudo-Hutt, A. S., Glenn, A. L., Soyfer, L., & Granger, D. A. (2015). Harsh discipline and behavior problems: The moderating effects of cortisol and alpha-amylase. *Biological Psychology*, *104*(2015), 19-27. doi:10.1016/j.biopsycho.2014.11.005

Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology*. *5*(7), 374-381. doi:10.1038/nrendo.2009.106

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders.

Overview of physical and behavioral homeostasis. *Journal of the American Medical Association, 267*(9), 1244-1252. doi:10.1001/jama.1992.03480090092034

Chrousos, G. P., Torpy, D. J., & Gold, P. W. (1998). Interactions between the hypothalamic-pituitary-adrenal axis and the female reproductive system: clinical implications. *Annals of Internal Medicine, 129*(3), 229-240. doi:10.7326/0003-4819-129-3-199808010-00012

Cohen, J. (1994). The earth is round (p<. 05). *American Psychologist, 49*(12), 997-1003. Doi: 10.1037/0003-066X.49.12.997

CompareIt [Computer Software]. (2006). Retrieved from https://www.grigsoft.com/wincmp3.htm

Davis, E. P., & Granger, D. A. (2009). Developmental differences in infant salivary alpha-amylase and cortisol responses to stress. *Psychoneuroendocrinology, 34*(6), 795-804. doi:10.1016/j.psyneuen.2009.02.001

De Kloet, E. R. (1991). Brain corticosteroid receptor balance and homeostatic control. *Frontiers in Neuroendocrinology, 12*(2), 95-164.

Derryberry, D., & Rothbart, M. K., (1984). Emotion, attention, and temperament In Izard, C. E., Kagan, J., & Zajonc, R. B. (Eds). *Emotions, cognition and behavior* (pp. 132-166). New York, NY: Cambridge University Press.

Dettling, A. C., Gunnar, M. R., & Donzella, B. (1999). Cortisol levels of young children in full-day childcare centers: Relations with age and temperament. *Psychoneuroendocrinology, 24*(5), 519-536. Doi: 10.1016/S0306-4530(99)00009-8

Dozier, M., Peloso, E., Lewis, E., Laurenceau, J. P., & Levine, S. (2008). Effects of an attachment-based intervention on the cortisol production of infants and toddlers in foster

care. *Development and Psychopathology*, *20*(3), 845-859. doi: 10.1017/S0954579408000400.

Dula, A. (1994). African American suspicion on the healthcare system is justified: What do we do about it? *Cambridge Quarterly of Healthcare Ethics*, *3*(3), 247–257. doi: 10.1017/s0963180100005168.

Eisenberg, N., Fabes, R. A., & Guthrie, I. K. (1997). Coping with stress: the roles of regulation and development. In S. A. Wolchik, & I. N. Sandler (Eds.), *Handbook of Children's Coping*, (pp. 41-70). New York, NY: Springer.

El-Sheikh, M., Arsiwalla, D. D., Hinnant, J. B., & Erath, S. A. (2011). Children's internalizing symptoms: The role of interactions between cortisol and respiratory sinus arrhythmia. *Physiology & Behavior*, *103*(2), 225-232. doi:10.1016/j.physbeh.2011.02.004

El-Sheikh, M., Erath, S. A., Buckhalt, J. A., Granger, D. A., & Mize, J. (2008). Cortisol and children's adjustment: the moderating role of sympathetic nervous system activity. *Journal of Abnormal Child Psychology*, *36*(4), 601-611. doi:10.1007/s10802-007-9204-6

Engert, V., Smallwood, J., & Singer, T. (2014). Mind your thoughts: Associations between self-generated thoughts and stress-induced and baseline levels of cortisol and alpha-amylase. *Biological Psychology*, *103*, 283-291. doi:10.1016/j.biopsycho.2014.10.004

Evans, G. W., & Kim, P. (2007). Childhood poverty and health: cumulative risk exposure and stress dysregulation. *Psychological Science, 18*(11), 953-957. Doi: 10.1111/j.1467-9280.2007.02008.x

Fabes, R. A., Eisenberg, N., & Eisenbud, L. (1993). Behavioral and physiological correlates of children's reactions to others in distress. *Developmental Psychology*, *29*(4), 655-663. doi:10.1037/0012-1649.29.4.655

Fisher, A. J., Granger, D. A., & Newman, M. G. (2010). Sympathetic arousal moderates self-reported physiological arousal symptoms at baseline and physiological flexibility in response to a stressor in generalized anxiety disorder. *Biological Psychology, 83*(3), 191-200. doi:10.1016/j.biopsycho.2009.12.007

Fortunato, C. K., Dribin, A. E., Granger, D. A., & Buss, K. A. (2008). Salivary alpha-amylase and cortisol in toddlers: Differential relations to affective behavior. *Developmental Psychobiology, 50*(8), 807-818. doi:10.1002/dev.20326

Franchimont, D., Kino, T., Galon, J., Meduri, G. U., & Chrousos, G. P. (2003). Glucocorticoids and inflammation revisited: the state of the art. *Neuroimmunomodulation, 10*(5), 247-260. doi:0.1159/000069969

Fox, N. A. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology, 25*(3), 364-372.

Gartstein, M. A., Putnam, S. P., & Rothbart, M. K. (2012). Etiology of preschool behavior problems: Contributions of temperament attributes in early childhood. *Infant Mental Health Journal, 33*(2), 197-211. doi:10.1002/imhj.21312

Granger, D. A., & Kivlighan, K. T. (2003). Integrating biological, behavioral, and social levels of analysis in early Child Development: Progress, problems, and prospects. *Child Development, 74*, 1058-1063. doi:10.1111/1467-8624.00590

Granger, D. A., Kivlighan, K. T., Blair, C., El-Sheikh, M., Mize, J., Lisonbee, J. A., et al. (2006). Integrating the measurement of salivary α-amylase into studies of child health, development, and social relationships. *Journal of Social and Personal Relationships, 23*, 267-290. doi:10.1177/0265407506062479

Granger, D. A., Kivlighan, K. T., El-Sheikh, Gordis, E. B., & Stroud, L. R. (2007). Assessment of salivary a-amylase in biobehavioral research. In L. Luecken & L. Gallo (Eds.) *Handbook of physiological research methods in health psychology*. (pp. 95 - 113) New York, NY: Sage.

Gold, P. W., Goodwin, F. K., & Chrousos, G. P. (1988). Clinical and biochemical manifestations of depression. *New England Journal of Medicine,* 319(6), 348-353. doi:10.1056/NEJM198808113190606

Goldsmith, H. H., & Rothbart, M. K. (1999). *Laboratory Temperament Assessment Battery, Prelocomotor Version 3.1.* Madison, WI: Univeristy of Wisconsin

Gordis, E. B., Granger, D. A., Susman, E. J., & Trickett, P. K. (2006). Asymmetry between salivary cortisol and α-amylase reactivity to stress: Relation to aggressive behavior in adolescents. *Psychoneuroendocrinology*, *31*(8), 976-987. doi:10.1016/j.psyneuen.2006.05.010

Gunnar, M. R. (1989). Studies of the human infant's adrenocortical response to potentially stressful events. *New Directions for Child and Adolescent Development*, 1989(45), 3-18. doi:10.1002/cd.23219894503

Gunnar, M. R. (1992). Reactivity of the hypothalamic-pituitary-adrenocortical system to stressors in normal infants and children. *Pediatrics*, *90*(3), 491-497.

Gunnar, M. R., & Cheatham, C. L. (2003). Brain and behavior interface: Stress and the developing brain. *Infant Mental Health Journal*, *24*(3), 195-211. doi:10.1002/imhj.10052

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, *27*(1), 199-220. doi:10.1016/S0306-4530(01)00045-2

Gunnar, M. R., Isensee, J., & Fust, L. S. (1987). Adrenocortical activity and the Brazelton Neonatal Assessment Scale: Moderating effects of the newborn's biomedical status. *Child Development*, *58*(6), 1448-1458.

Gunnar, M. R., Porter, F. L., Wolf, C. M., Rigatuso, J., & Larson, M. C. (1995). Neonatal stress reactivity: Predictions to later emotional temperament. *Child Development*, *66*(1), 1-13. doi:10.2307/1131186

Gunnar, M. R. & Vazquez, D. (2006). Stress neurobiology and developmental psychopathology. In D. Cicchetti, D. J. Cohen, (Eds.), *Developmental Psychopathology: Developmental Neuroscience* (pp. 533-577). Hoboken, NJ: John Wiley & Sons.

Habib, K. E., Gold, P. W., & Chrousos, G. P. (2001). Neuroendocrinology of stress. *Endocrinology and Metabolism Clinics of North America*. 30(3), 695-728. doi:10.1016/S0889-8529(05)70208-5

Hagan, M. J., Luecken, L. J., Modecki, K. L., Sandler, I. N., & Wolchik, S. A. (2016). Childhood negative emotionality predicts biobehavioral dysregulation fifteen years later. *Emotion*, *16*(6), 877-885. doi:10.1037/emo0000161

Haley, D. W., & Stansbury, K. (2003). Infant stress and parent responsiveness: regulation of physiology and behavior during still-face and reunion. *Child Development*, *74*(5), 1534-1546. doi:10.1111/1467-8624.00621

Heim, C., Owens, M. J., Plotsky, P. M., & Nemeroff, C. B. (1997). The role of early adverse life events in the etiology of depression and posttraumatic stress disorder. *Annals of the New York Academy of Sciences, 821*(1), 194-207. doi:10.1111/j.1749-6632.1997.tb48279.x

Henry, J. P. (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science*, *27*(1), 66-83. doi:10.1007/BF02691093

Herman, J. P., & Cullinan, W. E. (1997). Neurocircuitry of stress: central control of the hypothalamo–pituitary–adrenocortical axis. *Trends in Neurosciences, 20*(2), 78-84. doi:10.1016/S0166-2236(96)10069-2

Hertsgaard, L., Gunnar, M., Erickson, M. F., & Nachmias, M. (1995). Adrenocortical responses to the strange situation in infants with disorganized/disoriented attachment relationships. *Child Development, 66*(4), 1100-1106. DOI: 10.2307/1131801

Hill-Soderlund, A. L., Holochwost, S. J., Willoughby, M. T., Granger, D. A., Gariépy, J. L., Mills-Koonce, W. R., & Cox, M. J. (2015). The developmental course of salivary alpha-amylase and cortisol from 12 to 36 months: Relations with early poverty and later behavior problems. *Psychoneuroendocrinology, 52,* 311-323. doi:10.1016/j.psyneuen.2014.08.011

IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.

Jansen, J., Beijers, R., Riksen-Walraven, M., & de Weerth, C. (2010). Cortisol reactivity in young infants. *Psychoneuroendocrinology, 35*(3), 329-338. doi:10.1016/j.psyneuen.2009.07.008

Kagan, J. (1997). Temperament and the reactions to unfamiliarity. *Child Development, 68*(1), 139-143. doi:10.2307/1131931

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*(6), 1459-1473. doi:10.2307/1130685

Keller, P. S., & El-Sheikh, M. (2009). Salivary alpha-amylase as a longitudinal predictor of children's externalizing symptoms: respiratory sinus arrhythmia as a moderator of effects. *Psychoneuroendocrinology, 34*(5), 633-643. doi:10.1016/j.psyneuen.2008.12.016

Kivlighan, KT.; Granger, DA.; Blair, C. The Family Life Project Investigators. *Salivary alpha-amylase and cortisol: Levels and stress reactivity in 6-month-old infants and their mothers.* Paper presented at the biennial meeting of Society for Research in Child Development; Atlanta, GA. 2005 April.

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: recent developments and applications. *Psychoneuroendocrinology*, 19(4), 313-333. doi:10.1016/0306-4530(94)90013-2

Knitzer, J. & Perry, D. F. (2009). Poverty and infant and toddler development. In C. H. Zeanah (Ed,). *Handbook of Infant Mental Health* (pp. 135-152). New York, NY, US: The Guildford Press

Koo, T. K., & Li, M. Y. (2016). A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *Journal of chiropractic medicine*, *15*(2), 155-163. doi: https://doi.org/10.1016/j.jcm.2016.02.012

Larson, M. C., White, B. P., Cochran, A., Donzella, B., & Gunnar, M. (1998). Dampening of the cortisol response to handling at 3 months in human infants and its relation to sleep, circadian cortisol activity, and behavioral distress. *Developmental Psychobiology*, *33*(4), 327-337. doi:10.1002/(SICI)1098-2302(199812)33:4<327::AID-DEV4>3.0.CO;2-S

Laurent, H. K., Ablow, J. C., & Measelle, J. (2012). Taking stress response out of the box: Stability, discontinuity, and temperament effects on HPA and SNS across social stressors in mother–infant dyads. *Developmental Psychology*, *48*(1), 35-45. doi:10.1037/a0025518

Laursen, B. P., & Hoff, E. (2006). Person-centered and variable-centered approaches to longitudinal data. *Merrill-Palmer Quarterly*, *52*(3), 377-389. http://www.jstor.org/stable/23096200

Lewis, M., Alessandri, S. M., & Sullivan, M. W. (1990). Violation of expectancy, loss of control, and anger expressions in young infants. *Developmental Psychology*, *26*(5), 745-751. doi:10.1037/0012-1649.26.5.745

Little, R. J., & Rubin, D. B. (1989). The analysis of social science data with missing values. *Sociological Methods & Research*, *18*(2-3), 292-326. doi: https://doi.org/10.1177/0049124189018002004

Lo, Y., Mendell, N. R., & Rubin, D. B. (2001). Testing the number of components in a normal mixture. *Biometrika*, *88*(3), 767-778. http://www.jstor.org/stable/2673445

Lomax, R. G. (2001). *Statistical concepts: A second course for education and the behavioral sciences*. Mahwah, NJ: Lawrence Erlbaum Publishers.

Lovallo, W. R., & Thomas, T. L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 342-367). New York, NY, US: Cambridge University Press.Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: a meta-analysis. *Psychological Bulletin*, *130*(4), 531-552. doi:10.1037/0033-2909.130.4.531

Lord, C., Hall, G., Soares, C. N., & Steiner, M. (2011). Physiological stress response in postpartum women with obsessive–compulsive disorder: a pilot study. *Psychoneuroendocrinology*, *36*(1), 133-138. doi:10.1016/j.psyneuen.2010.04.014

Lummaa, V., Vuorisalo, T., Barr, R. G., & Lehtonen, L. (1998). Why cry? Adaptive significance of intensive crying in human infants. *Evolution and Human Behavior*, *19*(3), 193-202. doi:10.1016/S1090-5138(98)00014-2

Matousek, R. H., Pruessner, J. C., & Dobkin, P. L. (2011). Changes in the cortisol awakening response (CAR) following participation in mindfulness-based stress reduction in women who completed treatment for breast cancer. *Complementary Therapies in Clinical Practice, 17*(2), 65-70. Doi: 10.1016/j.ctcp.2010.10.005

McEwen, B. S. (2000). The neurobiology of stress: from serendipity to clinical relevance. *Brain Research Interactive, 886*(1), 172-189. doi:10.1016/S0006-8993(00)02950-4

McCorry, L. K. (2007). Physiology of the autonomic nervous system. *American Journal of Pharmaceutical Education, 71*(4), 78-89. doi:10.5688/aj710478

McLachlan, G., & Peel, D. (2004). *Finite mixture models*. New York, NY: John Wiley & Sons.

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience, 24*(1), 1161-1192. doi:10.1146/annurev.neuro.24.1.1161

Miller, R., & Plessow, F. (2013). Transformation techniques for cross-sectional and longitudinal endocrine data: application to salivary cortisol concentrations. *Psychoneuroendocrinology, 38*(6), 941-946.

Miner, J. L., & Clarke-Stewart, K. (2008). Trajectories of externalizing behavior from age 2 to age 9: Relations with gender, temperament, ethnicity, parenting, and rater. *Developmental Psychology, 44*(3), 771-786. doi: 10.1037/0012-1649.44.3.771

Moore, G. A., Cohn, J. F., & Campbell, S. B. (2001). Infant affective responses to mother's still face at 6 months differentially predict externalizing and internalizing behaviors at 18 months. *Developmental Psychology, 37*(5), 706-714. Doi: 10.1037/0012-1649.37.5.706

Munck, A., Guyre, P. M., & Halbrook, N. J. (1984). Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. *Endocrine Reviews, 5*(1), 25-44. doi:10.1210/edrv-5-1-25

Muthen, B., & Muthen, L. (2009). Categorical latent variable modeling with cross-sectional data. Retrieved from http://www.statmodel.com/course_materials.shtml

Muthén, L.K. & Muthén, B.O. (1998-2015). Mplus User's Guide. Seventh Edition. Los Angeles, CA: Muthén & Muthén

Nater, U. M., & Rohleder, N. (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology, 34*(4), 486-496. doi:10.1016/j.psyneuen.2009.01.014

Nederhof, E., Marceau, K., Shirtcliff, E. A., Hastings, P. D., & Oldehinkel, A. J. (2015). Autonomic and adrenocortical interactions predict mental health in late adolescence: The TRAILS Study. *Journal of Abnormal Child Psychology, 43*(5), 847-861. doi:10.1007/s10802-014-9958-6

Nesse, R. M., & Young, E. A. (2000). Evolutionary origins and functions of the stress response. In G. Fink (Ed.), *Encyclopedia of stress* (Vol. 2, pp. 79–84). New York, NY: Academic Press.

Nesse, R. M., Bhatnagar, S., & Young, E. A. (2007). Evolutionary origins and functions of the stress response. In B. McEwen, E. R. de Kloet, R. Rubin, G. Chrousos, A. Steptoe, N. Rose, I. Craig, & G. Feuerstein (Eds.), *Encyclopedia of Stress*, (pp. 965-970). Salt Lake City, UT: Academic Press

Pariante, C. M. (2003). Depression, stress and the adrenal axis. *Journal of Neuroendocrinology, 15*(8), 811-812. doi:10.1046/j.1365-2826.2003.01058.

Porter, C. L., Jones, B. L., Evans, C. A., & Robinson, C. C. (2009). A comparative study of arm-restraint methodology: differential effects of mother and stranger restrainers on infants' distress reactivity at 6 and 9 months of age. *Infancy*, *14*(3), 306-324.

Putnam, S. P., & Stifter, C. A. (2002). Development of approach and inhibition in the first year: Parallel findings from motor behavior, temperament ratings and directional cardiac response. *Developmental Science*, *5*(4), 441-451. doi:10.1111/1467-7687.00239

Quas, J. A., Hong, M., Alkon, A., & Boyce, W. T. (2000). Dissociations between psychobiologic reactivity and emotional expression in children. *Developmental Psychobiology*, *37*(3), 153-175. doi: 10.1002/1098-2302(200011)37:3<153::AID-DEV4>3.0.CO;2-Y

Ramsay, D., & Lewis, M. (2003). Reactivity and regulation in cortisol and behavioral responses to stress. *Child Development*, *74*(2), 456-464. doi: 10.1111/1467-8624.7402009

Reeves, J. W., Fisher, A. J., Newman, M. G., & Granger, D. A. (2016). Sympathetic and hypothalamic-pituitary-adrenal asymmetry in generalized anxiety disorder. *Psychophysiology*, 53(6), 951-957. doi: 10.1111/psyp.12634

Richardson, J. T. (2011). Eta squared and partial eta squared as measures of effect size in educational research. *Educational Research Review*, *6*(2), 135-147.

Roesch, S. C., Villodas, M., & Villodas, F. (2010). Latent class/profile analysis in maltreatment research: A commentary on Nooner et al., Pears et al., and looking beyond. *Child Abuse & Neglect*, *34*(3), 155-160. doi:10.1016/j.chiabu.2010.01.003

Rohleder, N., Nater, U. M., Wolf, J. M., Ehlert, U., & Kirschbaum, C. (2004). Psychosocial stress-induced activation of salivary alpha-amylase: an indicator of sympathetic activity? *Annals of the New York Academy of Sciences, 1032*, 258-263. doi:10.1196/annals.1314.033

Rothbart, M. K. (2007). Temperament, development, and personality. *Current Directions in Psychological Science, 16*(4), 207-212. doi:10.1111/j.1467-8721.2007.00505.x

Rothbart, M. K. (1989). Temperament in childhood: A framework. In G.A. Kohnstamm, J.E. Bates, & M.J. Rothbart (Eds.), *Temperament in Childhood* (pp. 59-73). Chichester, England: Wiley.

Rothbart M. K., & Derryberry D. (1981). Development of individual differences in temperament. In Lamb M.E., Brown A. (Eds.), *Advances in Developmental Psychology* (pp. 37–86). Hillsdale, NJ: Erlbaum

Rothbart, M. K., Derryberry, D., & Hershey, K. (2000). Stability of temperament in childhood: Laboratory infant assessment to parent report at seven years. In V. J. Molfese & D. L. Molfese (Eds.), *Temperament and Personality Development over the Life Span* (pp. 85–119). Hillsdale, NJ: Erlbaum.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews, 21*(1), 55-89. doi:10.1210/edrv.21.1.0389

Scerbo, A. S., & Kolko, D. J. (1994). Salivary testosterone and cortisol in disruptive children: relationship to aggressive, hyperactive, and internalizing behaviors. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*(8), 1174-1184. doi: 10.1097/00004583-199410000-00013

Scher, A., Hall, W. A., Zaidman-Zait, A., & Weinberg, J. (2010). Sleep quality, cortisol levels, and behavioral regulation in toddlers. *Developmental Psychobiology, 52*(1), 44-53. doi:10.1002/dev.20410

Schumacher, S., Kirschbaum, C., Fydrich, T., & Ströhle, A. (2013). Is salivary alpha-amylase an

indicator of autonomic nervous system dysregulations in mental disorders? A review of preliminary findings and the interactions with cortisol. *Psychoneuroendocrinology, 38*(6), 729-743. doi:10.1016/j.psyneuen.2013.02.003

Schwarz, G. (1978). Estimating the dimension of a model. *The Annals of Statistics, 6*(2), 461-464.

Selye, H. (1936). A syndrome produced by diverse nocuous agents. *Nature, 138*(3479), 32.

Selye, H. (1956). *The stress of life*. New York: NY. McGraw-Hill.

Sherman, L. J., Stupica, B., Dykas, M. J., Ramos-Marcuse, F., & Cassidy, J. (2013). The development of negative reactivity in irritable newborns as a function of attachment. *Infant Behavior and Development, 36*(1), 139-146. doi: 10.1016/j.infbeh.2012.11.004

Shirtcliff, E. A., Granger, D. A., Booth, A., & Johnson, D. (2005). Low salivary cortisol levels and externalizing behavior problems in youth. *Development and Psychopathology, 17*(01), 167-184. doi:10.1017/S0954579405050091

Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: uses in assessing rater reliability. *Psychological Bulletin, 86*(2), 420-428. doi:10.1037/0033-2909.86.2.420

Southwick, S. M., Bremner, J. D., Rasmusson, A., Morgan, C. A., Arnsten, A., & Charney, D. S. (1999). Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder. *Biological Psychiatry, 46*(9), 1192-1204. doi:10.1016/S0006-3223(99)00219-X

Spinrad, T. L., Eisenberg, N., Granger, D. A., Eggum, N. D., Sallquist, J., Haugen, R. G., ... & Hofer, C. (2009). Individual differences in preschoolers' salivary cortisol and alpha-amylase reactivity: Relations to temperament and maladjustment. *Hormones and Behavior, 56*(1), 133-139. doi:10.1016/j.yhbeh.2009.03.020

Stansbury, K., & Gunnar, M. R. (1994). Adrenocortical activity and emotion regulation. *Monographs of the Society for Research in Child Development, 59*(2-3), 108-134. Retrieved from http://www.jstor.org/stable/1166141

Stenberg, C. R., Campos, J. J., & Emde, R. N. (1983). The facial expression of anger in seven-month-old infants. *Child Development, 54*(1), 178-184. http://www.jstor.org/stable/1129875

Steudte-Schmiedgen, S., Stalder, T., Schönfeld, S., Wittchen, H. U., Trautmann, S., Alexander, N., ... & Kirschbaum, C. (2015). Hair cortisol concentrations and cortisol stress reactivity predict PTSD symptom increase after trauma exposure during military deployment. *Psychoneuroendocrinology, 59,* 123-133. doi:10.1016/j.psyneuen.2015.05.007

Stevens, J. P. (2009). *Applied multivariate statistics for the social sciences (5th edition).* New York, NY: Routledge.

Stifter, C. A., & Braungart, J. M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology, 31*(3), 448-455. doi:10.1037/0012-1649.31.3.448

Sturge-Apple, M. L., Davies, P. T., Cicchetti, D., & Manning, L. G. (2012). Interparental violence, maternal emotional unavailability and children's cortisol functioning in family contexts. *Developmental Psychology, 48*(1), 237. doi:10.1037/a0025419

Swingler, M. M., Perry, N. B., Calkins, S. D., & Bell, M. A. (2014). Maternal sensitivity and infant response to frustration: The moderating role of EEG asymmetry. *Infant Behavior and Development, 37*(4), 523-535. doi: 10.1016/j.infbeh.2014.06.010

Takai, N., Yamaguchi, M., Aragaki, T., Eto, K., Uchihashi, K., & Nishikawa, Y. (2004). Effect of psychological stress on the salivary cortisol and amylase levels in healthy young

adults. *Archives of Oral Biology, 49*, 963-968. doi:10.1016/j.archoralbio.2004.06.007

Tanaka, Y., Ishitobi, Y., Maruyama, Y., Kawano, A., Ando, T., Okamoto, S., ... & Hanada, H. (2012). Salivary alpha-amylase and cortisol responsiveness following electrical stimulation stress in major depressive disorder patients. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *36*(2), 220-224. doi:10.1016/j.pnpbp.2011.10.005

Tarullo, A. R., & Gunnar, M. R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behavior, 50*(4), 632-639. Doi: 10.1016/j.yhbeh.2006.06.010

Tarullo, A. R., Quevedo, K., & Gunnar, M. R. (2008). The LHPA system and neurobehavioral development. In C. A. Nelson & M. Luciana (Eds.), *Handbook of developmental cognitive neuroscience* (pp. 63-82). Cambridge, MA, US: MIT Press.

Thompson, B., & Snyder, P. A. (1997). Statistical significance testing practices in the Journal of Experimental Education. *Journal of Experimental Education, 66*(1), 75–83. Doi: 10.1080/00220979709601396

Thompson, B., & Snyder, P. A. (1998). Statistical significance and reliability analyses in recent Journal of Counseling & Development research articles. *Journal of Counseling & Development, 76*(4), 436–441. Doi: 10.1002/j.1556-6676.1998.tb02702.x

Tollenaar, M. S., Beijers, R., Jansen, J., Riksen-Walraven, J. M. A., & De Weerth, C. (2011). Maternal prenatal stress and cortisol reactivity to stressors in human infants. *Stress, 14*(1), 53-65. DOI: 10.3109/10253890.2010.499485

Towe-Goodman, N. R., Stifter, C. A., Mills-Koonce, W. R. Granger, D. A., & The Family Life Project Key Investigators. (2012). Interparental aggression and infant patterns of

adrenocortical and behavioral stress responses. *Developmental Psychobiology*, *54*(7), 685-699. doi:10.1002/dev.20618

Ursache, A., Blair, C., Granger, D. A., Stifter, C., Voegtline, K. & the Family Life Project Investigators (2014). Behavioral reactivity to emotion challenge is associated with cortisol reactivity and regulation at 7, 15, and 24 months of age. *Developmental Psychobiology*, *56*(3), 474-488. doi:10.1002/dev.21113

van Veen, J. F., Van Vliet, I. M., DeRijk, R. H., Van Pelt, J., Mertens, B., & Zitman, F. G. (2008). Elevated alpha-amylase but not cortisol in generalized social anxiety disorder. *Psychoneuroendocrinology*, *33*(10), 1313-1321. doi:10.1016/j.psyneuen.2008.07.004

West, S. G., Finch, J. F., & Curran, P. J. (1996). Structural equation models with nonnormal variables: Problems and remedies. In R. H. Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 56-75). Thousand Oaks, CA, US: Sage Publications, Inc.

Weston, R., & Gore Jr, P. A. (2006). A brief guide to structural equation modeling. *The Counseling Psychologist*, *34*(5), 719-751. doi: 10.1177/0011000006286345

Williams, G. A., & Kibowsky, F. (2016). Latent Class Analysis and Latent Profile Analysis. In Jason, L. A., & Glenwick, D. S. (Eds). *Handbook of Methodological Approaches to Community-Based Research: Qualitative, Quantitative, And Mixed Methods*. New York, NY: Oxford University Press.

Wilkinson, L. (1999). Task Force on Statistical Inference, American Psychological Association, Science Directorate.(1999). Statistical methods in psychology journals: Guidelines and explanations. *American Psychologist, 54*(8), 594-604. Doi: 10.1037/0003-066X.54.8.594

Yehuda, R., McFarlane, A., & Shalev, A. (1998). Predicting the development of posttraumatic

stress disorder from the acute response to a traumatic event. *Biological Psychiatry*, *44*(12), 1305-1313. doi:10.1016/S0006-3223(98)00276-5

Yohannan, J., Carlson, J. S., Shepherd, M., & Batsche-McKenzie, K. (2017). Exploring attrition, fidelity, and effectiveness of wraparound services among low-income youth of different racial backgrounds. *Families, Systems, & Health, 35*(4), 430-438. Doi: 10.1037/fsh0000313

Curriculum Vitae

MARIA LAUER

University of Oklahoma Health Sciences Center
The Children's Hospital at OU Medical Center
1200 Children's Ave, Oklahoma City, OK 73104
(302) 743 - 2578 / maria-lauer@ouhsc.edu

EDUCATIONAL TRAINING

Expected June 2018	University of Oklahoma Health Sciences Center Oklahoma City, Oklahoma **Pre-doctoral Clinical Psychology Internship** (APA accredited) Track: Pediatric Psychology
Expected August 2018	Lehigh University, Bethlehem, Pennsylvania **Doctor of Philosophy**, Counseling Psychology Dissertation: *A multi-systemic analysis of infant stress reactivity* Advisor: Susan S. Woodhouse, Ph.D.
2014	Lehigh University, Bethlehem, Pennsylvania **Master of Education**, Counseling and Human Services Advisor: Susan S. Woodhouse, Ph.D.
2011	University of Maryland, College Park, Maryland **Bachelor of Arts**, Psychology Minor: Religious Studies

CLINICAL EXPERIENCE

Current **University of Oklahoma Health Science Center; Oklahoma City, OK**
Pre-doctoral Psychology Intern, Pediatric Psychology Track
Primary supervisor: Ryan Blucker, Ph.D.

Child Clinical Psychology Outpatient Therapy (yearlong): providing psychological treatment to children, adolescents, and families with a wide array of presenting concerns, including: problems with treatment adherence, depression, selective mutism, separation anxiety, PTSD, and behavioral problems. **EBTs used: TF-CBT, PCIT, CBT, Exposure and Desensitization, Parent Management Training**.

OU Latino Clinic, Pediatric Primary Care: integrated pediatric primary care clinic providing Spanish-language services. Assessment, consultation, and treatment provided in Spanish. Received supervision in Spanish from licensed psychologist.

Specialty Clinics (Gastrointestinal and Hematology/Oncology): participated in two, four month rotations through two specialty clinics. Provided psychological assessment and individual/family treatment to children, adolescents, and young adults with a chronic hematological, oncological, or gastrointestinal medical condition. Primary presenting concerns included: behavior problems, anxiety, adjustment to

new diagnosis, adherence, sleep problems, academic concerns, food and feeding concerns, and mood difficulties.

Child Assessment (minor): administering, scoring, and interpreting psychological assessment and evaluation of children and adolescents referred by a primary care provider within a pediatric primary care clinic. Common presentations include ADHD, behavioral management/parenting issues, and learning difficulties. Additional responsibilities include writing integrative reports and providing diagnostic feedback to families.

Family Mental Health Program, OKC Veteran Affairs Medical Center (minor): provided Integrative Behavioral Couple's Therapy to veterans and their significant others. Co-conducted a family-oriented psychoeducational group about healthy relationships. Triage clinician, provided assessment and treatment planning for veterans initiating services.

2016 – 2017 **The Children's Hospital of Philadelphia; Philadelphia, PA**
Consulting and Liaison Psychology Extern
Supervisor: Leela Jackson, PsyD.

Provided consultation to medical teams regarding behavioral or mental health concerns for child and adolescent patients hospitalized with a primary medical diagnosis. Delivered brief interventions to patients during hospitalization and developed behavioral health recommendations for medical team. Attended team and family meetings and provided families with behavioral health recommendations and disposition plan following discharge. Primary presenting concerns: disordered eating, depression, anxiety, adjustment issues, conversion disorder, self-injury and suicidal risk. Attended C&L rounds. Didactics: Adolescent Medicine Grand Rounds.

2015 – 2016 **The Children's Hospital of Philadelphia; Philadelphia, PA**
Eating Disorder Assessment and Treatment Program Psychology Extern
Supervisors: Rachel Millner, PsyD, and Alix C. Timko, PhD.

Provided Family-Based treatment to children and adolescents diagnosed with a restrictive eating disorder. Provided family therapy in Spanish to a monolingual, Spanish-speaking family. Provided cognitive behavioral therapy for co-occurring disorders, primarily depression, OCD, generalized anxiety, and social anxiety. Assessed, treated, and monitored self-injury and suicidal ideation. Co-facilitated an interdisciplinary support group for adolescents preparing for bariatric surgery (Hospital of the University of Pennsylvania/CHOP partnership). Received supervision in Acceptance and Commitment Therapy, Family-Based Treatment for Anorexia, Cognitive Behavioral Therapy, Exposure and Response Prevention for OCD, and Acceptance-Based Separated Family Therapy.

2014 – 2015 **University of Delaware Center for Counseling and Student Development, Newark, DE**
Advanced Practicum Trainee
Supervisors: Jeremy Cohen, PhD., Kimberly Zahm, PhD., and Soonhee Lee, PhD

Delivered short-term psychotherapy to college students. Co-lead a DBT-informed, structured group for undergraduate students with anxiety and depression. Made referrals to and consulted with psychiatry as appropriate. Received group and individual supervision.

Summer 2014 **Lenape Valley Foundation, Doylestown, PA**

Partial Hospital Program Practicum
Supervisor: Phillip Braun, PhD.

Administered, scored, interpreted, and integrated psychodiagnostic assessments. Conducted intake assessments, developed initial treatment plan, and identified appropriate level of care (hospitalization, partial hospital, transitional program, or outpatient).

2013 – 2014 **Lehigh University Counseling and Psychological Services, Bethlehem, PA**
Clinical Practicum
Supervisors: Adrian Chiu, PhD.; MaryAnn Sutton, PhD.

Provided short-term psychotherapy to college students presenting with various concerns including: anxiety, depression, trauma, substance abuse, interpersonal conflicts, and adjustment to college.

EMPLOYMENT

2016 – 2017 **Community Voices Clinic; Bethlehem, PA**
Student Coordinator and Clinical Supervisor
Supervisor: Arpana Inman, PhD.

The Community Voices Clinic is a school-based integrated primary care clinic operating within two local community schools. Clinic provides cost-free services to students and to community members who are uninsured or underinsured. Responsibilities included providing psychological consultation and treatment to children and adolescents. Consulted and collaborated with school counselors. Provided outreach to middle and elementary school faculty, parents, and community.
Conducted brief assessment and intervention with primary care patients. Conduct universal psychosocial screenings during medical visits with primarily Spanish speaking, uninsured or underinsured individuals with low socioeconomic resources.

PROGRAM EVALUATION EXPERIENCE

2015 – 2016 ***AmeriCARES and Beckton Dickinson*, Integrated Care Clinic, El Salvador**
External Program Evaluator
Supervisor: Arpana Inman, PhD., Iveta Slova, PhD.

Lead external program evaluator for an international health education initiative to increase early detection and treatment of Diabetes Miletus and Hypertension in a rural community in El Salvador. Developed the evaluation framework, pre-post assessments, and knowledge acquisition assessments. Conducted evaluation on-site. Created framework for feedback post intervention in the form of a formal evaluation report. Created all contents in Spanish.

CLINICAL SUPERVISION EXPERIENCE

2016 - 2017 Community Voices Clinic, Bethlehem, PA
Coordinator and Clinical Supervisor
Supervisor: Arpana Inman, PhD.

Provided weekly clinical supervision to one internship and two practicum Master's level therapist-trainees. Co-lead weekly group supervision. Designed and provided didactic training on therapeutic interventions with children, adolescents, and adults to trainee cohort. Supervision provided in English and in Spanish, as appropriate.

2014 - 2015 Lehigh University, Bethlehem, PA
Master's Level Practicum and Internship Clinical Supervisor
Supervisor: Arpana Inman, PhD.

Provided weekly individual supervision to six Master's level, counseling trainees during two semester long practicums. Supervised work by trainees in child clinical, family, community, and college counseling settings. Provided weekly group supervision to five Master's level, school-counseling trainees completing practicum training at local schools.

PEER-REVIEWED PUBLICATIONS

Satterwhite, D. B., **Lauer, M.,** Bakaeva, G., & Hill, C.E. (2017). Disclosure and nondisclosure about being in counseling. *Counselling Psychology Quarterly*. *30*(2), 115-133.

Woodhouse, S. S., **Lauer, M.,** Beeney, J.R.S., & Cassidy, J., (2014). Psychotherapy process and relationship in the context of a brief attachment based mother infant intervention. *Psychotherapy*. *52*(1), 145-150.

Hill, C. E., Satterwhite, D. B., **Larrimore, M. L**., Mann, A. R., Johnson, V. C., Simon, R. E., ... & Knox, S. (2012). Attitudes about psychotherapy: A qualitative study of introductory psychology students who have never been in psychotherapy and the influence of attachment style. *Counselling and Psychotherapy Research*, *12*(1), 13-24.

In preparation

Lauer, M., Woodhouse, S. S., (in preparation). *Infant stress physiology at 6 months and later socio-emotional functioning at 12 months: A multi-systemic analysis of infant stress reactivity*

Lauer, M., Molenaar, C. M., & Inman, A.G. (In preparation, data collection and analysis complete). *Attachment style, shame proneness, and supervisee disclosure.*

Woodhouse, S. S., **Lauer, M.,** Jog, M. & Cassidy, J. (In preparation). *Mothers' representations of their infants' distress: Relations with maternal self-efficacy, perceptions of infant temperament, maternal sensitivity, and attachment.*

Woodhouse, S. S., **Lauer, M.,** Admoni, N., Jog, M., Hollander, T. (In preparation*). The Circle of CARE study: Integrating Mental Health and the Circle of Security-Parenting Intervention to Promote Positive Parenting and Child Behavior.*

Non-referred articles

Lauer, M. (2017). Identifying eating disorders in Latinas: Racial and ethnic bias in care. *Psychotherapy Bulletin, 52*(3), 41-48.

Lauer, M. (2013). Invisible Identities: Working with undocumented students in college counseling centers. *Psychotherapy Bulletin*. *48*(2), 43-46.

PEER REVIEWED PAPER PRESENATIONS

Lauer, M., & Woodhouse, S. S. (2018, July). Individual Differences in Infant Stress Reactivity: Identifying Homogenous Subgroups. Paper to be presented at the International Congress on Infant Studies, Philadelphia, Pennsylvania.

Lauer, M., & Woodhouse, S. S. (2017, June). *Maternal sensitivity and depression as predictors of infant's salivary alpha amylase levels in response to a stressor task.* Paper presented at the Annual Meeting of the International Attachment Conference, London, England.

Lauer, M., Woodhouse, S. S., Jog, M., Kim, M., & Cassidy, J. (2017, June). *Links between client adult attachment style and client perceptions of working alliance and group engagement.* Paper presented at the Annual Meeting of the Society for Psychotherapy Research, Toronto, Canada.

Satterwhite, D., Lauer, M., Bakaeva, G., & Hill, C. E. (2016, June). *Disclosure and nondisclosure about counseling.* In B. Farber (Chair), On client disclosures in psychotherapy: "Honesty is such a lonely word, everyone is so untrue." Panel presented at the International Society of Psychotherapy Research meeting, Jerusalem, Israel.

Lauer, M., & Woodhouse, S. S., (2016, March). *Salivary alpha-amylase reactivity to arm restraint in 6-month-old infants: links to maternal caregiving*. Paper presented at the biennial International Conference on Infant Studies in New Orleans, Louisiana.

Woodhouse, S. S., Lauer, M., Beeney, J. R. S., & Cassidy, J. (2014, July). *Mother-intervener relationship and the process of intervention: links to outcome in an attachment-based, mother-infant home visiting intervention.* In J. Ablow (Chair), Parent research in the news. Symposium presented at the biennial International Conference on Infant Studies, Berlin, Germany.

PEER REVIEWED POSTER PRESENTATIONS

Lauer, M., Mayes, S., Kahn, O. (2018, April). *A mixed methods examination of quality of life among children and adolescents living with a bleeding disorder and their siblings.* Poster presented at the Society of Pediatric Psychology Annual Conference in Orlando, Florida.

Lauer, M., Jog, M., Kim, M., Woodhouse, S. S. & Cassidy, J. (2017, March). *Links between client adult attachment style and client perceptions of group engagement.* Poster presented at the Annual Meeting of the Society for Research in Child Development in Austin, Texas.

Lauer, M., Hopp, S., Inman, A., & Silova. I. (2016, August). *Enhancing counseling psychology training: lessons learned from non-traditional training.* Poster presented at the annual conference of the American Psychological Association in Denver, Colorado.

Lauer, M., Inman, A. G. & Molenaar, C. M. (2016, August). *Shame proneness and supervisee disclosure: the mediating role of attachment to supervisor.* Poster presented at the annual conference of the American Psychological Association in Denver, Colorado.

Woodhouse, S.S., Lauer, M., Admoni, N., Kim, M., & Lee, M. (2016, March). *Maternal caregiving and infant respiratory sinus arrhythmia immediately after an episode of crying ends.* Poster to be presented at the biennial International Conference on Infant Studies in New Orleans, Louisiana.

Lauer, M., Satterwhite, D. B., Bakaeva, G., & Hill, C. E. (2015, August). *Disclosure of counseling usage by college students.* Poster presented at the annual conference of the American Psychological Association in Toronto, Ontario, Canada.

Woodhouse, S. S., Lauer, M., Hollander, T., Jog, M., Billings, K. (2014, July). *A systematic case comparison of eye contact and mutual gaze in secure vs. insecure mother-infant dyads in naturalistic interactions.* Poster presented at the biennial International Conference on Infant Studies, Berlin, Germany

Lauer, M., & Zelaya, D. G., (2014, March). *Indocumentados: Invisible identities.* Round table presented at the Society of Counseling Psychology Conference in Atlanta, GA.

Woodhouse, S.S., Beeney, J.R., & Larrimore*, M.L. (2013, April). *Attachment-related differences in both mother and infant emotion regulation and stress reactivity*. Poster presented at the Biennial meeting of the Society for Research in Child Development in Seattle, WA.

Larrimore*, M.L., Zelaya, D., Molenaar, C.M., Kwon, O., & Inman, A. (2013, January). *Understanding of human trafficking in the United States.* Workshop at the 30th annual Winter Roundtable Conference. New York, NY.

RESEARCH EXPERIENCE

Current **Research Assistant, OU Children's Hospital**
Comparison of quality of life between siblings with and without hemophilia.
Comparison between pediatric and adult primary care consults.
Supervised by Sunnye Mayes, PhD, ABPP., Stephen Gillaspy, PhD.

2012 – 2017 **Chief Graduate Assistant (assistantship funded), *Lehigh University***
Supervisor: Susan S. Woodhouse, PhD.
Project: Caregiving, Attachment, and Emotion Regulation (CARE). Funded by NIH, NICHD grant 5R01HD068594.

2014 – 2016 **Project Coordinator, Lehigh University**
Supervisor: Susan S. Woodhouse, PhD.
Project*: Community Engagement in Research to Promote Positive Parenting and Child Behavior: Integrating Mental Health and the Circle of Security.* Funded by Lehigh University Faculty Initiation Grant. Significant contributor to development of grant application.

2015 – 2016 **Principal Investigator**, *Lehigh University*
Supervisor: Susan S. Woodhouse, PhD.
Qualifying Project: *Salivary Alpha Amylase Reactivity to Arm Restraint in 6-Month-Old Infants: Links to Maternal Caregiving*

2010 –2013 **Research Assistant,** *University of Maryland*, College Park
Supervisors: Clara E. Hill, PhD, Katie Ehrlich, M.S., and Jingqing Liu, PhD.
Research assistant on two labs. One focused on adolescent and parent conflict and another conducting Consensual Qualitative Research.

TEACHING EXPERIENCE AND GUEST LECTURES

2016 **Invited Guest Speaker,** Lehigh University CAPS
Seminar in Supervision
Invited lecture on supervising mental health clinicians.

2016 **Invited Guest Lecturer,** *Consultation in Psychology Class*
Guest lecture about consultation in psychology.
Instructor of Record: Arpana Inman, PhD.

2013 **Graduate Teaching Assistant**, Introduction to Family Counseling
Lehigh University, Bethlehem, PA
Instructor of Record: Arnold Spokane, PhD.

2012 **Undergraduate Teaching Assistant**
University of Maryland, College Park, MD
Leading a two-hour weekly lab with undergraduate students.
Instructor of Record: Clara E. Hill, PhD.

GRANTS AND AWARDS

2017 College of Education, Diversity Committee Travel Fund - $575
2017 Doctoral Travel Grants for Global Opportunities - $1000
2015 Graduate Student Senate, Travel Grant - $150
2014 Scholarship to attend APA workshop, Women Counseling Men
2013 Lehigh University, Women's Center: Educator Award
2013 Pennsylvania Psychological Association Graduate Student Community Service Project
2012 College of Education: Community and Equity Grant - $1000
2012 Core Competencies Grant - $500

UNFUNDED GRANTS

2014 **Research Supplement to Promote Diversity in Health-Related Research**. National Institutes of Mental Health. *Project*: Minds of Mothers Study (MOMS). (PI of parent study: Susan Woodhouse, PhD). Total funds requested: $91,140.

PROFESSIONAL LEADERSHIP

2014 – 2016	Chair, Student Development Committee, Society for the Advancement of Psychotherapy. Voting Member of Board of Directors
2015	Student Member of Faculty Search Committee, Lehigh University
2013 – 2014	Campus Representative, Student Affiliates of Seventeen, Lehigh University
2012 – 2013	Unit Representative, Graduate Student Senate, Lehigh University

PROFESSIONAL MEMBERSHIP

American Psychological Association
Society of Counseling Psychology, Division 17
Society of Pediatric Psychology, Division 54
Society for the Advancement of Psychotherapy, Division 29
National Latino/a Psychology Association
Philadelphia Behavior Therapy Association
Psi-Chi National Honor Society

PROFESSIONAL DEVELOPMENT – TRAINING AND WORKSHOPS

2014 Circle of Security International. Certified COS-P interventionist

2013 Recognizing and Responding to Human Trafficking in a Child Welfare Context
2012 Heart Rate Variability (MindWare Inc.)