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# Cardiac Vagal Tone in Complex PTSD: A Polyvagal Perspective

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CARDIAC VAGAL TONE IN COMPLEX PTSD: A POLYVAGAL PERSPECTIVE

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## Abstract

Differences in functionality, impairment, and symptoms provide support for a nosological distinction between Complex PTSD and PTSD. Based on this information, the next step is to examine biological characteristics between the populations, thus bolstering the evidence supporting the uniqueness of these symptom expressions and associated etiologies. The proposed study is the first of its kind to do this, by examining theoretically derived potential differences between these populations. This investigation proposes the polyvagal theory as a comprehensive model through which one can examine psychological and physiological differences to demonstrate a systemic perspective of Complex PTSD. The primary distinguishing characteristic of Complex PTSD is the global symptom expression, disrupting multiple domains of functionality – relationships, emotions, and self-perception. From a polyvagal perspective, interpersonal problems and affect dysregulation are described by the same affective system – faulty neuroception, causing dysregulation in the hierarchical behavioral strategies. Physiologically, this dysregulation removes the neural inhibitory mechanism (cardiac vagal tone or vagal brake), while promoting one of the primal defensive strategies as autonomic regulator. Observations of the cardiopulmonary oscillator was expected to reveal differences between Complex PTSD, PTSD, and control samples. Hypotheses were confirmed, indicating that vagal brake does not reengage in the post-task resting period in the clinical groups, Complex PTSD and PTSD. Moreover, average RSA is lowest in the Complex PTSD group, as anticipated, given the severe affect dysregulation and social dysfunction evident in the symptomology.

# Dedication

I would like to dedicate this thesis to my past, for making me who I am now.

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First and foremost, I would like to acknowledge Dr. Caleb Lack for the hours of study design, implementation, and finally dissemination. He has been a pillar of hope in a barren wasteland of data and heart rhythms that is this thesis. Secondly, the Office of Research and Grants at UCO provided me the resources with which to implement the project via the RCSA grant offered to poor, young scientists. I also greatly appreciate the other members of my committee, and Stephanie, who spent countless hours providing feedback on a thesis manuscript of ungodly length. Thank you all. You are the essential building blocks to my empire of degrees, publications, and other masochistic endeavors.

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## Chapter 1: PTSD versus Complex PTSD

Natural disasters, domestic violence, sexual assault, combat – these unfortunate circumstances befall many at some point during their lifetime, and consistently for some. Estimates indicate that 89.7% of adults in the U.S. will have experienced a clinically significant traumatic event at some point in their life (Kilpatrick, Resnick, Milanak, Miller, Keys, & Friedman, 2013). Even though a large percentage of the population is likely to experience a traumatic event, only a relatively small percentage actually develops a mental disorder as result, with the most common being post-traumatic disorder (PTSD; Norris & Slone, 2013).Traumatic events can cover such a multitude of incidences and environments that a group of symptoms defining a single disorder may not be adequate to capture the complexity of all trauma psychopathology.

Individuals suffering with PTSD often experience functional difficulties and decreased quality of life. In fact, most PTSD diagnoses do not present alone; 80% of PTSD diagnoses are accompanied by at least one comorbid disorder, with depression being the most common (see Lack, 2013 for a review). Depressive symptoms associated with PTSD magnify the psychological, physical, social, and environmental quality of life deficits commonly associated with such cases (Araújo et al., 2014). Social relationships become difficult to maintain, as many alterations in behavior become apparent, interfering with intimate social interactions; hence, the increase divorce rate among this population. Suicidal ideation is also evidenced frequently.

Depression is not the only comorbidity evidenced alongside PTSD; 50% of PTSD diagnoses are accompanied by two or more comorbid disorders. Extensive investigation of PTSD – what it looks like, where it came from, and how to treat it – have revealed many nuanced complexities not represented in previous PTSD conceptualizations. More recently, research has

examined what's been called "Complex PTSD," which appears to have a unique set of symptoms and etiologies. This symptom set is ostensibly an extended version of PTSD, not a unique disorder (Cloitre et al., 2013). Early onset, prolonged trauma that is interpersonal in nature is commonly the trauma history described in Complex PTSD, which is characterized by self-organization dysregulation, such as interpersonal difficulties and lack of affect regulation (Cloitre et al., 2013; van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005).

Functional deficits associated with PTSD are quite impairing and influence a variety of aspects of one's life. Even so, the functional impairments in PTSD diagnoses associated with one-time incidents do not match the severity level observed in Complex PTSD. The longevity and developmental and relational aspects of Complex PTSD contribute to the dysregulation of self-organizing systems – behavioral, psychological, and physiological.

Maladaptive alterations in affect regulation, consciousness, somatization, self-perception, interpersonal relationships, and meaning systems perhaps summarize the impairment level. Individuals with Complex PTSD are more likely to become involved in risky behaviors (e.g., sexual promiscuity, substance use) and contemplate suicide frequently. More specifically, problems with affect regulation contribute to irrational responsiveness and angry outbursts; also, many self-destructive behaviors observed appear to be an attempt to dampen this emotional reactivity and irrationality. Social support and intimate relationships are scarce, unsurprising given the inability to trust and difficulties regulating emotion evidenced in those with Complex PTSD. Relationship strategies are organized based upon betrayal prevention, which is unconducive to healthy attachments. Physical health deficits are also pervasive in this population; specifically, digestive irregularity, chronic pain, cardiopulmonary complications, and conversion symptoms (see Herman, 1992 and van der Kolk et al., 2005 for reviews).

Differences in the functionality, impairment, and symptoms provide support for a nosological distinction between Complex PTSD and PTSD. Based on this information, the next step is to examine biological characteristics between the populations. This will bolster the evidence that currently supports the uniqueness of these symptom expressions and associated etiologies. The proposed study will be the first of its kind to do this, by examining theoretically derived potential differences between these populations. This investigation proposes the polyvagal theory as a comprehensive model through which one can examine psychological and physiological differences to demonstrate a systemic perspective on Complex PTSD.

The diagnosis of PTSD has a dense history, dating back to the First World War. Diagnostic criteria for PTSD in the DSM is noticeably colored with the social and political climates of each debut. Perhaps the temporary lull in high-deployment wars inspired research in populations other than combat veterans. Regardless, an increased research interest in this disorder illuminates the complexity and variability in post-trauma psychopathology. The distinctive symptom patterns and etiology associated with Complex PTSD have broad support (Cloitre et al., 2013; Courtois, 2004; Ford, Coutois, Steele, van der Hart, and Nijenhuis, 2005; van der Kolk et al., 2005) however, the impact of physiological regulatory systems on symptoms have not yet been examined in this population.

**History of the PTSD Diagnosis**

Prior to its official debut in the DSM-III, there were many terms used to describe what we now call PTSD, such as combat fatigue, shell-shock, battle stress, and gross-stress reaction (Andreasen, 2012). These combat-related terms reflect the era of war that encompassed the globe during their conception. First, World War I (WWI) prompted the term shell shock, which describes the heightened physiological arousal seen in combat vets during this time. Shell shock

was first thought to result from brain injury and neural damage from the artillery, similar to traumatic brain injury (TBI). Later, when uninjured soldiers began to present with similar symptoms, it became clinically recognized as an anxious reaction to the stress of combat (e.g., fatigue, neurotic, memory loss, dizziness). A dichotomy between the psychological and physical etiology developed, although both types of injury frequently co-occurred. This reflects the modern comorbidity and diagnostic similarities of TBI and PTSD (Jones, Fear, & Wessely, 2007). With the development of the DSM, the psychological narrative of trauma-induced symptoms gained ground, both clinically and research-wise (Andreasen, 2012).

In 1952, shortly after the Second World War (WWII), the first edition of the DSM emerged; this included the diagnoses of gross-stress reaction (Andreasen, 2012). Gross-stress reaction in the DSM-I was still prominently combat-focused, and included symptoms similar to current conceptualizations of trauma-induced stress (e.g., autonomic arousal, re-experiencing, sensitivity to trauma-related stimuli). Strangely enough, the diagnosis disappeared in the DSM-II, without any similar replacement. Its absence is speculated to be indicative of the momentary period of peace between WWII and the Vietnam War (Andreasen, 2010). Because of the combat-stress focus of gross-stress reaction and the intermediate peace, diagnoses probably decreased during this time.

By the time the third revision of the DSM was being designed, the psychological repercussions of the Vietnam War began to emerge. At this point, researchers had more clearly defined etiological bases and phenomenology of trauma-induced symptomology. The first conceptualization of PTSD appeared in the DSM-III and encompassed a variety of traumatic stressors in addition to combat, including (Nazi) death camps, industrial accidents, natural disasters, and mass catastrophes. Re-experiencing, avoidance, and increased arousal constituted

the main PTSD criteria. Traumatic event criterion specified that the experience must be so extreme that it would cause marked distress in almost anyone (3rd ed.; DSM-III; APA, 1980). This criterion is the primary differentiation between the DSM-III and current conceptualizations of PTSD.

The traumatic event criterion was broadened to include less severe traumas in a revision of the DSM-III, 7 years later (APA, 1980). DSM revisions continue to expand the event criterion. The most recent iterations of PTSD specify that simply witnessing a traumatic event happening to others or learning about a horrific event happening to loved ones is substantial enough to cause post-traumatic symptoms (4th ed., text rev.; DSM-IV-TR; APA, 2000). In the latest edition, the DSM-5 includes that the traumatic event can be the recurrent experience of trauma (e.g., police work, emergency room staff). Some express concern about the potential dilution of the PTSD diagnosis. However, assessment of the diagnosis reveals a valid condition with unique phenomenological symptoms that necessitate treatment (Institute of Medicine, 2006; 2007a; 2007b).

Recent conceptualizations of PTSD describe it as a psychological illness that develops following the experience or witness of life-threatening, sexually violating, or seriously injurious events. It is characterized by involuntarily re-experiencing the event(s), avoiding event-related situations, developing negative thought patterns and moods, and feeling excessively hyper-aroused (5th ed.; DSM-5; APA, 2013). Learning of such horrific events occurring to a loved one can also result in PTSD symptoms. In addition to these symptoms, some individuals experience dissociation from their environment, such as derealization of current surroundings or depersonalization. Some of the most common events associated with PTSD include sexual assault (experiencing and witnessing), accident or fire, violent death of a family member or

friend, and witnessing serious physical assault. In the general population (excluding members of the military) sexual assault is the most common traumatic event leading to PTSD (Kilpatrick et al., 2013). However, experiencing these types of trauma does not always lead to psychopathology; and PTSD, as it is currently defined, may not capture the variety of symptoms that accompany the unique experience of trauma.

**History of Complex PTSD**

Since the debut of PTSD in the third revision of the DSM post-Vietnam War, researchers and clinicians have gained a better understanding of the diversity in post-trauma characteristics beyond that of combat exposure. Examination of behavioral and physiological symptomology associated with specific traumatic histories reveal differences between long-term or recurring traumas and single-incident traumas (Herman, 1992; van der Hart, Nijenhuis, & Steele, 2005). Specifically, some researchers theorize that long-term, early-onset, interpersonal trauma is predictive of three distinctive symptoms clusters: somatization, dissociation, and affective changes; changes in personality, relationships, and identity; and vulnerability to repeated harmful behavior, self-mutilation, and re-victimization (Newman, Riggs, & Roth, 1997; Herman, 1992). Examples of such traumas can include domestic violence, child abuse, imprisonment, refugee status, and human trafficking (Courtios, 2004; Herman, 1992). Trauma beginning before the age of fourteen the largest risk factor for Complex PTSD (van der Kolk et al., 2005).

Abundant evidence argues that specific trauma types contribute to complex symptom outcomes (Cloitre, Miranda, Stoval-McClough, & Han, 2005; Herman, 1992); however, others suggest multiple traumas that accumulate over time are also predictors of symptom complexity (Briere, Kaltman, & Green, 2008; Cloitre et al., 2009). Herman (1992) describes a variety of traumatic experiences that could potentially result in Complex PTSD: imprisonment,

concentration/slave- labor camps, religious cults, battered women, and abused children. She implicates captivity and coercive control as the basis of complex trauma. However, childhood maltreatment is most frequently examined in terms of Complex PTSD (Cloitre et al., 2013; Cloitre et al., 2009; Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997; van der Kolk et al., 2005).

During the DSM-IV field trials, researchers proposed a new disorder, separate from PTSD, articulating these specific symptoms and trauma histories, called Disorders of Extreme Stress Not Otherwise Specified (DESNOS; Roth et al., 1997). Due to the frequent co-occurrence and overlap of PTSD with DESNOS, Complex PTSD became the common nomenclature to describe this unique presentation of symptoms. However, whether or not Complex PTSD constitutes a separate diagnostic category continues to be a controversial topic, as some argue that it is simply a more severe version of PTSD and not a separate disorder (Friedman, 2013). Again, some field trials preceding the most recent DSM edition proposed adding Complex PTSD as a unique disorder; however, it is still not included (Friedman, 2013; Pelcovitz et al., 1997). In a critical evaluation of Complex PTSD and its implications for the DSM-5, Resick and colleagues concluded that they could not support the addition but they did recognize the value of the recent acknowledgement of limitations in trauma literature (2012).

Although the DSM-5 did not include a separate disorder to distinguish Complex PTSD from traditional conceptualizations, the symptomology did change to incorporate more "complex" symptoms. Specifically, the three traditional features of PTSD were broadened to include "negative alterations in mood and cognitions" and a dissociative subtype (Friedman, 2013; APA, 2013). Broadening the definition to include additional symptoms caused concern because some complex symptoms are reflective of other mood disorders (e.g., anxiety and

depression; Brewin, Lanius, Novac, Schnyder, & Galea, 2009). However, recent research identifies the short-comings of the narrow definition in previous DSM versions (Cloitre, Garvert, Brewin, Bryant, & Maercker, 2013; Miller & Resick, 2007; Resick et al., 2012). In the DSM-5, experiencing "fear, helplessness, or horror" in response to trauma is no longer a required criterion for diagnosis, due the evidence suggesting such responses are not always present, particularly in on-going or long-term trauma (e.g., emergency responders, subjects of consistent sexual abuse; Friedman, Resick, Bryant, & Brewin, 2011). Another primary difference is the addition of dysphoric/anhedonic, externalizing, and dissociative symptoms. New criteria exemplify symptoms similar to Complex PTSD, including persistent distorted blame of self or others about the traumatic event(s), persistent negative emotional state, reckless of self-destructive behavior (APA, 2013; Friedman, 2013).

Efforts to empirically distinguish Complex PTSD from the current conceptualization of PTSD continue in field trials for the new edition of the International Classification of Diseases (ICD; Cloitre et al., 2013). Cloitre and colleagues posit that Complex PTSD consists of the three main traditional PTSD symptom clusters – re-experiencing, avoidance, and sense of threat – in addition to symptoms categorized under self-organization dysfunction (2013). The additional symptomology exemplify complex dysregulations of self-regulatory systems, such as affect dysregulation, negative self-concept, and interpersonal problems, reflective of the symptoms proposed by Herman (1992). For the ICD-11, Complex PTSD and PTSD are proposed to be "sibling disorders" in which the trauma(s), and resulting psychopathology, act as a gate, leading to one set of symptoms or the other (Cloitre, et al., 2013). While this clear cut distinction may be hard to find clinically, evidence does suggest that individuals who have experienced childhood abuse are twice as likely to develop Complex PTSD as opposed to PTSD only (Cloitre, et al.,

2013). Additionally, early-onset abuse predicts increased incidence of Borderline Personality Disorder (BPD) and Complex PTSD as opposed to PTSD only (McLean & Gallop, 2003). Complex PTSD and BPD share some etiology and symptomology (van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005).

Co-occurence of BPD and PTSD is frequent, particularly in cases involving childhood abuse and sexual assault (Clarke, Rizvi, & Resick, 2008; McLean & Gallop, 2003). For example, one study found that 83% of individuals with BPD reported traumatic histories involving physical and/or sexual abuse (Westphal et al., 2013). Additionally, evidence linking BPD to traumatic histories suggests that this personality disorder may often develop in response to long-term, interpersonal trauma (Westphal et al., 2013). In fact, some BPD symptomology resembles the "complex" symptoms that accompany PTSD diagnoses. The prominent characteristics of BPD are impairments in self-functioning, interpersonal functioning, negative affectivity, behavioral disinhibition, and antagonism (APA, 2013). National epidemiological data indicate that almost one-third (30.2%) of individuals with BPD also meet criteria for PTSD; and a quarter (24.2%) of PTSD cases are accompanied by BPD diagnoses. Moreover, BPD-PTSD comorbidity rates were higher (than individual diagnoses of BPD or PTSD) when repeated childhood traumas are involved; 65.9% of BPD-PTSD comorbid cases occurred in women (Pagura, Stein, Bolton, Cox, Grant, & Sareen, 2013).

Concerns have been raised as to whether Complex PTSD is actually a distinct disorder, due to its overlap with BPD (Resick et al., 2012). However, Cloitre and colleagues found distinct symptom-based subgroups of PTSD and Complex PTSD with and without comorbid BPD diagnoses (2013). The symptom profiles remained virtually the same in both cases. Specifically, trauma histories, sociodemographics, and symptom severity did not differ based on the presence

of BPD diagnoses. Individuals in the Complex PTSD subgroup highly endorsed affect dysregulation, negative self-concept, and dysfunctional relationship problems regardless of BPD diagnosis (Cloitre et al., 2013). Furthermore, in a group of sexually-abused women, all cases of childhood sexual abuse evidenced both BPD and Complex PTSD diagnoses, suggesting that a very specific subset of BPD belongs in a trauma-related classification – Complex PTSD (Mclean & Gallop, 2003).

**Epidemiology of Trauma-Related Psychopathology.** According to epidemiological studies conducted in the US, lifetime prevalence rates of PTSD are 6.6-8.3%; this estimate indicates the percentage of individuals who will develop PTSD at some point in their lifetime (Pagura et al., 2014). Risk for developing comorbid disorders is very high among PTSD cases; approximately 80% of individuals with PTSD also have one other disorder, and 50% have two or more. Major depression and substance abuse are the most commonly co-occurring issues. Substance abuse comorbidity with PTSD is higher among men than women – for alcohol (50% vs. 30%) and other drugs (34% vs. 27%). However, women are at much higher risk than men for developing PTSD; lifetime prevalence rates are almost three times as high for women (9.6 vs. 3.6%). Despite rates of PTSD diagnoses, men are actually more likely than women to experience a traumatic event(s) in their life (see Lack, 2013 for a review).

Although there is no epidemiological data on rates of Complex PTSD specifically, other data suggest the impact and severity of this trauma-related disorder. For example, the emotion dysregulation and interpersonal problems accompanying Complex PTSD cause significant functional impairments and poor quality of life (Cloitre et al., 2005). Additionally, functional impairments and poor quality of life are more impaired in cases of BPD-PTSD comorbidity than each disorder alone. Specifically, mood, anxiety, substance use, and other Axis I disorders were

significantly more common in comorbid cases than with individual diagnoses of either BPD or PTSD. Furthermore, these individuals were more likely to have poorer health, quality of life, and to have attempted suicide (Pagura et al., 2014).

Epidemiological studies reveal that nearly 90% of the US adult population has experienced at least one clinically significant trauma during their life. The most common traumas (meeting DSM-5 Criterion A) in descending order are physical or sexual assault (53.1), accident or fire (48.3%), violent death of a close loved-one (51.8%), disaster (50.5%), threat or injury to a close loved-one (32.4%), and witnessing physical or sexual assault (33.2%; Kilpatrick et al., 2013). Overlap in the percentages reflects the common experience of multiple traumas. It interesting to note that physical and sexual assault are the most frequently occurring traumas in addition to significantly increasing risk for complex symptoms. Specifically, childhood abuse is frequently the most reliable predictor of Complex PTSD symptoms (Cloitre et al., 2013; Harned, Korslund, Foa, & Linehan, 2012; McLean & Gallop, 2003).

National reports indicate that approximately one million children in the U.S. are confirmed victims of child maltreatment, and approximately 80% of the maltreatment cases are enacted by a parent or caregiver (see van der Kolk, 2005 for a review). The cost associated with child maltreatment is estimated to be one billion dollars per year; these costs are primarily from local, state, federal agencies and programs necessary for addressing these cases (see Cicchetti, 2013 for a review). In a national sample of adults, approximately 42% reported some type of interpersonal family trauma before the age of eighteen, 27% of which were described as sexual or physical abuse. Moreover, incidence of alcohol dependence is substantially higher in abused children than not (Fenton, Geier, Keyes, Skodol, Grant, & Hasin, 2013). When these children become adults, they are more likely to have an abusive relationship. Rates of intimate-partner

violence indicate that at it effects 1 out of 6 couples in the U.S. (see Finkel, in press, for a review).

**Impact of Interpersonal Trauma.** Much evidence strongly implicates early-onset, prolonged, and interpersonal trauma as a leading predictor of complex symptoms, as seen in child-maltreatment cases (Cloitre et al., 2013; Cloitre et al., 2009; Herman, 1992; van der Kolk, 2005; van der Kolk et al., 2005). In fact, early onset (vs. late onset) interpersonal abuse predicts increased lifetime prevalence of a Complex PTSD diagnosis. However, variety of traumatic experiences could potentially result in Complex PTSD. Interpersonal trauma describes physical, psychological, or emotional maltreatment that involves a perpetrator. This can include (but is not limited to) neglect, emotional manipulation, psychological control, and physical violence (Herman, 1992; van der Kolk, 2005).

Children raised in such tumultuous and unsafe environments suffer functionality deficits even into adulthood. Childhood maltreatment has been linked with increased suicide attempts, substance dependence, alcoholism, sexual promiscuity, and venereal diseases. Moreover, leading causes of death – heart disease, cancer, stroke, and diabetes – are also elevated in this population (Felitti et al., 1998 as reviewed in van der Kolk, 2005). Childhood sexual abuse, specifically, is linked with impairments in adulthood. In fact, it is a significant risk factor for sexual re-victimization (Classen, Palesh, & Aggarwal, 2005).

Long-term costs of childhood sexual abuse are products of emotional numbing and tension-reducing coping strategies, as evidenced in the high levels of self-destructive behaviors, such as substance use, suicide attempts, binge-eating, somatization, and sexual compulsivity, all of which are evidenced in Complex PTSD (see Polusny and Follette, 1995 for a review). These self-destructive behaviors and re-victimization in adulthood emphasizes the developmental

aspect of Complex PTSD, in that an unsafe environment and untrustworthy people set a precedent that remains consistent into adulthood.

## Chapter 2: Theoretical Perspectives on PTSD

Given that PTSD debuted around the cognitive revolution in the field of psychology, its etiology and development are often discussed in cognitive and behavioral terms. Developmental models are also implicated, but are primarily confined to descriptions of childhood trauma. Advancements in technology have provided a window into the physiological underpinnings of trauma that was not available prior. Each of these models contribute to understanding the etiology and course of PTSD. They also provide insights into the distinct aspects of Complex PTSD.

### Cognitive & Behavioral Theory

Traditional theories conceptualizing PTSD development include cognitive, behavioral, and developmental perspectives. Cognitive schemas prior to, surrounding, and after trauma encompass the basis of early cognitive conceptualizations of PTSD. Although many theorists disagreed on schema sets, they agreed on the premise that PTSD is a violation of pre-existing schemas causing many belief systems to be questioned. Violated schemas can be broadly categorized as self-worth, others' trustworthiness, and world-view (Epstein, 1991; Janoff-Bulman, 1992; McCann & Pearlman, 1990). Essentially, traumatic experiences impactful enough to challenge fundamental belief-sets will likely result in PTSD symptoms. Perceived threat and attributional assessments of trauma also contribute to PTSD symptoms.

Emotional processing theory focuses on the re-experiencing of traumatic events, suggesting that individuals typically habituate to unpleasant memories causing the associated emotional reactions to such memories to decrease; thus, when habituation does not occur, this

facilitates the development of PTSD or other psychopathologies (Foa, Skeketee, & Rothbaum, 1989). Additionally, avoidance of reminders, due to irrational fear and distorted beliefs about the event(s) contribute to PTSD symptomology. Specifically, vulnerability to developing chronic PTSD is based upon the following: original schemas about the self and world, memory of trauma and post-trauma experiences (see Elwood, Hahn, Olatunji, & Williams, 2009 for a review).

Improvements to the emotion processing theory are proposed through the behavioral-cognitive inhibition theory (BCIT), which describes behavioral and cognitive representation versus schematic-only representations of PTSD. This theory is hinged upon respondent-functional-appraisal memories. In PTSD, these memory types are dysfunctional and based upon faulty appraisals of trauma-centered memories that affect current appraisals, memories, and functionality (Paunovic, 2010). Essentially, current cognitive appraisals and behavioral responses are largely learned from earlier appraisals and responses of and around traumatic events, causing people to respond very dysfunctionally in their current environments. Therapeutically, according to BCIT, these learned dysfunctional-appraisal memories can be optimally inhibited through subsequent incompatible functional-appraisal-response contingencies, causing the new contingencies to "encode over" the dysfunctional ones.

Cognitive conceptualizations of dissociation in PTSD purport that symptoms emerge because of a lack of initial emotional processing. Thus, dissociation persists due to an attempt to suppress emotions associated with unaddressed memories. Individuals then attempt to distance themselves from those memories, sometimes resulting in depersonalization, emotional numbness, and derealization (Lanius et al, 2010). Accordingly, these coping mechanisms lead to PTSD symptomology. Research also suggests that peritraumatic dissociation – in which dissociation occurs during, or around the time of, traumatic events – can increase subsequent

psychopathology (Briere, Scott, & Weathers, 2005). Some suggest that dissociation during a traumatic event can increase the likelihood of subsequent development of dissociative symptoms (Van der Kolk & Van der Hart, 1989). These symptoms are often unrelated to trauma-specific triggers, suggesting pervasive disruptions of personal experiences (Breire et al., 2005).

More recent cognitive-processing and behavioral perspectives are more inclusive of the broad symptom base accompanying single event to long-term trauma. Violations of fundamental beliefs, as purported by early theorists, is more characteristic of single catastrophic events rather than long-term interpersonal trauma (Elwood et al., 2009).

**Developmental and Attachment Theories**

Ostensibly, developmentally-disruptive trauma could occur at any point in life, but children and adolescents are more malleable to its effects. The earlier the onset of trauma or victimization, the more detrimental subsequent functioning is, both during childhood and into adulthood. Developmental research has very specific relevance to Complex PTSD, as the disorder is considered to be a pathological response to developmentally-disruptive trauma. Developmental theorists would argue that a child's perception of the environment and people are molded by an untrustworthy, unsafe framework and so they learn to expect betrayal, victimization, and the absence of security. Behaviorally, these expectations often facilitate a lack of self-regulatory capacity, displayed as disorganized, anxious, angry and defiant behaviors (see van der Kolk, 2005 for a review). Dissociation with temporary behavioral and physiological withdrawal from the current environment is also a common response to this type of trauma (Liotti, 2004; van der Hart et al., 2005). This pattern of behavior among maltreated children is often discussed in terms of attachment theory (Hornor, 2009).

Accordingly, disorganized and insecure relationship attachment to a caregiver facilitates anxious and avoidant relational patterns. Caregiving behaviors that are inconsistent, neglectful, or violent can cause a child to be consistently hyper-vigilant and attentive as a self-preservation strategy (anxious attachment). Conversely, a child may become emotionally dismissing of her environment. Victims of child maltreatment often display both – disorganized attachment in addition to dissociation (Liotti, 2004). Institutionalized children (e.g., living in orphanages or group homes) display much higher rates of attachment insecurity than children living in non-institutionalized environments (Zeanah, Smyke, Koga & Carlson, 2005). However, in institutions where children had fewer and more consistent caregivers, attachment problems and behavioral disruptiveness are observed significantly less often. This evidence suggests that multiple caregivers provide inconsistent interactions, thus not allowing the child to form the bond necessary to facilitate trust and security (see Porges, 2003 for a review).

Attachment theorists argue that attachment patterns established during childhood provide a framework with which they interpret future relationships into adulthood. This perspective supports the trauma histories and symptoms commensurate with Complex PTSD, in that maladaptive patterns learned during young developmental years continue into adulthood (Allen, Coyne, & Huntoon, 1998; Pearlman & Courtois, 2005). As adults, similar patterns of behavior are displayed – lack of self-regulatory faculties, dissociative spells, and severe attachment insecurities (Liotti, 2004). In fact, child maltreatment victims are more likely to become perpetrators themselves, continuing the cycle (see van der Kolk, 2005 for a review).

**Physiological Perspectives**

***Neurological Correlates.*** Much recent research correlates specific brain areas and neural feedback loops with the symptomology PTSD. Brain areas implicated in emotion response and

regulation are commonly used for examining specific trauma-related psychopathology. Activity in the amygdala, which is a part of the limbic system, is widely implicated in fear responses and anxiety characteristic of PTSD (Garrett, Carrion, Kletter, Karchemskiy, Weems, & Reiss, 2012). Prefrontal areas of the brain, or the neocortex, are conceptualized as having regulatory influence over the basal areas of the brain, including the limbic system (see Frewen & Lanius, 2006 for a review).

For example, behavioral and physiological hyperactivity in PTSD is linked with decreased activation of the anterior cingulate cortex (ACC) and the prefrontal cortex (PFC; both medial and ventromedial areas). Ostensibly, when the higher-order regulatory brain areas are inhibited, the basal reactionary brain areas (amygdala) increase fear response via physiological arousal (see Etkin & Wager, 2007 and Frewen & Lanius, 2006 for reviews). This neural pattern is observed in conjunction with trauma-specific stimuli in PTSD populations.

In addition to the exploration of arousal systems in PTSD, researchers have begun to recognize the phenomenon of hypoarousal also occurring in response to trauma-specific stimuli. Hypoarousal is implicated in emotional numbing and dissociation, specifically in PTSD. During trauma-script driven imagery, PTSD patients displaying hypoarousal exhibited neurological and physiological "supersuppresion" in response, as indicated by neural and cardiac responses. Two brain areas associated with emotional regulation – the inferior frontal gyrus, the medial prefrontal cortex (mPFC), and the anterior cingulate cortex (ACC) – had increased activation. Decreased amygdala activity in the dorsal area is also implicated as an indicator of hypoactivity in PTSD (see Etkin & Wager, 2007 and Frewen & Lanius, 2006 for reviews).

***Hypothalamic-Pituitary-Adrenal Axis.*** Understanding aspects of the natural stress response is important for recognizing the pathological exemplifications of emotional regulation.

Internal and external stressors can activate the stress response, which is largely facilitated by endocrine system via the HPA axis. This stress response leads to physiological excitatory reactions. As such, these reactions lead to an increase in heart-rate, skin conductance, blood pressure, and blood-glucose levels (Elenkov, Wilder, Chrousos, & Vizi, 2000).

The hypothalamus, known as the master controller of the HPA axis, sends signals to the pituitary gland, and indirectly to the adrenal gland, when the body perceives stress. Essentially, the HPA axis is a neuro-endocrine communication system between brain and body. Specifically, when a stressor activates the periventricular nuclei (PVN) of the hypothalamus, corticotropin-releasing hormone is released, subsequently activating the pituitary gland, which then releases adrenal-corticotropin-releasing hormone (ACTH). ACTH activates the adrenal gland, which releases cortisol and epinephrine – adrenergic hormones also known as adrenaline. The pathway for the central control of the HPA axis differs slightly from the traditional pathway just outlined. The hypothalamus, via the PVN, activates the rostral ventrolateral reticular nuclei (RVRN), the excitation thereof releases glutamate. Glutamate activates the thoracic adrenal pathway that signals through the sympathetic chain and reaches the adrenal glands which secrete cortisol, epinephrine, DHEA, and norepinephrine. A noradrenergic neuron group, called the locus ceruleus, is also implicated as an HPA activator. The dorsal raphe, nuclei in the brain stem, is also an HPA activator and is the largest serotonergic region of the brain. More essential to the HPA, however, are the cortisol receptors located in the dorsal raphe. When cortisol is released from the adrenal glands, it binds to these cortical receptors in the RVRN (Elenkov et al., 2000). The pancreatic aspect of the HPA axis releases glucogen, which increases blood glucose levels, charging the body with energy for fighting or fleeing.

***Autonomic Arousal Systems.*** Much of the research in the realm of psychophysiology has focused on arousal systems (e.g., sympathetic nervous system) exclusively (Domschke, Stevens, Pfleiderer, & Gerlach, 2009; Kemp et al., 2009). Although the study of hyperarousal in PTSD has led to useful clinical insights (Orr & Roth, 2000), however, observing only one aspect of a system involving distinct visceral and behavioral components has limited utility (Porges, 2007).

Physiological correlates to PTSD symptomology are commonly studied by observing autonomic nervous system (ANS) functionality (Kemp, Felmingham, Falconer, Liddell, Bryant, & Williams, 2009). Communication between the ANS system and the body occurs neurally via the central nervous system, and is largely involuntary (Elenkov et al., 2000).

Autonomic functioning as a feature of PTSD has been examined via various indicators, such as heart activity, breathing patterns, and skin conductance. Specifically, attenuated respiration, decreased heart-rate variability (HRV), and increased skin conductance are indicative of sympathetic ANS activation. A healthy functioning ANS system regulates physiological arousal via an opponent-process method involving the sympathetic (excitatory) and parasympathetic (inhibitory) nervous systems. The sympathetic division arouses the body by increasing heart rate, attenuating respiration, and producing perspiration (Kreibig, 2010). Sympathetic activation is often called the "fight-or-flight" response. The parasympathetic division counterbalances the processes of the sympathetic division by depressing its excitatory functions and returning the body to baseline, or homeostasis. Diagnostic conceptions of PTSD focus heavily on sympathetic activation, or hyperarousal – a defining feature of the disorder (APA, 2000). Moreover, an abundance of PTSD literature focuses on sympathetic activation

alone (Fani et al., 2012; Orr & Roth, 2000; McTeague, Lang, Laplante, Cuthbert, Shumen, & Bradley, 2010).

Under current diagnostic criteria, a person must be experiencing hyperarousal that was not evident prior to the traumatic event (APA, 2013). Hyperarousal, as a symptom of PTSD, is characterized by difficulty falling or staying asleep, irritability or outbursts of anger, difficulty concentrating, hypervigilance, and an exaggerated startle response. Activation in the sympathetic division and a lack of regulation by the parasympathetic division over a long period of time can translate to autonomic dysregulation, which is commonly observed in PTSD populations (Norte et al., 2012; Tan, Dao, Farmer, Sutherland, & Gevirtz, 2010). For instance, one study indicated that in comparison to trauma-exposed groups (those who did not develop PTSD as a result of trauma), individuals with PTSD exhibit amplified heart-rates, attenuated respiration, and decreased HRV even at baseline. These indicators were especially apparent when individuals were exposed to trauma-specific stimuli. Furthermore, individuals with PTSD tend to remain physiologically aroused, and fail to return to baseline levels of physiological arousal (Norte, et al., 2012).

***Vagal Tone.*** Recent investigation of vagus-nerve influence on the ANS has inspired a significant amount of literature illuminating the significance of parasympathetic activity in psychophysiological systems (Dale, Carroll, Galen, Hayes, Webb, & Porges, 2009; Hauschildt, Peters, Moritz, & Jelinek, 2011; Park, Bavel, Vasey, & Thayer, 2012). The vagus nerve is the tenth of twelve cranial nerves originating in the brain or brain stem. The vagus nerve originates in the medulla oblongata of the brain stem, where various other autonomic and visceral components of the brain reside. From the medulla, the vagus nerve extends down through the pharyngeal and laryngeal branches, carotid sinus, lungs, heart, and some sub-thoracic renal and

gastric organs. The vagus nerve has a significant role in regulation of the ANS parasympathetic division (see Porges, 2011 for a review).

Inclusion of vagus influence and parasympathetic activation into the exploration of stress has led to a more comprehensive framework with which to understand trauma-related stress (Blechert, Michael, Grossman, Lajtman, & Wilhelm, 2007; Lewis, Furman, McCool, & Porges, 2012). The level of influence the vagus nerve exerts over the ANS is called vagal tone, and is a feature of parasympathetic influence. Increased vagal tone is associated with an increased capacity to deal with stress, emotionality, and attention (Bornstein & Suess, 2000; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996; Stifter & Fox 1990). Decreased vagal tone can lead to a decreased capacity to cope with stress and emotion. Vagal tone is a concept that originates from the polyvagal theory – a theory describing differentiated vagal systems within mammals that represent phylogenic adaptive reactions to challenge (Porges, 2011).

The regulatory mechanisms of the autonomic nervous system contribute to body's ability to protect itself as well as to maintain homeostasis. The polyvagal theory describes how and why autonomic processes correspond to affective experiences, thereby providing a visceral component of emotion. When these regulatory mechanisms become dysfunctional, abnormal affective and behavioral patterns parallel the dysfunction. Impairing difficulties with self-regulation are fundamental to trauma-related psychopathology. Cognitive and behavioral responses reflect the visceral dysregulatory patterns, as evidenced in trauma symptomology. The learning component that determines perceived threat and behavioral response to threat extends to the autonomic nervous system as well. This developmental component of trauma elucidates the global nature of cognitive, behavioral, and emotional experiences typical of Complex PTSD. The

polyvagal theory is unique in that it provides an outline into which other perspectives fit, offering a more comprehensive explanation for post-trauma experience.

**The Polyvagal Theory**

The polyvagal theory suggests that the orienting response is determined hierarchically based on three major adaptive stages in the vertebrate family tree now evident mammalian ANS development. These adaptive behavioral strategies, in chronological order, include immobilization, mobilization, and social engagement (Porges, 2001; Porges, 2007). The term polyvagal indicates that there are multiple vagus pathways, both motor and sensory. All three behavioral strategies are correspondent with a unique neural circuit, each representative of the environmental demands present during their respective developmental stages.

Thus, representing vagal tone as a unitary continuum from high to low is not congruent with differentiated vagal pathways and their origins (Porges, 1995a). Afferent vagal nerve fibers constitute up to 80% of the vagi (Agostoni, Chinnock, DeBurgh Daly, & Murray, 1957). The vagus nerve provides significant sensory information to the central nervous system. The theory focuses on understanding the bidirectional relationships between specific organs and their neural origins via vagal pathways, and how these relationships influence behavior. According to the polyvagal theory, the biological-behavioral relationship is best understood from a systemic perspective by exploring the feedback loops within motor output, sensory feedback, and central modulating mechanisms. The evolutionarily adaptive development of vagal pathways is also explored through this theory, providing insights into our phylogenic family tree and the nuanced autonomic responses unique to mammals.

Vagi innervate a variety of organs within the autonomic and immune systems (Berthoud & Neuhuber, 2000); however, in the current study, vagal influences on cardiac and pulmonary

functions are the focus. In mammals, there are two functionally distinguished vagal motor (efferent) systems – the right pathway originating in the nucleus ambiguous (NA), and the left in the dorsal motor nucleus of the vagus (DMNX); however, both pathways innervate the sinoatrial (SA) node of the heart, which is primary cardiac pacemaker. Degree of NA pathway influence on the cardiac pacemaker is what is most often referred to as vagal tone in the literature (e.g., Park, Bavel, Vasey, & Thayer, 2012; Smeets, 2010). It is theorized that the distinction between the DMNX and NA vagal pathways is evidence of phylogenic differences between reptilian and mammalian orienting response systems, representing an adaptive divergence between the phyla in which the NA pathway is the "social nerve," unique to mammals. (Porges, 2011).

The adaptive features of the ANS through the vagal system are also evident in the metabolic differences of mammals and reptiles. For example, stereotypic reptilian orienting response and predation is largely characterized by energy conservation, or a "sit and wait" strategy (Huey & Pianka, 1981; Porges, Riniolo, McBride, & Campbell, 2003); whereas, mammals have a defensive fight-flight response, are active foragers, and are highly adaptive to environmental changes (Else & Hulbert, 1985; Porges, 1995a). Reptilian and mammalian cardiac output differs quite significantly, as does the anatomy of the hearts. Mammals burn approximately 4 to 5 times more energy and 5 to ten times more oxygen than reptiles (Else & Hulbert, 1985; Bennett & Nagy, 1977). Oxygen consumption is the primary indicator of phylogenic differentiation, as mammals consume far more oxygen than reptiles (Else & Hulbert, 1985; Hubert & Dawson, 1974). Relative to body size, four-chambered mammalian hearts are larger than reptilian hearts (Else & Hulbert, 1985). The fight-flight response is correspondent with the sympathetic neural circuit, which does not receive efferent input from the vagus nerve, but is an essential part of mammalian autonomic response and developmental history. Within the

theoretical framework of polyvagal theory, it is theorized that the myelinated vagus pathway is a uniquely mammalian characteristic adapted to meet the evolving need for metabolic conservation and social interaction by mediating sympathetic activity via the NA pathway. This pathway is also known as the ventral vagal complex (Porges, 2011).

Influence from the ventral vagal pathway (right) produces Respiratory Sinus Arrhythmia (RSA) – a spontaneous breathing pattern primarily observed in mammals during a resting state (Porges et al., 2003). Cardiopulmonary oscillations between the bronchi and heart during a resting state produce a variable heart-rate pattern, such that inspiration increases heart rate and expiration subdues it. This pattern gives rise to the heart-rate variability captured with electrocardiogram assessments (Sack, Hopper, & Lamprecht, 2004).

RSA and heart period provide a prominent phylogenic distinction between the ventral and dorsal vagal complexes (Lewis et al., 2012; Porges, 1995a). In mammals, heart and respiratory patterns largely reflect each other's activity in an oscillatory pattern. However, when ventral vagal influence is not acting on the heart, the sympathetic nervous system and dorsal-vagal complex have more cardiac influence, resulting in a completely different autonomic response (Potter & McCloskey, 1986 as cited in Porges, 2007).

Dorsal-vagal influence (originating in the DMNX) on the heart facilitates an immobilized, freezing state characterized by "supersuppression" of the ANS, as observed by a decreased heart rate, slowed breathing, and lowered blood pressure. This autonomic reaction is characteristic of a reptilian orienting response to novel or threatening stimuli (Else & Hulbert, 1985). Prototypically, reptiles respond to challenge by freezing, both behaviorally and

physiologically (Huey & Pianka, 1981; Porges et al., 2003.[1] The reptilian vagal system does not include a myelinated ventral vagal pathway; only the unmyelinated pathway originating in the DMNX. The presence this unmyelinated pathway in mammals is theorized to be a vestigial feature of reptilian neuroanatomy that most vertebrates share (Porges, 2011; Porges, et al., 2003). Evidence from the polyvagal theory suggests that the development of the "new" ventral vagal complex in mammals is an adaption primarily to facilitate socialization (Porges, 2001; Porges, et al., 1996). This stance is well-supported by the evidence that consistent elevated sympathetic activity is not conducive to social interaction; for example, anxiety disorders are often characterized by elevated autonomic baselines and poor interpersonal relationships (see Lack, 2013 for a review).

Ventral vagal influence on the heart is a significant function of parasympathetic activity via cardiac innervation of the myelinated vagus pathway originating in the NA. As aforementioned, this type of influence is referred to as cardiac vagal tone (Park, et al. 2013). Cardiac vagal tone is characterized by a calm, variable heart rate and smooth, rhythmic breathing patterns – an ambient resting state. The presence of cardiac vagal tone is also called "vagal brake," illustrating its inhibitory influence on the heart and sympathetic activity (Porges, 1995a). Response to novelty or threat (orienting response) is accompanied by decreased vagal tone, and typically followed by sympathetic activation (Gorka et al., 2013). Although, it also can set the stage for DMNX vagal influence, causing a physiological and behavioral shut down. Because of

[1] Reptiles are heterogeneous in their foraging strategies, exemplifying both intensive and "sit and wait" foraging; however vagal regulation of the heart is still linked with the dorsal complex alone (Huey & Pianka, 1981; Porges et al., 2003).

its inhibitory control over sympathetic responses, vagal tone has a large role in autonomic regulation (Porges, 1992).[2]

**Neuroception and the Social Engagement System**

According to the polyvagal theory, risk evaluation and response strategy activation occurs before conscious recognition, mediated by the brain-stem circuitry – the "primal brain." This involuntary risk assessment is termed "neuroception," describing the executive neurophysiological role in perceiving the environment (Porges, 2011). Based on neuroceptive risk assessment, appropriate autonomic (and behavioral) responses are activated, beginning with the most recently developed response strategy – the social engagement system. This system is represented by ventral vagal influence, placing a "brake" on the cardiac pacemaker, facilitating an appropriate response to non-threatening stimuli (Gorka et al., 2013). When threat is perceived, this brake is released, facilitating sympathetic activation, or mobilization, the second and most common adaptive behavioral strategy. Immobilization, the most primitive response strategy, can also occur in response to perceived threat; this is primarily facilitated by dorsal vagal influence on various peripheral organs, but specifically the cardiac pacemaker. This typically results in neurogenic bradycardia (significant drop in heart rate), behavioral shut down, and occasional vaso-vagal syncope (sudden drop in heart rate, blood pressure, and brief loss of consciousness) (see Porges, 2007 for a review).

The adaptive behavioral strategies hierarchy in the ANS proposed by the polyvagal theory comes from the Jacksonian principle of dissolution, suggesting that phylogenetically

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[2] Vagal inhibition of sympathetic arousal may not be solely due to the vagus nerve, but rather synergistic interaction between sympathetic withdrawal and vagal brake; however, short latency decreases in heart rate indicate principle influence from the vagus. This reinforces the argument that the vagus is largely responsible for cardiac orienting responses (Porges, 1995b).

newer neural circuits have precedence over more primitive ones; this perspective argues that newer neural circuitry is activated more often (Jackson, 1886). It is important to note that the social engagement strategy is an inhibitory system, mediating the defensiveness of the two primitive strategies – mobilization and immobilization. Ostensibly, the primitive neural circuitry activates behaviors congruent with the environmental challenges occurring during their pinnacle of existence. The social engagement system attempts to facilitate behaviors necessary for mammalian survival – sociality. However, the primitive strategies remain adaptive for mammals during threatening situations. Dysregulation of this hierarchical order is presumed to have associated physiological and functional problems. Such autonomic dysregulation is argued to either be brought on by, or the cause of disease and illness (Jackson, 1886; Porges, 2007). Abundant evidence links autonomic dysfunction and maladaptive behaviors, both physiologically and functionally (Frewen & Lanius, 2006; Hauschildt, Peters, Moritz, & Jelinek, 2011; Sack et al., 2004).

**Cardiac Vagal Tone and Emotion Regulation**

Cardiac vagal tone and vagal brake, two indices proposed by the polyvagal theory, aptly illuminate autonomic mediators of human affect regulation and social interactions (Porges, 2001; Porges, Doussard-Roosevelt, & Maiti, 1994). For this reason, cardiac vagal tone and vagal brake are particularly useful for investigating psychopathologies involving affective and social regulatory dysfunctions, such as Complex PTSD. Affect dysregulation, negative self-concept, and interpersonal problems are prominent Complex PTSD features that differentiate it from PTSD (Cloitre et al., 2013).

Because of the large physiological component of emotion, the nervous system plays a significant role in affect regulation (see Amstadter, 2008 and Rottenberg & Gross, 2003 for

reviews). In fact, autonomic underpinnings of emotion are the basis of affective science (Rottenberg & Gross, 2003). Affect and emotion, although previously used interchangeably, are now differentiated in the literature. Affect is conceptualized as an overarching descriptor of all valenced experiences; emotion is a subtype thereof, consisting of experiential, behavioral, and physiological components, each describing an organism's appraisal of internal and external events (Amstadter, 2008; Lang, 1994). The psychophysiological approach to emotion provides significant and unique insight into the regulation of these systems. The experimental methodology with which to study cardiac vagal brake, as an inhibitory function of the vagus nerve, is the primary methodological contribution of the polyvagal theory, facilitating study of affect regulation and social behaviors. Degree of vagal brake on the heart can be observed non-invasively by mathematically mapping cardiopulmonary oscillations using electrocardiogram data; both RSA and heart period (HP) are observable with this data (Porges, 1985; Porges, 1986). Dependent upon age and health-status, specific frequency bands indicate a spontaneous breathing pattern (i.e. RSA). Cardiac vagal influence is apparent when RSA reliably predicts HP, demonstrating a cardiopulmonary oscillation and signifying that the vagal brake is activated (Porges, 1986).

Several recent studies applied this methodology to clinical populations struggling with psychopathologies characterized by affect dysregulation (Austin, Riniolo, & Porges, 2007; Dale et al., 2009; Sahar, Shalev, & Porges, 2001) and maladaptive social behaviors (Bal, Harden, Lamb, Vaughan van Hecke, Denver & Porges, 2009; Porges et al., 1996; Porges et al., 2013). For example, in response to emotionally charged stimuli, individuals with BPD displayed reliably different cardiopulmonary oscillations than controls during a resting period following emotionally-charged stimuli. Although the BPD and control groups had similar reactions to the

stimuli, the cardiopulmonary oscillation was no longer observable in the BPD group during the resting period, indicating a sustained absence of cardiac vagal brake. The control group returned to baseline (i.e., the vagal brake reactivated; Austin et al., 2007). Similarly, individuals with PTSD did not display indicators of vagal brake in a resting period following a challenging math task (Sahar, Shalev, & Porges, 2001).

With this methodology, evolutionary, biological, and psychological perspectives can be invoked to form a comprehensive framework with which to better understand the complexity of affect and its contribution to social behavior. The polyvagal theory offers such a framework, providing unique insight into affect regulation by incorporating a phylogenic history of nervous system development that includes sympathetic and parasympathetic functionality within the homeostatic regulatory system. Evolutionary perspectives of these adaptive purposes of nervous system developments facilitate a strongly empirical interpretation of observations, maximizing the utility thereof across scientific fields, and even clinically.

## Chapter 3: Self-Organizational Dysregulation in Complex PTSD: A Polyvagal Perspective

As with many psychological and physiological models, scientists work to understand adaptive systems to illuminate the maladaptive. Similarly, the polyvagal theory provides theoretical framework and experimental methodology with which to study typical autonomic functioning, thus illuminating pathological deficits. Specifically, this framework provides a structured, evidence-based description of the complexity that exemplifies specific trauma symptomology.

Interpersonal problems, affect dysregulation, and negative self-concept broadly describe the symptoms that extend traditional conceptions of PTSD to Complex PTSD (APA, 2000; Cloitre et al, 2013). More specific symptoms set are outlined in the literature for diagnostic

purposes, but each falls under the umbrella of self-organizational dysfunction (Cloitre et al., 2013; Herman, 1992; van der Kolk et al., 2005).

One distinguishing characteristic of Complex PTSD is global symptom expression, disrupting multiple domains of functionality – relationships, emotions, and self-perception (van der Kolk, Roth, Pelcovitz, Sunday, & Spinazzola, 2005). From a polyvagal perspective, interpersonal problems and negative self-concept are described by the same affective system – faulty neuroception, causing dysregulation in the hierarchical behavioral strategies. This dysregulation removes the neural inhibitory mechanism (cardiac vagal tone or vagal brake), promoting one of the primal defensive strategies as autonomic regulator – sympathetic (mobility) or immobility. Which primal strategy is employed and what individual variables regulate this is difficult to determine via cardiac vagal tone. Literature detailing physiological aspects of PTSD and affect dysregulation, including the polyvagal theory, suggests that the sympathetic neural circuit is most commonly activated, as it is the closest phylogenic strategy (Kreibig, 2010; Orr & Roth, 2000; Porges, 1995a). As of yet, there is limited (if any) literature on the physiological characteristics of Complex PTSD. However, given the physiological components of PTSD combined with affect dysregulation, there is likely a distinct autonomic regulatory pattern accompanying Complex PTSD.

Much evidence indicates that a tumultuous home life and lack of social support, leads to an abundance of maladaptive behaviors, such as dysfunctional relationships (see Cicchetti, 2013 for a review). Insecure attachment styles commonly develop, due to an untrustworthy or threatening environment, specifically caregiver interactions (Allen, Coyne, & Huntoon, 1998). A disorganized insecure attachment to a caregiver enables anxious and avoidant relational strategies. Caregiving behaviors that are inconsistent, neglectful, or violent can cause a child to

be consistently hyper-vigilant and attentive or approval-seeking. Also, children in these environments often become emotionally dismissing of their environment and other people. These attachment patterns often persist into adulthood (see Loitti, 2004 for a review). The polyvagal theory may attribute these relational patterns to developmentally disrupted neuroception caused by an untrustworthy environment – an adaptive reaction to an unsafe situation (Porges, 2011).

Maladaptive behaviors are inappropriate responses given the context that have negative consequences (see Rottenberg & Gross, 2003 for a review). Aspects of Complex PTSD provide examples of maladaptive and inappropriate responses to context. Conscious cognitive assessment of risk may not always congruent with neuroception, particularly when processing time differences are considered (Porges, 2011). Autonomic responses occur quickly and involuntarily, whereas conscious cognitive assessments are a bit more of a laborious task; thus, a polyvagal perspective argues that facilitation of behavior congruent with the initial autonomic response is likely, particularly when operating with a more primal strategy (Porges et al., 1996). For instance, some theories of emotion also suggest that cognitive awareness of emotion occurs subsequent to a physiological reaction (James, 1894; Lange, 1885); in an attempt to explain autonomic shifts, an emotion is assigned based on contextual cues (Schacter & Singer, 1962). Ostensibly, long-term exposure to an unsafe environment may foster a highly defensive response strategy, even in response to non-threatening stimuli. This proposition inspired the research questions examined here.

## Chapter 4: Current Study

This study examines the self-organization characteristics unique to Complex PTSD as proposed by Cloitre and colleagues (2013) and van der Kolk and colleagues (2005). Central to the current investigation are affect dysregulation and interpersonal problems. Although the

implications of the polyvagal theory can theoretically extend to all Complex PTSD symptomology, these specific symptom clusters can be more directly illuminated with polyvagal experimental methodology. This investigation is the first to examine physiological underpinnings of Complex PTSD, as there is no published literature on this topic. Moreover, these physiological indices have direct links to the etiology and developmental course of the disorder, in addition to corroborating the symptomology.

Observations of the cardiopulmonary oscillator will hypothetically reveal differences between Complex PTSD, PTSD, and control groups. This oscillator indicates the level of cardiac vagal tone from the brainstem NA (i.e., the vagal brake), which is expected to be very low or absent during laboratory-induced stress in all groups. However, during the recovery period following the stressful task, the control group's vagal brake should reengage in the post-stress resting period; whereas, vagal brake in both clinical groups is expected to remain unengaged even during the post-stress resting period. Furthermore, the Complex PTSD group should demonstrate lower vagal regulation than the PTSD group across all measurement phases.

## Method

### Participants and Procedure

Participants self-reported demographic, diagnostic, and screening measures via Qualtrics, an online survey system. The clinical sample was selected to participate in the laboratory protocol when they met or exceeded clinical cut-off scores on the PTSD-Checklist and the Complex PTSD screener (SIDES-SR). The control sample fell below clinical cut-off scores on all mental health assessments. Data from two control participants were not used, as they self-reported clinically significant trauma histories. Clinical and control samples were selected from the university subject pool via Sona-Systems, an online research participation recruitment site.

Students participated in research to earn credit toward their introductory psychology course and were provided a link to Qualtrics via Sona-Systems. The clinical sample was also recruited from local counseling clinics surrounding a Midwestern metropolitan university. Each clinic provided clients with a flyer providing basic information on how to participate in the screening portion of the study via Qualtrics.

Participant characteristics represent a virtually homogenous population, in terms of socio-demographics (see Table 1 more detail). Average age varied by group with the control group being the youngest ($M$ = 18.73) and the PTSD group the oldest ($M$ = 26.36). Over 87% of the clinical group participants were female, which was expected due to the much higher prevalence rates of PTSD in females overall (see Lack, 2013 for a review). All but two clinical participants (one Complex PTSD and one PTSD) were recruited from the university subject pool. This sample was somewhat ethnically diverse for a small sample with just over half (63%) being "White/Caucasian." As anticipated, the Complex PTSD group had higher severity scores on each PTSD assessment (detailed in Instrumentation). The clinical groups were more likely to being currently taking medication (40-55%) in comparison to the control group.

Table 1
*Socio-demographic and clinical characteristics for each group*

| | | Control ($n$ = 11) | C-PTSD ($n$ = 5) | PTSD ($n$ = 11) |
|---|---|---|---|---|
| **Age** | | *M(SD)*=18.73(.65)<br>Range: 18-20 | *M(SD)*=21.4(1.95)<br>Range=18-23 | *M(SD)*=26.36(12.1)<br>Range=18-55 |
| **Gender** | | | | |
| | Female | $n$ = 8 | $n$ = 5 | $n$ = 9 |
| | Male | $n$ = 3 | $n$ = 0 | $n$ = 2 |
| **Ethnicity** | | | | |
| White/Caucasian | | $n$ = 6 | $n$ = 3 | $n$ = 8 |
| African American | | $n$ = 1 | $n$ = 0 | $n$ = 1 |
| Asian | | $n$ = 0 | $n$ = 1 | $n$ = 0 |
| Pacific Islander | | $n$ = 0 | $n$ = 1 | $n$ = 0 |
| Native American | | $n$ = 1 | $n$ = 0 | $n$ = 0 |

| | | | |
|---|---|---|---|
| Hispanic/Latino | *n* = 0 | *n* = 0 | *n* = 0 |
| 2 or more ethnicities | *n* = 0 | *n* = 0 | *n* = 1 |
| **Education Level** | | | |
| High school | *n* = 2 | *n* = 1 | *n* = 2 |
| Some College | *n* = 6 | *n* = 4 | *n* = 2 |
| Two-Year Degree | *n* = 1 | *n* = 0 | *n* = 0 |
| Four-Year Degree | *n* = 0 | *n* = 0 | *n* = 1 |
| Some Graduate Work | *n* = 0 | *n* = 0 | *n* = 1 |
| Master's Degree | *n* = 0 | *n* = 0 | *n* = 0 |
| Doctorate | *n* = 0 | *n* = 0 | *n* = 1 |
| **Household Income** | | | |
| Less than $15k | *n* = 5 | *n* = 1 | *n* = 4 |
| $15-30k | *n* = 1 | *n* = 2 | *n* = 2 |
| More than $30k | *n* = 5 | *n* = 2 | *n* = 5 |
| **PTSD Checklist, DSM-IV-TR** | *M(SD)*=24.1(10.4) | *M(SD)*=58.4(10.2) | *M(SD)*=50.4(8.7) |
| Scale Range: 17-85 | Range: 17-52 | Range: 47-70 | Range: 42-70 |
| **PTSD Checklist, DSM-5** | *M(SD)*=4.9(6.5) | *M(SD)*=48.6(8.0) | *M(SD)*=36.7(13.8) |
| Scale Range: 0-80 | Range: 0-18 | Range: 37-56 | Range: 17-63 |
| **SIDES-SR** | *M(SD)*=12.67(11.72) | *M(SD)*=32.80(8.93) | *M(SD)*=10.22(6.02) |
| Scale Range: 0-135 | *(n* = 3) | *(n* = 5) | *(n* = 9) |
| **Psychotropic Meds** | | | |
| antidepressant | *n* = 0 | *n* = 2 | *n* = 5 |
| antianxiety | *n* = 0 | *n* = 0 | *n* = 2 |
| stimulants | *n* = 0 | *n* = 1 | *n* = 1 |
| sleepmeds | *n* = 0 | *n* = 1 | *n* = 3 |
| betablocker | *n* = 0 | *n* = 0 | *n* = 0 |
| antiseizure | *n* = 0 | *n* = 1 | *n* = 0 |
| Over-the-counter drowsy | *n* = 3 | *n* = 1 | *n* = 1 |
| Other Psychotropics | *n* = 0 | *n* = 1 | *n* = 1 |
| Smoking (< half pack a day) | *n* = 2 | *n* = 2 | *n* = 1 |
| Marijuana | *n* = 1 | *n* = 0 | *n* = 1 |
| Alcohol | *n* = 1 | *n* = 2 | *n* = 4 |

**Physiological Measures.** Once selected participants consented to participate, clinical and control groups went through the same laboratory protocol. Participants were hooked up to the

ECG amplifier (Biopac Systems, Inc., Santa Barbara, CA) using three Ag-AgCl electrodes with an 11mm diameter contact area attached below the clavicles and on the abdomen on the lower left rib. Participants were instructed to sit still and quiet while capturing resting baseline over five minutes. Subsequently, they completed a stress-inducing math task that lasted nine minutes and 15 seconds (PASAT; Gronwall, 1977) on a desktop computer in the lab. Post-stress resting activity was then assessed for five minutes.

Following ECG data acquisition, participants were unhooked from the Biopac and directed back to the computer where they completed the Trauma History Screen (Carlson, 2005). Lastly, participants were debriefed on the purpose and hypotheses of the study and thanked for their participation. The protocol took approximately an half an hour to complete. If the participant was still autonomically aroused at this point, the researcher would have conducted deep-breathing exercises with them before they left the lab. However, no participants required such intervention.

**Instrumentation**

**Demographic Information.** Participants began the screening assessment by answering questions regarding their biological sex, gender affiliation, race, age, socioeconomic status, and previous or current military affiliation. (If participants answered "yes" to military affiliation, they were asked to provide military branch, primary duties, and number of deployments). Questions regarding nicotine consumption, specific substances regularly used, and prescribed medications (being used currently) were addressed. All participants were 18 years old or older and spoke English.

**PTSD Assessments.** Given the recent change in DSM-5 PTSD criteria, assessments for both DSM-IV-TR and DSM-5 symptom sets were included.

***DSM-IV-TR PTSD Checklist.*** Participants completed the PTSD Checklist for Civilians (PCL-C), a self-report diagnostic screening measure assessing the level to which an individual meets DSM-IV-TR criteria for PTSD (Norris & Hamblen, 2003). Twenty items assess three primary symptoms clusters: Criterion B or re-experiencing the traumatic event (e.g., "Repeated, disturbing memories, thoughts, or images of a stressful experience from the past?"), Criterion C or avoidance of reminders and numbing of responsiveness (e.g., "Avoid activities or situations because they remind you of a stressful experience from the past?" and "Feeling emotionally numb or being unable to have loving feelings for those close to you?") and Criterion D or increased arousal (e.g., "Feeling jumpy or easily startled?"). Participants rate how much they have been bothered by each item during the last month on a 5-point Likert scale ranging from 1 (not at all) to 5 (extremely). To meet DSM-IV diagnostic criteria, an individual must score a three or higher on at least one Criterion B item, three Criterion C items, and two Criterion D items (APA, 2000). A total symptom severity score is obtained by summing all 17 items together. Cronbach's α coefficients, ranging from .73 to .85, indicate high internal consistency for the three symptom clusters (Weathers, Litz, Herman, Huska, & Keane, 1994). Scores range from 17 to 85, with a clinical cut-off score of 30 and above, in addition to meeting symptom pattern requirements outlined, to determine diagnostic significance.

***DSM-5 PTSD Checklist.*** This assessment is not differentiated between civilian and military populations, as with the DSM-IV PCL. The new checklist reflects the significant changes made to the diagnosis, except for those in Criterion A (Weathers, Litz, Keane, Palmieri, Marx, & Schnurr, 2013). Overall, wording has changed to reflect the possibility of multiple traumas. The primary change to Criterion B, intrusion symptoms, is the exclusion of the hallucinations and illusions as an intrusion symptom. Criterion C, avoidance symptoms, no

longer includes memory loss of the trauma, diminished interest in activities, feelings of detachment, restricted affect, and sense of foreshortened future. However, this symptom set was moved to Criterion D, which also includes symptoms involving negative alterations in cognitions or mood. This criterion is new to the DSM-5, but most of the symptoms come from DSM-IV Criterion C of except for negative beliefs and emotional state and blame. Criterion E, increased arousal symptoms, was previously Criterion D with the addition of reckless or self-destructive behavior. Verbal or physical have been added to specify the typical expression of irritable and angry outburst (APA, 2013). Finally, a dissociative subtype was added. Validity and reliability have not yet been formally examined in published literature. Participants rate how much they have been bothered by each item during the last month on a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely). To meet DSM-5 diagnostic criteria, an individual must score a two or higher on at least one Criterion B item, one Criterion C item, two Criterion D items, and two Criterion E items (APA, 2013). A total symptom severity score is obtained by summing all 20 items together. Scores range from 0 to 80, with a cut-off score of 38, in addition to meeting symptom pattern requirements, to determine diagnostic significance.

**Trauma History Screen.** The THS contains a list of traumatic events that fit Criterion A of the current PTSD diagnosis (APA, 2013; Carlson, 2005). Changes in Criterion A from DSM-IV include the broadening of qualifying traumas, such as sexual violence, vicarious traumatic experience, and repeated exposure to trauma. Vicarious trauma can be learning of or watching a loved one experience the threat of death, physical injury, or sexual violence. Because the trauma list has been broadened in the new DSM edition, this will be used to examine Criterion A with both PCL versions. This will be considered in the screening procedures. Participants will indicate whether or not (Yes or No) they have experienced any of the events. If they respond “Yes” on

any events, they will be asked to provide details about each event, including age of occurrence and a description of what happened in their own words. They will also respond to specific questions about the event, such as "When this happened, did anyone get hurt or killed?" and "After this happened, how long were you bothered by it?" In addition to its diagnostic utility, this measure also illuminates how types of trauma affect autonomic regulation and which types are associated with Complex PTSD versus PTSD.

**Complex PTSD Assessment.** The Structured Interview for Disorders of Extreme Stress-Self Report (SIDES-SR) was used to screen for symptoms most commonly expressed in Complex PTSD (Pelcovitz, et al., 1997). This 45-item measure corroborates the six symptom clusters proposed in the DSM-IV field trials: (1) disorders of affect regulation, (2) amnesia and dissociation, (3) somatization, (4) disruptions in self-perception, (5) disorders in relationships with others, and (6) disrupted systems of meaning (Pelcovitz et al., 1997; Roth et al., 1997; van der Kolk et al., 2005). Respondents answer "yes" or "no" to the 45 items; to every "yes" response, they also complete a rating scale indicating severity in the past month. This method allows for lifetime (yes/no) and past-month (rating scale) prevalence. The measure can be administered as a structured interview or self-report. The self-report method demonstrated consistency with the criterion of the structured interview (van der Kolk et al., 2005). The self-report method will be used in this study, as this measure will be a part of the online screener surveys. Internal consistency (full scale = .96; subscales = .76 to .90) and inter-rater reliability ($\kappa$ = .81) demonstrates dependability of the measure. However, criterion 6, somatization did not fit as tightly as the other 5 criteria ($\alpha$ = .68; Spinazolla & van der Kolk, nd). Participants were recruited for the Complex PTSD group if the demonstrated at least 5 out of the 6 criteria. Out of

the five Complex PTSD participants in this study, three did not score in the clinically significant range for somatization, corroborating the aforementioned findings.

**General Mental Health Assessment.** Participants completed the Behavioral Health Screening Measure (BHSM), a self-report measure designed to detect emotional problems in young adults (Zygowicz, & Saunders, 2003). This was used to screen out potential participants who have mental difficulties that would disqualify them from the healthy control group. The BHSM is a 22-item measure that detects a variety of DSM-IV defined symptoms, such a depression (e.g., "I feel unhappy, sad, or depressed"), anxiety (e.g., "I feel fearful, nervous, or anxious without knowing why"), and substance use problems (e.g., "I feel unhappy or guilty about my drinking or drug use"). Respondents indicate how much each item has distressed or bothered her or him in the past two weeks on a 5-point Likert scale (0 = None of the Time; 4 = All of the Time). Scores, ranging from 0-88, are added together to indicate the level of emotional problem. Scores of 16 or above indicate potential clinically significant problems, and was thus used as the clinical cut-off score. The BHSM has a very high internal consistency of .93 (Zygowicz & Saunders, 2003), and is sensitive to specific mental health difficulties.

**Generalized Anxiety Assessment.** The Penn State Worry Questionnaire (PSWQ) assesses the trait of worry as it relates to DSM-III-R criteria for Generalized Anxiety Disorder (GAD; Meyer, Miller, Metzger, & Borkovec, 1990). This measure was used to screen out potential participants who have mental difficulties that would disqualify them from the healthy control group. The PSWQ contains 16 items, with 11 items measuring characteristic (e.g., "I am always worrying about something") and five items measuring non-characteristic (e.g., "I do not tend to worry about things") traits. Participants respond with a 5-point Likert scale (1 = not typical at all of me; 5 = very typical of me). The five non-characteristic items are reverse scored

and the sum of scores indicate an individuals' level of worry, ranging from 16 to 80; score of 45 or above were considered clinically significant. Research indicates that the PSWQ measures the construct of worry as separate from other depressive and anxiety symptoms. Specifically, individuals meeting criteria for GAD have reliably higher PSWQ scores than individuals meeting criteria for PTSD. Additionally, internal consistency (.91) and test-retest reliability (.92) are shown to be very high for the PSWQ (Meyer et al., 1990).

**Depression Assessment.** Participants also completed the Zung Depression Scale (ZDS) – a 20-item scale assessing severity of depression (Zung, 1965). This measure was used to screen out potential participants who have mental difficulties that would disqualify them from the healthy control group. Affective, cognitive, behavioral, and physiological aspects of depression are represented in this scale. Participants rate the level to which each item was characteristic of them over the past week on a 4-point Likert scale (0 = none or a little of the time; 3 = most of the time). Depression severity is obtained when scores are summed. Scores between zero and 50 are normal, between 50 and 59 are minimal to mild, between 60 and 69 are moderate to severe, 70 or above is in the severe range; 65 was the clinical cut-off for this study. High split-half reliability of .73 and high internal consistency of .79 has been found (Knight, Waal-Manning, & Spears, 1983)

**Borderline Personality Disorder Assessment.** The abbreviated Borderline Symptom List (BSL-23) was used to briefly screen for BPD symptoms with 23 items (Bohus et al., 2008). This measure was derived from a 95-item BPD assessment (BSL-95), which aligns with BPD symptoms outlined in the DSM-IV-TR (2000). The BSL-95 demonstrates very high internal consistency between subscales (Cronbach's $\alpha$ = .97) and high test-retest reliability at one week ($r$ = .84; Bohus, Limberger, Frank, Chapman, Kuhler, & Stieglitz, 2007). The BSL-23 and BSL-95

sum scores are highly correlated (range of $r$ = 0.958-0.963). The BSL-23 internal consistency was also high (range of Cronbach's $\alpha$ = 0.94-0.96). Mean effect size for the BSL-23 in differentiating BPD patients from those with an axis-I diagnoses was 1.13, which was the clinical cut-off used in this study (Bohus et al., 2008).

**Paced Auditory and Serial Addition Task.** The PASAT was originally used to assess information processing rate in post-concussion patients as a neuropsychological measure (Gronwall, 1977). It has since been used to study its relation to intelligence, test cognitive dysfunction, and as a laboratory stressor. Recent versions of the PASAT are computerized, as is the one used in this study. Empirisoft experiment building software, MediaLab and DirectRT, were used in the creation and administration of the PASAT. The task consists of five trials in which single-digit numbers were presented consecutively. Participants must sum two consecutive numbers, while remembering the last number of the summation to add it to the next number. With each summation, the participant typed in their answer. Feedback responses were not given; numbers continued to be presented consecutively without intermission. After a practice trail with 10 consecutive digits presented at 3s each, the first of five trials began by presenting digits in 2.5s intervals, which were shortened by .5s each trial, making the digits on the fifth trial .5s each and totaling 9 minutes and 15 seconds (see Appendix A for detailed timing schemata used). These trials closely resemble those used as a laboratory stressor by Brindle, Ginty, and Conklin (2013). Correct answers and response times were calculated, as this was only a stressor.

## Data Reduction

Heart period and RSA were both derived from ECG data. Calculation and editing methods – outlined by Porges (1985) and Bohrer (1990) for mathematically deriving RSA

amplitude, expressed in the natural log of $ms^2$ – were applied using CardioEdit and CardioBatch MXedit software (Brain-Body Center, University of Illinois at Chicago).

First, inter-beat-intervals (IBIs), the time intervals between successive R-waves in milliseconds, from the ECG recordings were manually edited using the CardioEdit software. In CardioEdit, inter-beat-intervals (IBI) are displayed on a line graph mapping each interval between R-wave detections in milliseconds (y-axis) and measurement time (x-axis). Artifacts are determined individually and manually when there are significant deviations from the IBI pattern displayed. Integer arithmetic corrections are applied to such deviations by adding, dividing, and averaging IBIs that are missed or spuriously detected.

Heart period was derived from the IBIs in ms and averaged over 30-second epochs. Using CardioBatch software, RSA was derived from a high-frequency band of heart rate variability that reflects spontaneous breathing activity (0.12-.04 Hz for adults). The following calculation procedures are outlined by Heilman and colleagues (2013). Maturational differences in spontaneous breathing frequency are accounted for by using age-specific parameters for calculating RSA amplitude. For adult participants, the calculations of RSA are as follows:

1) timing sequential R-R intervals to the nearest millisecond
2) producing time-base data by resampling the sequential R-R intervals into 500 ms intervals
3) detrending the time-based series with a 21-point cubic moving polynomial (Porges & Bohrer, 1990) that is stepped through the data to create a smoothed template and the template is subtracted from the original time-based series to generate a detrended residual series

4) bandpass filtering the detrended time series to extract the variance in the heart period pattern associated with spontaneous breathing in adults (.12–.40 Hz)
5) transforming the variance estimates with a natural logarithm to normalize the distribution of RSA estimates (Riniolo & Porges, 1997).

**Statistical Analyses**

In order to test the four primary study hypothesis (as detailed again below), the following statistical procedures were employed. First, cardiac vagal influence was calculated by correlating the change scores (baseline to target point) for both heart period and RSA (i.e., when RSA-change and heart-period-change are significantly correlated, this indicates increased cardiac vagal regulation). The following four change scores were calculated: change from baseline to math-task stressor, change from baseline to post-task resting period for both RSA and HP (Austin et al., 2007).

Two within-participants multivariate analyses of variance were run to examine both RSA and HP differences in Complex PTSD, PTSD, and control groups across phases. The total ECG acquisition periods were used in analyses – 5 minute baseline, 9.25 minute math task, and 5 minute recovery period.

When cardiac vagal regulation is applied, RSA accounts for HP (and vice versa), demonstrating that HP and RSA are being influenced from the same origin; i.e., the vagus nerve. Correlations between RSA-change and HP-change, reveal differences in level of cardiac vagal regulation within Complex PTSD, PTSD, and control populations. An insignificant correlation would indicate that RSA cannot account for HP (or vice versa), implying that the heart is being influenced by something other than the ventral vagal pathway (e.g., sympathetic influence).

$H_1$ = Correlations between RSA-change and HP-change are expected to be insignificant post-task resting period in both clinical groups. Furthermore, the Complex PTSD group should demonstrate lower vagal regulation than the PTSD group across all measurement phases. During the post-task resting period, cardiac vagal tone was expected to reengage in the control group, indexed by a significant correlation between RSA and HP change scores.

Low RSA typically facilitates sympathetic activation, as it indicates release of the vagal brake, allowing sympathetic influences on the heart. HP can be an index of sympathetic activation; specifically, a decrease in heart period may indicate sympathetic activation (Austin et al., 2007). These effects are expected to be more pronounced in the Complex PTSD group, than in the PTSD and control groups; and more pronounced in the PTSD group, than in the control group.

$H_2$ = A main effect of group (Complex, PTSD, and control) across RSA and heart period measures was expected, such that downward trends in RSA and HP (shorter periods) would be most pronounced in the Complex PTSD group.

$H_3$ = We expected that a higher frequency of child maltreatment cases would be present in the Complex PTSD group than in the PTSD-only group. Because childhood maltreatment is most commonly related with Complex PTSD diagnoses, individuals such histories are expected to more frequently meet criteria for Complex PTSD as opposed to PTSD alone (Cloitre et al., 2013; van der Kolk et al., 2005).

Due to the recent changes to PTSD diagnostic criteria, the DSM-5 symptom assessment should more closely resemble the Complex PTSD assessment outcomes. These criterion changes reflect some complex symptoms (Friedman et al., 2011). This hypothesis was purely exploratory.

$H_4$ = In the whole sample, including all participants who completed screening measures (N=243), frequency of individuals meeting criteria for Complex PTSD and DSM-5 PTSD was expected to be higher than those meeting criteria for Complex PTSD and DSM-IV-TR PTSD.

## Results

### Cardiac Assessments

Data revealed expected results in that vagal brake, as determined by correlations between change scores, was removed during the math-task stressor in the control ($r$ = .46, *ns*), Complex PTSD ($r$ = -.29, *ns*), and PTSD ($r$ = .54, *ns*) groups. The control participants, but neither clinical group, then demonstrated vagal brake reengagement ($r$ = .64, $p$ = .03) in the post-task resting period (see Appendix B for change-score scatter plots). Although the correlation between RSA and HP in the post-task resting period was not higher in the PTSD group than the Complex PTSD group, these relationships should be cautiously interpreted because neither correlation was statistically significant. Visually examining RSA and HP at the post phase, provides more reliable information. Specifically, both RSA and HP are lower in the Complex PTSD group during the post phase, as anticipated (see Table 2).

Moreover, group comparisons using MANOVA demonstrated a main effect of group RSA across all measurement phases (baseline, math-task stressor, and resting period; see Figure 1), $F(2, 24) = 2.217$, $p$ = .011, $\eta^2_{partial}$ = .38, obs. power = .836, such that average RSA is lowest in the Complex PTSD group ($M$ = 5.23, $SD$ = 1.49), followed by the PTSD ($M$ = 5.68, $SD$ = 1.39) group, and the control group had the highest across phases ($M$ = 6.25, $SD$ = 0.92). Although mean RSA between groups was in the order expected, with Complex PTSD being the lowest, univariate post hoc comparisons did not reveal significant differences between groups across phases. However, baseline RSA differences by group were close to statistical significance,

*F* (2, 24) = 3.164, *p* = 0.06). Correlations between dependent variables (baseline, stressor, post) ranged from .76 to .90, implicating multicollinearity as a potential confound. However, according to Brace and colleagues (2010), correlation coefficients between dependent variables that fall within .30 and .09 are acceptable when interpreting a MANOVA.

Field (2010) recommends using planned comparisons for further examine main effects by comparing each clinical group to the control group. Accordingly, simple group contrasts with the control as the reference category (recommended by Field, 2010, p. 607) indicate that the Complex PTSD and control groups (*p* = .040) as well as the PTSD and control groups (*p* = .055) reliably differ during the baseline phase, but not in the stressor or post phases.

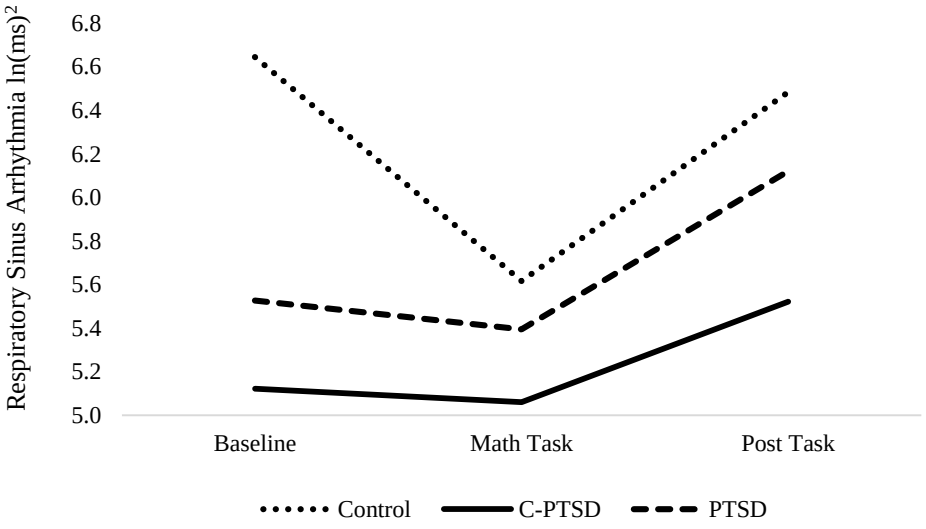


*Figure 1.* RSA distributions, natural logarithm of $ms^2$, by group across baseline, math task, and post task phases.

Trajectories of RSA and HP across phases slightly differ from expectation, in that post-task resting measures are higher than baseline in both clinical groups, but not the control group (Figures 1 and 2). Based on previous research, we anticipated that baseline measures would have

the highest indications of parasympathetic activity through vagal brake. There was no main effect of group in HP comparisons across phases, so HP means and trajectories are interpreted with discretion.

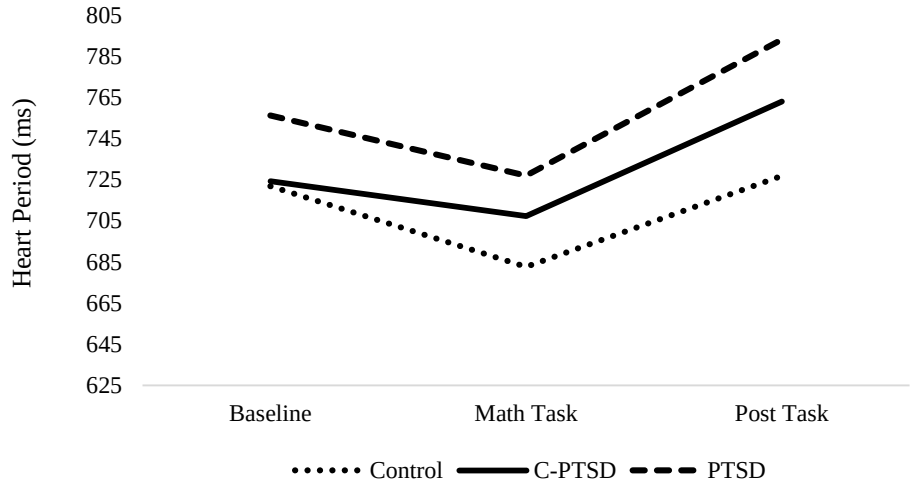


*Figure 2.* HP distributions in ms by group across baseline, math task, and post task phases.

Contrary to expectation, however, HP is lowest in the control group across all phases, indicating shorter IBIs (ms between R-waves). Sometimes, this can be an indication of greater sympathetic arousal, when outside of a normal range (700-900ms for adults). Control HPs were only slightly below this range during the math-task phase (see Table 2). Furthermore, RSA in the control group was consistently higher than the clinical groups, demonstrating increased heart-rate variability, which is a closer approximation of vagal tone than HP alone (Denver et al., 2007).

Table 2
Descriptive statistics of physiological variables for each group

| | RSA (SD) | HP (SD) | HR (SD) |
|---|---|---|---|
| Initial baseline | | | |
| Control | 6.65(0.99) | 721.89(65.24) | 84.30(7.99) |
| C-PTSD | 5.12(1.65) | 724.24(99.69) | 84.48(12.33) |
| PTSD | 5.53(1.40) | 756.18(106.06) | 80.92(10.32) |
| Math-task stressor | | | |
| Control | 5.62(0.78) | 682.61(80.02) | 89.72(11.61) |
| C-PTSD | 5.06(1.69) | 707.33(86.47) | 81.46(14.33) |
| PTSD | 5.39(1.36) | 726.97(85.47) | 83.83(9.02) |
| Post-stressor | | | |
| Control | 6.48(0.98) | 726.79(69.31) | 83.77(7.97) |
| C-PTSD | 5.52(1.13) | 762.92(117.30) | 80.53(12.24) |
| PTSD | 6.12(1.40) | 793.09(104.97) | 77.31(9.57) |

**Screening Assessments**

**Trauma History.** We predicted that a higher incidence of childhood maltreatment would be evident in the Complex PTSD group, as this symptomology typically accompanies such traumatic history (van der Kolk et al., 2005). Contrary to expectations, the PTSD group (45.5%) had a slightly higher rate of child maltreatment histories (childhood sexual and physical abuse) than the Complex PTSD group (40%; Table 3). Although, percentage of childhood physical abuse in the Complex PTSD (20%) group marginally exceeds that of the PTSD group (18.2%). This may be attributable to the disparity in group sample sizes. Although all control participants endorsed specific trauma histories, none of these were clinically significant, based on psychological distress level, duration of distress, and response to the event (APA, 2000). As detailed in the literature review, trauma exposure is very common, and so this level of exposure is expected.

Table 3

*Percentages of specific trauma histories*

| | C-PTSD | Control | PTSD |
|---|---|---|---|
| | (*n*= 5) | (*n*= 11) | (*n*= 11) |
| Vehicle Accident | 80.00% | 100.00% | 100.00% |
| Work/Home Accident | 0.00% | 27.30% | 27.30% |
| Natural Disaster | 40.00% | 54.50% | 36.40% |
| Childhood Physical Abuse | 20.00% | 18.20% | 18.20% |
| Adulthood Physical Abuse | 20.00% | 9.10% | 9.10% |
| Childhood Sexual Abuse | 20.00% | 9.10% | 27.30% |
| Adulthood Sexual Abuse | 0.00% | 0.00% | 18.20% |
| Attacked with Weapon | 20.00% | 0.00% | 27.30% |
| Military | 0.00% | 0.00% | 18.20% |
| Sudden Death | 20.00% | 81.80% | 54.50% |
| Witnessing Death/Injury | 0.00% | 36.40% | 36.40% |
| Scared/Helpless/Horrified | 60.00% | 9.10% | 63.60% |
| Sudden Move/Loss | 40.00% | 27.30% | 45.50% |
| Sudden Abandonment | 0.00% | 0.00% | 27.30% |

**Criteria Comparisons**. To investigate hypothesis 4, the entire dataset, collected via screener assessments (*N*=243), was used. In the Complex PTSD group, participants were just as likely to meet criteria on the DSM-IV-TR PCL-C as the DSM-5 PCL (Figure 3), contrary to hypotheses that Complex PTSD and DSM-5 PTSD symptomology would more often overlap.

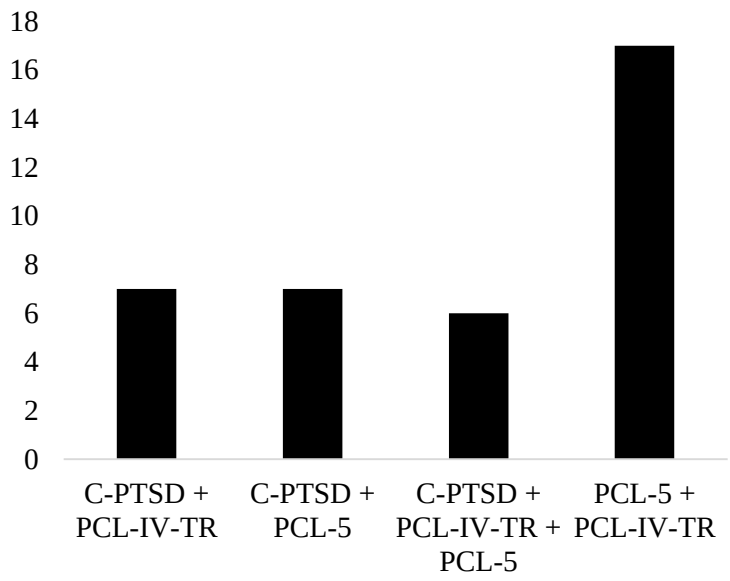


*Figure 3.* Overlapping of diagnostic clinical significance.

Correlational analyses ($n$=225; 22 participants did not endorse any Complex PTSD symptoms) indicate that severity scores on the SIDES-SR (the C-PTSD assessment) positively correlate with DSM-5 PTSD severity scores ($r$=.651, $p$<.001) slightly more than DSM-IV-TR PTSD severity scores ($r$=.650, $p$<.001). Moreover, regression analyses indicate that DSM-5 PTSD severity only predicts an additional 3.1% of variance in Complex PTSD ($\beta$=.368, $p$<.001) compared to severity than DSM-IV-TR PTSD severity ($\beta$=.337, $p$<.001).

## Discussion

These findings confirm the proposed relationship between autonomic and affective/social components of Complex PTSD. Austin and colleagues previously confirmed that emotion dysregulation and social dysfunction are corroborated by cardiac vagal regulation (2007). This study exemplifies that these symptoms provide not only nosological distinctions in trauma-related psychopathologies, but also neurophysiological ones. In each phase, RSA and heart period are lower in the Complex PTSD group, signifying less parasympathetic control over

respiration and heart activity. More importantly, absence of cardiac vagal regulation post-stressor task accompanied by lower RSA in Complex PTSD during this period strongly suggests that this disorder is characterized by extreme affect dysregulation that is not being captured in the current conceptualization of PTSD.

Unexpectedly, the greatest differences in RSA were observed during the baseline period, but groups did not differ reliably on the stressor or post measures. Research examining PTSD and RSA are inconsistent in terms of baseline difference. Blechert and colleagues, for example, had similar findings, such that RSA differed significantly between PTSD and control groups during baseline, but not during their stressor phase (2007). Cohen and colleagues also observed heart-rate variability differences between PTSD and control groups during baseline. However, they observed differences during the stressor, contrary to current findings. Sahar et al (PTSD; 2001) and Austin et al (BPD; 2007) did not observe differences in RSA during baseline or stressor phases. Austin et al (2007) did find differences in the post-resting phase, while Sahar et al (2001) did not. Current findings may indicate that Complex PTSD is characterized by a consistent autonomic state (i.e., absence of vagal regulation). Accordingly, repeated-measures comparisons with the Complex PTSD group reveal that RSA did not reliably differ by phase. Similar comparisons with the PTSD group indicate that baseline RSA does not reliably differ from stessor RSA, but there are differences from stressor to post measures, demonstrating an increase in RSA during the post-stressor resting period. RSA changes in the control group differ reliably across the phases, modeling the anticipated trajectory (refer to Figure 1).

A reductionist view of physiology singularly describing Complex PTSD is not being purported, despite the comprehensiveness of the polyvagal theory providing vast explanatory utility. Instead, the essence of the theory is interdisciplinary and multi-level, marrying the

explanations current within psychology to relevant biology and evolutionary history. This is, of course, highly reminiscent of the biopsychosocial model used in describing the etiology of many mental health problems. The implications of this study's results are not to solely point out physiological differentiation between Complex PTSD and current conceptions, so that yet more disorders can be added to the next DSM. Rather than providing experimental fodder for further distinction and classification, these preliminary results and future, related investigations should instead add to our understanding of both the similarities and differences between trauma-related psychopathologies and their experiential as well as physiological etiologies. This in turn should help guide novel treatment development that reflects a more dimensional rather than categorical understanding of trauma response.

Lower heart period in the control group than clinical groups may be explained by extraneous variables, such as current medications of both clinical groups. For example, 40% of participants in the Complex PTSD group were currently taking antidepressants, antiseizure medication, sleeping medication and/or over-the-counter drowsy medication, all of which can have a profound effect on heart rhythm (Julien, 2007). In the PTSD group, 55% of participants were currently taking antidepressants, antianxiety medication, sleeping medication, and over-the-counter drowsy medications. While such drug use is understandable, given that these participants are trying to alleviate the problems associated with their posttraumatic stress and other symptoms, this is a major drawback to studying heart rhythms in clinical populations.

**Clinical Implications**

The present study does not support the proposition that new DSM-5 criteria incorporates more "complex" symptoms into the revised PTSD criteria (Friedman, 2013). Participants meeting criteria for Complex PTSD were just as likely to meet criteria for the DSM-IV-TR PCL-

C as the DSM-5 PCL. Furthermore, the new PTSD criteria does not seem to be more comprehensive, even with its addition of the anhedonic/dissociative subtype and inclusion of a broader range of traumatic histories (Resick et al., 2011). Current results indicate that 18.1% of the total sample met DSM-IV-TR symptom criteria for PTSD, while only 9.5% met symptom criteria for DSM-5 PTSD. Of the population that met DSM-5 symptom criteria, 73% simultaneously met the DSM-IV-TR criteria, leaving over a quarter of that population that would not have been given a DSM-IV diagnosis. These results are similar to those presented in nationwide prevalence study by Kilpatrick and colleagues (2013). In the DSM-5, the PTSD diagnosis has undergone its greatest reconstruction since its introduction, incorporating broader trauma experiences and symptomology (Friedman, 2013). However, the current findings do not support the expectations that the new diagnostic symptomology would incorporate more "complex" symptoms and cast a wider net for the inclusion of various trauma types.

From a clinical perspective, the primary use for diagnostics is to provide structure with which to understand individual experiences and develop a treatment plan. Labels in the form of diagnoses can sometimes shape the pathology, as opposed to the pathology shaping the diagnoses. The literature review points out that PTSD has tended to resemble the present social and political climate and has largely been a war-related disorder. Investigation into post-trauma psychopathology continues to indicate the variety of traumatic experiences extending beyond combat exposure (Herman, 1992). The APA seems to have the right mindset, in that it has attempted to broaden the scope of PTSD as well as other trauma-related disorders, but these goals do not appear to be manifesting (Kilpatrick et al., 2013).

Increased understanding of cardiac vagal tone in trauma populations reveal distinctions within traumatized population, but more importantly it modify and augment treatment plans. For

example, mindfulness therapy incorporating meditation and Hatha yoga to treat combat veterans has been shown to significantly reduce PTSD symptomology (Bhatnagar et al., 2013). Using an animal model, vagal stimulation paired with extinction training resulted in rapid remission of fear behaviors (Peña, Engineer, & McIntyre, 2013). Vagal stimulation has also been shown to improve rehabilitation post injury (e.g., stroke) in an animal model (Khodaparast et al., 2014). But, despite the substantial amount of literature investigating cardiac vagal tone (Sack et al., 2004), especially in trauma populations, there is insufficient research examining vagal retraining in human samples (Cohen et al., 2000). This should be an area of focus in the future and some labs are already taking that step. For example, preliminary investigations are being conducted at Boston University examining how yoga can help retrain the vagus nerve in trauma populations.

**Limitations**

Sample size was likely the strongest threat to study validity, in that larger and equivalent control and PTSD samples sizes were used to examine potential differences with a much smaller Complex PTSD sample. In fact, Field suggests that multivariate group comparisons should have at least 20 cases per group to achieve adequate power (2010). However, Levene's test of normality indicated that all RSA measures had normal distributions and Box's M test for equality of covariance across measures proved to be true for the RSA measures. Both of these tests bolster the main effects and power of the MANOVA.

Another limitation is the forced grouping based on continuous assessment screeners. In order to effective study the distinction between trauma-related disorders, categories seemed the most reasonable method. For the purposes of this study, it was necessary to differentiate along a fine line between similar clinical groups to observe the potential autonomic differences. However, these results should be preliminary to further, and perhaps less categorical,

investigations of trauma-related psychopathologies that may more accurately reflect the true dimensionality of this disorder.

**Future Directions**

This study contributes significantly to the current literature, as it extends beyond the symptomological differences already established and examines whether autonomic function also differentiates Complex PTSD from PTSD. The potential therapeutic implications of this study will likely be most impactful. Additional investigation into Complex PTSD will continue to increase understanding and awareness of its phenomenological differences from PTSD as it is currently defined and highlight potential therapeutic approaches that would be more effective in treating it.

Furthermore, future research should investigate the contribution of the left vagus pathway, originating in the dorsal motor nucleus of the vagus (DMNX) and associated with the phylogenetically oldest defense system, to dissociative symptoms commonly observed in Complex PTSD (van der Hart, Nijenhuis, & Steele, 2005). Research has identified freezing in response to threat as tonic immobility (Volchan et al., 2011). It could be argued that immobility produced by the cardiac influence from the DMNX vagal pathway may contribute to this phenomenon. Presently, there is no literature on the relationship between trauma-related tonic immobility and DMNX vagal influence on the heart or other innervated organs.

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## Appendix A

## The PASAT Math Task

Empirisoft experiment building software, MediaLab and DirectRT, were used in the creation and administration of the PASAT. The task consists of four trials in which single-digit numbers presented consecutively. Participants must sum two consecutive numbers, while remembering the last number of the summation to add it to the following number. With each summation, the participant typed in their answer. After a practice trail with 10 consecutive digits presented at 3s each, the first of 5 trials began by presenting digits in 2.5s intervals, which were shortened by .5s each trial, making the digits on the fifth trial .5s each and totaling 9 minutes and 15 seconds

Table A1

*Timing schemata used for PASAT math task*

| Trial # | ms | Interim - ms | Trials | Trial ms | Total - ms | Total - min |
|---|---|---|---|---|---|---|
| 1 | 2,500 | 10,000 | 50 | 125,000 | 135,000 | 2.25 |
| 2 | 2,000 | 10,000 | 50 | 100,000 | 110,000 | 1.83 |
| 3 | 1,500 | 10,000 | 50 | 75,000 | 85,000 | 1.42 |
| 4 | 1,000 | 20,000 | 100 | 100,000 | 120,000 | 2.00 |
| 5 | 500 | 30,000 | 150 | 75,000 | 105,000 | 1.75 |
| | | 80,000 | 400 | 475,000 | 555,000 | 9.25 |
| | | | | ms | sec | min |
| interims between trials -200ms | | | total: | 555,000 | 555 | 9.25 |

# Appendix B

## Respiratory Sinus Arrhythmia by Heart Period Change-Score Scatter Plots

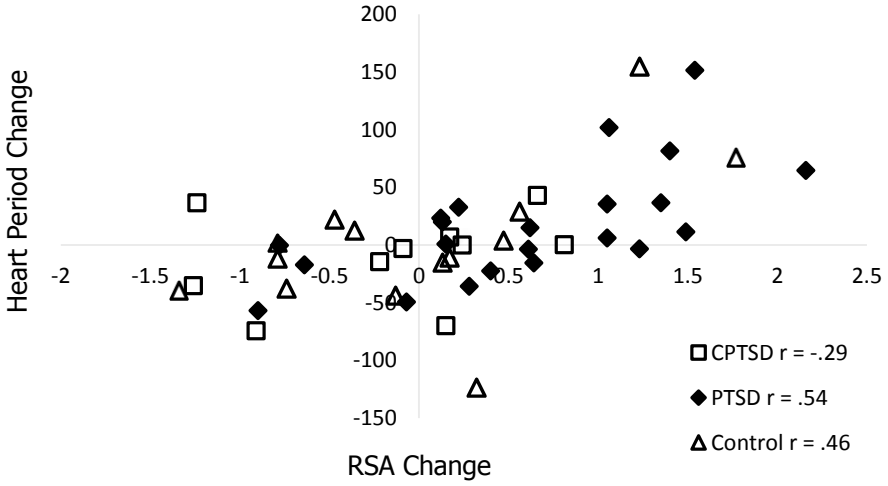


*Figure B1.* RSA and HP change scores, which are differences between baseline and math-task stressor values.

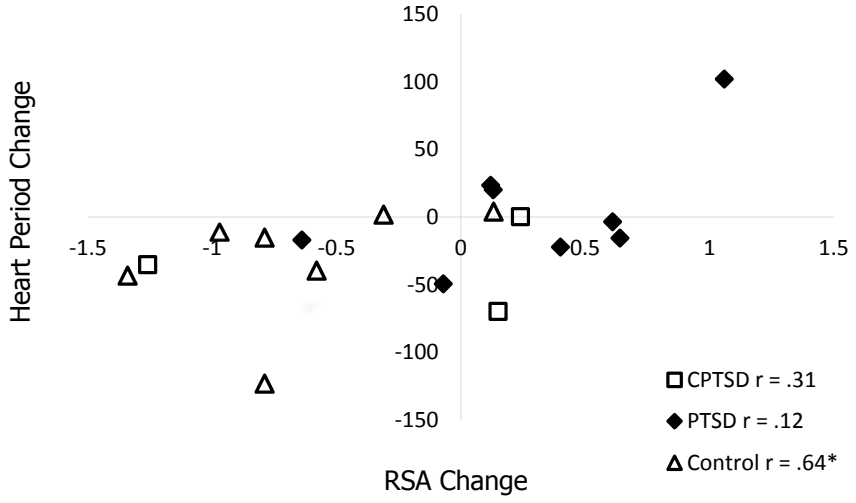


*Figure B2.* RSA and HP change scores, which are differences between baseline and post-task resting period values.