AN EXAMINATION OF THE SOCIAL SELF PRESERVATION MODEL AND THE PHYSIOLOGICAL RESONANCE OF SOCIAL STRESS

Christina Noel White, B.A.

An Abstract Presented to the Graduate Faculty of
Saint Louis University in Partial Fulfillment
of the Requirements for the Degree of
Master of Science

2013

Abstract

The social self preservation model posits that threats to the social self result in a unique and coordinated psychobiological response that evolved due to its adaptive benefits. Stressors that threaten the social self elicit feelings of shame and other negative self-conscious emotions, as well as increased hypothalamic-pituitary-adrenal (HPA) axis activity. The current study sought to test this model by exposing individuals to an acute stressor, and determining if they exhibit the emotional, physiological, and behavioral components proposed by the self preservation model. In addition, the physiological and emotional reactions of an observing participant were assessed to determine if they too exhibited a physiological and emotional reaction to observing an individual under social stress. Results supported the social self preservation model in that participants undergoing the acute stressor task exhibited significantly greater cortisol response and self-reported personal distress, as compared to observing participants. The social self preservation model was also extended by the current findings in that participant submissive nonverbal behavior, particularly gaze aversion, was related to their physiological response. Observing participants exhibited a significant salivary alpha-amylase (sAA) response, demonstrating the physiological effects of observing an individual experiencing social stress. In addition, observing participants with greater trait empathy levels exhibited significantly greater physiological reactivity as well as self-reported personal distress. These findings suggest that nonverbal behavior may be a mechanism of physiological resonance of stress.

AN EXAMINATION OF THE SOCIAL SELF PRESERVATION MODEL AND THE PHYSIOLOGICAL RESONANCE OF SOCIAL STRESS

Christina Noel White, B.A.

A Thesis Presented to the Graduate Faculty of
Saint Louis University in Partial Fulfillment
of the Requirements for the Degree of
Master of Science

2013

COMMITTEE IN CHARGE OF CANDIDACY:

Associate Professor Tony Buchanan,
Chairperson and Advisor

Professor Ronald Kellogg

Assistant Professor Lisa Willoughby

Acknowledgements

I would like to thank all the great teachers I've had throughout my life. First and foremost, I am eternally grateful to my mentor, Dr. Tony Buchanan, Ph.D., for providing me with the opportunity to attend Saint Louis University, and for his constant guidance and insight. He has truly been an inspiration in my life, and this accomplishment would not have been possible without him. I would also like to express my great appreciation for Dr. Lisa Willoughby, Ph.D. and Dr. Ronald Kellogg, Ph.D. for taking the time to be on my thesis committee, and for their invaluable advice throughout this process.

It is with immense gratitude that I acknowledge the years of hard work and dedication of Mary Kralemann and Anh Tran. Their assistance with collecting and analyzing data has made this thesis possible. I would also like to thank the Department of Psychology at Saint Louis University for taking a chance on me and providing me with an extraordinary education. In addition, I am so grateful for the encouragement and counsel of Dr. Terry Cooper, Ed.D, Ph.D. during my early years in college. He is a remarkable educator, and I credit him with inspiring me to pursue a career in psychology. Last but certainly not least, I am forever indebted to the most important and influential teachers I've ever had: my mother and father. Thank you both for always supporting me and for making me the person I am today.

Table of Contents

List of Figures

List of Tables

List of Abbreviations

Hypothalamic-Pituitary-Adrenal Axis: HPA

Autonomic Nervous System: ANS

Sympathetic Nervous System: SNS

Parasympathetic Nervous System: PNS

Paraventricular Nucleus: PVN

Salivary Alpha-Amylase: sAA

Arginine Vasopressin: AVP

Corticotrophin-Releasing Hormone: CRH

Adrenocorticotropic Hormone: ACTH

Trier Social Stress Test: TSST

Social Anxiety Disorder: SAD

Perception-Action Model: PAM

Empathic Trier Social Stress Test: eTSST

Interpersonal Reactivity Index: IRI

Emotional Response Questionnaire: ERQ

Expectation Maximization: EM

Area Under the Curve with Respect to Increase: AUC*i*

Electromyography: EMG

CHAPTER 1: LITERATURE REVIEW & DESCRIPTION OF STUDY

Introduction

Although stress was once thought to be a global response that is identical across all contexts, more recent stress research supports an integrated specificity model of stress. This perspective is based on the premise that stress responses are actually context-specific, thus different types of stressors elicit unique physiological and behavioral stress responses (Dickerson, Gruenewald & Kemeny, 2004). The diversity of stress responses is considered more adaptive, since this allows an organism to more effectively cope with the distinct characteristics of each situation (Kemeny, 2008; Weiner, 1992). An integrated specificity model of stress is also based on the premise that emotions play an integral role in stress responses, and are therefore also diverse and dependent upon context. For instance, the emotions elicited by losing a loved one might include sadness and despair, while experiencing social rejection might elicit shame and embarrassment. The unique emotional response to different types of stressors is believed to be an important component of psychobiological stress responses to specific types of stressors, perhaps by providing motivation to adaptively respond (Dickerson, et al., 2004).

The coordinated physiological, behavioral, and emotional responses to threats to the social-self seem to be unique when compared to threats to the physical self. Because physical and social threats exert differential demands on an organism, an integrated specificity model of stress would predict that these two types of stressors would elicit differential stress responses that are adaptive for coping with each specific type of stressor. Threats to the social self occur when an individual is in a situation in which there is the potential for a loss of social status, self-esteem, or social acceptance. Contexts that threaten the social self are often characterized by the potential for negative evaluation from others. Dickerson, Gruenewald, and Kemeny (2004) proposed the social self preservation model, which posits that threats to the social self are associated with a unique set of physiological, behavioral and emotional responses. In particular, stressors that threaten the social self are related to activation of the hypothalamus-pituitary-adrenal (HPA) axis, which is a context-specific physiological stress response that is adaptive in situations in which the social-self has been threatened. In addition to HPA axis activation, threats to the social self also elicit negatively self-evaluative emotions, particularly shame, along with submissive nonverbal behaviors, which are also considered adaptive in such contexts.

Stress responses affect more than just the individual who directly experiences the stressor. Recent research suggests that those who are exposed to an individual exhibiting a stress response may also show a stress response. A basic component of empathy is called empathic physiological resonance, in which individuals who are exposed to someone who is in pain or under stress exhibit similar physiological reactions, even though the observer may not be directly affected (Decety & Jackson, 2006; Decety & Meyer, 2008; Preston & de Waal, 2002). In this way, physiological stress can be contagious, spreading to those who are in the presence of someone experiencing a stress response. Recent evidence has emerged suggesting that a crucial component of the coordinated psychobiological response to threats to the social self may be contagious, specifically activation of the HPA axis. Buchanan, Bagley, Stansfield and Preston (2012) first demonstrated that simultaneously-interacting participants can exhibit similar HPA axis activation, and this similarity in physiological stress response was related to trait empathy levels. The proposed study will assess the social self preservation model through the examination of physiological stress responses, changes in emotions, as well as nonverbal behaviors associated with shame and threats to the social self. The current study will attempt to replicate the findings of Buchanan et al. (2012), and extend them to include the behavioral and emotional components of the unique psychobiological response to social self threats. Using the same simultaneously-interacting participants design, this study will examine whether the physiological and emotional components of the psychobiological response to a social threat in one target individual can resonate in another individual who is observing the target under social stress. In addition, submissive nonverbal behaviors in stressed participants will be measured as a potential mechanism of physiological resonance of social stress.

Human Stress Responses

Stress responses involve the activation of various physiological regulatory systems of the body (Kudielka & Kirschbaum, 2007). These physiological responses are believed to have evolved due to their utility in allowing an organism to cope with environmental and internal threats. When an organism is exposed to a stressor, those systems that facilitate adaptation to a threat are activated, while unnecessary systems are suppressed in order to more efficiently utilize energy (Kemeny, 2008). The particular physiological systems that are activated can be context-specific or more generalized in nature. The autonomic nervous system (ANS) is a global physiological stress system that plays a role in the regulation of a variety of functions, including responding to general stressors. In particular, the ANS coordinates activity throughout the body, including cardiovascular, respiratory, and other visceral functions (Lovallo, 2005). It is composed of the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS functions to increase the state of physiological activation, while the PNS functions to decrease activation. The concerted actions of these two components of the ANS ultimately work to maintain homeostasis within the body (Thayer & Sternberg, 2006). Arousal of the SNS begins with the paraventricular nucleus (PVN) of the hypothalamus activating the brainstem nucleus of the solitary tract. Neural signals are carried by sympathetic preganglionic fibers to the paravertebral chain ganglia in the spinal cord, which in turn synapses with postganglionic fibers that connect to the adrenal glands located above the kidneys. From here, cholinergic transmission to the adrenal medulla, the innermost portion of the adrenal gland, causes the release of epinephrine, which can act as both a hormone and a neurotransmitter.

Immediately upon release of epinephrine, the SNS activates various physiological systems in preparation to cope with the demands of the current stressor. In particular, blood vessels are constricted, increasing heart rate and blood pressure, which results in a rush of oxygen and glucose to skeletal muscles, in preparation for movement. Additionally, epinephrine stimulates a rise in blood glucose levels and initiates the breakdown of lipids, both of which provide the additional energy needed to cope with the stressor (Lovallo, 2005). The SNS is activated within seconds of exposure to any type of stressor, including both physical and psychological stressors, as well as positively- and negatively-valenced stressors. Since the SNS is activated after exposure to a wide array of different stressors (Lovallo & Thomas, 2000), measures of SNS activity are used as general-arousal indicators in research.

Commonly used measures of SNS activity include cardiovascular measures, such as heart rate and blood pressure. Another physiological measure of SNS activity is salivary alpha-amylase (sAA), an enzyme found in saliva. Produced in the salivary glands, the main function of sAA is the initiation of starch digestion in the mouth. It is released upon activation of the ANS and correlates with response patterns of other physiological measures of the SNS, including cardiovascular parameters, and is reduced by antagonists of the adrenergic system (Granger, Kivlighan, el-Sheikh, Gordis, & Stroud, 2007; Rohleder, Wolf, Maldonado, & Kirschbaum, 2006).

Since sAA corresponds with SNS activity, it is considered another general-arousal indicator that occurs when an individual is presented with either physical or psychological stressors. In fact, it has been suggested that sAA is actually a more sensitive and reliable measure of SNS activity than cardiovascular measures like heart rate, since it is less affected by confounding variables like posture (Nater & Rohleder, 2009). In laboratory settings, saliva samples are simple, convenient, and noninvasive measures of sAA levels that can be taken before and after the introduction of a stressor (Nater & Rohleder, 2009; Rohleder, et al., 2006).

The Hypothalamic-Pituitary-Adrenal Axis (HPA)

Another primary physiological stress response is the hypothalamic-pituitary-adrenocortical (HPA) axis, which is a more specific index of stress reactivity than activation of the SNS. The HPA axis begins with activation of the PVN, which leads to the secretion of arginine vasopressin (AVP) and corticotrophin-releasing hormone (CRH) into the portal circulation between the hypothalamus and anterior pituitary gland. These hormones then stimulate the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH), which circulates through the bloodstream to the adrenal cortex, causing the release of cortisol. Cortisol is a steroid hormone that is instrumental in many metabolic processes and can be measured in saliva, blood, and urine (Kemeny, 2008). This series of neurohormonal processes leading to the secretion of cortisol is much slower than those leading to SNS activation, occurring over the course of minutes rather than seconds, resulting in a peak of cortisol in saliva between 20 to 40 minutes after the onset of a stressor (Dickerson & Kemeny, 2004; Kemeny, 2008; Kirschbaum, Pirke, & Hellhammer, 1993). Like sAA, cortisol levels are most easily measured in laboratory settings using saliva samples taken before and after the introduction of a stressor. Due to convenience, salivary cortisol is the preferred method of measuring HPA activation in human research.

The Trier Social Stress Test (TSST).

HPA axis activity in humans occurs only in specific contexts, particularly those involving social-evaluation and uncontrollability (Dickerson, Mycek & Zaldivar, 2009). Activation of the HPA axis varies considerably among individuals, and eliciting a cortisol response in a laboratory setting is notoriously difficult (Dickerson & Kemeny, 2004; Dickerson, Mycek & Zaldivar, 2009). A meta-analysis of 208 acute psychological stressor tasks indicated certain essential characteristics of acute stressor tasks that successfully elicited HPA axis activation, as indicated by a cortisol response (Dickerson & Kemeny, 2004). Specifically, they found that stressors most likely to elicit a cortisol response include motivated behavior, uncontrollability, and were characterized by a socially-evaluative component in which performance is negatively judged by others. In fact, individuals who had undergone a stressor task that included a socially-evaluative component exhibited cortisol responses that were greater than four times the magnitude of those exhibited by individuals who had undergone a stressor without any socially-evaluative component. Those stressor tasks that included *both* uncontrollability and social-evaluation aspects elicited the strongest cortisol response. The Trier Social Stress Test (TSST) is one of the few psychological stressors that Dickerson and Kemeny (2004) conclude satisfies these essential components, eliciting strong HPA axis activation. The TSST is a motivated performance task in which the participant must perform a public speaking and mental arithmetic task in front of a negatively-evaluative, unfamiliar audience (Kirschbaum, Pirke, & Hellhammer, 1993). The TSST is an ideal acute stressor task to induce HPA axis activation, which the social self preservation model asserts is uniquely associated with threats to the social self.

Social stress & HPA axis activity.

There is a wealth of evidence that both acute and chronic social-rejection are related to activation of the HPA axis in humans (Gruenewald, Kemeny, Aziz, & Fahey, 2004). Nonhuman research also supports this relationship, showing an association between HPA activation and the experience of social stress in many different species, particularly in primates (Avitsur, Stark & Sheridan, 2001; Czoty, Gould, & Nader, 2008; Sassenrath, 1970; Shively, Laber-Laird & Anton, 1997). Based on this research, Dickerson, Gruenewald & Kemeny (2009) propose that the release of cortisol in contexts that threaten the social self is adaptive by regulating crucial immunological and metabolic processes to allow an organism to more effectively cope with the social self threat. More specifically, animal research suggests that cortisol's regulation of immune system activity is related to behavioral disengagement and submissive nonverbal behaviors (Avitsur, Stark, & Sheridan, 2001), which is often an adaptive response to social self threats. The regulation of metabolic processes may also be an adaptive function of HPA axis activation, since cortisol leads to the release of glucose, which provides energy for the brain and peripheral nervous tissue to prepare the organism for action (Dickerson, Gruenewald & Kemeny, 2009). In this way, Dickerson, Gruenewald, and Kemeny (2009) suggest that activation of the HPA axis may be an evolutionarily adaptive response to contexts in which the social self is threatened.

Shame & Submissive Nonverbal Behaviors

Research has consistently found that threats to the social self are not only related to HPA axis activity, but also to shame-related emotions. Circumstances that threaten the social-self, including negative social-evaluation or rejection, are related to shame and other negative self-conscious emotions (see Leary, 2007; Tracy & Robins, 2004 for review). Shame is experienced when one judges themselves to be inferior or inadequate compared to others (Tangney, 1995). The idea that shame is a socially-relevant emotion can be traced back to Charles Darwin, who recognized that shame seems to only be evoked by the judgment of others (Darwin, 1872). Gilbert (1989) suggests that shame is aroused in an individual when their social status is threatened, and they feel inferior or powerless in comparison to others. Even in the absence of an audience, shame is thought to be the result of evoking an imagined negatively-evaluative other (Dickerson & Kemeny, 2004).

Lewis and Ramsay (2002) found that children as young as three years of age who were exposed to negative social evaluation exhibited a significant cortisol response. Relatedly, previous research has found abnormally high cortisol levels in shy children, compared to their non-shy peers (Dettling et al., 1999; Schmidt, Fox, Sternberg, Gold, Smith & Schulkin, 1999), and shyness has been suggested to be a precursor to the development of social anxiety (Rettew, 2000). This research demonstrates that, even early in development, the human experience of shame seems to be uniquely related to HPA activation. In adults, Gruenewald, Kemeny, Aziz and Fahey (2004) found that those who underwent a version of the TSST with a negatively-evaluative audience not only exhibited the greatest increase in shame and the greatest decrease in social self-esteem, but also the greatest increase in cortisol levels, as compared to participants who underwent a version of the TSST *without* the presence of an evaluative audience. Importantly, the conditions did not differ on other negative emotions including anxiety or sadness, or on perceived task difficulty, performance, and effort, further supporting the notion that shame was uniquely affected by negative social evaluation.

Individuals who hold negative beliefs about themselves, including low self-esteem and low social competencies, exhibit augmented cortisol responses to the TSST (Pruessner, Hellhammer & Kirschbaum, 1999; Schmidt, et al., 1999). In a review of the literature, Zoccola and Dickerson (2012) concluded that stressors that included a socially-evaluative component showed a significant relationship between rumination and cortisol. Specifically, those individuals who reported greater rumination also exhibited greater cortisol reactivity to stress as well as delayed recovery of cortisol levels. Since research also shows that individuals who experience greater shame after a social stressor also tend to experience greater rumination as well, perhaps rumination about the event that threatens one's social self and the experience of shame-related emotions are both components of the psychobiological response to social self threats (Zoccola, Dickerson & Lam, 2012). These studies suggest that self-conscious emotions, particularly shame, might be uniquely affected by negative social-evaluation, and may be specifically related to HPA axis reactivity. The social self preservation model proposes that shame is an important affective component of the coordinated psychobiological response to threats to the social self (Dickerson, et al., 2004).

Shame is related to distinct nonverbal displays, specifically submissive behaviors and postures, including slumped posture, lowered head, and gaze aversion (Gilbert, 2000; Keltner, 1995). Gilbert, Pehl & Allan (1994) provide additional evidence of a unique relationship between shame and submissive nonverbal behaviors by finding a strong positive correlation between submissive behaviors and self-reported feelings of shame, but not feelings of guilt, a related self-conscious emotion. The social self preservation model accounts for this relationship by proposing that these responses are evolutionarily advantageous for group cohesion and ultimately survival (Dickerson, Gruenewald, & Kemeny, 2009). Research suggests that submissive nonverbal behaviors are cross-culturally recognized (Keltner, 1995), and submissive behaviors have been observed in many other animal species including several primate species, rats, wolves, domestic dogs, and seals (de Waal & Luttrell, 1985; Cafazzo, Valsecchi, Bonanni & Natoli, 2010; Czoty, Gould, & Nader, 2008; Keltner & Busswell, 1997; Maslow, 1936; Roswell, 1974). More recently, Tracy and Matsumoto (2008) found evidence of nonverbal displays of shame in congenitally blind humans in a competitive context. Evidence of the conservation of submissive behaviors across cultures and even across species implies that these nonverbal behaviors may have evolved because they serve a crucial adaptive function. The social self preservation model asserts that this function is to reestablish social status and group cohesion after experiencing a threat to the social self (Dickerson, et al., 2004).

It has been theorized that submissive behaviors evolved as a means to convey subordination when an organism is threatened by a more dominant other. Exhibiting submissive behaviors signals the acceptance of another's power over them, or conveys appeasement after committing a social transgression (Keltner & Buswell, 1997). Appeasement is a vital behavior for the maintenance of social relationships and one's social status. Submissive nonverbal behaviors may serve as appeasement to others by displaying the shame and regret one feels for their own misconduct in order to maintain trust among group members, thereby minimizing the severity of the negative response from others (Fessler, 2007; Gilbert, 2007). Not only this, but choosing appeasement strategies, such as exhibiting shame through the use of submissive behaviors, instead of fighting after a social transgression saves the organism's energy, which can then be utilized for other fitness-enhancing endeavors such as resource acquisition (Gangestad & Simpson, 2000). The social self preservation model proposes that submissive nonverbal behaviors are a component of the coordinated psychobiological response to threats to the social self (Dickerson, et al., 2004), though it is currently unclear exactly which submissive behaviors are involved.

In support of the premise that submissive behaviors function to communicate shame to others in order to reestablish social bonds, research has found that individuals are able to identify when others are experiencing shame by analyzing their nonverbal behavior, and this recognition elicits feelings of sympathy for the shamed individual (Keltner, 1995). Similarly, Giner-Sorolla, Castano, Espinosa and Brown (2008) found that apologies that expressed more shame, as compared to simply guilt, evoked significantly less recipient insult. These studies support the view that shame expressions serve to promote the reestablishment of group cohesion after a social transgression, perhaps by lessening the severity of negative social consequences due to one's own misconduct. More recent research has also shown that individuals who underwent the TSST subsequently exhibited more prosocial behaviors compared to control participants (von Dawans, Fischbacher, Kirschbaum, Fehr & Heinrichs, 2012), which could be an attempt on the part of the shamed individual to restore their place within the social hierarchy through the use of positive gestures towards others. Dickerson, Gruenewald & Kemeny (2009) propose that shame and submissive nonverbal behavior serve a critical adaptive function when the social self is threatened. Shame provides a strong motivation to modify one's behavior in order to reestablish one's social status, while accompanying submissive nonverbal behaviors signal to others that the individual is indeed shameful and accepts their own transgression, which helps to reduce group conflict. Expressions of shame may also elicit conciliatory behaviors in others, further facilitating the reestablishment of group cohesion.

Nonhuman research has consistently found a relationship between submissive behaviors and HPA axis reactivity, revealing that increased socially-avoidant behaviors in primates are associated with increased cortisol levels (Sapolsky, 1990). However, there is currently very little research investigating the relationship between HPA axis reactivity, and submissive or avoidant nonverbal behaviors in humans. Aside from the current study, the only known research investigating nonverbal behaviors and cortisol reactivity of participants completing the TSST was conducted by Lerner, Dahl, Hariri, and Taylor (2007), although only facial expressions of speakers was examined. These researchers found that individuals who exhibited more fearful facial expressions also showed significantly larger cardiovascular and cortisol responses (Lerner, Dahl, Hariri & Taylor, 2007). Expressions of fear are theoretically similar to submissive and shameful nonverbal behaviors, which may also be associated with exaggerated HPA reactivity.

Individuals with social anxiety disorder (SAD) tend to show exaggerated cortisol reactivity in response to the TSST, which is correlated with the social avoidance behaviors they subsequently exhibit (Roelofs, van Peer, Berretty, de Jong, Spinhoven & Elzinga, 2009). SAD, also known as social phobia, is a psychological condition characterized by a fear of social situations, particularly of negative evaluation from others. Individuals with SAD experience chronically elevated feelings of shame and self-consciousness (Weeks, Rodebaugh, Heimberg, Norton & Jakatdar, 2009). In addition, clinical research suggests that a very common behavioral symptom of SAD is avoidance of eye contact (Schneier, Kent & Hirsch, 2011), which is a prototypical submissive behavior. Individuals diagnosed with SAD also avoid eye contact during social interactions (Marks & Gelder, 1969; Ohman, 1986). Moukheiber, Rautureau, Perez-Diaz, Soussignan, Dubal, Jouvent and Pelissolo (2010) found through the use of eye tracking technology that individuals with SAD engage in significantly less fixations and dwell time in the eye area of photos of faces, as compared to healthy controls. Studies that have found an association between submissive nonverbal behaviors and cortisol reactivity, along with clinical research investigating the anxiety disorder SAD provides support for the social self preservation model by demonstrating a reliable relationship among submissive nonverbal behaviors, shame, and HPA axis activity.

Empathy

In order for shame and submissive nonverbal behaviors to serve the proposed evolutionary functions, individuals must be able to determine the psychological state of another so they can recognize when an individual is experiencing shame. Most theorists describe empathy as an other-focused state of mind, in which an individual experiences congruent or similar emotions and thoughts as another. It has been proposed that empathy evolved as a mechanism for promoting prosocial behavior and group cohesion (Decety & Jackson, 2006). Empathy is multidimensional, including affective, cognitive, and physiological components in response to witnessing the emotional state of another (Decety & Meyer, 2008). Empathic physiological resonance occurs when an observing-individual is exposed to a target individual experiencing an emotional state including a physiological response, and in response, the observing individual exhibits similar physiological activity (Decety & Jackson, 2006; Decety & Meyer, 2008).

The perception-action model (PAM) of empathy is often used to explain the mechanism of these effects (Preston & de Waal, 2002). The PAM states that an automatic, underlying component of empathic responses is similar or even overlapping neural activity between individuals. This overlap in neural representations for experiencing a particular state produces similar physiological responses between individuals, which is referred to as empathic physiological resonance. PAM predicts that the physiology of an individual experiencing an empathic response will correlate with that of the other, since their underlying neural activation is so similar (Preston & de Waal, 2002). The process of physiological resonance is automatic and can occur without conscious awareness (Decety & Meyer, 2008). Previous research has demonstrated physiological resonance between individuals using various measures including heart rate, skin conductance, facial muscle activity, pupil dilation, and neural activity (e.g., see Decety & Jackson, 2006; Eisenberg & Fabes, 1990; Sze, Gyurak, Goodkind & Levenson, 2012). The current study expands on this research by investigating the physiological resonance of the psychobiological response to social self threats.

eTSST & physiological resonance.

A modified version of the TSST, referred to as the empathic TSST (eTSST), was introduced by Buchanan, Bagley, Stansfield, and Preston (2012), in order to study the physiological resonance of social stress. These authors simultaneously examined the physiological responses of 2 participants at a time, one of whom was the speaker and the other served as the observer in a modified TSST paradigm. Observers' cortisol responses were greater when their associated speaker exhibited a large cortisol response, and those observers with higher trait empathy exhibited greater cortisol and sAA reactivity (Buchanan et al., 2012). While physiological resonance using the eTSST has been demonstrated, research has not yet identified possible mechanisms by which the observer picks up on the speaker's physiological stress response in order to resonate their response. The current study investigates the hypothesis that the speakers' submissive nonverbal behavior is one factor allowing observers to sense the physiological stress response in the speakers, leading to empathic physiological resonance.

Nonverbal behavior accounts for a significant and salient portion of human communication, conveying a great deal of information between individuals (Baesler & Burgoon, 1987; Burgoon, Beutler, Le Poire, Engle, Bergan, Salvio & Mohr, 1993; Kleinke, 1986). There is a long history of research on nonverbal behaviors related to anxiety (although there is little research on nonverbal behavior specifically related to stress), which include random movements and lack of body coordination (Burgoon, Kelley, Newton, & Keeley-Dyreson, 1989; Burgoon, Le Poire, Beutler, Bergan, & Engle, 1992; Burgoon et al., 1993; Finn, Sawyer, & Behnke 2003; Fuller, Horii, & Conner, 1992; Mulac & Sherman, 1974; Sparks & Greene, 1992), fidgeting and trembling (Finn, et al., 2003), as well as self-touching (Burgoon et al., 1992; Burgoon & Le Poire, 1999; Finn, et al., 2003; Fuller, Horii, & Conner, 1992; Shreve, Harrigan, Kues, & Kagas, 1988), among others. In addition, there is evidence that individuals are very accurate in distinguishing between nonverbal displays of similar emotions, including shame and embarrassment (Keltner & Buswell, 1997). This implies that humans have evolved a keen sensitivity to the nonverbal behavior of others, allowing us to infer very specific psychological states from these behaviors.

Perhaps, during the eTSST, a speaker's nonverbal expression of social stress is used by the observer to determine the speaker's stress response, which then elicits an automatic empathic physiological response in the observer. The resonance of social stress could expand on the social self preservation model, by providing evidence that nonverbal behavior allows others to infer when someone is experiencing shame and social stress, thus allowing the psychobiological response to social self threats to serve its evolutionary function.

Significance

Although the coordinated response to threats to the social self may be adaptive in acute contexts, prolonged exposure to threats to the social self may actually be maladaptive, negatively impacting physical and mental health. Stressful life events coupled with individual differences in stress reactivity may result in chronic activation of the response to social self threats, or even "misfires" in which activation of this response is occurring unnecessarily. The negative health effects of chronic or misfiring psychobiological responses are believed to be mediated by dysregulation of critical stress systems (Chrousos & Gold, 1992; Matthews, Gump & Owens, 2001; McEwen, 2004). In the case of chronic social stress or shame, the result may be dysfunction of the HPA axis, as indicated by an exaggerated or a blunted cortisol response to social stressors (Dickerson, Gruenewald, & Kemeny, 2009). There is evidence of individual differences in shame-proneness and cortisol reactivity in children as young as three years of age (Mills, Imm, Walling, & Weiler, 2008), suggesting these differences may develop quite early in life. Dickerson, Gruenewald, and Kemeny (2009) propose that individual differences in the psychobiological response to social threats can lead to differential vulnerability to dysregulation of the HPA axis, making certain individuals more susceptible to various diseases.

Dysfunction of the HPA axis is related to a variety of different mental and physical ailments. Abnormal cortisol reactivity is related to numerous psychological and physiological illnesses including depression, cardiovascular disease, diabetes, and even death (Harris, Ferrucci, Tracy, Corti, Wacholder & Ettinger, 1999; Matthews et al., 2001; McEwen, 2004). Not only has abnormal HPA activity been linked to the development of many diseases, but so has social stress and shame. Nonhuman primate research has found evidence that chronic social threat is related to increased depression and anxiety-related behaviors, and abnormal cortisol activity (Shively, Laber-Laird & Anton, 1997), as well as increased vulnerability to a variety of disorders (Sapolsky, 1990). In humans, shame and other negative self-conscious emotions are thought to be a central component of depression and social anxiety (Gilbert, 2000), and shame-proneness is associated with psychological maladjustment in general (Tangney, Wagner, Gletcher & Gramzow, 1992). The development of a wide range of psychopathology, including personality disorders and alcoholism, are linked to the experience of excessive social stress and shame.

Research has also found that increased social inhibition (Cole, Kemeny, Fahey, Zack & Naliboff, 2003) and prolonged experiences of shame and guilt (Dickerson, Gruenewald & Kemeny, 2009) are correlated with poor immunological and other negative health outcomes in HIV-positive patients. Along with individual differences, experiencing trauma early in life, particularly child abuse, has been linked to abnormal cortisol reactivity as well as various psychological illnesses (Elzinga, Spinhoven, Berretty, de Jong & Roelofs, 2010). Research such as this suggests that the psychobiological response to social self threat may be detrimental to the health of some individuals, and may be related to various pathologies, both physical and psychological. Therefore, a better understanding of the relationships among social stress, HPA axis activation, shame, and submissive nonverbal behaviors could eventually lead to new treatments for diseases related to HPA axis dysregulation and shame-related emotions.

In particular, social anxiety disorder (SAD) is uniquely related to the social self preservation model. As previously discussed, individuals with SAD exhibit an exaggerated cortisol reactivity in response to social stress (Furlan, DeMartinis, Schweizer, Rickels, & Lucki, 2001; Roelofs, et al., 2009). In addition, submissive behaviors (Russell, Moskowitz, Zuroff, Bleau, Pinard & Young, 2011), particularly gaze aversion or avoidance of eye contact, is a reliable behavioral tendency in these individuals (Marks & Gelder, 1969; Ohman, 1986; Schneier, Kent & Hirsch, 2011). Eye contact and gaze are essential social signals used to regulate interactions (Kleinke, 1986), and the tendency for individuals with SAD to avoid eye contact could negatively affect their interactions with others. Dysfunction of the HPA axis has long been implicated in the pathology of various anxiety disorders, especially SAD (Chrousos & Gold, 1992). Research such as this suggests that SAD may be uniquely related to the psychobiological response to social threats. SAD is the fourth most common psychiatric disorder and the most common anxiety disorder (Kessler et al., 2005). The symptoms of SAD are often socially-debilitating in many areas of life, including employment, education, romantic relationships, friendships, etc. (Stein & Stein, 2008). Weeks, Rodebaugh, Heimberg, Norton & Jakatdar (2009) suggest that SAD may represent a psychobiological response that is adaptive in highly competitive contexts, but has become maladaptive in modern times in which cooperative contexts are now more common. Researching the coordinated psychobiological response to social threats could lead to a better understanding of the etiology of SAD, perhaps contributing to the development of more effective treatments for this disorder.

Recent evidence of the physiological resonance of social stress (Buchanan et al., 2012) has made it apparent that the negative health effects of social stress could also affect individuals who are simply in the presence of someone experiencing social stress. If the physiological consequences of social stress can be contagious, the health of those who are regularly exposed to stressed individuals could be compromised as well. For instance, mental health professionals, social workers, health care providers, and emergency first responders are commonly exposed to individuals experiencing extreme negative emotions, which could lead to chronic physiological and psychological stress. Research investigating physiological resonance could help to explain job burnout in professions that involve regular exposure to stressed individuals. Determining the exact mechanisms by which we infer that an individual is experiencing social stress could help to reduce the potentially negative effects of resonating the social stress of another.

Specific Aims & Hypotheses

The current study is the first to examine speaker whole-body nonverbal behaviors in the context of the TSST, in addition to measures of salivary cortisol and alpha-amylase, heart rate, and emotional responses.

Study 1.

In order to determine which nonverbal behaviors previously associated with anxiety and/or stress should be included as measures of submissive nonverbal behavior exhibited by participants performing the TSST, study 1 was conducted by analyzing archival TSST speech videos (White, McErhney & Buchanan, 2012).

- **Hypothesis 1 (H1)**: It is predicted that the nonverbal behaviors exhibited by the participants will be related to their salivary cortisol response.

Study 2.

In study 2, the aim is to investigate the relationships among the nonverbal behaviors of speaker participants performing the eTSST, the physiological reactivity of these speaker participants, as well as the physiological reactivity of observer participants watching the speaker participants to determine how these components may be affected by threats to the social self.

- **Hypothesis 2.1 (H2.1)**: Based on the social self preservation model (Dickerson, Gruenewald, & Kemeny, 2009), it is predicted that those speaker participants will exhibit significantly greater salivary cortisol responses and great self-reported personal distress compared to observers.
- **Hypothesis 2.2 (H2.2)**: Speakers who exhibit submissive nonverbal behaviors identified in study 1, particularly gaze aversion, will also show greater physiological reactivity to social stress in the form of greater salivary cortisol, sAA, and heart rate changes.

Study 2 will also seek to replicate findings from Buchanan et al. (2012) in which physiological resonance in observers of stressed individuals was first demonstrated.

- **Hypothesis 2.3 (H2.3)**: Thus, it is predicted that (the physiological stress of the speakers will resonate in the paired observer in the form of similar salivary cortisol, sAA, and heart rate changes.
- **Hypothesis 2.4 (H2.4)**: In addition, it is predicted that those observers with higher trait empathy measures will show greater increases in cortisol, sAA, and heart rate in response to watching the stressed speakers.

CHAPTER 2: METHODS

Study 1

Participants.

Study 1 was conducted in order to assess which nonverbal behaviors previously associated with anxiety and/or stress should be included as measures of submissive nonverbal behavior exhibited by participants performing the TSST. Archival videos of 46 (26 female, 20 males; mean age ± s.d. = 19.19 ± 1.33) participants performing the speech portion of the TSST during a recent study were coded for a battery of nonverbal behaviors indicated by previous nonverbal research as being related to anxiety, stress, and submission.

Measures.

Prior to conducting the study, a battery of various nonverbal behaviors was constructed from previous literature on stress and anxiety. The nonverbal items, which can be found in Appendix A, include slumped posture, blinking, gaze aversion, and speechless rigidity. The battery of nonverbal behaviors was used by raters to code archival videos of TSST participants. Trained research assistants time sampled the videos of the speech portion of the eTSST, coding minutes 1, 3 and 5 of each speech for the frequency and duration of various nonverbal behaviors. As recommended by Dael, Mortillaro, & Scherer (2012), raters were given very specific instructions, including that (1) participants can exhibit multiple nonverbal behaviors simultaneously, (2) audio should be disabled during coding, (3) unclear behaviors should not be coded, (3) Code for nonverbal behaviors exhibited by one body part at a time (e.g. head, trunk, arms, etc.).

Salivary cortisol measurements were taken in a multiple time series design. The pre-stressor cortisol measurement was subtracted from the cortisol measurement taken 10 minutes after the TSST for each participant, in order to determine their cortisol response to the TSST. The cortisol response variable was converted to a dichotomous categorical variable. Those participants who exhibited a positive cortisol response were categorized as *responders*, while participants who exhibited a negative or no cortisol response were categorized as *nonresponders*.

Procedure.

The participants underwent the TSST in which they were instructed to imagine that they have been falsely accused of shoplifting while in a store and must defend themselves in front of the store managers in the other room. Before the task begins, the participant underwent a 5 minute preparatory period. The participant was then instructed to step into another room and give a speech to defend themselves in front of one unfamiliar research assistants, a microphone, and a video camera. After speaking for 5 minutes, the participant was then instructed to perform a mental arithmetic task, which also lasted 5 minutes. During the mental arithmetic task, the speaker participant was instructed to count backwards from 1,022, in 13-number steps, as quickly and accurately as possible. Following the mental arithmetic task, saliva samples were taken in order to determine participant cortisol response.

Subsequently, raters were asked to time sample each speech video, coding minute 1, minute 3, and minute 5 of each TSST speech for the frequency and duration of the nonverbal behaviors listed in Appendix A. The resulting values from each minute were added together to form a composite score for each nonverbal item.

Study 2

Participants.

The sample consisted of 100 total participants, forming 50 speaker-observer pairs. Table 1 includes the ages and gender breakdowns of the sample, which were roughly equivalent for both speaker and observer participants.

Table 1. *Description of Ages and Genders of Sample*

	Age		Gender	
	Mean	**SD**	**Males**	**Females**
Speakers	21.66	3.17	26	24
Observers	21.52	3.26	22	28
Total	21.59	3.21	48	52

The sample was comprised of Saint Louis University students ($N = 80$), as well as healthy volunteers recruited from the general population of Saint Louis, Missouri ($N = 20$) through the use of online ads and flyers. Participants were screened for neurological and psychiatric conditions, as well as medications known to affect cortisol reactivity, such as oral contraceptives. Data collected during screening and testing were kept completely confidential. Student participants were given course credit, while community members were given $20 as reimbursement. All participants provided informed consent to participate in the study, and all procedures were approved by the Institutional Review Board of Saint Louis University.

Measures.

Saliva sample measurement of cortisol and sAA

Salivary alpha-amylase and cortisol levels were measured in a multiple time series design in order to assess fluctuations over time, yielding a total of 4 saliva samples collected in Salivette tubes (Sarstedt, Rommelsdorf, Germany). The Salivette tubes contain cotton rolls that the participants were instructed to chew on for 1.5 minutes and then place back into the tube. As recommended by previous research (Rohelder & Nater, 2009), the samples were stored in -20°C before being assayed. The quantitative enzyme kinetic method was used to assess salivary alpha-amylase levels, while cortisol was measured using a commercial immunoassay kit with chemiluminescence detection (CLIA; IBL Hamburg, Germany). Assays were conducted by Dresden LabService, Dresden, Germany.

Heart rate measurement.

In order to measure the heart rates of both speaker and observer participants simultaneously, electrocardiograph electrodes were placed on each participants' right side of the neck, as well as just below their left rib cage. Heart rate was recorded using BioPac Systems MP150 and AcqKnowledge software (BioPac Systems, Santa Barbara, CA) connected to Apple Macintosh laptop computers (Apple Cupertino, CA). The mean heart rate of each participant was recorded separately for the ten-minute baseline period, as well as for the five-minute speech preparation period. Heart rate of both speaker and observers were simultaneously recorded during the ten-minute speech and math test portion of the eTSST.

Emotional Response Questionnaire (ERQ).

Emotional response was measured the Emotional Response Questionnaire (ERQ; based on Batson, 1997). The ERQ is a self-report measure containing a total of 52 Likert scale items, ranging from 1 (not at all) to 7 (extremely). The first section of the ERQ instructs the participant to indicate the degree to which they felt 26 specific emotions during the eTSST, such as happy, troubled, alarmed, distressed, compassionate, and upset. The second section of the ERQ instructs the participant to indicate the degree to which they think the other participant felt the same 26 emotions during the eTSST. Two subscales can be calculated from the ERQ, each of which are meant to distinguish between two distinct aspects of empathy: empathic concern (EC) and personal distress (PD). This measure was completed by all participants after the eTSST, as a measure of their own emotional response, as well as a measure of inferred emotional reaction of the other participant in the pair.

Nonverbal behavior measurement.

The dependent measures for nonverbal behavior was comprised of the battery of nonverbal behaviors chosen based on the findings of study 1. After testing, trained research assistants time sampled the videos of the speech portion of the eTSST, coding minutes 1, 3 and 5 of each speech for various nonverbal behaviors. Appendix A shows the items of the nonverbal battery, as well as previous literature citing these nonverbal behaviors as being related to anxiety or fear.

Interpersonal Reactivity Index (IRI).

Trait empathy levels were measured using the Interpersonal Reactivity Index (IRI; Davis, 1983), which was designed to capture the multidimensional nature of empathy. The IRI is composed of twenty-eight 5-point Likert scale items, ranging from A (does not describe me well) to E (describes me very well). For each item, participants were asked to read a statement and then choose the appropriate letter to indicate how well the statement describes them. Examples of statements from the IRI include, “I daydream and fantasize, with some regularity, about things that might happen to me,” “I sometimes find it difficult to see things from the ‘other guy’s’ point of view,” and “Other people’s misfortunes do not usually disturb me a great deal.” Four subscales comprise the IRI, each of which captures a particular component of empathy identified by theory and previous research. The four subscales include (1) the Perspective-Taking (PT) subscale, which assesses an individual’s tendency to adopt the point of view of another, (2) the Fantasy (F) subscale, which is designed to measure an individual’s tendency to imagine the thoughts and feelings of others, even fictitious characters, (3) the Empathic Concern (EC) subscale, which assesses an individual's feelings of empathy, sympathy, concern, and compassion for others, and finally (4) the Personal Distress (PD) subscale, which is designed to measure feelings of anxiety or unease an individual may feel in interpersonal contexts.

Procedure.

eTSST procedure.

Testing took place between 1200 and 1600 in order to control for natural diurnal cycles of cortisol and salivary alpha-amylase. Upon arrival, the speaker and observer participants were separated into different rooms to obtain informed consent, and to collect baseline saliva samples, heart rate, and ERQ measurements. At this point, the procedure for the observer and the speaker participants diverge. The speaker participant was told by the first experimenter that they will be performing the speech and math test. The speaker participant was instructed to imagine that they have been falsely accused of shoplifting while in a store and must defend themselves in front of the store managers in the other room. During this time, the observer participant in the other room was told by the second experimenter that they will be observing someone who is defending themselves against false accusations of shoplifting. The observer participant was told to remain as stoic as possible during the speech and math test. Before the task begins, both participants undergo a 5 minute preparatory period, still in separate rooms, in which their heart rate was measured. The speaker participant was then instructed to step into the other room and give a speech to defend themselves in front of the second experimenter, the observer participant, a microphone, and a video camera. After speaking in front of the unfamiliar audience for 5 minutes, the speaker participant was then instructed to perform a mental arithmetic task, which also lasted 5 minutes. During the mental arithmetic task, the speaker participant was instructed to count backwards from 1,022, in 13-number steps, as quickly and accurately as possible. When the speaker participant made an error, they were informed of this and told to start back at 1,022. Following the mental arithmetic task, the speaker participant was taken back into the original room and saliva samples from both speaker and observer participants were immediately collected. Both participants completed the post-task ERQ, followed by a filler task involving recognition of facial expressions, and then a third saliva sample was taken. Next, the speaker and observer participants completed the IRI, and a final saliva sample was taken. Lastly, the participants were debriefed together and paid, and any questions or concerns they may have had were addressed. Table 2 describes the procedure chronologically. Including the informed consent process and the debriefing process, each testing session lasted 1.5-2 hours.

Table 2. *Procedures for Simultaneous Testing of 2 Participants Using eTSST Paradigm*

Approximate Duration Since Testing Session Began (minutes)	**Approximate Duration of Specific Task (minutes)**	**Speaker Procedures**	**Observer Procedures**
5	5	Obtained informed consent & HIPAA	Obtained informed consent & HIPAA
15	10	Baseline heart rate measurement	Baseline heart rate measurement
25	10	Performed filler task	Performed filler task
27	2	Saliva sample #1	Saliva sample #1
29	2	Given eTSST instructions	Given eTSST instructions
34	5	Prep heart rate measurement	Prep heart rate measurement
44	10	Performed eTSST speech and math test	Observed speaker performing eTSST speech and math test
46	2	Saliva sample #2	Saliva sample #2
56	10	Filled outs ERQ	Filled out ERQ
66	10	Performed filler task	Performed filler task
68	2	Saliva sample #3	Saliva sample #3
103	35	Performed filler task	Performed filler task
113	10	Filled out IRI	Filled out IRI
115	2	Saliva sample #4	Saliva sample #4
120	5	Debriefed and reimbursed	Debriefed and reimbursed

Procedure for coding nonverbal data of speaker participants speech videos.

Two trained research assistants time sampled the speaker participants' videos of the speech portion of the eTSST (see Baesler & Burgoon, 1987) for both micro- and macrobehaviors. The data recording form, which can be found in Appendix B, was used by the raters to record the frequency and duration of various nonverbal behaviors previously associated with stress (refer to Appendix A). As recommended by Dael, Mortillaro, & Scherer (2012), raters were given very specific instructions, including that (1) participants can exhibit multiple nonverbal behaviors simultaneously, (2) audio should be disabled during coding, (3) unclear behaviors should not be coded, (3) Code for nonverbal behaviors exhibited by one body part at a time (e.g. head, trunk, arms, etc.).

Data Management

Missing physiological data were regarded as missing at random (MCAR, X^2 (68) = 75.91, $p>.05$). When possible, missing physiological data were replaced using Expectation Maximization (EM). EM is one of many methods for estimating missing-completely-at-random values by assembling a missing data correlation matrix based upon underlying distributions (Tabachnick & Fidell, 2007). In cases of missing heart rate data *during* the speech and math test portion of the eTSST, these values were replaced using EM only if four or fewer minutes were missing. However, if a participant was missing more than four minutes of the heart rate values during the speech and math test of the eTSST, they were not included in analyses of heart rate. For participants missing mean baseline or prep heart rate values, EM was used to replace them only if 1 of these values was missing, otherwise the case was removed from analyses. Only 0.0025% (1 out of 400) of the total heart rate data was replaced using EM, although 10% (10 out of 100) of the cases were removed due to missing heart rate data. For sAA, only 0.75% (3 out of 400) of all measurements were replaced, and only 1% (4 out of 400) of the cases had to be removed from analyses due to missing sAA data. No missing cortisol values could be replaced, and only 1 case was removed due to missing cortisol data. All statistical analyses were performed using SPSS 20.0 (Predictive Analytics SoftWare; Chicago, IL).

Summary parameters of repeated measurements can take the form of either measures of overall magnitude, or measures of the response patterns over time. In order to capture the dynamic nature of the physiological measurements, response pattern summary parameters were calculated for all physiological variables for use in analyses examining relationships between physiology and behavior. Area under the curve with respect to increase (AUC*i*) was chosen because this method allows for analysis of changes in values over repeated measures in comparison to a baseline measurement in the form of one statistical value (see Fekedulegn, Andrew, Burchfiel, Violanti, Hartley, Charles & Miller, 2007). This method controls for individual differences in baseline physiology, and captures the extent of change in the variable over time in one summary measure (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003).

CHAPTER 3: RESULTS

Study 1

Study 1 results revealed that several nonverbal items were not reliably coded, including the frequency and duration of arm movements, therefore these items were removed from the nonverbal battery. Several nonverbal items had insufficient variability or even failed to occur at all, including hands in pockets and crossed arms, and these items were also removed from the battery. In total, 17 nonverbal items were removed from the nonverbal battery based on the findings of study 1, leaving 24 items (listed in Table 4 and Appendix A and B).

In order to assess whether the nonverbal behaviors exhibited by the participants was related to their salivary cortisol response, a profile analysis was conducted. Results indicated that cortisol responders ($M = 21.99$; $SD = 1.17$) averted their gaze significantly more often than non-responders ($M = 11.92$; $SD = 1.17$) (White, McErhney & Buchanan, 2012). These findings suggest a potential relationship between gaze aversion and HPA axis reactivity, providing support for H1 which predicted that nonverbal behavior exhibited by the participants will be related to their salivary cortisol response.

The results of this study were then used to construct a battery of nonverbal behaviors used in study 1 measuring submissive behaviors in participants completing the eTSST. Aside from gaze aversion, the revised battery, which can be found in Appendix A and B, includes other frequency and duration measures of nonverbal behaviors from study 1 that showed fair interrater reliability (assessed using Pearson's r) and/or near-significant differences between cortisol responders and nonresponders (assessed using independent t-tests).

Study 2

Cortisol data.

In order to determine if the eTSST successfully induced physiological stress, profile analyses were conducted on the dependent physiological measures, comparing speakers and observers. A multivariate approach to analyzing the physiological data was chosen due to violation of the repeated measures univariate assumption of sphericity. These findings should be considered with caution however, due to the nonnormal distribution of the raw physiological data.

The cortisol data obtained from each of the four saliva samples consisted of the pre-stressor baseline sample, along with 3 post-stressor samples all measured on the same scale. The profile analysis performed on the four measurements of salivary cortisol values revealed that the test of between-subjects effects, which compared roles (speaker or observer), was significant, F (1, 97) = 10.38, $p < 0.05$, partial $\eta^2 = 0.1$, indicating that the pattern of change in cortisol levels between speakers and observers was not parallel. There was also a significant interaction effect, Wilk's Lambda = 0.8, F (3, 95) = 8.03, $p < 0.001$, partial $\eta^2 = 0.2$, indicating that the mean salivary cortisol levels significantly differed between speakers and observers, as can be seen in Figure 1. In addition, there was a significant main effect of time, Wilk's Lambda = 0.5, F (3, 95) = 32.17, $p < 0.001$, partial $\eta^2 = 0.5$, indicating that the change in cortisol levels of all participants was not flat, meaning their cortisol levels significantly changed over time.

Post-hoc analyses of variances (ANOVAs) were conducted to determine at which specific points during testing that the speaker and observer participants exhibited significantly different salivary cortisol responses. Results indicated that, for all samples except the baseline, speakers exhibited significantly greater salivary cortisol levels than observers in the sample taken just after the speech and math test, F (1, 98) = 10.49, p = 0.002, partial η^2= 0.098, the saliva sample taken 20 minutes after the speech and math test, F (1, 98) = 19.05, p < 0.001, partial η^2 = 0.164, and the sample taken 60 minutes after the speech and math test, F (1, 98) = 10.23, p < 0.01. The baseline samples did not significantly differ in cortisol levels between speaker and observers. These findings support H2.1 by demonstrating that the eTSST successfully induced HPA activation in speaker participants, and that the speakers' cortisol responses over time were significantly different from observers.

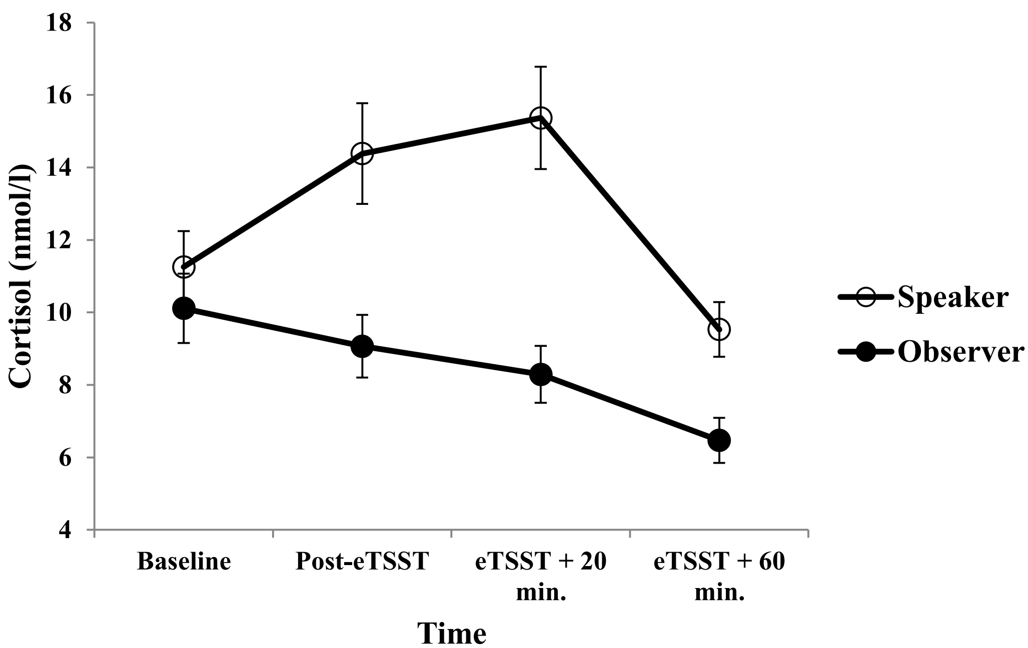

Figure 1. *Raw Cortisol Data.* Salivary cortisol reactivity to eTSST in speaker and observer participants. Values are ±S.E.M.

sAA data.

The sAA data obtained during each of the four saliva samples consisted of the pre-stressor baseline sample, along with 3 post-stressor samples. The profile analysis performed on the four measurements of sAA values showed that the test of between-subjects effects, which compared roles (speaker or observer), was not significant, F (1, 95) = 1.24, p = *n.s.*, partial η^2 =

0.013, indicating that the pattern of change in sAA levels between speakers and observers was parallel. The interaction effect was also nonsignificant, Wilk's Lambda = 1, F (3, 93) = 0.63, p = *n.s.*, partial $\eta^2 = 0.2$, indicating that the mean salivary sAA levels did not significantly differ between speakers and observers. However, there was a significant main effect of time, Wilk's Lambda = 0.69, F (3, 93) = 13.65, $p < 0.001$, partial $\eta^2 = 0.31$, indicating that the change in sAA levels of all participants was not flat, meaning their sAA levels significantly changed over time. As can be seen in Figure 2, observers actually exhibited greater sAA levels at every sample, although these differences were not significant and the general distributions of sAA reactivity were similar for speakers and observers.

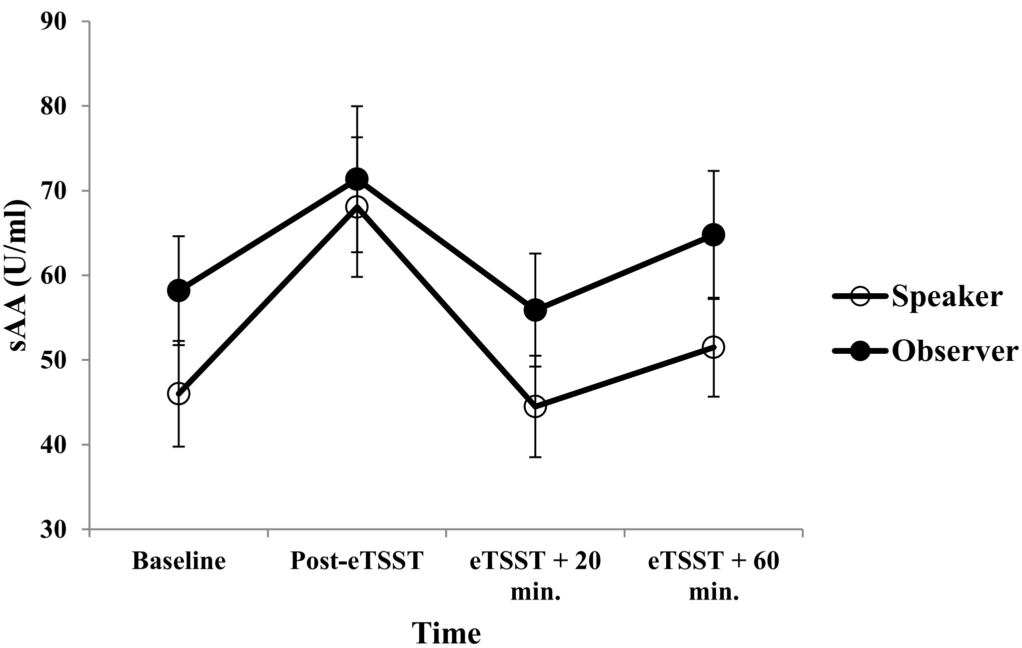

Figure 2. *Raw sAA Data.* Salivary alpha–amylase reactivity to eTSST in speaker and observer participants. Values are ±S.E.M.

Heart rate data.

The profile analysis performed on the four measurements of heart rate values revealed that the test of between-subjects effects, which compared roles (speaker or observer), was significant, $F(1, 88) = 16.59$, $p < 0.001$, partial $\eta^2 = 0.16$, indicating that the pattern of change in heart rate between speakers and observers was not parallel. There was also a significant interaction effect, Wilk's Lambda = 0.48, $F(4, 85) = 23.48$, $p < 0.001$, partial $\eta^2 = 0.53$, indicating that the mean heart rate significantly differed between speakers and observers, as can be seen in Figure 3. In addition, there was a significant main effect of time, Wilk's Lambda = 0.7, $F(4, 85) = 9.34$, $p < 0.001$, partial $\eta^2 = 0.31$, indicating that the change in heart rate of all participants was not flat, meaning their heart rates significantly changed over time. Post-hoc analyses of variances (ANOVAs) were conducted to determine at which points during testing that the speaker and observer participants exhibited significantly different mean heart rates. Results indicated that speaker participants exhibited significantly higher heart rates compared to observers, but only during the speech and math test portion of the eTSST. This included the first 5 minutes of the eTSST, corresponding to the speech portion of the task, $F(1, 90) = 76.55$, $p < 0.001$, partial $\eta^2 = 0.47$, and for minutes 6-10, which correspond to the mental arithmetic portion $F(1, 90) = 37.5$, $p < 0.001$. As seen in Figure 3, the baseline and prep heart rate measurements did not significantly differ, but the mean heart rate of speakers were significantly higher during the eTSST.

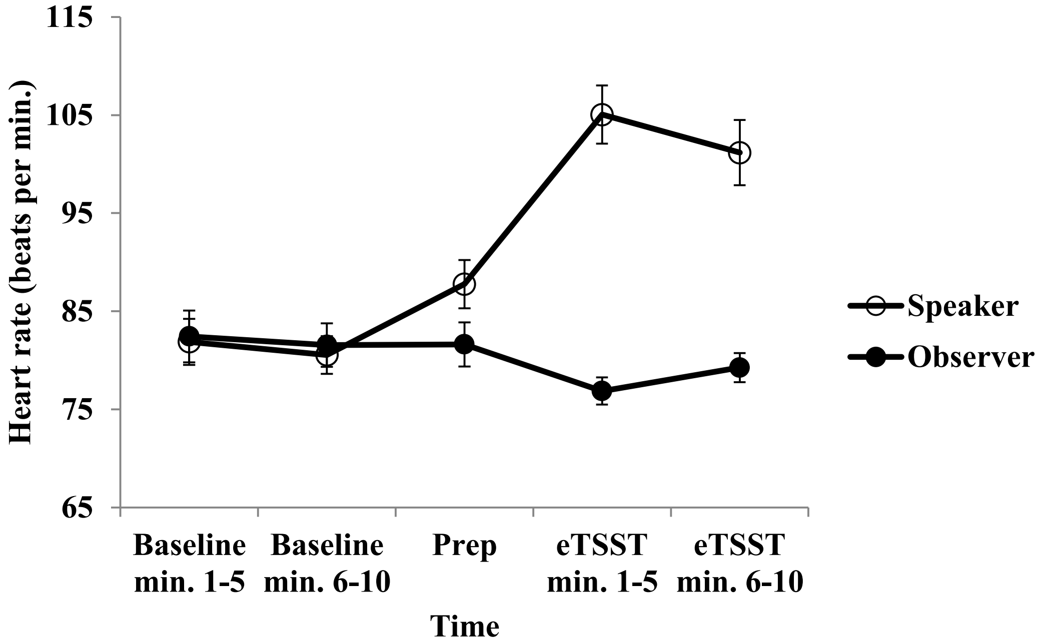

Figure 3. *Raw Heart Rate Data.* Heart rate reactivity to eTSST in speaker and observer participants. Values are ±S.E.M.

ERQ data.

The ERQ data showed a very high Cronbach α value of 0.93, which may be indicative of redundancies in the questionnaire. As can be seen in Table 3, the mean scores for speakers and observers on the 2 subscales of the ERQ, personal distress (PD; indexed by emotions related to personal distress, including alarmed, troubled, and panicked) and empathic concern (EC; emotions including sympathetic, softhearted, and tender) differed.

Table 3. *ERQ Subscale Scores for Speakers and Observers*

	Speakers (*N* = 50)			Observers (*N* = 50)			Total		
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
PD	3.28	1.49	5.63	2.07	1.1	4.75	2.67	1.44	5.63
EC	2.19	1.08	3.83	3.16	1.23	3.83	2.68	1.25	5.33

ANOVAs comparing speaker and observer scores on the subscales of the ERQ showed that the speaker participants reported greater PD compared to observers (a significant main effect for PD, F (1, 98) = 21.40, $p < 0.001$, $\eta^2 = 0.179$). In contrast, observer participants reported feeling significantly more EC compared to speakers (a significant main effect for EC, F (1, 98) = 17.64, $p < 0.001$, $\eta^2 = 0.153$). These findings partially support H2.1 by demonstrating that the eTSST successfully induced greater personal distress in speakers compared to observers.

Nonverbal data.

Only three participants exhibited lip biting behavior once during their speeches, therefore biting lip frequency was removed from subsequent analyses due to insufficient variability. The remaining nonverbal measures, along with means, standard deviations, and measure of interrater reliability are shown in Table 4.

Interrater reliability was assessed by having 20% (10 out of 50) of the speaker participants coded by two raters independently. Intraclass correlation coefficients (ICC) with raters treated as fixed effects, along with Spearman's Rho (ρ) correlation coefficients are presented in Table 4 as measures of interrater reliability due to the presence of both frequency and duration measures (see Rousson, Gasser, & Seifert, 2002). Ratings of the nonverbal measures between raters resulted in a highly significant intraclass correlation coefficient, ICC = 0.83, $p < 0.001$, as well as a highly significant Spearman's Rho value, $\rho = 0.94$, $p < 0.001$, indicating that nonverbal behavior was very reliably coded in general. As Table 4 shows, only 5 out of the 24 nonverbal items resulted in nonsignificant ICC and Spearman's Rho values. Brow lowering duration was not significantly correlated between raters, ICC = 0.33, p = *n.s.*, $\rho = 0.58$, p = *n.s.*, suggesting that brow lowering behaviors were not reliably coded. In addition, the two raters did not reliably code leaning posture frequency, ICC = 0.4, p = *n.s.*, $\rho = 0.26$, p = *n.s.*, or leaning posture duration, ICC = 0.39, p = *n.s.,* $\rho = 0.22$, p = *n.s.* The subjective measures also exhibited low interrater reliability, including global nonverbal stress, ICC = 0.37, $p = n.s.$, ρ = 0.3, $p = n.s.$, and positive arousal, ICC = -0.2, $p = n.s.$, ρ = -0.22, $p = n.s.$

Table 4. *Descriptives and Interrater Reliability Statistics for Nonverbal Items*

Nonverbal Item	Mean	SD	ICC	ρ
Head Aversion Frequency	22.43	11.24	0.98**	0.97**
Smiling Frequency	3.41	2.83	0.91**	0.96**
Smiling Duration	14.76	22.31	0.99**	0.9**
Licking Lips Frequency	1.63	1.84	0.88**	0.75*
Swallowing Frequency	0.63	1.27	0.94**	0.97**
Brow Lowering Duration	1.73	2.57	0.33	0.58
Gaze Aversion Frequency	53.29	71.31	0.62*	0.85**
Gaze Aversion Duration	56.61	36.91	0.98**	0.97**
Body Movements Frequency	7.22	5.14	0.98**	0.92**
Body Movements Duration	60.04	91.23	0.93**	0.88**
Speechless Rigidity Frequency	0.88	3.67	0.53*	0.67*
Speechless Rigidity Duration	0.64	1.69	0.94**	0.68*
Leaning Posture Frequency	2.39	3.41	0.4	0.26
Leaning Posture Duration	2.59	3.61	0.39	0.22
Averted Trunk Frequency	7.94	6.24	0.75**	0.85**
Averted Trunk Duration	19.37	75.71	0.98**	0.86**
Slumped Posture Frequency	0.52	0.86	0.96**	0.77*
Slumped Posture Duration	0.61	1.08	0.92**	0.93**
Self-Adaptors & Self-Touch Frequency	5.88	8.47	0.95**	0.79**
Self-Adaptors & Self-Touch Duration	30.67	80.94	0.99**	0.83**
Global Nonverbal Stress Rating	6	1.19	0.37	0.3
Positive Arousal Rating	4.63	1.26	-0.2	-0.22

Note: $*p \leq .05$, $**p \leq .01$. N = 49 for all analyses.

Correlations between nonverbal behavior and physiology.

Due to the positively skewed distributions of all nonverbal data, as well as the presence of frequency data, the nonparametric Spearman's rho (ρ) was utilized to determine the relationship between the remaining nonverbal items and the physiological measurements. As can be seen in Table 5, speaker cortisol response was significantly related to swallowing frequency, ρ = 0.37, $p < 0.01$. In addition, gaze aversion frequency exhibited a significant correlation with cortisol response at minute 3, ρ = -0.36, $p < 0.05$, and gaze aversion duration exhibited a near-significant correlation with speaker cortisol response at minutes 3 and 5, ρ =

0.37, p = *n.s.* Head aversion frequency was also significantly correlated with cortisol response, $\rho = 0.31$, $p < 0.05$. These results suggest that speaker participants who exhibit a larger cortisol response also exhibit more swallowing, gaze aversion, and head aversion.

Observer cortisol response was most strongly related to speechless rigidity, $\rho = 0.35$, $p < 0.05$, although gaze aversion frequency during minute 5 and swallowing frequency during minute 1 were near significant. These results demonstrate that observers' cortisol responses were higher when their associated speaker exhibited instances of speechless rigidity, and that gaze aversion duration and swallowing frequency may also be related to observer cortisol response.

Table 5. *Spearman's Rho Correlations Among Nonverbal Behaviors Exhibited by Speakers During Minutes 1, 3, and 5 of Speech, and Cortisol Measures.*

Nonverbal Measure	Speaker Cortisol				Observer Cortisol			
	Minute 1	Minute 3	Minute 5	Sum	Minute 1	Minute 3	Minute 5	Sum
Head Aversion Frequency	0.14	0.028	0.31*	0.16	0.14	0.16	0.051	0.14
Smiling Frequency	-0.13	0.041	0.046	-0.1	-0.014	0.016	0.011	0.035
Swallowing Frequency	0.02	0.38**	0.44**	0.37**	0.28	0.01	0.016	0.15
Gaze Aversion Frequency	0.035	-0.36*	-0.14	-0.088	0.23	-0.39	-0.26	0.032
Gaze Aversion Duration	-.12	-0.25	-0.25	-0.22	0.092	-0.14	-0.21	-0.094
Body Movements Frequency	0.037	0.17	0.053	0.053	-0.1	-0.058	-0.18	-0.158
Body Movements Duration	-0.047	0.012	0.002	0.037	0.074	0.051	-0.72	0.055
Speechless Rigidity Frequency	0.22	0.071	0.071	0.065	0.19	0.31*	0.35*	0.36*
Speechless Rigidity Duration	0.22	0.071	0.083	0.077	-0.17	0.31*	-0.33*	0.35*
Slumped Posture Duration	-0.16	0.18	0.006	-0.06	-0.13	-0.2	0.009	-0.14
Self-Adaptors and Self-Touch Frequency	0.046	0.083	0.053	0.078	-0.49	0	-0.072	0.096
Self-Adaptors and Self-Touch Duration	0.135	-0.019	-0.052	0.097	-0.051	0.023	-0.033	0.021
Global Nonverbal Stress Rating	-0.16	-0.12	-0.10	-0.1	0.023	0.48	0.084	0.062

Note: *$p \leq .05$, **$p \leq .01$. N = 49 for all analyses.

The correlations in Table 6 indicated that the speaker's sAA response was most strongly related to swallowing frequency, ρ = 0.37, $p < 0.01$. Gaze aversion duration was also significantly related to speaker sAA, but only during minute 1, ρ = -0.4, $p < 0.01$, and minute 3, ρ = -0.31, $p < 0.05$. In addition, speaker body movement duration during minute 1 was also significant, ρ = -0.32, $p < 0.05$. Global nonverbal stress ratings for the speakers were also correlated with observer sAA response during minute 1, $\rho = -0.45$, $p < 0.01$. These results suggest that the speaker participants sAA responses are related to the speakers' swallowing frequency, gaze aversion duration, body movements duration, and global nonverbal stress ratings.

Speaker gaze aversion frequency was significantly correlated with observer sAA response, $\rho = 0.4$, $p < 0.05$, but only for minute 3. In addition, speaker body movement duration was significantly correlated with observer sAA response, $\rho = -0.32$, $p < 0.05$, although only for minute 3. Slumped posture was also significant, though only in minute 5, $\rho = 0.31$, $p < 0.05$ These results suggest that observer sAA responses are somewhat related to the duration of gaze aversion, slumped posture, and whole body movements

Table 6. *Spearman's Rho Correlations Among Nonverbal Behaviors Exhibited by Speakers During Minutes 1, 3, and 5 of Speech, and sAA Measures.*

Nonverbal Measure	**Speaker sAA**				**Observer sAA**			
	Minute 1	Minute 3	Minute 5	Sum	Minute 1	Minute 3	Minute 5	Sum
Head Aversion Frequency	0.23	0.074	0.31*	0.16	-0.054	0.29	0.084	-0.025
Smiling Frequency	-0.024	0.007	0.046	-0.1	-0.12	-0.024	0.2	0.13
Swallowing Frequency	-0.23	0.1	0.44**	0.37**	-0.096	-0.17	-0.06	-0.05
Gaze Aversion Frequency	-0.18	0.001	-0.14	-088	0.17	-0.39	0.13	0.092
Gaze Aversion Duration	-0.4**	-.31*	0.53	-0.22	0.005	0.4**	0.002	0.032
Body Movements Frequency	0.02	-0.099	-0.25	0.053	-0.021	0.2	0.07	0.09
Body Movements Duration	-0.32*	-0.32*	0.002	0.037	-0.15	-0.32*	0.17	-0.037
Speechless Rigidity Frequency	-0.07	-0.072	0.071	0.065	-0.021	-0.07	0.11	0.12
Speechless Rigidity Duration	-0.07	-0.072	0.083	0.077	-0.021	-0.07	0.11	0.12
Slumped Posture Duration	-0.11	0.11	0.11	0.014	0.09	-0.1	0.31*	0.2
Self-Adaptors and Self-Touch Frequency	0.046	0.014	0.107	0.05	-0.49	-0.061	-0.177	0.096
Self-Adaptors and Self-Touch Duration	0.023	0.05	-0.052	0.097	-0.17	-0.042	-0.033	-0.17
Global Nonverbal Stress Rating	-0.45**	-0.34*	-0.10	-0.1	0.12	0.187	0.17	0.12

Note: **p* ≤ .05, **p ≤ .01. N = 49 for all analyses.

The correlations in Table 7 indicated that the speaker's heart rate was only related to body movement duration, $\rho = -0.36$, $p < 0.05$, and only during minute 1. In addition, head aversion duration was near significant. These results suggest that the speaker participants' heart rates are related to them exhibiting whole body movements. Head aversion duration may also be related to speaker heart rate.

Observer heart rate response was related to quite a few speaker nonverbal items. Specifically, head aversion duration during minute 1 was correlated with observer heart rate, $\rho = 0.36$, $p < 0.05$. Speaker smiling frequency was also significantly correlated with observer heart rate at minute 3, $\rho = 0.37$, $p < 0.05$, at minute 5, $\rho = 0.33$, $p < 0.05$, and for the sum of minutes 1, 3 and 5, $\rho = 0.4$, $p < 0.01$. Self-touch and self-adaptor frequency, $\rho = 0.32$, $p < 0.05$, and duration, $\rho = 0.32$, $p < 0.05$, were both significantly correlated with observer heart rate as well. In addition, speaker general body movements and speechless rigidity exhibited near significant correlations with observer heart rate. These findings suggest that the observers heart rate was related to speaker smiling frequency and self-touch or self-adaptor behaviors, but also possibly to speaker head aversion, general body movements, and speechless rigidity.

Table 7. *Spearman's Rho Correlations Among Nonverbal Behaviors Exhibited by Speakers During Minutes 1, 3, and 5 of Speech, and Heart Rate Measures.*

Nonverbal Measure	Speaker Heart Rate				Observer Heart Rate			
	Minute 1	Minute 3	Minute 5	Sum	Minute 1	Minute 3	Minute 5	Sum
Head Aversion Frequency	0.23	0.25	0.25	0.27	0.36*	0.21	0.18	0.27
Smiling Frequency	-0.082	-0.15	0.021	-0.098	0.1	0.37*	0.33*	0.4**
Swallowing Frequency	-0.23	0.2	0.19	-0.034	-0.068	-0.006	0.003	-.047
Gaze Aversion Frequency	-0.066	-0.083	-0.13	-0.079	-0.037	0.11	-0.013	-0.015
Gaze Aversion Duration	-0.093	-0.006	-0.15	-0.11	-0.062	0.056	-0.006	0.006
Body Movements Frequency	-0.11	0.036	-0.007	-0.016	-0.14	-0.26	-0.14	-0.23
Body Movements Duration	-0.36*	-0.27	-0.25	-0.3	-0.15	-0.17	-0.072	-0.17
Speechless Rigidity Frequency	-0.006	-0.024	-0.15	-0.3	-0.21	-0.27	-0.069	-0.068
Speechless Rigidity Duration	-0.006	-0.024	-0.15	-0.14	-0.21	-0.27	-0.058	-0.068
Self-Adaptors and Self-Touch Frequency	0.08	0.003	-0.043	0.012	0.28	0.33*	0.31*	0.32*
Self-Adaptors and Self-Touch Duration	0.028	-0.053	-0.29	0.004	0.23	0.33*	0.35*	0.3*
Global Nonverbal Stress Rating	-0.14	-.132	-0.1	-0.14	-0.17	-0.18	0.021	-0.102

Note: *$p \leq .05$, **$p \leq .01$. N = 49 for all analyses

Hierarchical logistic regression analyses predicting speaker physiology.

Three separate hierarchical logistic regression analyses were performed to determine if the speakers' physiology predicted their nonverbal behavior, which would support H2.1 that those speakers who exhibit submissive nonverbal behaviors will also show greater physiological reactivity in the form of greater salivary cortisol, sAA, and heart rate changes. Logistic regression was chosen due to the severe positive skew of the nonverbal data (see Tabachnick & Fidell, 2007). In addition, logistic regression is useful when the relationships between the variables may not be linear (Tabachnick & Fidell, 2007), and, due to the exploratory nature of this research, it is not known if the relationships are linear. The physiological variables were reduced to categorical variables by splitting each into high and low groups, in order to create binary dependent variables. The first model predicted high and low cortisol responders, the second predicted high and low sAA responders, and the third predicted high and low heart rate responders. None of the analyses resulted in significant Hosmer-Lemeshow values, indicating there were no issues with the assumption of linearity of the logit.

Hierarchical logistic regression analysis predicting speaker cortisol response

Table 8 shows the resulting values from the first hierarchical logistic regression analysis in which high and low speaker cortisol AUC*i* was the dependent variable. Speakers' sex was the only predictor in block 1. This variable was included because it is known to be related to physiological stress responses, particularly cortisol responses (Kirschbaum, Wust, & Hellhammer, 1992). At block 1, the model was nonsignificant, X^2 (1) = 2.47, *p* = *n.s.* Pseudo R^2 values indicated that the model accounted for approximately 4.9% (Cox and Snell) to 6.6% (Nagelkerke) of the variance. The overall prediction success rate at block 1 was 61.2%. These results indicate that speaker sex does not reliably distinguish between speakers with high or low cortisol AUC*i* speaker participants.

Block 2 consisted of sums of speaker nonverbal items from minute 1, 3, and 5 of their speech, specifically gaze aversion duration, speechless rigidity, self/adaptors and self-touch, and global nonverbal stress rating. These variables were chosen for both theoretical and empirical reasons, and certain nonverbal items could not be included in the analysis due to issues with multicollinearity, as well as large parameter estimates and standard errors (see Tabachnick & Fidell, 2007). Although both block 2 and the entire model were nonsignificant, the overall prediction success rate improved to 71.4%. In addition, the inclusion of block 2 resulted in a larger effect size, with 18% (Cox and Snell) to 24% (Nagelkerke) of the variance in speaker cortisol estimated to be explained by the durations of speaker gaze aversion, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress nonverbal stress rating. Gaze aversion exhibited the only significant Wald statistic, 4.47, $p < 0.05$. Speaker sex showed a near-significant Wald statistic, 3.32, $p =$ *n.s.* However, the adjusted odd ratio of 0.98 indicates that the practical significance is extremely small. These results demonstrate that gaze aversion may be somewhat related to the cortisol response of speakers, but not the other nonverbal behaviors.

Table 8. *Hierarchical Logistic Regression Model Predicting High/Low Speaker Cortisol AUC*i *Response*

	Variable	B	S.E.	Adjusted OR	95% CI for OR Lower	95% CI for OR Upper
Block 1	Sex	-0.91	0.59	0.40	0.127	1.27
Block 2	Sex	-1.2	0.66	0.3	0.083	1.1
	Gaze Aversion Duration	-0.024*	0.011	0.98	0.96	1
	Speechless Rigidity Duration	0.15	0.25	1.17	0.71	1.9
	Self-Adaptors/Self-Touch Duration	0.005	0.007	1.01	0.99	1.02
	Global Nonverbal Stress Rating	0.2	0.36	1.22	0.61	2.47

Note: $*p \leq .05$, $**p \leq .01$. N = 49 for all analyses.

Hierarchical logistic regression analysis predicting speaker sAA.

Table 9 shows the resulting values from the second hierarchical logistic regression analysis in which high and low speaker sAA AUC*i* was the dependent variable. Speakers' sex was the only predictor in block 1. At block 1, the model was nonsignificant, $X^2(1) = 0.023$, $p =$ *n.s.*. Pseudo R^2 values indicated that the model accounted for approximately 0% (Cox and Snell) to .001% (Nagelkerke) of the variance. The overall prediction success rate at block 1 was 53.1%. These results indicate that speaker sex could not reliably distinguish between high and low sAA speaker participants.

Block 2 consisted of sums of speaker nonverbal items from minute 1, 3, and 5 of their speech, specifically gaze aversion duration, speechless rigidity, self/adaptors and self-touch, and global nonverbal stress rating. Although the whole model was near-significant, X^2 (5) = 10.24, p = 0.069, block 2 was indeed significant, X^2 (4) = 10.22, $p < 0.05$. Block 2 also showed a larger prediction success rate of 71.4%, as well as a larger effect size of 18.9% (Cox and Snell) to 25.2% (Nagelkerke). Although no predictors were significant, gaze aversion duration exhibited a near-significant Wald statistic, 3.71, $p = 0.05$. However, the adjusted odd ratio of 0.98 indicates that the practical significance may be very small. These finding suggest that these nonverbal behaviors could reliably distinguish between high and low sAA speaker participants, particularly gaze aversion duration.

Table 9. *Hierarchical Logistic Regression Model Predicting High/Low Speaker sAA AUC*i *Response*

	Variable	B	S.E.	Adjusted OR	95% CI for OR Lower	95% CI for OR Upper
Block 1	Sex	0.087	0.57	1.09	0.36	3.35
Block 2	Sex	0.13	0.65	1.14	0.32	4.06
	Gaze Aversion Duration	-0.025	0.013	0.98	0.95	1
	Speechless Rigidity Duration	0.21	0.28	1.23	0.71	2.12
	Self-Adaptors/Self-Touch Duration	0.002	0.01	1	0.98	1.02
	Global Nonverbal Stress Rating	3.93	0.41	0.64	0.29	1.42

Note: $*p \leq .05$, $**p \leq .01$. N = 49 for all analyses.

Hierarchical logistic regression analysis predicting speaker heart rate.

Table 10 shows the resulting values from the third hierarchical logistic regression analysis in which high and low observer heart rate AUC*i* was the dependent variable. Observers' sex was the only predictor in block 1. At block 1, the model was nonsignificant, X^2 (1) = 0.5, p = *n.s.* Pseudo R^2 values indicated that the model accounted for approximately 1% (Cox and Snell) to 1.3% (Nagelkerke) of the variance. The overall prediction success rate at block 1 was 55.1%.

These results indicate that speaker sex cannot reliably distinguish between high and low heart rate speaker responders.

Block 2 consisted of sums of speaker nonverbal items from minute 1, 3, and 5 of their speech, specifically gaze aversion duration, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress rating. Both the whole model, X^2 (5) = 5.14, p = *n.s.*, and block 2 were nonsignificant, X^2 (4) = 4.64, p = *n.s.* The prediction success rate of did not improve from block 1, though model did explain 9.9% (Cox and Snell) to 13.3% (Nagelkerke) of the variance in speaker sAA. These finding suggest that speaker gaze aversion, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress ratings cannot reliably distinguish between high and low heart rates in speaker participants.

Table 10. *Hierarchical Logistic Regression Model Predicting High/Low Speaker Heart Rate AUC*i *Response*

	Variable	B	S.E.	Adjusted OR	95% CI for OR Lower	95% CI for OR Upper
Block 1	Sex	-0.41	0.58	0.67	0.22	2.07
Block 2	Sex	-0.37	0.61	0.69	0.21	2.3
	Gaze Aversion Duration	-0.003	0.009	1	0.98	1.02
	Speechless Rigidity Duration	0.11	0.23	1.11	0.70	1.76
	Self-Adaptors/Self-Touch Duration	-0.003	0.007	1	0.98	1.01
	Global Nonverbal Stress Rating	-0.63	0.4	0.54	0.24	1.17

Note: *$p \leq .05$, **$p \leq .01$. N = 49 for all analyses.

Correlations among speaker and observer physiological measures.

In order to assess whether the physiological activity of the speakers and observers were related, Pearson r's were calculated. All AUC*i* physiological data met the assumption of normality. The only significant correlation was between speaker sAA AUC*i* and speaker cortisol AUC*i*, r = 0.29, p = 0.05, and between observer sAA AUC*i* and speaker heart rate AUC*i*, r = 0.34, $p < 0.05$.

Correlations among participant physiological and emotional measures.

Spearman's rho correlation coefficients were calculated between the physiological and emotional measures, as well as the observer's trait empathy levels. Table 11 shows the resulting correlation coefficients, along with significance levels. Spearman's rho was chosen due to the highly nonnormal distribution of the ERQ and IRI subscales. Observers' empathic concern (EC) ERQ scores was significantly correlated with their personal distress (PD) ERQ scores, $\rho = 0.49$, $p < 0.01$, suggesting that observers' empathic concern for the speaker was related to the personal distress they experienced. Results also indicated that those observers who scored high on the IRI PD subscale scored significantly higher on the ERQ EC subscale, $\rho = 0.39$, $p < 0.01$, indicating that the observers who reported experiencing more personal distress for others in general also reported experiencing more personal distress for the speaker. These findings suggest a relationship between observers' empathic concern and personal distress for others.

There was a significant correlation between observers' ratings of the speakers' personal distress and speakers' self-reports of personal distress, suggesting that the negative emotional responses of speakers were experienced by the observers ($\rho = 0.284$, $p < 0.05$). In addition, speakers' self-reported personal distress was highly correlated with speakers' ratings of their observer's empathic concern ($\rho = 0.519$, $p < 0.001$), suggesting that those speakers who experienced the greatest personal distress also inferred the most empathic concern in their observers. These results coincide with the proposed function of shame-related emotions, which is to elicit empathic responses from others in order to reestablish group cohesion. The resulting correlations fit well with the theoretical basis of physiological resonance as a component of empathic responses.

Table 11. *Spearman's Rho Correlations Among Physiological and Observer Trait Empathy Measures.*

Measure	Speaker Cortisol AUC*i*	Speaker sAA AUC*i*	Speaker HR AUC*i*	Speaker ERQ-PD	Observer Cortisol AUC*i*	Observer sAA AUC*i*	Observer HR AUC*i*	Observer ERQ-PD	Observer ERQ-EC	Observer IRI-PT	Observer IRI-F	Observer IRI-EC	Observer IRI-PD
Speaker Cortisol AUC*i*													
Speaker sAA AUC*i*	0.14												
Speaker HR AUC*i*	0.27	0.14											
Speaker ERQ-PD	0.041	-0.13	0.1										
Observer Cortisol AUC*i*	0.14	0.03	-0.057	-0.14									
Observer sAA AUC*i*	-0.02	0.09	0.18	0.004	-0.11								
Observer HR AUC*i*	0.05	0.053	0.1	0.15	0.11	0.11							
Observer ERQ-PD	-0.09	-0.093	0.11	0.15	-0.25	0.26	0.071						
Observer ERQ-EC	0.18	0.18	0.13	0.14	-0.1	0.029	0.021	0.49**					
Observer IRI-PT	-0.07	-0.07	0.027	-0.22	0.2	-0.079	0.16	0.015	0.078				
Observer-IRI-F	-0.06	0.2	-0.007	-0.13	-0.066	0.26	0.15	0.1	0.19	0.056			
Observer IRI-EC	-0.025	0.07	-0.036	-0.33*	-0.09	0.061	-0.047	0.18	0.16	0.39**	0.43**		
Observer IRI-PD	0.11	-0.15	0.071	-0.074	-0.2	0.007	-0.096	0.31*	0.39**	-0.037	0.18	0.29*	

Note: *$p<0.05$, **$p<0.01$. $N = 100$ for all analyses. Cases excluded pairwise.

Hierarchical logistic regression analyses predicting observer physiology.

Three separate hierarchical logistic regression analyses were performed to determine if the observers' physiology predicted their nonverbal behavior, which would partially support H2.2 that those speakers who exhibit submissive nonverbal behaviors will also show greater physiological reactivity in the form of greater salivary cortisol, sAA, and heart rate changes. The physiological variables were reduced to categorical variables by splitting each into high and low groups, in order to create binary dependent variables. The first model predicted high and low cortisol responders, the second predicted high and low sAA responders, and the third predicted high and low heart rate responders. None of the analyses resulted in significant Hosmer-Lemeshow values, indicating there were no issues with the assumption of linearity of the logit.

***Hierarchical logistic regression analysis predicting observer cortisol response*.**

Table 12 shows the resulting values from the first hierarchical logistic regression analysis in which high and low observer cortisol AUC*i* was the dependent variable. Observers' sex was the only predictor in block 1. At block 1, the model was nonsignificant, X^2 (1) = 0.51, $p = n.s.$ Pseudo R^2 values indicated that the model accounted for approximately 1% (Cox and Snell) to 2.1% (Nagelkerke) of the variance. The overall prediction success rate at block 1 was 89.8%. These results indicate that speaker sex weakly related to observer cortisol response.

Block 2 consisted of sums of speaker nonverbal items from minute 1, 3, and 5 of their speech, specifically gaze aversion duration, speechless rigidity, self/adaptors and self-touch, and global nonverbal stress rating. Both block 2 and the entire model were nonsignificant, Block: X^2 (1) = 5.09, $p = n.s.$; Model: X^2 (1) = 5.6, $p = n.s.$, and the overall prediction success rate did not improve. The inclusion of block 2 resulted in a larger effect size, however, with 18% (Cox and Snell) to 24% (Nagelkerke) of the variance in observer cortisol explained by the durations of speaker gaze aversion, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress nonverbal stress rating. Although no predictors reached significance, global nonverbal stress rating did reach near-significance, Wald = 3.58, $p < n.s.$ While the adjusted odds ratio of 4 indicates medium effect size, the wide range between confidence intervals demonstrates a lack of precision in the global stress ratings data. These results suggest that that these nonverbal behaviors may be related observer cortisol response.

Table 12. *Hierarchical Logistic Regression Model Predicting High/Low Observer Cortisol AUC*i *Response*

					95% CI for OR	
	Variable	**B**	**S.E.**	**Adjusted OR**	**Lower**	**Upper**
Block 1	Sex	-0.68	0.96	0.51	0.077	3.34
Block 2	Sex	-0.62	1.08	0.54	0.065	4.46
	Gaze Aversion Duration	-0.019	0.019	0.98	0.95	1.02
	Speechless Rigidity Duration	0.25	0.3	1.28	0.71	2.32
	Self-Adaptors/Self-Touch Duration	0.011	0.009	1.01	0.99	1.03
	Global Nonverbal Stress Rating	1.38	0.73	3.97	0.95	16.52

Note: $*p \leq .05$, $**p \leq .01$. N = 49 for all analyses.

Hierarchical logistic regression analysis predicting observer sAA.

Table 13 shows the resulting values from the second hierarchical logistic regression analysis in which high and low observer sAA AUC*i* was the dependent variable. Observers' sex was the only predictor in block 1. At block 1, the model was near-significant, $X^2\,(1) = 3.47$, $p = 0.062$. Pseudo R^2 values indicated that the model accounted for approximately 6.8% (Cox and Snell) to 9.1% (Nagelkerke) of the variance. The odds ratio for observer sex was 3, indicating a medium to small effect size, and the overall prediction success rate at block 1 was 63.3 %. These results indicate that observer sex may be weakly related to sAA.

Block 2 consisted of sums of speaker nonverbal items from minute 1, 3, and 5 of their speech, specifically gaze aversion duration, speechless rigidity, self/adaptors and self-touch, and global nonverbal stress rating. Although the whole model was near-significant, X^2 (5) = 10.37, p = 0.066, block 2 was nonsignificant, X^2 (4) = 6.89, p = *n.s.* Block 2 showed a larger prediction success rate than block 1 of 71.4%, as well as a larger effect size of 19.1% (Cox and Snell) to 25.4% (Nagelkerke). No predictors showed significant Wald statistics. These finding suggest that speaker gaze aversion, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress ratings may possibly be related to observer sAA response.

Table 13. *Hierarchical Logistic Regression Model Predicting High/Low Observer sAA AUC*i *Response*

	Variable	B	S.E.	Adjusted OR	95% CI for OR Lower	95% CI for OR Upper
Block 1	Sex	1.09	0.6	2.98	0.93	9.57
Block 2	Sex	1.55*	0.71	4.68	1.17	18.75
	Gaze Aversion Duration	-0.004	0.009	1	0.98	1.01
	Speechless Rigidity Duration	0.044	0.29	1.05	0.6	1.86
	Self-Adaptors/Self-Touch Duration	-0.1	0.01	0.99	0.97	1.01
	Global Nonverbal Stress Rating	-0.85	0.5	2.34	0.88	6.21

Note: $*p \leq .05$, $**p \leq .01$. N = 49 for all analyses.

Hierarchical logistic regression analysis predicting observer heart rate.

Table 14 shows the resulting values from the third hierarchical logistic regression analysis in which high and low observer heart rate AUC*i* was the dependent variable. Observers' sex was the only predictor in block 1. At block 1, the model was nonsignificant, X^2 (1) = 1.51, p = *n.s.* Pseudo R^2 values indicated that the model accounted for approximately 3% (Cox and Snell) to 4.1% (Nagelkerke) of the variance. The odds ratio for observer sex was 2.04, indicating a small effect size, and the overall prediction success rate at block 1 was only 59.2%. These results indicate that observer sex weakly related to heart rate, if at all.

Block 2 consisted of sums of speaker nonverbal items from minute 1, 3, and 5 of their speech, specifically gaze aversion duration, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress rating. Both the whole model, X^2 (5) = 3.38, p = *n.s.*, and block 2 were nonsignificant, X^2 (4) = 1.83, p = *n.s.* However, the prediction success rate improved from block 1 to 67.3%. In addition, the model explained 6.6% (Cox and Snell) to 8.8% (Nagelkerke) of the variance in speaker heart rate, although no predictors reached significance. These finding suggest that speaker gaze aversion, speechless rigidity, self-adaptors and self-touch, and global nonverbal stress ratings may not be related to speaker heart rate.

Table 14. *Hierarchical Logistic Regression Model Predicting High/Low Observer Heart Rate AUC*i *Response*

					95% CI for OR	
	Variable	**B**	**S.E.**	**Adjusted OR**	**Lower**	**Upper**
Block 1	Sex	0.71	0.59	2.04	0.65	6.42
Block 2	Sex	0.8	0.61	2.22	0.67	7.39
	Gaze Aversion Duration	-0.006	0.009	0.99	0.98	1.01
	Speechless Rigidity Duration	-0.11	0.23	0.9	0.58	1.4
	Self-Adaptors/Self-Touch Duration	-0.003	0.006	1	0.99	1.01
	Global Nonverbal Stress Rating	-0.062	0.33	0.94	0.49	1.79

Note: $^*p \leq .05$, $^{**}p \leq .01$. N = 49 for all analyses.

CHAPTER 4: DISCUSSION

The social self preservation model posits that threats to the social self result in a unique and coordinated psychobiological response that evolved due to its adaptive benefits in these contexts (Dickerson, Gruenewald, & Kemeny, 2004). Specifically, stressors that threaten the social self elicit feelings of shame and other negative self-conscious emotions. In addition, threats to the social self are associated with increased HPA activity. The current study sought to test this model by exposing individuals to an acute stressor, and determining if they exhibit the emotional, physiological, and behavioral components proposed by the self preservation model. In addition, the physiological and emotional reactions of an observing participant were assessed to determine if they too exhibited a physiological and emotional reaction to observing an individual under social stress. Using the same stressor task developed by Buchanan and colleagues (2012) in which physiological resonance of stress was first demonstrated, this study also investigated whether trait empathy levels of the observing participant were related to their physiological reactivity. To our knowledge, this is the first study to examine whole-body nonverbal behaviors in the context of the TSST, in addition to salivary cortisol, sAA, and heart rate, providing a more comprehensive view of the psychobiological response to social threats.

The eTSST elicited a significantly larger cortisol response in speaker participants compared to observer participants, as well as significantly greater heart rate responses. These findings coincide with ample previous research demonstrating that the TSST reliably induces physiological stress (see Dickerson & Kemeny, 2004). Previous analyses revealed that cortisol levels of speakers were significantly higher than those of observers, demonstrating greater HPA activity in speakers. In addition, the ERQ data suggested that the eTSST also induced significantly greater emotions related to personal distress in speakers compared to observers, and personal distress includes shame and other self-conscious emotions. These findings support H2.1 in that they indicate greater HPA activation in those participants exposed to negatively-evaluative social stress, as well as the elicitation of shame and other negative self-conscious emotions, which is consistent with the social self preservation model. While speakers did exhibit significantly higher heart rates during the eTSST, the sAA patterns for speaker and observers did not significantly differ, although speakers sAA levels were consistently higher than observers. These findings provide evidence that SNS activation occurred in both speaker and observer participants, but that speakers showed greater SNS activation during the speech and math test portion of the eTSST, which is also consistent with previous literature (Hellhammer & Schubert, 2012).

Nonverbal Behavior During Stress

The nonverbal data also partially supported the social self preservation model in that submissive nonverbal behaviors, particularly gaze aversion, were somewhat related to the speakers' cortisol responses. Gaze aversion was the strongest predictor of speaker and observer cortisol and sAA responses in several separate analyses. In addition, speaker speechless rigidity and self-touch/self-adaptor behaviors were near significant predictors of speaker and observer cortisol and sAA responses. Swallowing frequency was also significantly correlated with speakers' cortisol responses. However, the effect sizes for these analyses were small, suggesting the practical significance of using submissive behavior to predict physiological stress reactivity is diminished. These results are the first to demonstrate that the speakers' nonverbal behaviors during the eTSST may be related to the physiological stress reactivity of the both the speakers and observers.

Strong interrater reliability was found for coding of nonverbal behaviors, suggesting that nonverbal behaviors can be reliably measured through visual analysis of eTSST speech videos. However, certain nonverbal items showed very poor interrater reliability, including brow lowering duration, and the subjective measures of global nonverbal stress and positive arousal. These findings indicate that brow lowering activity may not be reliably coded visually, which may be partly due to the presence of hair or glasses which obstructed the view of certain speakers' foreheads. In the future, perhaps facial electromyography (EMG) would be a more reliable measure of muscle activity in the forehead. This study also provides evidence that ratings of subjective measures of nonverbal stress behaviors should be avoided in future research, due to poor interrater reliability.

Only one other study has examined nonverbal behavior in relation to stress (Lerner et al., 2007). This study found that the more fear a speaker's face exhibits during the TSST, the greater their cortisol and cardiovascular stress responses. The current results expanded on this finding to show that sAA may also be related to speaker nonverbal behaviors, and that an observing participant's physiology may also be related speaker nonverbal behavior. It is possible that facial expression of fear and social stress share underlying psychological and physiological substrates.

Empathy-Related Hypotheses

Not only did speaker participants show a physiological stress response, but observer participants also exhibited a significant sAA response to observing the speaker perform the eTSST. Thus, this study did not fully support H2.3 that the physiological stress of the speaker would resonate in the observer in the form of similar salivary cortisol, sAA, and heart rate changes. Additionally, this study did not directly replicate the findings from Buchanan and colleagues (2012) in which physiological resonance in observers of stressed individuals was first demonstrated. Buchanan et al. (2012) found that observer cortisol levels also increased during the eTSST, which was not found in the current study. However, there were several methodological differences between Buchanan and colleagues (2012) and the current study. For instance, the current study included an additional saliva sample compared to Buchanan and colleagues (2012). In addition, Buchanan et al. (2012) converted the physiological data to difference scores, while the current study converted physiological data to AUCi summary parameters. Also, their study utilized observers who were research assistants as opposed to participants, as they are in the current study. The research assistants were exposed to multiple participant speeches over the course of the study, which may have contributed to the greater cortisol reactivity of the observers in that study. Perhaps more chronic exposure to others who are undergoing social stress leads to greater HPA axis reactivity. Future research should test this possibility to determine how regular exposure to stressed individuals differs from infrequent exposure. While the physiological variables between speakers and observers were not significantly correlated with one another, the observers did exhibit a significant physiological stress response to observing the speaker perform the eTSST in the form of similar sAA responses to the speakers. Thus, observers showed physiological resonance in the form of increased SNS activity. PAM suggests that this similarity in SNS activity is the result of congruent neural activation between speakers and observers (Preston & de Waal, 2002). Perhaps similarity in neural activation causes observers to resonate the SNS activity of the speakers.

Greater observer trait empathy measures were related to greater physiological reactivity as well as greater self-reported personal distress, which supports H2.4 and extends the findings of Buchanan and colleagues (2012). The current study demonstrated that certain nonverbal behaviors of speaker participants during the eTSST was related to observer and speaker cortisol and sAA responses, suggesting nonverbal behaviors as a potential mechanism of physiological resonance. Specifically, speaker gaze aversion was the strongest predictor of participant cortisol response and for sAA, both speakers and observers. This finding corresponds with previous literature linking gaze aversion with social anxiety (Marks & Gelder, 1969; Moukheiber, et al., 2010; Ohman, 1986; Roelofs, et al., 2009; Schneier, et al., 2011; Weeks, et al., 2009), as well as findings that cortisol reactivity is related to submissive behaviors in primates (Sapolsky, 1990). The current study supports the premise that observers are inferring the stress response in the speakers through the speakers' nonverbal behaviors, particularly speaker gaze aversion.

The emotion literature has established that individuals can experience *vicarious shame*, in which they feel negatively self-conscious emotions for another individual (see Welten, Zeelenberg & Breugelmans, 2012). Recent research has found that vicarious shame can be the result of two distinct psychological processes. If an individual identifies with another as an ingroup member, they can feel vicarious shame for them due to feeling as if their own social identity is being threatened. However, individuals can also feel vicarious shame for others due to imagining themselves under the same threat, which is referred to as *empathic shame* (Welton, et al., 2012). In the current study, the speakers and observers' self-reported personal distress measures were significantly correlated, and observers' empathic concern was related to experienced personal distress for the speakers during the eTSST. These findings support the premise that observers may feel vicarious shame in the form of empathic shame for the speakers during the eTSST, and this emotional response is accompanied by a physiological stress response in the form of SNS reactivity.

One proposed evolutionarily adaptive benefit of exhibiting shame is appeasement after a social transgression, which promotes group cohesion (Dickerson, Gruenewald, & Kemeny, 2009). However, very few researchers have theorized as to why observing and identifying shame in others might also be adaptive. Martens, Tracy, and Shariff (2012) propose that displays of shame can be used by others to quickly determine who is trustworthy and cooperative. Identifying those who are more trustworthy and cooperative could provide an evolutionary advantage, allowing individuals to reap the most benefits from their social interactions. For instance, primate research has found that groups who cooperate with one another are more successful in hunting and gathering food, which promotes evolutionary fitness (Boesch, 2005). In addition, recognizing shame in others allows group members to infer the social hierarchy of a group, which facilitates coordinated group behaviors (Van Vugt, Hogan & Kaiser, 2008). Thus, the ability to identify shame in others may have evolved due to its adaptive benefits in social contexts.

Limitations

All participants were prescreened for the presence of existing psychological, neurological, or endocrinological conditions. Sex differences were also controlled for in the regression models, due to ample evidence indicating sex differences in HPA axis activity and stress reactivity in general (see Bourke, Harrell & Neigh, 2012 for review), as well as evidence that females are more vulnerable to experiencing social stress (see Troisi, 2001 for review). There are, however, a number of limitations of the current study that need to be taken into consideration. Having one speaker paired with one observer meant that each observer was exposed to a different speaker. Therefore, each observer was not exposed to the same level of stress or nonverbal behavior. Future research could develop standardized stimuli to expose observers to, such as a videotaped speech, so that the manipulation is consistent across observers.

Another limitation is that various individual differences known to affect HPA axis activity and affective responses to stress were not assessed in the current study. For example, individual differences in the use of emotion regulation strategies correlate with HPA reactivity (Lam, Dickerson, Zoccola & Zaldivar, 2009). Also, cognitive appraisals of one's own behavior influence the experience of shame and other self-conscious emotions (Tracy & Robins, 2006). Rumination is another cognitive process that is correlated with cortisol response to the TSST (Zoccola, Quas & Yim, 2012). The current study, however, did not control for these psychological differences in participants, and these factors may have affected their physiological and emotional response to the eTSST. Recent research has also found that an individual's level of physical activity can act as a protective agent against the negative effects of rumination on cortisol reactivity and recovery (Puterman, O'Donovan, Adler, Tomiyama, Kemeny, Wolfowitz & Epel, 2011), however the participants' regular levels of physical activity were not recorded in this study. In addition, due to the use of a convenience sample of mostly university students, the generalizability of these findings is also limited. The nonnormality of the data is another limitation that should be considered.

Future Directions & Conclusions

In the future, more experimental research regarding the relationship among shame, submissive behaviors, and HPA activation should be conducted. Due to the clear association between various physical and psychological diseases and HPA dysfunction, it is recommended that clinical populations also be researched, particularly in repeated-measures or even longitudinal studies, to determine if clinical samples respond to social stress similarly. An improved understanding of the unique psychological and biological differences that affect one's reaction to threats to the social self could eventually allow a more nuanced approach to treating many medical conditions, both physical and psychological.

The social self preservation model posits that threats to the social self result in a unique and coordinated psychobiological response that evolved due to its adaptive benefits in these contexts (Dickerson, Gruenewald, & Kemeny, 2004). The current study supported and expanded on this model by exposing individuals to an acute stressor, and finding that they exhibited HPA reactivity, SNS reactivity, shame-related emotions, and submissive nonverbal behaviors, particularly gaze aversion. In addition, the physiological and emotional reactions of an observing participant were assessed, and it was found that observing participants exhibited significant sAA responses, along with increases in shame-related emotions. High observer trait empathy levels were related to the self-reported increase in personal distress in both observers and speakers, suggesting an emotional empathic response on the part of the observer participants. This is the first study to examine the effects of social stress on both the speaker and observer participants of the eTSST, investigating the physiological and psychological effects of participants experiencing social stress and other participants observing them under stress.

References

Avitsur, R., Stark, J. L., & Sheridan, J. F. (2001). Social stress induces glucocorticoid resistance in subordinate animals. *Hormones and Behavior, 39*, 247-57.

Baesler, E. J., & Burgoon, J. K. (1987). Measurement and reliability of nonverbal behavior. *Journal of Nonverbal Behavior, 11*(4), 205-233.

Batson, C. D. (1997). Self-other merging and the empathy-altruism hypothesis: Reply to Neuberg et al. (1997). *Journal of Personality and Social Psychology, 73*(3), 517-522.

Behnke, R. R., Sawyer, C. R., & King, P. E. (1987). The communication of public speaking anxiety. *Communication Education, 36*(2), 138-141.

Boesch, C. (2005). Joint cooperative hunting among wild chimpanzees: Taking naturalistic observations seriously. *Behavioral and Brain Sciences, 28*, 692-693.

Boone, R. T., & Cunningham, J. G. (2001). Children's expression of emotional meaning in music through gesture. *Journal of Nonverbal Behavior, 25,* 21- 40.

Bourke, C. H., Harrell, C. S., & Neigh, G. N. (2012). Stress-induced sex differences: Adaptations mediated by the glucocorticoid receptor. *Hormones and Behavior, 62*, 210-218.

Buchanan, T. W., Bagley, S. L., Stansfield, R. B., & Preston, S. D. (2012). The empathic, physiological resonance of stress. *Social Neuroscience*, *7(2)*, 191-201.

Burgoon, J. K., Kelley, D. L., Newton, D. A., & Keeley-Dyreson, M. P. (1989). The nature of arousal and nonverbal indices. *Human Communication Research, 16*(2), 217-255.

Burgoon, J. K., Le Poire, B. A., Beutler, L. E., Bergan, J., & Engle, D. (1992). Nonverbal behaviors as indices of arousal: Extension to the psychotherapy context. *Journal of Nonverbal Behaviors, 16*(3), 159-178.

Burgoon, J. K., Beutler, L. E., Le Poire B. A., Engle, D., Bergan, J., Salvio, M., & Mohr, D. C. (1993). Nonverbal indices of arousal in group psychotherapy. *Psychotherapy, 30*(4), 635-645.

Burgoon, J. K., & Le Poire B. A. (1999). Nonverbal cues and interpersonal judgments: Participant and observer perceptions of intimacy, dominance, composure, and formality. *Communication Monographs, 66*, 105-125.

Cafazzo, S., Valsecchi, P., Bonanni, R., & Natoli, E. (2010). Dominance in relation to age, sex, and competitive contexts in a group of free-ranging domestic dogs. *Behavioral Ecology, 21*(3), 443-455.

Carney, D. R., Cuddy, A. J. C., & Yap, A. J. (2010). Power posing: Brief nonverbal displays affect neuroendocrine levels and risk tolerance. *Psychological Science, 21*(10), 1363-1368.

Castellano, G., Mortillaro, M., Camurri, A., Volpe, G., & Scherer, K. (2008). Automated analysis of body movement in emotionally expressive piano performances. *Music Perception: An Interdisciplinary Journal, 26*(2), 103-119.

Chrousis, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *The Journal of the American Medical Association, 267*(9), 1244-1252.

Clevenger, T., Jr. & King, T. T. (1961). A factor analysis of the visible symptoms of stage fright. *Speech Monographs, 28*, 296-298.

Cole, S. W., Kemeny, M. E., Fahey, J. L., Zack, J. A., & Naliboff, B. (2003). Psychological risk factors for HIV pathogenesis: Mediation by the autonomic nervous system. *Biological Psychiatry, 54*, 1444-1456.

Czoty, P. W., Gould, R. W., & Nader, M. A. (2008). Relationship between social rank and cortisol and testosterone concentrations in male Cynomolgus monkeys. *Journal of Neuroendocrinology, 21*, 68-76.

Dael, N., Mortillaro, M., & Scherer, K. R. (2012). Emotion expression in body action and posture. *Emotion, 12*(5), 1085-1101.

Dahl, S., & Friberg, A. (2007). Visual perception of expressiveness in musicians' body movements. *Music Perception, 24*(5), 433-454.

Daly, S. (1978). Behavioral correlates of social anxiety. *British Journal of Social and Clinical Psychology, 17*(2), 117-120.

Davis, M. H. (1983). Measuring individual differences in empathy: Evidence for a multidimensional approach. *Journal of Personality and Social Psychology, 44*(1), 113-126.

Darwin, C. (1872). *The expression of the emotions in man and animals*. New York: Oxford University Press.

de Waal, F. B. M., & Luttrell, L. M. (1985). The formal hierarchy of rhesus macaques: An investigation of the bared-teeth display. *American Journal of Primatology, 9*, 73-85.

Decety, J. & Jackson, P. L., (2006). A social-neuroscience perspective on empathy. *Current Directions in Psychological Science, 15*(2), 54-58.

Decety, J. & Meyer, M. (2008). From emotion resonance to empathic understanding: A social developmental neuroscience account. *Development and Psychopathology,* 20, 1053-1080.

Dettling, A. C., Gunnar, M. R., Donzella, B. (1999). Cortisol levels of young children in full-day childcare: relation with age and temperament. *Psychoneuroendocrinology, 24*, 519-536.

Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality, 72*(6), 1191-1216.

Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2009). Psychobiological responses to social self threat: Functional or detrimental? *Self and Identity, 8*, 270-285.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*(3), 355-391.

Dickerson, S. S., Mycek, P. J., & Zaldivar, F. (2009). Negative social evaluation, but not mere social presence, elicits cortisol responses to a laboratory stressor task. *Health Psychology, 27*(1), 116-121.

Eisenberg, N., & Fabes, R. A. (1990). Empathy: Conceptualization, measurement, and relation to prosocial behavior. *Motivation and Emotion, 14*, 131-149.

Ekman, P., & Friesen, W. V. (1972). Hand movements. *Journal of Communication, 22*, 353-374.

Ekman, P. & Friesen, W. V. (1974). Detecting deception from the body or face. *Journal of Personality and Social Psychology, 29*(3), 288-298.

Elzinga, B. M., Spinhoven, P., Berretty, E., de Jong, P., & Roelofs, K. (2010). The role of childhood abuse in HPA-axis reactivity in social anxiety disorder: A pilot study. *Biological Psychology, 83*, 1-6.

Fekedulegn, D. B., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., Hartley, T. A., Charles, L. E., & Miller, D. B. (2007). Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosomatic Medicine, 69*(7), 651-659.

Fessler, D. M. T. (2007). *The self-conscious emotions: Theory and research*. New York: Guilford.

Finn, A. N., Sawyer, C R., & Behnke, R. R. (2003) Audience-perceived anxiety patterns in public speaking. *Communication Quarterly, 51*(4), 470-481.

Frijda, N. H. (2007). Emotion experience and its varieties. *Emotion Review, 1*(3), 264-271.

Fuller, B, F., Horii, Y., & Conner, D. A. (1992). Validity and reliability of nonverbal voice measures as indicators of stressor-provoked anxiety. *Research in Nursing & Health,* 15, 379-389.

Furlan, P.M., DeMartinis, N., Schweizer, E., Rickels, K., Lucki, I. (2001). Abnormal salivary cortisol levels in social phobic patients in response to acute psychological but not physical stress. *Biological Psychiatry 50*, 254–259.

Gangestad, S. W., & Simpson, J. A. (2000). The evolution of human mating: Trade-offs and strategic pluralism. *Behavioral and Brain Sciences, 23*, 573-644.

Gilbert, P. (1989). *Human nature and suffering*. London: Erlbaum.

Gilbert, P. (2000). Varieties of submissive behavior as forms of social defense: Their evolution and role in depression. In L. Sloman & P. Gilbert (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy*. Mahwah, NJ: Lawrence Erlbaum Associates.

Gilbert, P., Pehl, J., & Allan, S. (1994). The phenomenology of shame and guilt: An empirical investigation. *British Journal of Medical Psychology, 67*, 23-36.

Giner-Sorolla, R., Castano, E., Espinosa, P., & Brown, R. (2008). Shame expressions reduce the recipient's insult from outgroup reparations. *Journal of Experimental Social Psychology, 44*, 519-526.

Granger, D. A., Kivlighan, K. T., el-Sheikh, M., Gordis, E. B., & Stroud, L. R. (2007). Salivary alpha-amylase in biobehavioral research: Recent developments and applications. *Annals of the New York Academy of Science, 1098*, 122-144.

Gruenewald, T. L., Kemeny, M. E., Aziz, N., & Fahey, J. L. (2004). Acute threat to the social self: Shame, social self-esteem, and cortisol activity. *Psychosomatic Medicine, 66*, 915-924.

Harris, T., Ferrucci, L., Tracy, R., Corti, M., Wacholder, S. & Ettinger, W. J., et al. (1999). Association of elevated interleukin-6 and C-reactive protein levels with mortality in the elderly. *American Journal of Medicine, 106*, 506-512.

Jurich, A. P. & Jurich J. A. (1974). Correlations among nonverbal expressions of anxiety. *Psychological Reports, 34*, 199-204.

Keltner, D. (1995). Signs of appeasement: Evidence for the distinct displays of embarrassment, amusement, and shame. *Journal of Personality and Social Psychology, 68*, 441-545.

Keltner, D. & Busswell, B. N. (1997). Embarrassment: Its distinct form and appeasement functions. *Psychological Bulletin, 122*, 250-270.

Kemeny, M. E. (2008). Psychobiological responses to social threat: Evolution of a psychological model psychoneuroimmunology. *Brain, Behavior, and Immunity,* 23, 1-9.

Kirschbaum, C, Pirke, K. M., & Hellhammer, D. H. (1993) The 'Trier Social Stress Test' A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76-81.

Kirschbaum, C., Wust, S., & Hellhammer, D.H. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine, 54,* 648-657.

Kleinke, C. L. (1986). Gaze and eye contact: A research review. *Psychological Bulletin, 100*(1), 78-100.

Kret, M. E. & De Gelder, B. (2012). A review on sex differences in processing emotional signals. *Neuropsychologia, 50*, 1211-1221.

Kudielka, B. M., & Kirschbaum, C. (2007). Biological bases of the stress response. In A. Mustafa (Eds.), *Stress and addiction: Biological and psychological mechanisms* (pp. 3-19). San Diego, CA: Elsevier Academic Press.

Lam, S., Dickerson, S. S., Zoccola, P. M., & Zaldivar, F. (2009). Emotion regulation and cortisol reactivity to a social-evaluative speech task. *Psychoneuroendocrinology, 34*, 1355-1362.

Leary, M. R. (2007). Motivational and emotional aspects of the self. *Annual Reviews of Psychology, 58*, 317-344.

Lerner, J. S., Dahl, R. E., Hariri, A. R., & Taylor, S. E. (2007). Facial expressions of emotion reveal neuroendocrine and cardiovascular stress responses. *Biological Psychiatry, 61*(2), 253-260.

Lewis, M., & Ramsay, D. (2002). Cortisol response to embarrassment and shame. *Child Development, 66*, 657-670.

Lovallo, W. R. (2005). *Stress and health: Biological and psychological interactions* (2nd ed.). Thousand Oaks, California: Sage Publications, Inc.

Lovallo, W. R. & Thomas, T. L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In J. T. Cacioppo & L. G. Tassinary (Eds.), *Handbook of psychophysiology* (2nd ed.) (pp. 342–367), New York, NY: Cambridge University Press.

Marks, I. M., & Gelder, M. G. (1969). Controlled trials in behavior therapy. *International Journal of Psychiatry in Clinical Practice, 6*(1), 68-86.

Martens, J. P., Tracy, J. L., & Shariff, A. F. (2012). Status signals: Adaptive benefits of displaying and observing the nonverbal expressions of pride and shame. *Cognition & Emotion, 26*(3), 390-406.

Maslow, A. H. (1936). The role of dominance in the social and sexual behavior of infrahuman primates. *The Journals of Gerontology, 49*, 161-198.

Matthews, K. A., Gump, B. B., & Owens, J. F. (2001). Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially in men. *Health Psychology, 20*, 403-410.

McEwen, B. S. (2004). Protection and damage from actue and chonric stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences, 1032,* 1-7.

Mills, R. S. L., Imm, G. P., Walling, B. R., & Weiler, H. A. (2008). Cortisol reactivity and regulation associated with shame responding in early childhood. *Developmental psychology, 44*(5), 1369-1380.

Moukheiber, A., Rautureau, G., Perez-Diaz, F., Soussignan, R., Dubal, S., Jouvent, R., & Pelissolo, A. (2010). Gaze avoidance in social phobia: Objective measure and correlates. *Behaviour Research and Therapy, 48*, 147-151.

Mulac, A., & Sherman, A. R. (1974). Behavioral assessment of speech anxiety. *Quarterly Journal of Speech,* 60, 48-58.

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*, 486-496.

Ohman, A. (1986). Face the beast and fear the face: Animal and social fears as prototypes for evolutionary analyses of emotion. *Psychophysiology, 23*(2), 123-145.

Preston, S. D. & de Waal, F. B. M. (2002). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences, 25*(1), 1-71.

Pruessner, J. C., Hellhammer, D. H., & Kirschbaum, C. (1999). Low self-esteem, induced failure and the adrenocortical stress response. *Personality and Individual Differences, 27*, 477-489.

Pruessner, J.C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D.H. (2003). Two formulas for the computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology, 28,* 916-931.

Puterman, E., O'Donovan, A., Adler, N. E., Tomiyama, J., Kemeny, M., Wolfowitz, O. M., & Epel, E. (2011). Physical activity moderates effects of stressor-induced rumination on cortisol reactivity. *Psychosomatic Medicine, 73*, 604-611.

Rettew, D. C. (2000). Avoidant personality disorder, generalized social phobia, and shyness: Putting the personality back into personality disorder. *Harvard Review of Psychiatry, 8*, 283-297.

Roelofs, K., van Peer, J., Berretty, E., de Jong, P., Spinhoven, P., & Elzinga, B. M. (2009). Hypothalamic-pituitary-adrenal axis hyperresponsiveness is associated with increased social avoidance behavior in social phobia. *Biological Psychiatry, 65*, 336-343.

Rohleder, N., Wolf, J. M., Maldonado, E. F., & Kirschbaum, C. (2006). The psychosocial stress-induced increase in salivary alpha-amylase is independent of saliva flow rate. *Psychophysiology, 43,* 645-652.

Roswell, T. E. (1974). The concept of social dominance. *Behavioral Biology, 11*, 131-154.

Rousson, V., Gasser, T., & Seifert, B. (2002). Assessing intrarater, interrater and test-retest reliability of continuous measurements. *Statistics in Medicine, 21*(22), 3431-3446.

Russell, J. J., Moskowitz, D. S., Zuroff, D. C., Bleau, P., Pinard, G. & Young, S. N. (2011). Anxiety, emotional security and the interpersonal behavior of individuals with social anxiety disorder. *Psychological Medicine, 41*(3), 545-554.

Sapolsky, R. M. (1990). Adrenocortical function, social rank, and personality among wild baboons. *Biological Psychiatry, 28*, 862-878.

Sassenrath, E. N. (1970). Increased adrenal responsiveness related to social stress in rhesus monkeys. *Hormones & Behavior, 1*, 283-298.

Schmidt, L. A., Fox, N. A., Sternberg, E. M., Gold, P. W., Smith, C. C., & Schulkin, J. (1999). Adrenocortical reactivity and social competence in seven year-olds. *Personality and Individual Differences, 26*, 977-985.

Schneier, F. R., Kent, J. M., & Hirsch, J. (2011). Neural circuitry of submissive behavior in social anxiety disorder: A preliminary study of response to direct eye gaze. *Psychiatry Research, 173*, 248-250.

Shively, C. A., Laber-Laird, K., Anton, R. F. (1997). Behavior and physiology of social stress and depression in female cynomolgus monkeys. *Biological Psychiatry, 758*, 871-882.

Shreve, E. G., Harrigan, J. A., Kues, J. R., & Kagas, D. K. (1989). Nonverbal expression of anxiety in physician-patient interactions. *Psychiatry, 51*, 378-384.

Sparks, G. G., & Greene, J. O. (1992). On the validity of nonverbal indicators as measures of physiological arousal. *Human Communication Research, 18*(3), 445-471.

Stein, M. B., & Stein, D. J. (2008). Social anxiety disorder. *Lancet, 371*, 1115-1125.

Sze, J. A., Gyurak, A., Goodkind, M. S., & Levenson, R. W. (2012). Greater emotional empathy and prosocial behavior in late life. *Emotion, 12*(5), 1129-1140.

Tabachnick, B. G. & Fidell, L. S. (2007). *Using multivariate statistics: Fifth edition.* Needham Heights, MA: Allyn & Bacon.

Tangney, J. P. (1995). Recent advances in the empirical study of shame and guilt. *American Behavioral Scientist, 38*, 1132-1145.

Tangney, J. P., Wagner, P., Gletcher, C. & Gramzow, R. (1992). Shamed into anger? The relation of shame and guilt to anger and self-reported aggression. *Journal of Personality and Social Psychology, 62*, 669-675.

Thayer, J. & Sternberg, E. (2006). Beyond heart rate variability: Vagal regulation of allostatic systems. *Annals of the New York Academy of Sciences, 1088*, 361-372.

Tracy, J. L., & Robins, R. W. (2004). Putting the self intro self-conscious emotions: A theoretical model. *Psychological Inquiry, 15*, 103-125.

Tracy, J. L., & Robins, R. W. (2006). Appraisal antecedents of shame and guilt: Support for a theoretical model. *Personality and Social Psychology Bulletin, 32*(10), 1339-1351.

Tracy, J. L., & Matsumoto, D. (2008). The spontaneous expression of pride and shame: Evidence for biologically innate nonverbal displays. *Proceedings of the National Academy of Sciences, 105*(33), 11655-11660.

Troisi, A. (1999). Ethological research in clinical psychiatry: The study of nonverbal behavior during interviews. *Neuroscience and Biobehavioral Reviews, 23*, 905-913.

Troisi, A. (2001). Gender differences in vulnerability to social stress: A Darwinian perspective. *Physiology & Behavior, 73*, 443-449.

Van Vugt, M., Hogan, R., & Kaiser, R. (2008). Leadership, followership, and evolution: Some lessons from the past. *American Psychologist, 63*, 182-196.

von Dawans, B., Fischbacher, U., Kirschbaum, C., Fehr, E., & Heinrichs, M. (2012). The social dimension of stress reactivity: Acute stress increases prosocial behavior in humans. *Psychological Science, 6*, 651-660.

Weeks, J. W., Rodebaugh, T. L., Heimberg, R. J., Norton, P. J., & Jakatdar, T. A. (2009). “To avoid evaluation, withdraw”: Fears of evaluation and depressive cognitions lead to social anxiety and submissive withdrawal. *Cognitive Therapy and Research, 33*, 375-389.

Weeks, J. W., Heimberg, R. G., & Heuer, R. (2011). Exploring the role of behavioral submissiveness is social anxiety. *Journal of Social and Clinical Psychology, 30*(3), 217-249.

Weiner, H. (1992). *Perturbing the organism: The biology of stressful experience*. Chicago: University of Chicago Press.

Weisbuch, M., Seery, M. D., Ambady, N., & Blascovich, J. (2009). On the correspondence between physiological and nonverbal responses: Nonverbal behavior accompanying challenge and threat. *Journal of Nonverbal Behavior, 33*, 141-148.

Welton, S. C. M., Zeelenberg, M., & Breugelmans, S. M. (2012). Vicarious shame. *Cognition and Emotion, 26*(5), 836-846.

White, C. N., McEnerney, K., Buchanan, T. W. (2012, April). *Nonverbal behavior as a mechanism of physiological resonance*. Poster presented at the Sigma Xi Symposium, Saint Louis, MO.

Zoccola, P. M., & Dickerson, S. S. (2012). Assessing the relationship between rumination and cortisol: A review. *Journal of Psychosomatic Research, 73*, 1-9.

Zoccola, P. M., Dickerson, S. S., Lam, S. (2012). Eliciting and maintaining ruminative thought: The role of social-evaluative threat. *Emotion, 12*(4), 673-677.

Appendix A. *Nonverbal Items Included in Battery of Nonverbal Behaviors Used to Code Speakers During Speech Portion of eTSST during Study 2.*

Nonverbal Items	Descriptions	References
Head Aversion Frequency	Frequency of turning head in any direction.	Boone & Cunningham, 2001; Castellano, Mortillaro, Camurri, Volpe, & Scherer, 2008; Dahl & Friberg, 2007; Troisi, 1999
Smiling Frequency & Duration	Frequency and duration of smiling, either with or without teeth.	Burgoon & Le Poire, 1999; Burgoon et al., 1989; Burgoon et al., 1992
Biting Lip Frequency	Frequency of biting upper or lower lips	Troisi, 1999
Licking Lips Frequency	Frequency of licking upper or lower lips	Troisi, 1999
Swallowing Frequency	Frequency of swallowing	Troisi, 1999
Brow Lowering Duration	Duration of lowering or furrowing brows	Weisbuch, Seery, Ambady, & Blascovich, 2009
Gaze Aversion Frequency & Duration	Frequency and duration of averting one's eyes in any direction; avoiding eye contact	Burgoon & Le Poire, 1999; Clevenger & King, 1961; Daly, 1978; Finn, et al., 2003; Jurich & Jurich, 1974; Mulac & Sherman, 1974; Troisi, 1999
Blinking Frequency	Frequency of blinking both eyes	Baesler & Burgoon, 1987; Weisbuch, Seery, Ambady, & Blascovich, 2009
General Body Movements Frequency & Duration	Frequency and duration of general body movements that included the whole body	Behnke, Sawyer, & King, 1987; Burgoon, et al., 1989; Burgoon, et al., 1992; Burgoon, et al., 1993; Clevenger & King, 1961; Finn et al., 2003; Mulac & Sherman, 1974; Sparks & Greene, 1992
Speechless Rigidity Frequency & Duration	Frequency and duration of exhibiting speechless rigidity	Finn et al., 2003; Mulac & Sherman, 1974; Sparks & Greene, 1992
Averted Trunk Frequency & Duration	Frequency and duration of averting whole trunk in any direction	Dael, Mortillaro, & Scherer, 2012
Leaning Posture Frequency & Duration	Frequency and duration of exhibiting a leaning posture in any direction, in which the spine remains straight	Dael, et al., 2011; Frijda, 2007
Slumped Posture Frequency & Duration	Frequency and duration of exhibiting a slumped posture in shoulders are slumped forward/inward	Carney, Cuddy, & Yap, 2010; Weeks, Heimberg, & Heuer, 2011
Self-Touch & Self-Adaptors Frequency & Duration	Frequency and duration of touching oneself, or adjusting oneself (ex. adjusting one's own clothing, touching one's own face, etc.)	Burgoon et al., 1989; Burgoon et al., 1992; Burgoon & Le Poire, 1999; Ekman & Friesen, 1972; Ekman & Friesen, 1974; Finn et al., 2003; Shreve et al., 1988; Sparks & Greene, 1992
Subjective Global Nonverbal Stress Estimate	Subjective rating of intensity of nonverbal stress	Baesler & Burgoon, 1987
Subjective Positive Arousal Estimate	Subjective rating of positive arousal or enthusiasm	Baesler & Burgoon, 1987

Appendix B. *Data Collection Form Used by Raters to Code the Nonverbal Behavior Related to Stress Exhibited by Speaker Participant in Study 2.*

Nonverbal Coding Sheet

Coder Name:______________________________________
Participant #:____________

Instructions

1) This time, please code even *very subtle* movements. So, for example, even if the participant's head barely shakes at all, please still code it as a shake. Throughout coding try to count even **subtle *hard-to-see* behaviors**.
2) There is no need to record the exact times of each instance of the behaviors anymore (ex. 2 min 3 sec - 2 min 5 sec). Only record the ***overall frequency*** within that minute and the ***overall duration*** for that minute.
    a. When asked to give the *frequency*, record the number of times something occurs within that minute
    b. When asked to give the *duration*, record the duration of the behavior in *seconds* within that minute
3) Do **NOT** listen to the sound when coding. This may make timing a little more difficult, but you can just time the beginning of the speech as when the participant **visibly begins talking**
4) Code each *section* at a time (ex. head, mouth, etc.)
5) Be sure to thoroughly read the **description of each item** so we are all coding the exact same way
6) Use the space to the right of each nonverbal item as scratch paper for tallying and note-taking
7) It is possible for a participant to simultaneously exhibit more than one type of behavior at a time
8) If the video cuts off a behavior, only code it if the participant has almost completed the motion or if the posture is clearly achieved before the video stops

Minute #0-1:

Minute 1:______________to_____________

Head

Averted Head Orientation: code when participant's head is facing any direction *OTHER* than straightforward; include even subtle turns of the head

1) **HEAD frequency**:___________________

Mouth

Smiling: code when participant smiles, either with or without displaying teeth; code subtle smiles as well

2) **SMILING frequency**:_________________
3) **SMILING duration**:_______________

Biting Lips: code when participant bites lip(s)

4) **BITING frequency**:_________________

Licking Lips: code when participant licks lip(s)

5) **LICKING frequency**:_________________

Swallowing: code when participant swallows

6) **SWALLOW frequency**:_______________

Eyebrows

Brow Lowering: code when participant clearly *lowers* or *furrows* their brow

7) **BROW duration**:__________________

Gaze Aversion

Gaze Aversion: code when the participant's gaze appears to be averted (*NOT* straightforward at the camera or experimenters); for *frequency*, each time the participant's gaze changes directions (*REGARDLESS* of whether they return to a straightforward position) should be counted as *separate instances* of gaze aversion; for *duration*, record the total number of *seconds* that the participant appears to *NOT* be looking at the experimenters or camera

8) **GAZE frequency**:____________________
9) **GAZE duration**:_____________________

Eyelids

Eye Blinking: tally and record the number of times the participant blinks

10) **BLINK frequency**:____________________

General Body Movements

General Body Movements: code when participant exhibits any movements of *at least their trunk if not their whole body*; examples include shifting positions, swaying, fidgeting, rocking, twisting, trembling, or erratic movements

11) **BODY frequency**:____________________
12) **BODY duration**:___________________

Speechless Rigidity: code when participant freezes, exhibiting complete motionlessness, a lack of gestures, and tension of facial and body muscles, as well as *no speech*

13) **RIGID frequency**:____________________
14) **RIGID duration**:___________________

Averted Trunk Orientation: code when the participant's trunk is twisted and facing either direction *OTHER* than straightforward

15) **TRUNK frequency**:___________________
16) **TRUNK duration**:_________________

Leaning Posture: code when participant's trunk, while still vertically erect (*with no discernible bend in the spine*), is clearly leaning forwards, backwards, or to either side

17) **LEAN frequency**:____________________
18) **LEAN duration**:___________________

Slumped Posture: code when participant exhibits a slumped, contractive, and closed posture in which the shoulders and spine appear *bent inward*, with a *bent spine*

19) **SLUMP frequency**:___________________
20) **SLUMP duration**:_________________

Self-Touch & Self-Adaptors

Self-Touch & Self-Adaptors: code when participant touches and/or adjusts themselves in some way, such as grooming behaviors, adjusting clothing, touching one's own face, scratching oneself, rubbing one's arm, etc.; code when participant is adjusting any leads or other physiological equipment

21) **SELF frequency**:______________________
22) **SELF duration:**________________________

Global Stress & Positive Arousal Estimates

23) **Global Stress Estimate**: please circle the number below to indicate the degree to which you think the participant was exhibiting *stress or anxiety*; 0 means they exhibited *no stress or anxiety at all*, 5 means they exhibited an *average or expected amount of stress*, and 9 means they exhibited *the most stress or anxiety possible*

1 2 3 4 5 6 7 8 9

24) **Global Positive Arousal Estimate**: please circle the number below to indicate the degree to which you think the participant was exhibiting positive arousal, which refers to *positive excitement or enthusiasm*; 0 means they exhibited *no positive excitement or enthusiasm* at all, 5 means they exhibited an *average or expected amount of positive excitement or enthusiasm*, and 9 means they exhibited *the most positive excitement or enthusiasm possible*

1 2 3 4 5 6 7 8 9

Additional Notes on Minute #1

Repeat for minutes 3 and 5

Vita Auctoris

Christina Noel White was born in Cleveland, Ohio on November 4th, 1985. In May of 2010, she graduated from Webster University in Saint Louis, Missouri with a Bachelor of Arts degree in Psychology. Following college, she gained a position at Saint Louis University as a research and teaching graduate assistant studying cognitive neuroscience. After completion of her Master's degree, Christina will begin working on her Ph.D. in experimental psychology with a concentration in cognitive neuroscience.