Resting respiratory sinus arrhythmia and posttraumatic stress disorder: A meta-analysis

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Abstract:

Respiratory sinus arrhythmia (RSA) has been examined as a psychophysiological marker of stress vulnerability. Research indicates that low resting RSA is associated with physical and mental health problems, including posttraumatic stress disorder (PTSD). Some research suggests that people diagnosed with PTSD have lower RSA than people without PTSD, but findings have been mixed and the overall magnitude of this effect is unknown, indicating the need for a comprehensive meta-analysis. This meta-analysis examined the association between PTSD and baseline RSA in 55 studies, including 12 unpublished studies, with a total sample size of 6689. Studies were included if they used a PTSD measure, a baseline measure of RSA, and involved humans. Studies were excluded if they were not available in English, did not present quantitative data, presented duplicate data, were a case series, or did not provide results required for computing an effect size. The meta-analysis indicated there is a small but significant association between PTSD and RSA ($g = -0.26$; 95% CI = −0.35, −0.16) with moderate heterogeneity. Moderator analyses suggested that effects are larger for adults than for children and for *DSM-5* PTSD measures than for non-*DSM* referenced measures. We found some evidence for publication bias among the meta-analysis findings. Overall, there is a small but reliable association between PTSD and lower resting RSA, providing support for further research examining the complex relationship between parasympathetic activity and PTSD.

Keywords: Post-traumatic stress disorder | Posttraumatic stress disorder | PTSD | Respiratory sinus arrhythmia | RSA | Meta-analysis | Resting respiratory sinus arrhythmia | Psychophysiology | Parasympathetic nervous system | PSNS | Heart rate variability | HRV | Trauma | Quantitative review | Baseline RSA | High frequency heart rate variability | HF-HRV | Vagal control | Vagal tone | Traumatic stress

Article:

1. Introduction

Respiratory sinus arrhythmia (RSA)—the beat-to-beat variability in heart rate associated with respiration (Grossman & Taylor, 2007)—is associated with many processes in stress,

health, and psychopathology. In the present research, we examined the relationship between RSA and posttraumatic stress disorder (PTSD). PTSD, a psychological disorder resulting from trauma, is characterized by trauma intrusions (e.g., intrusive trauma memories, nightmares), avoiding internal and external trauma reminders, negative changes in cognition or mood, and alterations in arousal and reactivity. Research to date suggests a link between baseline RSA and PTSD—namely, that people diagnosed with PTSD have lower baseline RSA than people without the disorder (*Blechert, Michael, Grossman, Lajtman, & Wilhelm, 2007; Chang et al., 2013; Cohen et al., 1997) and that RSA is inversely associated with PTSD symptom severity (Song et al., 2011)—but the literature's complexity and heterogeneity suggest a need for a quantitative synthesis. Our findings offer support for a link between RSA and PTSD and offer guidance for future work in this field.

1.1. Respiratory sinus arrhythmia

RSA is used to estimate cardiac vagal control (i.e., the level of activity of the vagus nerve to the sinoatrial node, which serves as the heart's natural pacemaker; Berntson, Cacioppo, & Quigley, 1993). High RSA, when accounting for possible confounds like respiration and residual inspiratory vagal activity, indicates a healthy parasympathetic nervous system (PSNS) in the sense that the PSNS is adaptively responding to internal and external cues through changes in heart rate and respiration (Porges, 1995). Researchers agree that RSA can be an estimate of cardiac vagal control, or how much influence the PSNS has on cardiac regulation (Grossman, Wilhelm, & Spoerle, 2004). Many factors, however, can influence the accuracy of RSA as an estimate of cardiac vagal control. Within an individual, RSA changes with age, posture, activity level, and respiration (Grossman & Taylor, 2007). Respiration may be particularly confounding as fast breathing can decrease RSA amplitude, whereas deep breathing can increase RSA amplitude (Hirsch & Bishop, 1981). Thus, RSA should be considered an estimate, not a direct measure, of cardiac vagal control, and care must be taken to control for other factors that also affect RSA.

Theories, such as Porges' Polyvagal Theory (Porges, 1995) and Thayer's Model of Neurovisceral Integration (Thayer & Lane, 2000), propose that RSA is an index of stress resilience, and suggest that low resting RSA is a risk factor for stress vulnerability (e.g., Porges, 1995). The foundation of each model is that cortical regulation of heart rate is mediated by the vagus nerve (Porges, 2011; Thayer & Lane, 2000), which acts as a "brake" that inhibits sympathetic activity in safe environments (Porges, 2011). Porges' theory further suggests that bidirectional connections between the vagus nerve and heart evolved uniquely in mammals to support flexible social behavior based on environmental conditions (e.g., safe, dangerous).

Grossman and Taylor (2007) conceptualize RSA differently, critiquing Porges' notion that RSA is distinct to mammalian species. Grossman and Taylor (2007) critique the foundation of polyvagal theory, noting that the myelinated vagal pathway—the second vagal pathway proposed by Porges to have developed uniquely in mammals—is present in other species (e.g., fish, reptiles, amphibians, birds) that also have RSA. They also critique Porges' work, noting that many studies did not consider the influence of the respiratory system when measuring RSA. This may have led to a potentially unsubstantiated conclusion that vagal control is connected to social

behavior. Grossman and Taylor (2007) instead present RSA not as a direct measure of cardiac vagal control, but as a potential reflection of it when controlling for important confounds.

Grossman and Taylor (2007) theorize that RSA influences changes in cardiovascular and respiratory patterns to match behavior and metabolic demands in each context. Changes in RSA from rest may suggest adaptive flexibility across physiological and psychological contexts for optimal energy exchange. Atypical RSA reactivity would then suggest disturbed physiological (e.g., cardiovascular, respiratory, autonomic) or psychological functioning, or a maladaptive dynamic between physiological and psychological functioning (Grossman & Taylor, 2007).

Despite these differences in theories, a common assertion across appraisal-based models of RSA and environmental stress is that the vagus nerve increases parasympathetic activity (e.g., slowed heart rate) under safe conditions and decreases parasympathetic activity under dangerous conditions when acting optimally. Low resting RSA, indicating suppressed parasympathetic activity at rest, has been characterized as a general marker of stress vulnerability. Consistent with this conceptualization, low resting RSA has been associated with a wide variety of stress-related health outcomes including cardiovascular disease (Dekker et al., 2000), higher rates of depression (Rottenberg, 2007), anxiety (Chalmers, Quintana, Abbott, & Kemp, 2014), borderline personality disorder (Austin, Riniolo, & Porges, 2007). Thus, RSA is a marker with wide relevance across a range of physical and mental health problems.

1.2. RSA and posttraumatic stress disorder (PTSD)

Recent evidence has also suggested that low resting RSA is associated with posttraumatic stress disorder (PTSD). The distinct physiological symptom presentation of PTSD implies an association between the disorder and lowered RSA. Although the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) criteria for PTSD have evolved over the years, physiological symptoms have been an essential feature since PTSD was first added to the *DSM* in its third edition (3rd ed.; *DSM–III*; American Psychiatric Association, 1980). Indicators of sympathetic nervous system (SNS) response (e.g., skin conductance) or nonspecific indicators of autonomic arousal (e.g., heart rate) have traditionally been examined in PTSD research (Butler et al., 1990; Shalev et al., 2000). However, these measures tend to be interpreted as evidence for SNS over-activation, ignoring influences from the parasympathetic nervous system (PSNS). The PSNS is of particular interest because of its influence on resting heart rate. Resting heart rate is one indicator of cardiovascular health: an elevated resting heart rate puts an individual at greater risk for cardiovascular problems later in life (Fox et al., 2007). The PSNS influences resting heart rate independently of the SNS (Berntson, Cacioppo, & Quigley, 1991). Indeed, the PSNS has been found to influence heart rate more than the SNS (Katona, McLean, Dighton, & Guz, 1982), affecting cardiac control with a ratio of 7:1 in humans (Berntson et al., 1993), increasing the value of examining PSNS functioning in PTSD.

A comprehensive meta-analysis of the psychophysiology of PTSD informs our understanding of PSNS functioning in this disorder (Pole, 2007). The meta-analysis synthesized the literature examining markers of the autonomic nervous system, and it found that individuals with PTSD display both higher resting arousal (higher resting heart rate [HR] and skin conductance [SC]) and heighted physiological reactivity to startling sounds and trauma cues. Importantly, the

association between PTSD and resting HR was larger than the association between PTSD and resting SC. Because resting SC is a means of assessing the SNS, whereas resting HR is affected by both the SNS and PSNS (1993, Berntson et al., 1991; Katona et al., 1982; although see also Mackersie & Calderon-Moultrie, 2016 for a contradictory view of SC), Pole's finding suggests that resting arousal in PTSD is influenced by both the SNS and PSNS, instead of solely the SNS (Pole, 2007). This pattern of findings provides further support for examining PSNS markers, such as RSA, in PTSD.

The current literature suggests that PTSD may be associated with lower resting RSA, although findings are mixed. Two types of study designs—between-groups and correlational—have been commonly used to examine the association between RSA and PTSD. The between-groups design examines RSA in participants with PTSD compared to control groups. Studies suggest that individuals with PTSD have a lower RSA than healthy individuals or trauma-exposed individuals without PTSD (Blechert et al., 2007; Chang et al., 2013; Cohen et al., 1997), although some studies have not found significant differences between groups (Bertram et al., 2014; *Kirsch, Wilhelm, & Goldbeck, 2015). The correlational design measures PTSD severity continuously and correlates symptom severity with RSA. The severity of symptoms is typically on a continuum from trauma-exposed without PTSD to severe PTSD. Some research suggests PTSD symptom severity is negatively correlated with RSA (Song et al., 2011), whereas other studies have not found this result (*Keary, Hughes, & Palmieri, 2009; *Sahar, Shalev, & Porges, 2001).

Although low RSA is sometimes discussed as a risk factor for PTSD (Kamkwalala et al., 2012), low RSA may be a physiological symptom of PTSD instead of a risk factor. Shah et al. (2013) examined high-frequency HRV (HF-HRV; a measure of RSA) in 459 male veteran twins who were discordant for combat PTSD. The study found that only the twins with PTSD had lower HF-HRV, and the inverse relationship between combat exposure and HF-HRV was reduced when controlling for PTSD. These findings suggest low RSA may be a result of PTSD, not a risk factor or biological predisposition to trauma exposure. Treatment outcome studies have also found an increase in RSA following various treatments modalities including mindfulness, relaxation training, biofeedback, cognitive-behavioral therapy, prolonged exposure, stress inoculation training, psychodynamic therapy, eye movement desensitization, and psychotropic medication (Bhatnagar et al., 2013; Cohen et al., 2000; D'Andrea & Pole, 2012; Farina et al., 2015; Lewis et al., 2015; Nishith et al., 2003; Reyes, 2014; Sack, Lempa, & Lamprecht, 2007; Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009). We should note, however, that interventions that result in slower and deeper breathing can change RSA independent of vagal activity (Grossman & Taylor, 2007).

2. The present research

Due to the mixed findings, the literature would benefit from a quantitative synthesis. To date, there has been one published meta-analysis examining RSA as a psychophysiological indicator of PTSD (Nagpal, Gleichauf, & Ginsberg, 2013). Nagpal et al. (2013) examined HF-HRV along with other RSA measures (i.e., root mean square of successive differences (RMSSD), standard deviation of beat-to-beat intervals (SDNN)). The meta-analysis found HF-HRV, RMSSD, and SDNN to be significantly associated with PTSD, with effect sizes of $g = -2.27$, -2.94, and -0.61, respectively. Their meta-analysis was limited, however, by a relatively small sample size

($n = 491$) and number of studies ($k = 19$). The studies were limited to between-group studies with control groups, thus excluding all bivariate correlational data between RSA and PTSD symptom severity. Finally, the meta-analysis did not include unpublished data, so their findings might overestimate the effect size due to publication bias. This could explain the unusually large observed effect sizes. Nagpal et al. (2013) also did not examine potential moderators of the association between RSA and PTSD.

The present meta-analysis expands upon previous work to offer a more comprehensive meta-analysis that includes unpublished data, includes correlational data, and examines potential moderators of this relationship. Given our focus on resting RSA, we included only baseline measures of RSA. We did not include measures of RSA change in response to stress-inducing stimuli (e.g., trauma script, loud tone, or other stress-inducing tasks).

2.1. Potential moderators

Many factors affect RSA, so it is crucial to examine potential variables that may moderate the association between RSA and PTSD. One potential moderator is the type of control group used in between-groups studies. Research suggests that individuals with other anxiety disorders have lower RSA than healthy controls. Pittig, Arch, Lam, and Craske, (2013) found that people with panic, generalized anxiety, social anxiety, and obsessive-compulsive disorder all displayed lower RSA compared to healthy controls. The literature suggests that people with anxiety disorders or depression display lower RSA and cardiac vagal control (Friedman & Thayer, 1998; Kemp et al., 2010; Rottenberg, 2007). Due to these findings, we predict that there will be a larger difference in RSA between participants with PTSD and healthy controls than between participants with PTSD and participants with other mental health disorders.

Consideration of trauma-exposed control groups is also necessary because these individuals experienced a traumatic event but did not develop PTSD, indicating resilience. When examining RSA in PTSD compared to trauma-exposed controls, some studies found no difference between groups (Martinez & Eliez, 2008; Sahar et al., 2001), suggesting that a trauma might reduce RSA even without the development of PTSD. Similarly, we predict that there will be a larger difference in RSA between individuals with PTSD and healthy controls than between individuals with PTSD and trauma-exposed controls.

Another moderator to consider is trauma type, which may be strongly connected to PTSD. Specifically, people who experience interpersonal traumas (e.g., sexual assault, abuse, combat) have a greater probability of developing PTSD than people who experience non-interpersonal traumas (e.g., motor vehicle accidents, natural disasters; Schumm, Briggs-Phillips, & Hobfoll, 2006). Survivors of interpersonal trauma may also have more distinct fear conditioning (Forbes et al., 2011) and emotion regulation difficulties (Ehring & Quack, 2010). Emotion regulation difficulties may be most salient in childhood interpersonal trauma, perhaps due to disruption of relationships with attachment figures (Diamond & Hicks, 2005), suggesting that not only trauma type, but also the developmental period of the trauma, may affect later functioning. The effects of trauma type or timing on resting RSA are unclear, as they have seldom been examined. But given the associations of these variables with PTSD, we thought it important to examine trauma type and timing of trauma exposure as potential moderators.

Another important moderator to consider is age. RSA is higher at younger ages (Hirsch & Bishop, 1981) and decreases over time (Masi, Hawkley, Rickett, & Cacioppo, 2007). Gender may be another moderator, as female gender is a risk factor for developing PTSD (Brewin, Andrews, & Valentine, 2000). Women also have higher RSA than men (Dishman et al., 2000), further highlighting the need to examine gender differences.

2.2. Goals and hypotheses

The first goal of this meta-analysis is to combine existing findings to estimate the overall size of the association between PTSD and RSA and to determine whether PTSD is significantly related to RSA. The second goal of this study is to examine moderators of the relationship between PTSD and RSA (e.g., age, control group). Specifically, we predict that the type of control group will serve as a significant moderator of the association between PTSD and RSA. It is expected that when PTSD groups are compared to healthy control groups, there will be a larger effect size than when the PTSD group is compared to other control groups (i.e., trauma-exposed controls, other mental health disorders).

The study will examine many other exploratory moderators that have been associated with psychophysiological responses in PTSD in prior research. Categorical moderators include age group, age group at time of trauma, trauma type (interpersonal, noninterpersonal, or mixed), measure used to quantify RSA (time vs. frequency domain), measure used to assess PTSD severity (i.e., clinical interview, self-report), *DSM* version of PTSD measure, presence of trauma cue or other stressful task in study protocol following baseline assessment, presence of accommodation period, position of baseline RSA measurement (i.e., sitting, supine, unknown), study design (i.e., categorical or correlational), treatment study (i.e., treatment design, no treatment), psychophysiology setting (i.e., lab, ambulatory), medication exclusions (i.e., whether the study excluded for cardiovascular medication or psychoactive medication), and use of paced breathing. In addition, the following exploratory continuous moderators will be examined: percent female, length of baseline RSA measurement, mean years since trauma occurred, mean resting HR, and comorbid depression (either mean BDI score or percent of the sample with a depression diagnosis, depending on which is reported). Additionally, for studies with mixed trauma type (both interpersonal and non-interpersonal traumas included), the percent of interpersonal trauma will be examined as a continuous moderator. Examining the overall effect size and moderators of this effect will help scientists and clinicians better grasp the association of PTSD and RSA and clarify how strong this effect is. The final goal is to evaluate the extent of publication bias in the literature to determine the accuracy of the observed effect size.

3. Method

The methods used to conduct this meta-analysis were in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA Transparent Reporting of Systematic Reviews & Meta-analyses., 2015).

3.1. Study retrieval

Several methods were used to ensure an exhaustive literature search. First, a computer search was conducted on July 31, 2017 across the following databases: PsycINFO (Psychological Information Database), PubMed, and PILOTS (Published International Literature on Traumatic Stress) databases. No time period restriction was set on the search. The following search terms were used: "PTSD," "post traumatic stress disorder," "post-traumatic stress disorder," or "trauma," combined with "RSA," "respiratory sinus arrhythmia," "HRV," "heart rate variability," "vagal control," or "parasympathetic nervous system."

3.2. Study inclusion and exclusion criteria

At the full-text review stage, the first and fourth authors assessed all studies for eligibility based on the following inclusion criteria: the study included a measure of PTSD, the study included a baseline RSA measure, and the study involved human participants (either adult or child/adolescent). A baseline measure was defined as a measure of RSA (lab-based or ambulatory) of any duration one minute or longer so long as no stressful or emotion-inducing stimuli were presented (neutral visual or auditory stimuli were allowed).

Studies were excluded for the following reasons: the article was not available in English, no data were presented (e.g., literature review), the study was a case study or case series ($n < 5$), or the study did not report findings needed to calculate an effect size. In cases of duplicate data (i.e., multiple studies included the same participant sample), the study that provided the data necessary to compute an effect size was used. If more than one article met that criterion, the article that was published first was included in analysis.

3.3. Unpublished data

To reduce and evaluate the effects of publication bias, we collected unpublished data using the same inclusion and exclusion criteria. Unpublished data were collected through the following methods: unpublished dissertation/thesis databases, contacting individual research labs or researchers involved in PTSD research, and listserv postings. A literature search was performed under the Global Dissertation/Thesis database for any unpublished studies that fit the inclusion criteria. Prominent authors who appear regularly among the published articles were contacted and asked for possible unpublished data. Finally, requests for unpublished data were posted to professional organization listservs, including the Association for Behavioral and Cognitive Therapies and the Anxiety and Depression Association of America.

3.4. Variables

Measures of PTSD and RSA served as the variables of interest in this meta-analysis. The following RSA measures were included in analysis: SDNN, RMSSD, pNN50 (proportion of RR intervals that differ by more than 50 ms), peak-to-valley method (mean difference of maximum heart period during inhalation and minimum during exhalation), Porges' method (see Porges & Bohrer, 1990 for a description), and HF-HRV. For our meta-analysis, the only HRV measures that were excluded from coding were very-low frequency HRV, low-frequency HRV, high frequency percentage (the percent of high-frequency bands over total bands), and the low-frequency to high-frequency HRV ratio. These controversial measures were excluded because

they appear to confound SNS and PSNS information (1993, Berntson et al., 1991; Heathers, 2007).

For PTSD measures, we included any measure that assesses PTSD symptoms, including measures linked to the *DSM* criteria for PTSD, measures linked to *ICD* criteria, or measures that pre-date the *DSM* but are well-established measures of PTSD (e.g., the Impact of Event Scale). At final analysis of included articles, there were 15 different PTSD measures. The clinical interview measures used were the Clinician Administered PTSD Scale (CAPS; $n = 23$), the Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorder-IV* (*DSM-IV*) PTSD module ($n = 9$), *International Classification of Diseases (ICD-9)* PTSD module ($n = 1$), Research Diagnostic Interview for Psychological Disorders (F-DIPS; $n = 1$), and the Childhood Trauma Interview (CTI; $n = 1$). The rest of the PTSD measures were self-report questionnaires and consisted of the PTSD Checklist (PCL; $n = 6$), Davidson Trauma Scale (DTS; $n = 2$), Youth Symptom Survey Checklist (YSSC; $n = 1$), PTSD Symptom Scale (PSS; $n = 1$), Child PTSD Symptom Scale (CPSS; $n = 2$), Trauma Symptom Checklist for Children (TSCC-A; $n = 1$), Minnesota Multiphasic Personality Inventory (MMPI-PTSD) PTSD scale ($n = 1$), Detailed Assessment of Posttraumatic Stress (DAPS; $n = 1$), Harvard Trauma Questionnaire (HTQ; $n = 2$), and Impact of Event Scale-Revised (IES-R; $n = 1$).

3.5. Study coding procedures

A comprehensive study coding spreadsheet was created to collect data across domains within included studies. The coding spreadsheet included basic study information (authors, year, whether data were published); sample characteristics (e.g., sample size, age, age at trauma, ethnicity, gender, trauma type, population type, cardiovascular disease, smoking, medication, depression, resting heart rate); study methods and procedures (e.g., whether the PTSD measure was self-report or structured clinical interview, specific PTSD questionnaire or interview measure used, measure of RSA, type of control group, accommodation period present, trauma or stressful task present after the baseline, study design, position of baseline reading, time length of baseline reading, whether the study implemented a treatment condition, psychophysiology setting, *DSM* measure, paced breathing present). The first author coded all effect sizes and moderator variables for analyses. The fourth author then independently coded a random selection of 40% of all effect sizes and moderator variables to examine inter-rater agreement.

4. Data analysis

Hedges' *g* was selected as the effect size because it provides a better estimate of the standardized mean difference than Cohen's *d* in small samples (Borenstein, Hedges, Higgins, & Rothstein, 2009), which are common in this literature. The effect size was interpreted with the standard Hedges' *g* cut-points of 0.2 as small, 0.5 as medium, and 0.8 as large. Hedges' *g* can be negative or positive depending on the nature of the association. In the context of this meta-analysis, a negative Hedges' *g* would be interpreted as individuals with PTSD have lower RSA than control participants, or that PTSD symptoms are negatively correlated with RSA. Several studies had multiple effect sizes calculated because they had more than one RSA measure or more than one control group, but only one pooled or average effect size per study was used to estimate the overall mean effect size. Where multiple effect sizes are computed for several independent

subgroups of a study, we used the Comprehensive Meta-Analysis (CMA; Comprehensive Meta-Analysis, 2017) Version 3 software to calculate a pooled effect size, weighted for the subgroup sample sizes (see Borenstein et al., 2009, pp. 26–28, for details). Where multiple effect sizes were computed for multiple measures within the same sample, an average effect size was computed across all measures (Borenstein et al., 2009, pp. 28–30).

We used a random effects model, which assumes error is systematic and that there is no one true effect size, but rather a population distribution of effect sizes which observed effects are drawn from (Borenstein, Hedges, Higgins, & Rothstein, 2010). In psychological research, random-effects models are used more often than fixed-effects models, where variance in the effect size is a result of sampling error alone, due to the assumption that variation in effects is not solely due to sampling error. We also assessed for study heterogeneity using I^2. Thresholds for I^2 can be interpreted as 0–40 suggesting non-relevant heterogeneity, 30–60 suggesting moderate heterogeneity, 50–90 suggesting substantial heterogeneity, and 75–100 suggesting considerable heterogeneity (Higgins & Green, 2011).

Moderator analyses investigated whether several variables act as moderators in the association between PTSD and RSA. Each study was coded to include each of these variables, and we calculated moderator analyses in CMA to determine each moderator's significance. Categorical moderators were assessed in an approach similar to ANOVA (Borenstein et al., 2009, p. 164–183), and continuous moderators were assessed in an approach similar to regression (Borenstein et al., 2009, p. 196–203). Several studies included more than one measure of RSA. For the moderator analysis of type of RSA measure, measures were disaggregated to have more power for analysis. For categorical moderators, we estimated the effect size and 95% confidence interval around the effect size at each level of each moderator variable. Because the levels of the moderators typically had different numbers of *k* studies, and some levels typically had much smaller numbers of studies than others, we adopted a conservative approach to evaluating the confidence intervals (Schenker & Gentleman, 2001). Estimates with non-overlapping upper and lower interval limits were interpreted as having a statistically significant difference; estimates that were included in another estimate's interval were interpreted as not statistically significant; and estimates with overlapping limits were interpreted as ambiguous regarding significance, given the disparities in *k* sizes and low *k* values in many cases. To calculate categorical moderator analyses, we required at least three effect sizes ($k = 3$) for each level of the moderator variable (Sumner, Griffith, & Mineka, 2010). The continuous moderators were run using meta-regression methods in CMA (see Borenstein et al., 2009, p. 196–203). Each continuous moderator was run in a separate model where the variable was entered as a covariate for the overall effect size. The meta-regression produced a coefficient value, standard error, 95% confidence interval, and 2-sided *p*-value for each moderator test. We required at least six effect sizes ($k = 6$) for each moderator variable assessed through meta-regression (Huizenga, Visser, & Dolan, 2011).

We also calculated several analyses to assess possible publication bias. We used funnel plot diagrams, trim and fill analyses (Duval & Tweedie, 2000), and Egger's test (Egger, Smith, Schneider, & Minder, 1997) as indicators of publication bias. The more symmetrical the funnel plot distribution, the less likely publication bias affected results. The trim and fill analysis reconstructs the funnel plot diagram to create a more symmetric distribution by trimming studies

with more extreme effect sizes and filling missing studies to have symmetry. This analysis then recomputes the effect size with the new data accounting for studies that have either been removed or added. Egger's test is a linear regression where the standard normal deviate is regressed on precision (i.e., the opposite of standard error). The test produces an intercept that relates to the slope of the effect size on standard error. The further the intercept lies from zero, the larger the bias and greater evidence for small-study effects (Sterne, Gavaghan, & Egger, 2000). Egger's test is more sensitive to small study effects and is thus a useful test for publication bias in this literature. Finally, we compared the effect sizes of published and unpublished studies.

5. Results

The meta-analysis included studies published or datasets collected between 2000 and 2017. The final analysis included 55 studies and a total sample size of 6689 participants.

5.1. Excluded studies

The initial keyword search yielded 3975 articles. All articles retrieved from this search were then merged into Zotero, a reference management program (Puckett, 2011). Zotero automatically co-selects items identified as potential duplicates and puts them into a duplicate file folder. The first author then reviewed this folder manually, removing duplicate articles. The next step was to screen articles based on their titles to evaluate whether they were to be included in full-text review. This step yielded 269 articles to be included for full-text review. Using our a priori article inclusion and exclusion criteria, the final number of studies included in the meta-analysis was 55 (see online supplemental figure). The kappa agreement between first and fourth author for study inclusion was almost perfect (kappa = .93; McHugh, 2012). It should be noted that an additional 22 studies met inclusion criteria; however, the articles did not include the information needed to compute an accurate effect size. The corresponding authors for these articles were contacted, and they either did not respond to our inquiries or were unable to provide the requested information. Twelve of the 55 studies came from unpublished datasets generously provided by colleagues.

5.2. Characteristics of the studies included

The studies were quite heterogeneous, as many of the studies' primary research question was not to investigate the association between PTSD and RSA. Most of the studies were conducted in the lab (85.5%) as opposed to ambulatory research (14.5%). The ambulatory studies' methodology varied greatly, with some studies conducted in home settings, others in hospitals, and others not specifying the location. Studies were evenly divided between trauma cue or another stressful paradigm following the baseline assessment (51%) compared to no stress-inducing task after the baseline (49%). Sample type varied, with community sample the most prevalent (62%), followed by a military and/or veteran sample (34.5%), then undergraduate student sample (3.5%). Most studies included adult samples (90.9%) compared to child/adolescent samples (9.1%). Most of the studies recruited a mixed trauma type sample (63%), followed by interpersonal trauma (33%), then non-interpersonal trauma (4%). Studies had an average baseline collection time of 6.5 min, ranging from 2 min to 20 min in length. The majority (84%) collected baseline data for

at least three minutes, the recommended minimum time for accurate baseline RSA (Berntson et al., 1997). The most common position for collecting baseline RSA data was sitting or reclining (50.91%), followed by supine position (12.72%), then standing (1.82%), with several studies (34.55%) not reporting body position. The online supplement provides a detailed description of all studies included and their characteristics.

5.3. Mean effect size and effect sizes of moderators

Study coding kappa agreement between first and fourth author was excellent (kappa = .84). The random effects model yielded a small but significant mean effect size ($g = -0.26$; 95% CI = −0.35, −0.16; $p < 0.001$) for the association between RSA and PTSD. Studies' effect sizes ranged from -1.84 to 0.54, with 18 of the 55 studies indicating significant effect sizes at $\alpha = .05$. The overall heterogeneity measure indicated significant substantial heterogeneity among studies ($I^2 = 55.65$; $p < 0.001$).

For the categorical moderators, age group was significant with adults showing a more pronounced effect ($g = -0.30$; 95% CI = −0.39, −0.20) compared to child/adolescent samples ($g = 0.09$; 95% CI = −0.10, 0.28). Adult trauma also had a more pronounced effect ($g = -0.40$; 95% CI = −0.57, −0.23) than child trauma ($g = 0.07$; 95% CI = -0.13, 0.26). The type of control group moderator was not significant and yielded similar effect sizes for healthy controls ($g = -0.44$; 95% CI = −0.62, −0.25), trauma exposed controls ($g = -0.20$; 95% CI = −0.41, 0.01), and other mental health controls ($g = -0.30$; 95% CI = −0.55, −0.05). Contrary to our hypothesis that there would be a larger effect among healthy controls than trauma exposed controls or other mental health controls, we did not find a significant effect.

We examined several other exploratory categorical moderator variables with significant effects across a few variables. The type of RSA measure used had some significant distinctions. Specifically, there were effect size differences when comparing SDNN ($g = -0.40$; 95% CI = −0.59, −0.21) and pNN50 ($g = -0.66$; 95% CI = −0.98, −0.34) against Porges' method ($g = 0.09$; 95% CI = -0.20, 0.39). We also found a significant difference between DSM-5 measures ($g = -0.56$; 95% CI = −0.84, −0.28) compared to a non-DSM measure of PTSD ($g = -0.05$; 95% CI = −0.21, 0.11). All other exploratory moderator variables were non-significant (Table 1). Several of these non-significant variables contained zero in their confidence intervals (trauma exposed control group, peak-to-valley RSA measure, no exclusion for cardiovascular medicine, paced breathing present) (Fig. 1).

For the continuous moderators, age ($\beta = -0.01$; 95% CI = −0.01, 0.00) significantly moderated the association between RSA and PTSD, indicating that for every one year increase in age the inverse association between PTSD and RSA strengthens by 0.01 units. Mean HR also significantly moderated the association between RSA and PTSD ($\beta = 0.02$; 95% CI = 0.00, 0.03), indicating that for every one beat increase in mean heart rate the inverse association between PTSD and RSA weakens by 0.02 units. No other continuous moderator variables were significant (Table 1).

Table 1. Moderator Analyses.

Categorical Moderators	**N[a]**	**Hedges'g**	**95% CI**
Age Group			
Adult	**50**	**−0.3**	**−0.39, -0.20**
Child/Adolescent	**5**	**0.09**	**−0.10, 0.28**
Age at Trauma			
Adult	**15**	**−0.4**	**−0.57, -0.23**
Child	**6**	**0.07**	**−0.13, 0.26**
Mixed	13	−0.22	−0.43, -0.01
Unknown	21	−0.26	−0.42, -0.11
Control Group			
Healthy Control	18	−0.44	−0.62, -0.25
Trauma Exposed Control	18	−0.2	−0.41, - 0.01
Other Mental Health Control	10	−0.3	−0.55, -0.05
Trauma Type			
Interpersonal	19	−0.25	−0.34, -0.16
Mixed	28	−0.35	−0.41, -0.24
RSA Measure			
HF HRV	49	−0.26	−0.36, -0.16
SDNN	**18**	**−0.4**	**−0.59, -0.21**
RMSSD	14	−0.39	−0.59, -0.19
Peak-to-Valley	5	−0.18	−0.37, 0.01
pNN50	**3**	**−0.66**	**−0.98, -0.34**
Porges' Method	**3**	**0.09**	**−0.20, 0.39**
PTSD Measure			
Clinical Interview	38	−0.28	−0.41, -0.16
Self-Report	17	−0.21	−0.35, -0.07
Trauma/Stress Task			
Trauma/Stress Task Present	28	−0.17	−0.29, -0.05
Not Present	27	−0.35	−0.49, -0.21
Accommodation Period			
Present	14	−0.34	−0.54, -0.14
Not Present	41	−0.23	−0.33, -0.12
Study Design			
Categorical	35	−0.32	−0.45, -0.19
Correlational	20	−0.13	−0.23, -0.03
Position of Baseline Reading			
Sitting/reclining	28	−0.21	−0.32, -0.09
Supine	**7**	−0.48	−0.75, -0.21
Treatment Study			
Treatment Study	5	−0.66	−1.29, -0.03
Not Treatment Study	50	−0.24	−0.33, -0.14
Psychophysiology Setting			
Lab	47	−0.24	−0.57, -0.21
Ambulatory	8	−0.39	−0.54, -0.23
DSM Measure			
DSM-IV	43	−0.28	−0.39, -0.16
DSM-5	**3**	**−0.56**	**−0.84, -0.28**
Non-DSM Measure	**7**	**−0.05**	**−0.21, 0.11**
Cardiovascular Medicine			
Excluded For	19	−0.29	−0.50, -0.09
Not Excluded For	11	−0.15	−0.31, 0.01
Psychoactive Medicine			
Excluded For	17	−0.24	−0.46, -0.02
Not Excluded For	19	−0.26	−0.37, -0.15
Paced Breathing			
Present	3	−0.55	−1.48, 0.39
Not Present	52	−0.25	−0.34, -0.16
Published Status			
Published	43	−0.31	−0.42, -0.20
Unpublished	12	−0.08	−0.22, 0.05

Continuous Moderators	**N**	**β[b]**	**95% CI**	**p**
Age	**49**	**−0.01**	**−0.01, 0.00**	**0.02**
Gender	36	0	0.00, 0.00	0.46
Length of Baseline Reading	43	0	−0.03, 0.02	0.76
Mean Years Since Trauma	14	0	−0.02, 0.02	0.98
Mean Resting HR	**24**	**0.02**	**0.00, 0.03**	**0.04**
Percent Smoking Status (PTSD)	10	0	−0.01, 0.01	0.68
Mean BDI (PTSD)	12	0.01	−0.02, 0.05	0.44
Percent MDD (PTSD)	7	−0.01	−0.01, 0.00	0.13
Percent Interpersonal Trauma	11	0	−0.01, 0.01	0.71

Note. Categorical moderator significance is found if 95% confidence intervals are nonoverlapping. Continuous moderator significance is found if $p < 0.05$.

[a] N's do not always sum up to total of 55 studies due to some studies not providing necessary moderator information to run analyses.

[b] Raw beta weights provided.

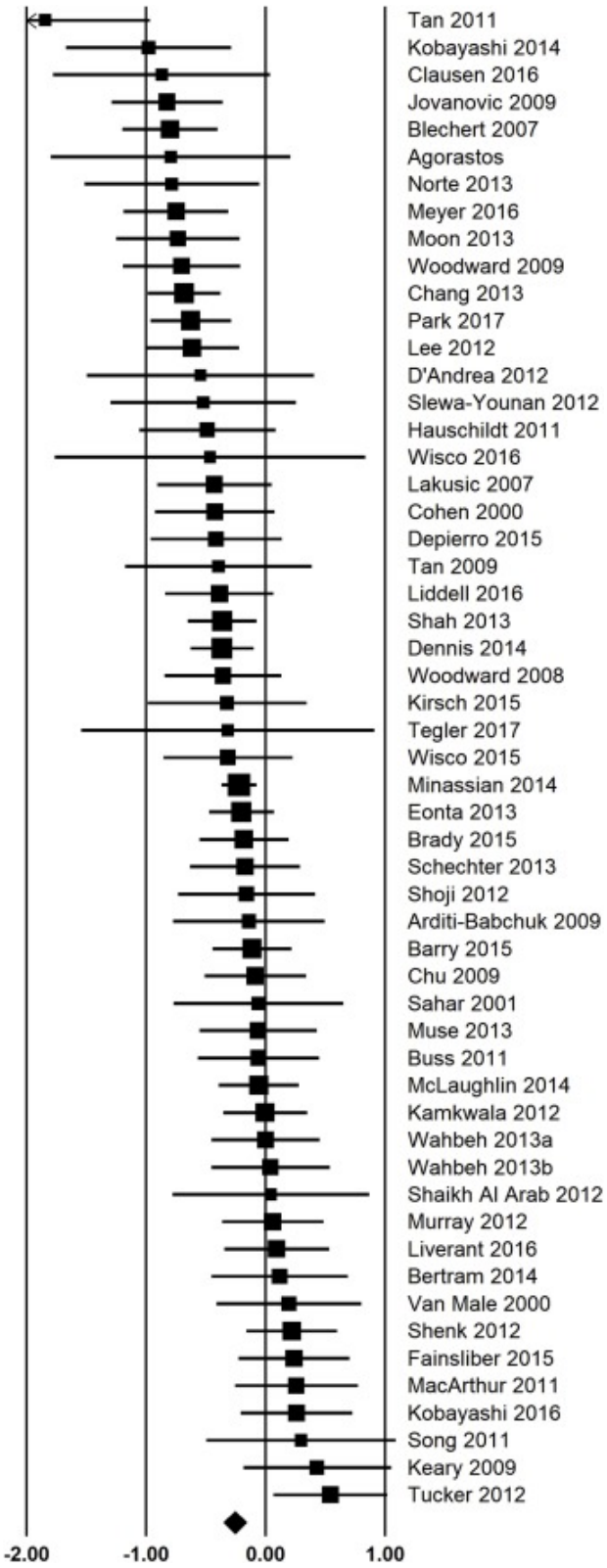

Figure 1. Forrest Plot of all studies included in meta-analysis.[1]

[1] Studies are sorted from largest negative to largest positive effect size. The size of the square reflects the sample size, with larger squares indicative of larger samples.

5.4. Publication Bias

Several analyses were run to evaluate publication bias. First, we evaluated the effect sizes of published (g = -0.31; 95% CI = -0.42, -0.20) and unpublished (g = -0.08; 95% CI = -0.22, 0.05) studies. The disparity between the effect sizes is consistent with a file-drawer effect, in that the effect size for published studies is significant but the effect of unpublished studies is not. Second, we graphed a funnel plot of standard error by Hedges' g (Fig. 2). The plot yielded some asymmetry on the left side of the distribution, suggesting there were more studies indicating a significant association between PTSD and RSA. Third, we calculated Egger's regression test, showing limited publication bias (b = −0.23; 95% CI = −1.23, 0.76), with a slope (b) near zero indicating low publication bias. Finally, we calculated Duval and Tweedie's trim and fill, which indicated limited publication bias (g = −0.25; 0 adjusted values; 95% CI = −0.31, −0.19), with the reconstructed Hedges' g being similar to original Hedges' g found, and with no adjusted values, indicating no need to adjust Hedges' g because of significant publication bias.

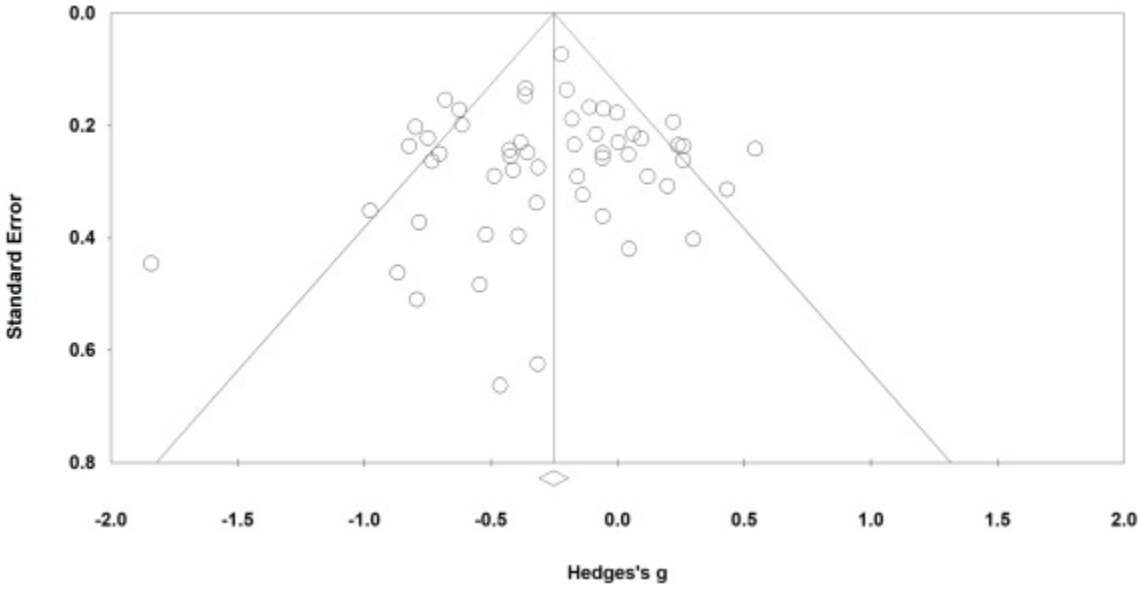

Figure 2. Funnel plot of publication bias.
This figure illustrates where individual study effect sizes fall. The more symmetrical the funnel plot, the less publication bias present.

6. Discussion

This meta-analysis of 55 studies found a small but significant association between PTSD and RSA. This finding provides clarity to the mixed results in the literature and indicates that a reliable association exists. It is possible that the studies that did not report significant findings may have been underpowered to detect an effect of this size, leading to mixed findings in the literature. However, the effect size should be interpreted cautiously due to the effect size's heterogeneity. The heterogeneity of the observed effect size was significant, and the sources of that heterogeneity were only partially explained by significant moderators.

Results indicated age is a significant moderator in explaining effect size heterogeneity. Specifically, as age increases the association between PTSD and RSA becomes stronger, and the

association between PTSD and RSA is significantly stronger among adults than among children. This finding suggests that the association between PTSD and RSA may grow stronger as individuals age, although longitudinal data are needed to test this possibility. In future research on PTSD and RSA, we recommend that researchers constrict the age range of participants to decrease variability. Results also indicated a significant effect of age at time of trauma, such that there was a larger effect when the trauma occurred in adulthood. However, this effect may have been driven by age, and there were few studies of traumas occurring during childhood ($k = 5$), so this moderator should be interpreted cautiously.

Mean resting HR was also significant in explaining effect size heterogeneity. As mean HR increases, the association between PTSD and RSA weakens. Many confounding variables influence resting HR (e.g., exercise, medication), but this finding may suggest that the lower resting HR allows for less variability, leading to a stronger association. While this effect is difficult to interpret, it does reinforce the importance of measuring and controlling for resting HR when examining RSA.

Finally, the type of *DSM* measure also differed in effect size values, with *DSM-5* measures producing a significantly larger effect than non-*DSM* measures, with *DSM-IV* measures producing an effect size that fell between the other two measures (and was not significantly different from either). These findings suggest the importance of using *DSM-5* constructed measures when examining the association between RSA and PTSD. Additionally, we did not find that type of control group moderated the association between RSA and PTSD, suggesting there may not be a large difference in RSA among these different control groups.

6.1. Comparisons with past meta-analyses

The only meta-analysis that also investigated the association between RSA and PTSD was published in 2013 (Nagpal et al., 2013). Nagpal et al. (2013) had a wider scope than our study, examining heart rate and low-frequency heart rate variability alongside parasympathetic activity. However, their meta-analysis was limited by a smaller sample size ($n = 491$, compared with $n = 6725$ in our meta-analysis) and smaller number of studies ($k = 19$, compared with $k = 55$ in our meta-analysis). Several of the studies analyzed in Nagpal et al. were excluded from our analysis due to a priori exclusion criteria (e.g., case series, percent high frequency HRV values, non-baseline RSA values). Additionally, their meta-analysis excluded correlational data and unpublished data. Our meta-analysis expanded upon their work by extending study inclusion criteria to correlational studies, incorporating unpublished data into our results, and including studies that have been published in the years between meta-analyses.

Nagpal et al. (2013) did not compute an overall mean effect size, and instead provided separate mean effect sizes for different physiological measures. In their meta-analysis, effect size measures examining RSA ranged from a Hedges' g of −.61 to −2.94 (Nagpal et al., 2013). The present meta-analysis overlapped Nagpal et al. in three measures (i.e., HF-HRV, RMSSD, SDNN). Their findings show much larger effects than our results. The most extreme differences were for HF-HRV and RMSSD. For HF-HRV we found an effect of $g = −0.26$, whereas they found $g = −2.27$. For RMSSD, we found an effect of $g = −0.39$, whereas they found $g = −2.94$. The findings for SDNN were more comparable with $g = −0.40$, compared to Nagpal et al. $g =$

−0.61. The difference in findings may be attributed to the smaller number of studies, the design of studies included, and the lack of unpublished data in their meta-analysis.

Pole's (2007) meta-analysis examining other psychophysiology of PTSD found similar effect sizes to our meta-analysis. After converting Pole's *r* coefficients of resting physiology results into Hedges' *g*, effect sizes ranged from $g = 0.22$ to $g = 0.41$. Pole (2007) examined resting HR ($g = .41$), resting skin conductance ($g = 0.26$), resting systolic blood pressure ($g = 0.22$), and resting diastolic blood pressure ($g = 0.41$). These effect sizes are in the opposite direction of what we found, as would be expected for measures of sympathetic arousal and PTSD, but of a similar magnitude to our findings. Comparing our results to those of Pole (2007), these findings indicate that RSA has an association with PTSD of similar magnitude to some more established physiological indicators of PTSD, such as skin conductance and systolic blood pressure. Our results also support Pole's speculation that PSNS activity may explain the relatively larger effect size observed for HR, affected by both PSNS and SNS activity, than for SC, affected primarily by the SNS. Specifically, we found evidence for lowered PSNS activity at rest associated with PTSD, offering support for the notion that PSNS and SNS activity may have an additive effect on resting HR in this population.

6.2. Publication Bias

Evidence for publication bias was mixed. The effect size for published studies ($g = -0.31$) was larger than for unpublished studies ($g = -0.08$), and the effect size for unpublished studies was not significantly different from zero. There was considerable variability in effect sizes for unpublished studies, from $g = -0.45$ to $g = 0.26$, with five unpublished studies having effect sizes between $g = -0.45$ to $g = -0.16$. Duval and Tweedie's trim and fill also showed relatively little evidence for publication bias, with other tests (funnel plot, Egger's regression test) indicating possible publication bias present in this meta-analysis. We attempted to target the problem of publication bias by including unpublished findings ($k = 12$). It is also possible that the "file drawer effect" was not as strong in our analyses because of the nature of the published studies included. Many published studies included were treatment studies, or studies primarily examining other variables associated with PTSD (e.g., sleep, aggression, attention bias, marital health) that happened to include a baseline measure of RSA. These studies, whose primary outcome was not RSA, are less likely to be influenced by the "file drawer effect."

6.3. Limitations

This meta-analysis had limitations both general to all meta-analyses as well as unique to this specific design. First, many studies did not have clean samples and did not exclude participants for criteria like medication use, smoking, and medical conditions. Pole's meta-analysis (2007) found no moderation between psychoactive medications and psychophysiological effects, although it did not examine RSA. However, best practice is to exclude psychoactive medications when examining physiological variables to reduce heterogeneity. Smoking may have confounded the results because smoking is common among individuals with PTSD and has been found to affect RSA (Barutcu et al., 2005). Finally, medical conditions including cardiovascular disease were not always accounted for in included studies, which would lead to decreased RSA independent of psychopathology (Thayer, Yamamoto, & Brosschot, 2010). We initially hoped to

include all of these variables as moderators, but coding indicated that too few studies reported on these variables to include them.

Additionally, we had hoped to include respiration rate as a moderator, as this variable has been shown to influence RSA (Grossman & Taylor, 2007). However, only four studies reported data on respiration rate. Due to low power, we were unable to examine to what extent RSA accurately estimates vagal control regardless of respiration. In theory, there could be respiration differences among PTSD samples driving this effect. Fitness level also contributes to baseline RSA (Grossman & Taylor, 2007). We were also unable to examine fitness variables (e.g. body mass index, weight, physical activity) due to lack of reporting. It is particularly important to examine this variable in PTSD samples due to the connection between PTSD and poorer physical health (Pacella, Hruska, & Delahanty, 2013). We recommend that future studies examining RSA in PTSD report data on respiration, heart rate, and fitness level.

Another limitation in this meta-analysis was the exclusion of 22 studies because authors did not respond to inquiries related to computing an accurate effect size. This affected overall power, along with power to detect significant moderators. A final limitation was our decision to exclude articles not available in English. Of 3970 articles identified, this criterion excluded 34 articles (1%) from full-text evaluation.

6.4. Future directions

Our findings have provided some answers to the relationship between PTSD and RSA but many questions remain. Due to the heterogeneity of our results, it is important to consider other possible moderators of this association. Specifically, sample exclusion criteria (e.g., smoking status, medical conditions) and trauma type (e.g., sexual assault, military combat). could be examined more closely. To examine trauma type, it will be necessary for studies to recruit specific trauma types instead of the mixed trauma samples commonly used.

Our main finding indicates that RSA and PTSD are correlated, clearing up the past ambiguity of this association. This finding cannot determine whether low RSA is a risk factor for developing PTSD or if low RSA is a result of PTSD. However, if low RSA were to be a risk factor, practitioners could target this physiological abnormality by implementing simple interventions like increasing exercise or decreasing smoking (Grossman et al., 2004). If low RSA is a result of PTSD, future research could examine whether decreases in RSA due to PTSD explain some of the comorbidity between PTSD and physical health problems, and whether increases in RSA following PTSD treatment lead to physical health benefits (Fox et al., 2007; Grossman et al., 2004; Masi et al., 2007).

Given the demonstrated small but significant association between resting RSA and PTSD, we hope that researchers will more consistently measure RSA in PTSD samples. This would allow for future research to uncover how RSA may change across established evidence-based treatments or in more novel therapeutic approaches. Because PTSD includes a number of heterogeneous symptoms, we also recommend that researchers examine RSA in relation to specific PTSD symptoms (e.g., flashbacks, nightmares, negative cognitions, avoidance, numbing) to see whether RSA is related to all PTSD symptoms or whether the observed

association is driven by specific symptoms or symptom clusters. Additionally, *DSM*-5 introduced a dissociative subtype of PTSD that may be characterized by a distinct physiological profile. Future research should examine potential differences in RSA between PTSD and the dissociative subtype of PTSD. Finally, as the literature continues to grow, it will be necessary to conduct further moderator analyses with adequate power to best explain effect size heterogeneity.

Overall, our meta-analysis provides some clarity to the mixed literature surrounding the association between RSA and PTSD. Moderator findings suggest that the association between RSA and PTSD is larger for adults than for children, and that future studies should consider restricting the age range of their participants to reduce variability due to age. Moderator findings also suggest the importance of using DSM-5-referenced PTSD measures. Historically, sympathetic markers of psychophysiology have been more extensively studied in PTSD than parasympathetic markers. However, the significant small association between RSA and PTSD reinforces the increased need to examine parasympathetic measures in psychological research in the hopes of better understanding the physiological correlates of mental illness.

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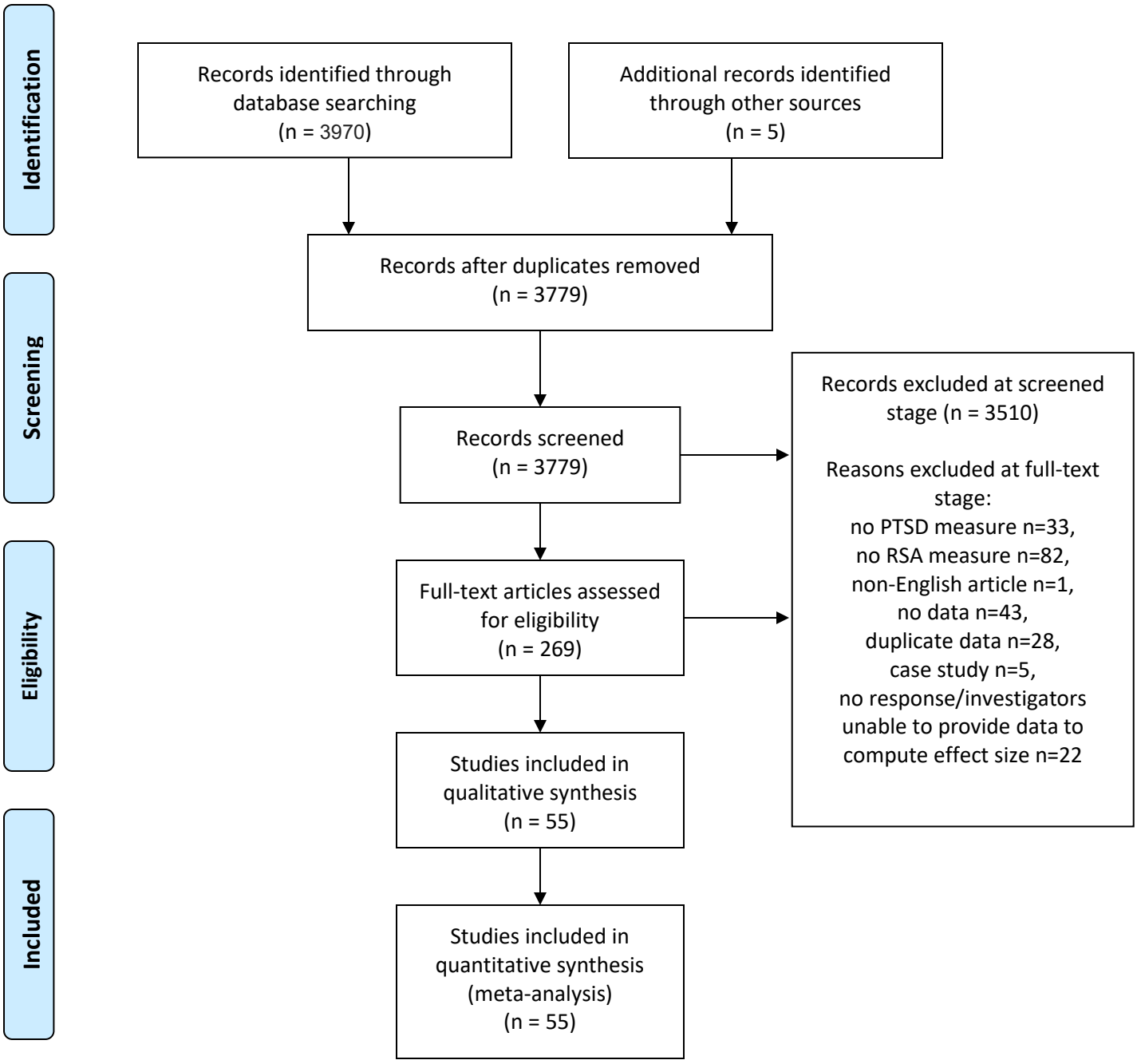
Appendix A. Supplementary data

Supplemental Figure. Article inclusion PRISMA flow diagram



Supplemental Table. Characteristics of studies included in meta-analysis

First Author	Year	Sample Size	Categorical vs Continuous	Number of Control Groups	Average Age	Percent Female	Trauma Type	Control Group(s)	RSA Measure	PTSD Measure	Publication Status	Effect Size
Agorastos	2013	15	Categorical	1	28.7	0	Interpersonal	Trauma-Exposed Control	RMSSD, SDNN	Clinical Interview	Published	-0.15
Cohen	2000	64	Categorical	2	34.14	60	Mixed	Healthy Control, Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.05
D'Andrea	2012	20	Correlational	0	38	100	Mixed	Not Applicable	Peak-to-Valley	Self-Report	Published	-0.54
Dennis	2014	227	Categorical	1	29.32	57	Mixed	Not Specified	HF-HRV, SDNN	Clinical Interview	Published	-0.36
DePierro	2015	55	Correlational	0	35.08	63	Interpersonal	Not Applicable	HF-HRV	Self-Report	Unpublished	-0.41
Eonta	2013	218	Correlational	0	21.5	72	Mixed	Not Applicable	Peak-to-Valley	Self-Report	Unpublished	-0.20
Barry	2015	147	Correlational	0	19.02	53	Mixed	Not Applicable	Peak-to-Valley	Self-Report	Published	-0.12
Hauschildt	2011	44	Categorical	1	35.11	75	Interpersonal	Healthy Control	HF-HRV, RMSSD	Clinical Interview	Published	-0.49
Jovanovic	2009	78	Categorical	1	39.38	0	Interpersonal	Healthy Control	HF-HRV	Clinical Interview	Published	-0.82
Kamkwalala	2012	141	Categorical	1	39.80	61	Mixed	Trauma Exposed Control	HF-HRV	Self-Report	Published	-0.00
Bertram	2014	46	Categorical	1	54.16	0	Mixed	Healthy Control	HF-HRV	Clinical-Interview	Published	0.12
Fainsilber	2015	75	Correlational	0	9.33	51	Not Specified	Not Applicable	HF-HRV	Self-Report	Published	0.24
Keary	2009	40	Categorical	1	37.75	100	Mixed	Healthy Control	HF-HRV	Clinical Interview	Published	0.43
Kirsch	2015	34	Categorical	1	12.90	63	Mixed	Trauma Exposed	HF-HRV	Clinical Interview	Published	-0.32

Kobayashi	2014	37	Categorical	1	23.05	68	Mixed	Control Trauma Exposed Control	HF-HRV	Clinical Interview	Published	-0.98
Lakusic	2007	68	Categorical	1	49.0	0	Interpersonal	Healthy Control	HF-HRV, pNN50, SDNN, RMSSD	Clinical Interview	Published	-0.43
Lee	2012	125	Categorical	1	48.22	100	Mixed	Healthy Control	RMSSD, SDNN	Clinical Interview	Published	-0.61
MacArthur	2011	61	Correlational	0	15.70	44.30	Mixed	Not Applicable	RMSSD, SDNN	Self-Report	Unpublished	0.26
Minassian	2014	2430	Categorical	2	22.80	0	Interpersonal	Healthy Control, Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.22
Moon	2013	51	Categorical	1	37.21	62.00	Mixed	Healthy Control	HF-HRV, RMSSD, SDNN	Clinical Interview	Published	-0.73
Murray	2012	87	Categorical	1	21.70	0	Not Specified	Healthy Control	HF-HRV, RMSSD, SDNN	Clinical Interview	Unpublished	0.06
Muse	2013	66	Correlational	0	36.02	0	Not Specified	Not Applicable	SDNN	Self-Report	Unpublished	-0.06
Blechert	2007	81	Categorical	2	41.92	71.62	Mixed	Healthy Control, Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.80
Sahar	2001	29	Categorical	1	41.75	0	Mixed	Trauma Exposed Control	Porges' Method	Clinical Interview	Published	-0.06
Schechter	2013	54	Categorical	2	30.68	100	Interpersonal	Trauma Exposed Control, Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.17
Brady	2015	115	Correlational	0	32.90	9.70	Interpersonal	Not Applicable	HF-HRV, SDNN	Clinical Interview	Published	-0.18

Shah	2013	459	Categorical	2	55.15	0	Interpersonal	Healthy Control, Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.36
Shaikh al arab	2012	21	Categorical	1	24.50	Not Specified	Traffic Accident	Trauma Exposed Control	RMSSD	Clinical Interview	Published	0.05
Shenk	2012	110	Correlational	0	17.00	100	Interpersonal	Not Applicable	Porges' Method	Clinical Interview	Published	0.22
Slewa-Younan	2012	25	Categorical	0	Not Specified	48.12	Mixed	Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.52
Song	2011	24	Categorical	1	50.20	71.90	Mixed	Trauma Exposed Control	HF-HRV, RMSSD, SDNN	Self-Report	Published	0.30
Tan	2009	28	Correlational	0	31.68	7.1	Interpersonal	Not Applicable	SDNN	Clinical Interview	Published	-0.39
Tan	2011	30	Categorical	1	38.8	10	Interpersonal	Healthy Control	SDNN	Clinical Interview	Published	-1.84
Tucker	2012	64	Categorical	2	37.6	Not Specified	Hurricane Katrina	Healthy Control, Trauma Exposed Control	HF-HRV	Clinical Interview	Published	0.54
Buss	2011	62	Correlational	0	38.19	64.5	Mixed	Not Applicable	SDNN	Clinical Interview	Unpublished	-0.06
Wahbeh	2013	81	Categorical	1	53.90	0	Interpersonal	Trauma Exposed Control	HF-HRV	Clinical Interview	Published	0.00
Wahbeh	2013	45	Categorical	2	Not Specified	Not Specified	Interpersonal	Healthy Control, Trauma Exposed Control	HF-HRV	Clinical Interview	Published	0.04
Woodward	2008	77	Categorical	1	49.00	8.0	Interpersonal	Trauma Exposed Control	HF-HRV	Clinical Interview	Published	-0.35

Woodward	2009	48	Categorical	2	42.00	Not Specified	Mixed	Healthy Control, Other Mental Health Control	HF-HRV	Clinical Interview	Published	-0.70
Liddell	2016	80	Correlational	0	39.00	62.00	Mixed	Not Applicable	RMSSD	Self-Report	Published	-0.38
McLaughlin	2013	168	Correlational	0	14.90	56.00	Mixed	Not Applicable	HF-HRV	Self-Report	Published	-0.06
Meyer	2016	68	Categorical	2	31.29	Not Specified	Mixed	Healthy Control, Other Mental Health Control	HF-HRV, pNN50, RMSSD, SDNN	Clinical Interview	Published	-0.75
Kobayashi	2016	71	Categorical	1	22.75	59.49	Mixed	Trauma Exposed Control	HF-HRV	Clinical Interview	Published	0.26
Wisco	2015	10	Categorical	1	29.00	80.00	Mixed	Trauma Exposed Control	HF-HRV	Clinical Interview	Unpublished	-0.46
Wisco	2015	56	Correlational	0	54.04	3.60	Mixed	Not Applicable	HF-HRV	Clinical Interview	Unpublished	-0.31
Liverant	2016	79	Categorical	1	51.17	12.50	Mixed	Other Mental Health Control	HF-HRV	Clinical Interview	Unpublished	0.10
Chang	2013	256	Categorical	2	36.79	41.00	Mixed	Healthy Control, Trauma Exposed Control	HF-HRV	Self-Report	Published	-0.68
Park	2017	141	Categorical	1	63.70	8.50	Mixed	Trauma Exposed Control	SDNN, RMSSD	Self-Report	Published	-0.62
Arditi-Babchuk	2009	40	Correlational	0	25.00	100	Mixed	Not Applicable	Porges' Method	Self-Report	Published	-0.14
								Healthy				

Van Male	2000	30	Categorical	2	18.50	100	Interpersonal	Control, Other Mental Health Control	HF-HRV	Clinical Interview	Unpublished	0.20
Shoji	2012	49	Correlational	0	22.49	87.80	Mixed	Not Applicable	Peak-to-Valley	Self-Report	Unpublished	-0.16
Clausen	2016	24	Correlational	0	32.75	0	Interpersonal	Not Applicable	HF-HRV	Clinical Interview	Published	-0.87
Tegeler	2017	12	Correlational	0	47.99	58.00	Mixed	Not Applicable	SDNN	Self-Report	Published	-0.32
Chu	2009	88	Correlational	0	30.70	100	Interpersonal	Not Applicable	HF-HRV	Self-Report	Unpublished	-0.09
Norte	2013	35	Categorical	1	40.67	45.55	Mixed	Trauma Exposed Control	RMSSD	Clinical Interview	Published	-0.78

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