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Emotion suppression and acute physiological responses to stress in healthy populations: a quantitative review of experimental and correlational investigations

Alexandra T. Tyra,
Thomas A. Fergus,
Annie T. Ginty
Department of Psychology and Neuroscience, Baylor University, Waco, TX, USA

Abstract

Emotion suppression may be linked to poor health outcomes through elevated stress-related physiology. The current meta-analyses investigate the magnitude of the association between suppression and physiological responses to active psychological stress tasks administered in the laboratory. Relevant articles were identified through Medline, PsychINFO, PubMed, and ProQuest. Studies were eligible if they (a) used a sample of healthy, human subjects; (b) assessed physiology during a resting baseline and active psychological stress task; and (c) measured self-report or experimentally manipulated suppression. Twenty-four studies were identified and grouped within two separate random effects meta-analyses based on study methodology, namely, manipulated suppression ($k = 12$) and/or self-report ($k = 14$). Experimentally manipulated suppression was associated with greater physiological stress reactivity compared to controls ($H_g = 0.20$, 95% CI [0.08, 0.33]), primarily driven by cardiac, hemodynamic, and neuroendocrine parameters. Self-report trait suppression was not associated with overall physiological stress reactivity but was associated with greater neuroendocrine reactivity ($r = 0.08$, 95% CI [0.01, 0.14]). Significant moderator variables were identified (i.e., type/duration of stress task, nature of control instructions, type of physiology, and gender). This review suggests that suppression may exacerbate stress-induced physiological arousal; however, this may differ based upon the chosen methodological assessment of suppression.

Keywords

Emotion regulation; suppression; psychological stress; physiological stress reactivity; meta-analysis

Emotions play a critical role in mental and physical health outcomes, particularly cardiovascular health (Appleton & Kubzansky, 2014). Previous reviews have established

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CONTACT Alexandra T. Tyra, Alexandra_Tyra1@baylor.edu.

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negative emotions to be detrimental to cardiovascular health (Chida & Steptoe, 2009; Matthews, 2013; Roest et al., 2010; Suls, 2018; Suls & Bunde, 2005), whereas positive emotions may be protective (Boehm & Kubzansky, 2012; DuPont et al., 2020). In fact, it is suggested that the tendency to experience negative emotion (e.g., anger, depression, anxiety) poses proportionate levels of health risk as compared to conventional factors (e.g., smoking, physical inactivity, inadequate diet, obesity; Rozanski, 2014). However, evidence from emotion regulation research suggests that risk may not solely be based on the existence of a negative emotion, but also the inability to effectively regulate that emotion (Appleton & Kubzansky, 2014; Haukkala et al., 2010).

Although many definitions of emotion regulation have been put forward, one widely accepted definition posits that emotion regulation is the modification of which emotion is being experienced, when it manifests, and how it is expressed (Gross, 1998b; 2014). It involves a combination of heritable processes and the use of strategies that are learned through socialisation and experience over time, particularly during childhood; thus, it is not entirely an ‘inborn trait’, but also a developed pattern of regulatory tendencies (Appleton et al., 2014). This perspective is supported by the sociogenomic approach to personality traits, which suggests that a trait is an identifiable pattern of repeated moment-to-moment behavioural states, and as such, could be modified through behavioural interventions to improve health and well-being (Roberts et al., 2017). While there are many different types of emotion regulation strategies, empirical findings suggest that the habitual use of emotional suppression may be a risk factor for later cardiovascular disease (CVD; Appleton et al., 2013; Appleton et al., 2014; Appleton & Kubzansky, 2014; Mauss & Gross, 2004). As such, the current review will focus primarily on identifying a possible link that connects emotional suppression to CVD endpoints.

The process model of emotion regulation

The process model of emotion regulation (Gross, 1998b; 2015) identifies five families of emotion regulation processes that occur in temporal order along the continuum of emotion generation: situation selection (i.e., directly seeking out or avoiding situations that may lead to certain emotions), situation modification (i.e., altering a situation to change its emotional impact), attentional deployment (i.e., distraction), cognitive change (i.e., adjusting how one thinks about a situation or one’s capacity to handle it), and response modulation (i.e., attempts to decrease or suppress the experiential, physiological, and behavioural aspects of an ongoing emotion; Gross, 2014; 2015; see Figure 1). These five processes can be grouped based on whether they are employed before (antecedent focused) or after (response focused) the emotional response is fully developed (Gross, 1998a; 2015; Gross & John, 2003).

It is argued that strategies employed earlier in the emotion generative process, such as reappraisal, are more effective than strategies employed later in the process, such as suppression (Gross, 2001). According to the process model, suppression involves the conscious inhibition of emotionally expressive behaviour *after* becoming emotionally aroused (Gross, 1998a; Gross & Levenson, 1993; 1997). While there are some contexts where the use of suppression could be considered beneficial (Bonanno & Keltner, 1997;

Mesquita et al., 2014), regular use of this type of response-focused strategy is often regarded as maladaptive (Gross, 2014).

Effects of suppression

A greater propensity to use suppression is associated with decreased positive emotions and increased negative emotions (e.g., Gross, 1998a; Gross & John, 2003; Gross & Levenson, 1997; Moore et al., 2008), poorer memory (e.g., Richards & Gross, 2000), and worse social relationships (e.g., Butler et al., 2003; English et al., 2013). Additionally, a one standard deviation increase in use of suppression significantly associates with a 22% increase in levels of circulating C-reactive protein (CRP), which is an inflammatory risk marker of CVD (Appleton et al., 2013), and a 10% increase in estimated likelihood of developing CVD in 10 years (Appleton et al., 2014). To understand how emotional suppression can have so many varying consequences, one must appreciate the fact that emotions are multifaceted phenomena capable of influencing changes in the domains of subjective experience, behavioural activation, and physiological responding (Gross, 2014; 2015; Mauss et al., 2005). It would thus stand to reason that the inhibition of emotions would have significant repercussions in each of these areas.

With respect to physiology, it has long been thought that the concealment of emotion can have detrimental bodily consequences. In psychodynamic theory, this concept was described as ‘strangulated affect,’ with emphasis placed on the importance of cathartic release (Freud, 1923/1961). Early theorists in the field of psychosomatic medicine believed that the inhibition of emotional expression could lead to increased risk of somatic illnesses, such as hypertension (Alexander, 1939; Menninger, 1938), asthma (Alexander, 1950; Halliday, 1937), and ulcers (Alexander, 1950). While many of these early theories are no longer supported due to methodological concerns[1], it is still widely believed that emotional suppression is linked to poor health outcomes, and that this may be facilitated through increased physiological activation (Gross, 1998b; Traue et al., 2016). For instance, prior laboratory-based research has found that participants who were instructed to suppress the expression of various film-induced emotional experiences (e.g., disgust, sadness, amusement) displayed greater sympathetic nervous system activation compared to control groups (Gross, 1998a; Gross & Levenson, 1993; 1997). This physiological activation was observed despite the fewer metabolic demands of reduced behavioural expression (Gross1 & Levenson, 1997). One possible explanation is that the active effort required to inhibit behavioural expression in the face of an intense emotional experience is both cognitively and physiologically demanding (Appleton & Kubzansky, 2014; Butler et al., 2003; Johns et al., 2008; Richards & Gross, 2000; 2006). If an individual is consistently inhibiting behavioural expression, this cognitive exertion and associated patterning of physiology could be considered a type of chronic stress exposure and may eventuate in pathophysiology over time (Appleton & Kubzansky, 2014; Consedine et al., 2002; Mauss et al., 2006; Pennebaker & Beall, 1986).

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[1] F. Alexander’s anger and hypertension theory is still popularly supported through later 20th century research, revealing that inhibition of anger and hostility may indeed be linked to hypertension and coronary heart disease (Brosschot & Thayer, 1998; Friedman & Booth-Kewley, 1987; Smith, 1992).

Physiological stress reactivity

This is in line with the well-established stress reactivity hypothesis, which states that individuals who demonstrate exaggerated physiology (i.e., cardiovascular, neuroendocrine) in response to psychological stress are at a greater risk of developing poor health outcomes, such as CVD (Carroll et al., 2009; Obrist, 1981; Phillips & Hughes, 2011; Treiber et al., 2003; Turner et al., 2020). Exaggerated stress reactivity can be identified as metabolically disproportionate increase in heart rate and blood pressure levels, increase in cortisol release, and decrease in heart rate variability for a given context (Sapolsky et al., 2000). Indeed, a robust body of literature has demonstrated exaggerated physiological responses to laboratory stress tasks, above and beyond what would be expected given the minimal metabolic demand of these mental tasks (Balanos et al., 2010; Carroll et al., 1986a; 1986b; 2009; Lambiase et al., 2012; Sherwood et al., 1986; Turner & Carroll, 1985). Exaggerated physiological responses to psychological stress are predictive of risk factors for CVD, such as atherosclerosis (Barnett et al., 1997; Everson et al., 1997; Jennings et al., 2004; Matthews et al., 1998), hypertension (Carroll et al., 2011; Chida & Steptoe, 2010; Gerin et al., 2000; Markovitz et al., 1998), increased left ventricular mass (Allen et al., 1997; Georgiades et al., 1997; Kapuku et al., 1999), and CVD mortality (Carroll et al., 2012).

Suppression and physiological stress reactivity

Stressful experiences also induce negative emotions, and how individuals chose to regulate these negative emotions will impact the above-mentioned physiological changes (Gross, 2014). Thus, the possible consequences of experiencing a stressful event may be further compounded by the additional stress of suppressing one’s emotional response to it. Researchers have attempted to examine this relationship using a straightforward laboratory paradigm, in which participants are instructed to suppress their emotional responses during an acute psychological stress task (e.g., Jentsch & Wolf, 2020; Quartana & Burns, 2010). Physiological measurements (e.g., blood pressure, heart rate, cortisol) are recorded during the task and then compared to an uninstructed control group. Other researchers have taken a different methodological approach by examining the correlation between self-report suppression and physiological stress responses (e.g., Herhaus et al., 2020; Lam et al., 2009). So far, regardless of the approach, the findings have been relatively mixed and inconclusive, with some studies reporting no relationship between suppression and stress reactivity (e.g., Richardson et al., 2014; Shapero et al., 2016), other studies reporting a positive relationship (e.g., Jentsch & Wolf, 2020; Lam et al., 2009; Quartana & Burns, 2010), and others reporting a negative relationship (e.g., Cundiff et al., 2019; Yuan et al., 2014).

A recent meta-analysis supports this inconsistency of results, such that during various emotion-inducing laboratory tasks, suppression was found to be significantly related to decreased responses on some, but not all, physiological parameters during passive stress tasks (e.g., sitting and watching a film, viewing pictures; Zaehringer et al., 2020). To our knowledge, no one has yet attempted to systematically quantify the relationship between emotion suppression and physiological reactivity to only active laboratory stress tasks (i.e., mental arithmetic, speech delivery under social evaluative threat), nor has anyone attempted to examine whether this relationship differs across study methodologies (i.e., self-report versus experimental manipulation). Given the robust link between individual differences in acute stress responses to active stress tasks and later CVD outcomes, this is a highly important area of research that could inform possible routes of intervention.

It is also important to note that there are substantial discrepancies in how suppression is defined across the literature (for review see Webb et al., 2012). Unlike Gross and colleagues, some researchers have defined it as conscious inhibition of the *experience* of emotion itself (Quartana & Burns, 2007), while others define it as control over *thoughts* regarding an emotion-eliciting event (Burns, 2006; Dalgleish & Yiend, 2006). In some instances, the definition is mixed, including instructions to suppress both emotional expression and experience (Braams et al., 2012; Vohs & Schmeichel, 2003). Prior research has drawn attention to possible distinctive differences between the various forms of suppression and physiological responses to stress (Quartana & Burns, 2010). For instance, while *expressive* suppression is believed to require cognitive and physiological exertion, *experiential* suppression may not require such effort (Quartana & Burns, 2007; 2010). It is for these reasons that the current review adopts Webb and colleagues (2012) taxonomy of suppression, which includes the following four subtypes: 1) suppress the expression of emotion, 2) suppress the experience of emotion, 3) suppress thoughts regarding the emotion-eliciting event, and 4) suppress both emotional experience and expression. This taxonomy has been validated and used in other emotion regulation reviews (see Zaehringer et al., 2020).

Study aims

The objective of the present review was to consolidate and quantify the findings of all available existing laboratory studies regarding the association between emotion suppression and physiological reactions (e.g., cardiovascular; neuroendocrine) to *active* laboratory tasks designed to induce psychological stress. In doing so, we provide novel and comprehensive understanding of a possible pathway between use of suppression as an emotion regulation strategy and cardiovascular disease outcomes. Research has consistently highlighted the importance of investigating mechanisms that link psychological constructs (e.g., suppression) to biomarkers of disease, so that clinicians may develop more precise and effective health interventions. Laboratory paradigms are advantageous for examining acute physiological stress reactivity, as they allow for precise experimental control and manipulation of conditions, such as the type of stress task employed, the duration of the task, and multiple physiological measurements before (i.e., baseline) and during the task (i.e., reactivity). This review is strengthened by the fact that differences in study methodologies were examined in two separate meta-analyses to further delineate their effects on outcomes, namely self-report suppression versus experimentally manipulated suppression. Additional moderation analyses were conducted to examine the effects of various demographic characteristics, stress task type and duration, study quality, and when applicable, type of suppression manipulation and type of control instructions. Lastly, publication bias was assessed to recognise the potential implications of unpublished null results.

Method

Literature search

All procedures and results are reported in line with the guidelines contained in the PRISMA Statement (Moher et al., 2009). Potentially relevant studies were identified through a systematic literature search using three online databases (Medline, PsycINFO, PubMed). All database searches were performed on November 4th, 2020. An additional database search was conducted on May 27th, 2021, using ProQuest Dissertation and Theses in efforts to minimise publication bias. The search strategy consisted of carefully selected key words and Boolean logic to maximise sensitivity of article identification. Publication date was unrestricted. The exact search terms can be found in the supplementary materials. All database search results were exported and uploaded to Rayyan QCRI (https://rayyan.qcri.org; Ouzzani et al., 2016), which is a web application for systematic reviews. Rayyan QCRI displays the title and abstract of each uploaded study and provides a detailed record of duplicate removals and inclusion/exclusion rationale during the initial screening process. In addition, the reference lists from all eligible studies as well as prior systematic reviews were thoroughly screened for titles that may have been previously overlooked.

Eligibility criteria

Identified studies were assessed based on the following eligibility criteria: (a) written in English and peer-reviewed; (b) used a sample of healthy, human subjects of any age range without any known medical or psychiatric conditions; (c) included an acute laboratory psychological stress paradigm that adhered to the requirements specified below; (d) included a true, resting baseline period administered prior to the stress exposure; (e) measured either self-report (i.e., questionnaire) or experimentally manipulated suppression (can include either experiential or expressive suppression instructions); (f) if suppression was experimentally manipulated, then also included a control group (e.g., between-groups design with a no-suppression group); and (g) collected physiological data during both the baseline period and acute psychological stress task (i.e., reactivity).

Definitions for the term ‘acute psychological stress’ tend to vary significantly across the literature (Chida & Steptoe, 2010). As such, we restricted our definition of acute psychological stress to an active, but metabolically undemanding psychological task that is time-limited and performed under controlled conditions in a laboratory environment (Brindle et al., 2014). This includes active cognitive stressors (i.e., unsolvable puzzles, mental arithmetic), active social stressors (i.e., dyadic interactions, preparing and presenting a speech to a panel of judges), or active emotional stressors (i.e., recall of an emotional event). We excluded physical laboratory tasks that have little to no psychological component (i.e., physical exercise). However, the cold pressor was still included because it is reported to evoke a psychological response along with the physical components (Lovallo, 1975). Passive emotion induction tasks (i.e., film-viewing, picture viewing) were also excluded, given that active stress tasks are found to engage the sympathetic nervous system to a stronger degree than passively sitting and watching a monitor (e.g., Fechir et al., 2008). Startle probe tasks (i.e., auditory startle tones) were also excluded on the basis that the startle response is primarily driven by reflex circuits in the brainstem and does not evoke active appraisal of psychological stimuli via activation in forebrain regions (Gianaros et al., 2017; Ginty et al., 2017; Richter et al., 2011; Wager et al., 2009).

Due to a wide range of constructs that could arguably fit under the description ‘emotion suppression’, we narrowed our search criteria to only include studies that defined emotion suppression in a way that was consistent with the taxonomy established by Webb and colleagues’ (2012). Studies were included if suppression was defined according to one of the following four subtypes: 1) suppress the expression of emotion, 2) suppress the experience of emotion, 3) suppress thoughts regarding the emotion-eliciting event, or 4) suppress both emotional experience and expression. Experimental manipulations of suppression should include scripted instructions that closely resemble the procedures laid out by Gross and colleagues in prior studies (Gross, 1998a; Gross & Levenson, 1997; John & Gross, 2004; Richards & Gross, 2000). For example, manipulated *expressive* suppression instructions should include language that closely resembles the following script, ‘… if you have any feelings, try your best not to let those feelings show. In other words, try to behave in such a way that a person watching you would not know you were feeling anything’. Alternatively, manipulated *experiential* suppression instructions should include language that resembles a separate script, ‘… it is extremely important that you suppress all of your feelings. That is, do not think about any of your feelings at all; push them out of your mind’ (Quartana & Burns, 2010). Self-report assessments of suppression should include reporting of instrument validation and reliability. Similar constructs that were either too broad or inconsistently defined across studies (i.e., effortful control, anger-in, repression, internalising problems) as well as articles that incorrectly included the use of alternative regulation strategies in the definition of emotion suppression (i.e., reappraisal, avoidance, distraction) were not included in the present study (see Zaehringer et al., 2020).

Data collection

Approximately 7,001 records were identified through database searches. After all duplicates were removed ($n = 660$ removed), the first author and last author independently screened the remaining titles and abstracts for eligibility based on the criteria described above. This was completed using the blind review option in Rayyan QCRI, in which the decisions and labels of collaborators are not visible to each other. Afterwards, the two authors consulted with each other to discuss any disagreements for article eligibility. The first author further identified 178 potential records through manual inspection of relevant article reference sections. After the initial title and abstract screening, 6,322 records were removed due to ineligibility, resulting in the full-text qualitative assessment of 197 remaining articles. If articles appeared to be eligible but did not publish the necessary data of interest, the study’s corresponding author was contacted via email for access to the unpublished data ($n = 14$ were contacted, but only $n = 7$ responded). A final total of 26 studies met the criteria for inclusion in the current review and meta-analyses (see Figure 2 for a PRISMA flow diagram; Moher et al., 2009).

Data extraction and coding

Prior to data extraction, the proposed meta-analysis was registered on PROSPERO on April 4th, 2021 (Registration number: CRD42021240920;https://www.crd.york.ac.uk/prospero/).

All data were extracted and coded by the first author and checked twice for accuracy. The data were manually extracted from published data and email communications using DistillerSR (Evidence Partners, Ottawa, Canada), which is a web-based systematic review software. Online coding forms were customised and piloted prior to beginning data extraction. The following variables were extracted: article reference, country where study was conducted, study design, sample size, age (*M, SD*), sex (% female), race (% white), type of laboratory stress task (including duration), type of self-report measurement or experimental manipulation instructions for suppression, type of control instructions, type of physiology assessed during baseline and active phases of the stress task, and any other covariates included in the original study analyses. For correlational studies, data were extracted in the form of correlation coefficients. For between groups studies, data were extracted either in the form of means/standard deviations from baseline and stress, or reactivity change scores (stress – baseline). Some of the data were only presented in figures; in these cases, means and standard deviations were extracted using an open-source Web plot digitiser (version 4.4; https://automeris.io/WebPlotDigitizer), which is specifically designed to extract underlying numerical data from published figures.

Data were coded such that larger, positive values indicated higher use of suppression and greater physiological stress reactivity. Most physiological markers are already positively correlated with sympathetic activation, such that higher values indicate greater sympathetic arousal. However, some parameters of physiology associated with sympathetic engagement, such as a shorter pre-ejection period (PEP) and interbeat interval (IBI), are negatively correlated with sympathetic outflow (e.g., lower values indicate greater sympathetic activity). And although the sympathetic and parasympathetic arms of the autonomic nervous system are not regulated along a continuum of reciprocal control (i.e., they can be uncoupled or coactivated; Berntson et al., 1994), putative markers of cardiac parasympathetic activity (e.g., respiratory sinus arrythmia [RSA] and high-frequency heart rate variability [HF-HRV]) are often decreased by laboratory stressors, sometimes in conjunction with increased sympathetic engagement depending on the particular stressor (Brindle et al., 2014). Accordingly, to aid in interpretation and comparability with other outcomes, these markers were reverse coded (multiplied by – 1; DuPont et al., 2020). Thus, a negative effect size for these markers indicates *less* sympathetic arousal/parasympathetic withdrawal during stress exposure. For completeness of reporting and owing to the possibility of qualitative variation in response patterning across parameters of autonomic physiology, we also present supplemental sensitivity tests examining the influence of task type and measure. Data were separated into two broad categories for analysis: experimental manipulations of suppression (e.g., between-groups studies) and self-report measures of suppression (e.g., correlational studies). All data are publicly available online (https://osf.io/aw8h4/).

Quality assessment

The first author assessed the quality of each study. The NHLBI quality assessment tool for observational cohort and cross-sectional studies was used as a starting template, which includes a series of questions designed to direct focus to key concepts for evaluating the internal validity of a study as good, fair, or poor (https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools). However, this checklist was originally designed to assess the quality of epidemiological research. As such, we used a previously adapted version of the checklist specifically customised for smaller-scale, laboratory paradigms (DuPont et al., 2020). We further modified the checklist to meet the quality assessment needs of the current review (see Table S1). This tool was not designed to be a checklist that gets tallied up to provide a quality ‘score’ for each study, but rather it was originally designed to simply assist with the critical appraisal of each individual study. As such, scores were not awarded to each study. Instead, studies received an overall rating of good, fair, or poor based on the perceived risk of bias. It should be noted that quality ratings were not considered during inclusion decisions.

Statistical analyses

Analyses were conducted separately across two broad categories of study design (either experimental manipulation or self-report suppression). For between-groups experimental manipulation studies (no within-group studies were eligible), effect sizes were calculated from either reported reactivity change scores (stress-baseline) or from the means and standard deviations of baseline and stress phases for both suppression manipulation and control groups. Hedge’s g was chosen as the effect size measure in the between-groups meta-analyses, as it corrects for small sample sizes and is thus a more conservative measure of effect size than Cohen’s d (Borenstein et al., 2011). We were unable to obtain exact group sample sizes for two studies; in this case, equal group sample sizes were assumed by taking the total sample size and dividing it by the number of groups/conditions in the study.

For correlational self-report studies, reported correlation coefficients (r) served as the effect size index. For the most part, correlation coefficients were obtained directly from the articles or via email communication. However, for a few studies, effects sizes were estimated from either standardised beta coefficients or p-values. Standardised beta coefficients were converted to Pearson’s r using the formula $r = \beta * 0.98 + 0.05\lambda$, with λ equal to 1 if the coefficient was positive and 0 if the coefficient was negative (Peterson & Brown, 2005). This method was chosen because standardised beta coefficients and Pearson’s r values are robustly correlated when Pearson’s r is between the range of – 0.50–0.50 (Peterson & Brown, 2005), as was the case with all correlations reported in this meta-analysis. As recommended by Lipsey and Wilson (2001), studies that reported null findings, but no supporting statistics were still represented in the analyses with a conservative zero correlation. All correlations were then individually transformed into Fisher’s z, with variance and confidence intervals calculated for each study’s effect size. The summary effect sizes were transformed back into correlations (r) for ease of interpretation. Studies differed in their measurement of trait (i.e., in general; habitual) or state suppression (i.e., during the task). As such, two separate quantitative and qualitative analyses were performed, one examining self-report trait suppression and the other examining self-report state suppression. This method helped ensure independence of samples within each analysis.

Random effects modelling was employed in the present analyses because it was expected that studies would not share a common effect size due to variation across sample characteristics and study design (Borenstein et al., 2011). To ensure independence of samples when calculating the summary effect sizes, if a study included multiple assessments of an outcome across more than one stress task or examined various physiological outcomes, we aggregated across stress tasks and physiological outcomes such that only the mean of the multiple effect sizes was used (Chida & Hamer, 2008). The summary effect size was calculated by weighting studies using the inverse variance method (Lipsey & Wilson, 2001), such that studies with more precision (small variance) have greater weight than studies with less precision (greater variance). Subsequent subgroup meta-analyses were performed for each type of physiological outcome. Physiological outcomes were grouped into four overarching categories: miscellaneous autonomic (e.g., pre-ejection period, indices of heart rate variability, skin conductance response, salivary alpha-amylase), cardiac (e.g., heart rate, cardiac output, interbeat interval), hemodynamic (e.g., systolic blood pressure, diastolic blood pressure, mean arterial pressure, total peripheral resistance, stroke volume) and neuroendocrine (e.g., cortisol). Heterogeneity in study effects was assessed by calculating the Q-statistic ($p < .05$ means significant heterogeneity), along with the Higgin’s I^2 statistic, which provides a percentage of variance attributable to true heterogeneity in effect sizes across the studies. Unlike our calculations of the summary effect size, which required the aggregation of multiple reported effects within a single study, each reported effect size was treated as independent when conducting tests of heterogeneity. If significant heterogeneity was observed in the separate meta-analyses, random effects meta-analytic regression moderator analyses were employed to examine whether other variables may be moderating the strength of the observed effects. Age, race, sex, baseline physiology, type of stress task, duration of stress task, and quality assessment were included as potential moderators. In the correlational analyses, specific method of calculating reactivity (e.g., delta change scores, residualized change score, area under the curve, etc.) was examined when applicable. In the between-groups analyses, type of suppression manipulation and type of control instructions were also examined when applicable (see Table S2 for coding of specific instructions).

Publication bias was assessed using Egger’s unweighted regression asymmetry test (Egger et al., 1997) as well as Rosenthal’s fail-safe N (Rosenberg, 2005; Rosenthal, 1979). If publication bias is low, the Eggers asymmetry test will provide a p-value that is greater than 0.05. A funnel plot was used to graphically depict the distribution of studies around the summary effect size; greater symmetry reveals lower risk of publication bias. Rosenthal’s fail-safe N provides an estimate of how many nonsignificant studies would need to be included for the results of the meta-analyses to be nullified. As such, a small Fail-safe N reveals greater concern for publication bias compared to a larger number.

All statistical analyses were conducted using the metaphor package from R (version 2.4; Viechtbauer, 2010) and SPSS version 27 (IBM Corp, USA). Results were reported as statistically significant if p values were $\leq .05$.

Results

Descriptive analyses

This systematic review included a total of 26 studies (see Figure 2). Twelve studies examined self-report trait (i.e., habitual) suppression, with three of these studies using only child/adolescent samples. Ten between-groups studies examined experimental manipulation of suppression compared to an unmanipulated control group. Two studies examined both trait self-report and experimental manipulation of suppression. Given so few studies examined self-report state measures of suppression ($n = 4$), these were omitted from the quantitative meta-analyses. However, a qualitative review of these studies can be found in the online supplementary materials. As such, a final total of 24 studies were included in the meta-analyses, which were further broken down into 62 individual samples based on multiple reported samples, measures, or outcomes (see Table 1). On average, studies reported sample sizes that were larger than 100 participants; however, sample sizes varied from small ($n = 26$) to large ($n = 307$). Across all studies, a grand total of 3,254 participants were examined in the current analyses.

All self-report studies assessed the habitual use of expressive suppression ($n = 14$). Expressive suppression was also the most frequently utilised manipulation in the experimental studies ($n = 9$), with two studies manipulating both expressive and experiential suppression, and only one study manipulating thought suppression.[2] Cortisol was the most frequently reported physiological outcome in studies examining self-report suppression, whereas heart rate was the most frequently reported outcome in studies examining experimental manipulation of suppression (see Table 1). Dyadic interactions were the most common stress task in manipulation studies, followed by mental arithmetic tasks. In contrast, the Trier Social Stress Test (TSST) was the most cited stress task in self-report studies. Average stress task duration was between 8–11 min. Almost all studies provided sample age and sex, with most studies reporting on young adult samples. Fewer studies reported data on race/ethnicity. Refer to Table 1 for a summary of all samples included in this meta-analysis.

Experimental manipulation of suppression

Overall physiological reactivity: Experimental manipulation of suppression was significantly associated with greater overall physiological reactivity to acute laboratory stressors when compared to control groups, $H_g = 0.20$, 95% CI [0.08, 0.33], $p = 0.002$ (Figure 3). There was significant heterogeneity, such that 62.1% of the detected variation in effect sizes could be attributed to true differences in the effect sizes (Table 2). As such, further analyses were performed to examine each category of physiological outcome separately.

Autonomic reactivity (Misc.): Manipulation of suppression was not significantly associated with overall miscellaneous autonomic reactivity to acute stress when compared to control groups, $H_g = -0.20$, 95% CI [−0.54, 0.15], $p = 0.26$ (Figure 3). However, heterogeneity was still observed, such that 65.89% of variance could be attributed to true differences in effect sizes (Table 2). Upon closer qualitative inspection, it was observed that Yuan et al. (2014) examined the effects of manipulated suppression on skin conductance response in an exclusively Chinese sample. This is important to note, as research has indicated that the consequences of suppression may be moderated by cultural context (e.g., Butler et al., 2007; Mauss & Butler, 2010; Soto et al., 2011). For example, emotion

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[2] This lack of heterogeneity in the types of suppression assessed prevented any follow-up moderation analyses in that regard.

suppression may have negative consequences in people from American/European cultures that value individualistic expression, but these adverse effects may be attenuated in people from East-Asian cultures who value emotional restraint as part of their collectivistic lifestyle.

Given that Yuan et al. (2014) was the only study in this review to examine an East-Asian sample, further analyses were performed to investigate whether the outcomes changed when this sample was removed. Interestingly, manipulation of suppression was still not significantly associated with overall miscellaneous autonomic reactivity, $H_g = -0.12$, 95% CI [−0.47, 0.24], $p = 0.52$. However, additional moderation analyses did reveal that studies with a greater percentage of women demonstrated a stronger negative association between manipulation of suppression and miscellaneous autonomic reactivity (Table S4). Measures that typically decrease in response to sympathetic arousal (i.e., pre-ejection period, heart rate variability) were reverse coded to ease interpretation and comparability with other physiological markers, such that a positive effect size would reveal *greater* sympathetic arousal/parasympathetic withdrawal, and a negative effect size would reveal *less* sympathetic arousal/parasympathetic withdrawal. Keeping this in mind, the current findings indicate that samples with only women or a greater percentage of women demonstrated *less* sympathetic arousal/parasympathetic withdrawal compared to controls. It was also found that the results were moderated by type of physiology, such that manipulation of suppression was significantly associated with *less* sympathetic arousal/parasympathetic withdrawal when only examining parameters of heart rate variability (i.e., smaller decrease in HRV; Table S4), but not when examining only pre-ejection period (PEP), $H_g = 0.14$, 95% CI [−0.19, 0.46], $p = 0.41$.

Cardiac reactivity: Manipulation of suppression was significantly associated with overall cardiac reactivity when compared to controls, $H_g = 0.22$, 95% CI [0.01, 0.44], $p = 0.043$ (Figure 3). Significant heterogeneity was still observed, such that 57.7% of variance could be ascribed to true differences in effect size across the studies (Table 2). This heterogeneity was explained by type of stress task and control instructions. Manipulation of suppression was significantly associated with greater cardiac reactivity during cognitive tasks (i.e., mental arithmetic), $H_g = 0.63$, 95% CI [0.38, 0.87], $p < .001$, compared to non-cognitive tasks (e.g., cold pressor, social only), $H_g = -0.02$, 95% CI [−0.18, 0.15], $p = 0.85$ (Table S5). When studies with no control instructions were included only, manipulation of suppression was not associated with cardiac reactivity, $H_g = -0.08$, 95% CI [−0.31, 0.14], $p = 0.46$, nor was suppression associated with cardiac reactivity when studies with instructions to respond naturally were included only, $H_g = 0.06$, 95% CI [−0.18, 0.29], $p = 0.63$ (Table S5). However, suppression manipulation was associated with greater cardiac reactivity when control groups were specifically instructed not to regulate in a specific manner, $H_g = 0.63$, 95% CI [0.38, 0.87], $p < .001$ (Table S5). That said, it should be noted that four samples within a single study, although independent, were primarily driving these moderation results (Quartana & Burns, 2010). This may also explain the observed risk for possible publication bias in these outcomes (Table 3; Figure S1).

Hemodynamic reactivity: A significant association was found between manipulation of suppression and hemodynamic reactivity when compared to control groups, H_g= 0.34, 95% CI [0.19, 0.49], $p < .001$ (Figure 3), such that participants who were instructed to suppress during the laboratory stress tasks demonstrated greater hemodynamic stress reactivity compared to uninstructed controls. This relationship was not heterogeneous (Table 2) with low risk of publication bias (Table 3; Figure S1), thus precluding moderation analyses.

Neuroendocrine reactivity: There was a significant association between manipulation of suppression and HPA-axis reactivity when compared to controls, H_g = 0.33, 95% C1 [0.12, 0.54], $p = .002$ (Figure 3); manipulation of suppression was related to greater HPA-axis reactivity to the laboratory stressors. There was no significant heterogeneity in this association (Table 2), thus eliminating the need for further moderation analyses. Even though a nonsignificant Egger's regression test revealed low risk of publication bias, Rosenthal's Fail-Safe statistic predicted only twelve nonsignificant studies would be needed to nullify these results (Table 3; Figure S1).

Self-report trait measures of suppression

Overall physiological reactivity: Self-report trait suppression was not significantly associated with overall physiological reactivity to acute laboratory stress tasks, $r = 0.03$, 95% CI [−0.04, 0.10], $p = 0.42$ (Figure 4). That said, significant heterogeneity (I^2 = 66.96%; Table 2) justified subsequent examination of separate physiological outcomes.

Autonomic reactivity (Misc.): No significant association was found between self-report trait suppression and miscellaneous autonomic stress reactivity, $r = -0.09$, 95% CI [−0.23, 0.05], $p = 0.22$ (Figure 4). However, significant heterogeneity was still observed, such that 72.78% of variance could be attributed to true differences in effect size across the studies (Table 2). This heterogeneity was not explained by age, sex, or study quality, but it was significantly impacted by stress task duration, such that studies with longer tasks reported a more negative association between trait suppression and autonomic stress reactivity (Table S6). In other words, participants with higher trait suppression demonstrated *less* sympathetic arousal/parasympathetic withdrawal during longer laboratory stress tasks. This is similar to what was reported above for experimental manipulation of suppression with autonomic stress reactivity. However, only five studies were included in these analyses, so these findings should be treated as provisional.

Cardiac reactivity: There was no significant association between self-report trait suppression and cardiac reactivity to acute laboratory stress, r = 0.07, 95% CI [−0.23, 0.35], $p = 0.67$ (Figure 4). There was still significant heterogeneity, with 91.49% of variance resulting from true differences in effect size (Table 2). However, the small number of studies ($n = 3$) in this subgroup precluded moderator analyses. A qualitative review of these studies revealed some interesting differences between the studies (see Figure 4). Shapero et al. (2016) was unique in that it examined a sample of adolescents, unlike the other two studies which examined adult samples only. Cundiff et al. (2019) was unique in that it only examined a sample of males, whereas the other two studies examined mixed samples; however, Cundiff et al. (2019) had a significantly larger sample size compared to the other studies. All three studies employed different types of stress tasks (e.g., TSST, dyadic conversation, anger recall) with varying durations (4 min to 30 min). That said, all three studies were similar in quality assessments. These qualitative observations should be approached with caution but could be directions for future research.

Hemodynamic reactivity: Only one study examined the relationship between trait suppression with hemodynamic stress reactivity (Cundiff et al., 2019). As such, subgroup meta-analyses were not possible to examine with this outcome (see Figure 4).

Neuroendocrine reactivity: Interestingly, there was a significant association between self-report trait suppression and HPA-axis reactivity, $r = 0.08$, 95% CI [0.01, 0.14], $p = 0.02$ (Figure 4), with no significant heterogeneity (Table 2) and no significant risk of publication bias (Table 3; Figure S1). More specifically, greater self-report trait suppression was related to increased cortisol reactivity across 11 studies.

Ancillary sensitivity analyses

Ancillary sensitivity testing of cardiac-autonomic parameters of physiology and associated variation in task types suggested no significant differences in the above reported outcomes. Details regarding these ancillary analyses can be found in the online supplemental materials.

Discussion

Emotion suppression may be a risk factor for adverse health outcomes, such as CVD (e.g., Appleton & Kubzansky, 2014); however, little is known about the underlying mechanisms linking suppression to poor health. One potential mechanism is that suppression may exacerbate the negative effects of psychological stress on health through heightened stress-related physiology. As such, the purpose of this review was to assess whether the prevalent literature supports an association between suppression and physiological stress reactivity. This study extends a prior review (Zaehringer et al., 2020) by focusing solely on suppression, including only active laboratory stress tasks, and separately investigating the effects of self-report suppression and experimentally manipulated suppression across two independent meta-analyses. Our findings demonstrated that experimental manipulation of suppression, when compared to control groups, was associated with greater overall physiological reactivity to acute laboratory stress tasks. In contrast, self-report trait suppression was not related to overall physiological stress reactivity, however, self-report trait suppression was specifically associated with greater neuroendocrine reactivity in subsequent subgroup analyses. There were not enough studies to conduct a quantitative review of self-report state suppression with physiological stress reactivity. Nonetheless, a qualitative summary of these studies can be found in the supplementary materials.

Participants who were instructed to actively suppress during laboratory acute stress tasks demonstrated greater cardiac, hemodynamic, and neuroendocrine reactivity compared to controls. These findings demonstrate that the active use of suppression increases arousal of multiple stress response systems (i.e., the SAM and HPA axes) above the influence of the stressor itself. The relationship between manipulated suppression and hemodynamic reactivity was particularly robust (e.g., high quality studies, low risk of publication bias). This suggests that the effort required to suppress during a highly stressful situation leads to increased blood pressure and/or total peripheral resistance (TPR), which over time, can increase the risk of pre-clinical and clinical CVD (e.g., Chida & Steptoe, 2010; Jennings et al., 2004; Matthews et al., 2006; Turner et al., 2020). Additionally, actively suppressing during a stress task may increase cortisol levels, which has also been implicated in precursors of CVD, such as progression of coronary artery calcification and hypertension (Hamer et al., 2012; Hamer & Steptoe, 2012; Turner et al., 2020). However, these findings with cortisol are provisional, given only four studies were included in this subgroup analysis. Caution should also be used when interpreting the relationship between experimentally manipulated suppression and cardiac reactivity due to an observed risk for publication bias. It appears that four independent samples within the same study were primarily driving the significance of these results (Quartana & Burns, 2010), such that when they were removed, manipulated suppression was no longer associated with greater cardiac reactivity.

Even so, it is worth noting a few important characteristics of the studies included in the cardiac subgroup that may have led to the significant findings between manipulated suppression and cardiac reactivity. First, the study by Quartana and Burns (2010) utilised a cognitive stress task (i.e., mental arithmetic), whereas the other studies utilised non-cognitive stress tasks (i.e., speech task, dyadic interaction, cold pressor). It is well established that there is a cognitive cost associated with the use of suppression (Johns et al., 2008; Richards & Gross, 2000; 2006; Schmeichel et al., 2003). It is possible that suppressing *during* higher order cognitive functioning uniquely depletes executive resources by requiring effortful engagement in both the act of suppression and the performance of the cognitive task itself. Literature has demonstrated that task engagement is associated with increased task-related cardiovascular reactivity (Cohen et al., 2000; Maier et al., 2003; Waldstein et al., 1997). Unfortunately, the studies in this analysis did not include task engagement (e.g., did not report task engagement or performance scores), thus preventing assessment of how suppression may have differentially impacted task effort or performance.

Second, the studies by Quartana and Burns (2010) and Peters et al. (2014) included unique control instructions that may have influenced the significance of the between-groups results. Indeed, when control groups were instructed not to suppress (Quartana & Burns, 2010), or instead to express outwardly (Peters et al., 2014), manipulated suppression was associated with greater cardiac reactivity. In contrast, when studies included no control group instructions (e.g., Braams et al., 2012; Mauersberger et al., 2018; Oveis et al., 2020) or instructions to respond naturally (e.g., Burns, 2006; Butler et al., 2006), suppression was no longer associated with cardiac reactivity. This is partially supported by the recent meta-analysis from Zaehringer et al. (2020), who also found the effects of suppression on various electrodermal and cardiovascular measures to be null when instructions to respond naturally were given. One explanation for the difference in significant findings is that without specific instructions *not* to regulate, control participants may have been naturally suppressing their responses to the stress tasks, unbeknownst to the study researchers. Surprisingly, only two studies included a post-task, manipulation check questionnaire, in which participants were asked to indicate what type of emotion regulation strategy they employed during the task (Braams et al., 2012; Mauersberger et al., 2018). However, it is unclear whether these manipulation checks were also given to the control participants. These results demonstrate the importance of explicit control instructions, along with the use of manipulation checks to ensure *all* groups are engaging in the appropriate response. For example, researchers conducting experimental manipulation studies should always consider including a self-report measurement of trait/state suppression, not only to identify individuals in the control group who may be inclined to naturally suppress, but also to examine possible interaction effects between self-reported trait suppression and the suppression manipulation. Only two of the included studies took this approach and found that high trait suppressors demonstrated worse physiological outcomes when additionally instructed to suppress (Jentsch & Wolf, 2020; Mauersberger et al., 2018).

Interestingly, manipulated suppression was not associated with miscellaneous parameters of autonomic reactivity. However, these results were found to be moderated by gender and type of physiological outcome, such that *less* sympathetic arousal/parasympathetic withdrawal was observed in primarily female samples with indicators of HRV (i.e., RSA, RMSSD) as the outcome of interest. Typically, HRV decreases in response to a stress exposure, which is a sign of vagal withdrawal and/or increased sympathetic activation (Balzarotti et al., 2017; Kim et al., 2018). However, the present results reveal that the active use of suppression during a stressor may be associated with *less* of a decrease in HRV as compared to controls (i.e., less vagal withdrawal or less sympathetic activation). It has been proposed that phasic increases in HRV may reflect attempts at emotional self-regulation (for review, see Balzarotti et al., 2017). However, if engagement in emotion suppression was truly increasing cardiac vagal tone over and above any sympathetic activation, one would expect to also see decreases in heart rate, which was not observed in this review (see Brindle et al., 2014). Additionally, only two studies examined the relationship between manipulated suppression and HRV, and both studies utilised only female samples, thus raising concerns about the robustness and generalizability of these outcomes. In contrast, the observed lack of association between manipulated suppression and PEP supports our similar findings regarding cardiac reactivity, thus suggesting that actively suppressing during a stress task may not have any additive impact on cardiac responding, other than the threat of the stress exposure itself.

In contrast to experimental manipulations of suppression, self-report trait suppression was not associated with overall physiological stress reactivity. However, it was specifically associated with neuroendocrine reactivity, such that greater trait suppression was associated with greater cortisol responses to the stress exposures. This finding is unsurprising, given that suppression has previously been associated with increased amygdala activation and delayed prefrontal cortex activation (Goldin et al., 2008), two regions known to influence HPA axis functioning (Diorio et al., 1993; Herman et al., 2012; Lupien et al., 2009). Unfortunately, there were too few studies available to quantitatively examine the relationship between trait suppression and hemodynamic reactivity ($n = 1$) and barely enough studies to examine trait suppression with cardiac reactivity ($n = 3$). The relationship between trait suppression and miscellaneous parameters of autonomic reactivity was moderated by task duration, such that participants with greater trait suppression demonstrated less autonomic reactivity during longer stress tasks (e.g., higher HRV). As mentioned above, it is possible that engaging in emotional self-regulation can result in increased HRV (for review, see

Balzarotti et al., 2017). That said, it is also possible that autonomic reactivity simply had enough time to habituate during longer stress tasks, which, if averaged across the entire task, can result in what appears to be less of a decrease in HRV. Research has identified fluctuations in physiological responding over the course of prolonged stress exposures (e.g., Kelsey et al., 1999; Ring et al., 2002). A review by Hughes and colleagues (2018) recommends the use of cardiovascular measurements that improve granularity, such as beat-to-beat or minute-by-minute data collection, which can then be quantitatively examined using slope or trajectory analyses. Given the small number of samples included in this subgroup analysis ($n = 5$), more research is needed to elucidate these effects. Indeed, the lack of studies examining trait suppression with physiological outcomes other than cortisol was surprising. This may be a 'file drawer problem', so we urge future research to examine self-report trait (and state) suppression with various parameters of physiological stress reactivity, and to report the results even if they are null.

Prior research has argued that suppressing emotions during a stress exposure may elicit a pattern of physiological arousal that is characteristic of 'threat responding' (Peters et al., 2014; Peters & Jamieson, 2016). According to the biopsychosocial model of challenge and threat, an individual's response to a stressful situation is dependent upon the following appraisals: 1) how demanding the situation is, and 2) whether there are sufficient personal resources available to cope with it (Blascovich, 2008; Blascovich & Mendes, 2010; Blascovich & Tomaka, 1996). In a *challenge* state, an individual perceives themselves to have ample resources to handle the demands of the situation. On the other hand, in a *threat* state, an individual perceives themselves as having inadequate resources to cope with the situational demands. These challenge and threat appraisals can be identified by distinct physiological response patterns. While both challenge and threat states evoke sympathetic arousal via the SAM axis, the experience of threat also mobilises the HPA axis (for review see Seery, 2011). More specifically, a challenge response is marked by increased cardiac efficiency (i.e., greater cardiac output, CO; greater stroke volume, SV), and simultaneous dilation of peripheral vasculature (i.e., reduced total peripheral resistance, TPR). This allows for the heart to pump more oxygenated blood to the brain and large skeletal muscles in preparation for physical action, if necessary. In contrast, a threat response is marked by decreased cardiac efficiency (i.e., reduced CO and SV) and concurrent constriction of vasculature (i.e., greater TPR), as well as the delayed release of cortisol from the adrenal glands. The result is a heart that is working harder with no increase in blood flow to the periphery.

Indeed, this threat-like pattern of physiological responding has been previously observed in individuals instructed to suppress during a stress task (Peters et al., 2014; Peters & Jamieson, 2016), and is partially supported by the findings of the present review, such that suppression activated both the SAM and HPA axes, with a particularly robust effect on hemodynamic parameters (including TPR). It is possible that the active effort required to monitor and maintain inhibition of emotionally expressive behaviour increases the perceived situational demands, thus pushing suppressors closer to threat appraisal (Peters & Jamieson, 2016). However, caution is advised when applying this interpretation, as many of these studies only report the calculated end products of physiological functioning (e.g., PEP, CO, TPR, SV), rather than the raw underlying mechanisms involved (e.g., HRV, HR, BP). This severely limits the ability to understand and elucidate the exact physiological determinants of these outcomes (e.g., TPR is calculated as MAP divided by CO; however, CO can be influenced by a wide variety of underlying factors such as HR, SV, preload, and afterload effects). As such, future research should attempt to examine a more comprehensive profile of reactivity.

Limitations

While the present study is the first quantitative review of the relationship between emotion suppression and physiological stress reactivity, it is not without limitations. First, almost all data were drawn from cross-sectional studies, thus preventing casual inferences to be made. Second, all subgroup and moderation analyses should be treated as preliminary due to a limited number of studies included in these analyses, particularly for trait suppression. Third, more research is needed to understand the relationship between self-report state suppression and physiological stress reactivity. Fourth, the present results may not be generalisable to stressful events in everyday life, or to other types of laboratory tasks, such as passive film/picture viewing. Additionally, most of the included studies consisted of American/European samples, except for one East-Asian sample (i.e., Yuan et al., 2014). It is well known that the consequences of suppression may be moderated by cultural context (e.g., Butler et al., 2007; Mauss & Butler, 2010; Soto et al., 2011). As such, we encourage future researchers to investigate these same relationships in other cultural contexts, such as those that place value on collectivism and interdependence rather that individualism and independence. Fifth, it should be noted that while researchers frequently examine the contrast between suppression and reappraisal, the current study focused solely on the relationship between suppression and physiological stress reactivity. The focus on suppression in this study is because a prior study has already systematically quantified the relationship between reappraisal and stress physiology, but did not examine suppression (see Liu et al., 2019). Sixth, not all types of physiological parameters were equally represented in these analyses; in particular, associations between suppression and skin conductance, cardiac output, stroke volume, and salivary-alpha amylase were grossly underrepresented, making it difficult to build a fully comprehensive profile of how suppression relates to all types of stress reactivity. Seventh, it is unclear how many studies controlled for baseline physiology in their analyses, and unfortunately, including baseline physiology as a moderator variable in the present study was not possible due to a lack of reported values. Eighth, the present analyses do not consider other variables that may influence these associations such as jobs that have high affective demands, but also require maintaining a level of professionalism that encourages expressive suppression (e.g., healthcare workers, service workers, educators). Unfortunately, there is a lack of reporting on the same covariates across studies, thereby limiting conclusions that can be drawn at this time on which third variables could be most relevant to consider. We encourage future research to address this gap in the literature.

Lastly, only two studies included both experimental manipulation and self-report assessment of suppression. This is important to note, because habitual suppressors may (or may not) be better at implementing the strategy during an experimental manipulation, as compared to participants who habitually utilise other types of emotion regulation strategies. Further, without including an assessment of self-report trait/state suppression, there is no way to know whether habitual suppressors exist in the control group and use it as their ‘natural’ response, thus contaminating the manipulation conditions. We recommend always including a self-report assessment of trait suppression alongside any experimental manipulation of suppression, to ensure that differences between habitual and non-habitual suppressors are controlled. It would also be beneficial to include brief self-report assessments of state suppression during the laboratory task, which can act as a manipulation check.

Conclusion and future directions

The present study represents the first attempt to systematically quantify the relationship between emotion suppression and physiological responses to active acute laboratory stress. Existing evidence suggests that suppression may exacerbate stress-induced physiological arousal, particularly through heightened hemodynamic and neuroendocrine responses. These findings advance emotion regulation research in two primary ways: 1) we identify a potential pathway through which suppression impacts poor health outcomes, and 2) we reinforce the popular belief that predominant use of response-focused strategies (i.e., suppression) is maladaptive, and is often associated with a wide range of negative consequences. Future research should continue to examine the psychological, physiological, and behavioural pathways through which suppression impairs healthy stress responding, and how this compares to other types of emotion regulation strategies. Identifying these pathways will provide insight into how we can specifically modify stress management interventions to include training in adaptive emotion regulation techniques. Moving forward, we encourage researchers to consider the following set of directions for future research:

1. There is a dearth of studies examining self-report trait or state suppression with physiological stress responses other than cortisol, thus preventing adequate comparison of the effects of different types of suppression assessments (e.g., self-report, experimental manipulation) on stress reactivity. Researchers should aim to report these findings, even if they are null.
2. Multiple assessments of suppression within a single a study, such as including a self-report trait measurement with an experimental manipulation will afford better control and allow for examination of more complex questions. For instance, more research is needed to understand how high or low trait suppression impacts the instructed use of suppression (or other emotion regulation strategies) and subsequent physiological outcomes during stress.
3. How does instructed suppression during a laboratory stress task influence cognitive processes, such as arousal, attention, task engagement, or performance? Does this differ depending upon the type of task or the content of the suppression instructions?
4. Most studies only examine a limited number of physiological variables at a time, thus preventing a comprehensive picture of how suppression impacts overall stress reactivity. Additionally, there are many physiological variables that have yet to be examined with suppression (e.g., what is the relationship between suppression and markers of inflammatory responses to stress, such as IL-6?).

5. The relationship between suppression and physiological stress reactivity should be further examined in other contexts, such as during chronic stress exposures (i.e., bereavement) or in other cultural samples (i.e., East-Asian). In doing so, we can begin to understand whether the use of suppression to cope with stress is universally maladaptive, or only in certain contexts.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data availability statement

The data that support the findings of this study are openly available in Open Science Framework at (https://osf.io/aw8h4/). The search strategy, list of included study references, summary of included studies, and additional tables/figures can be found in the online supplemental materials.

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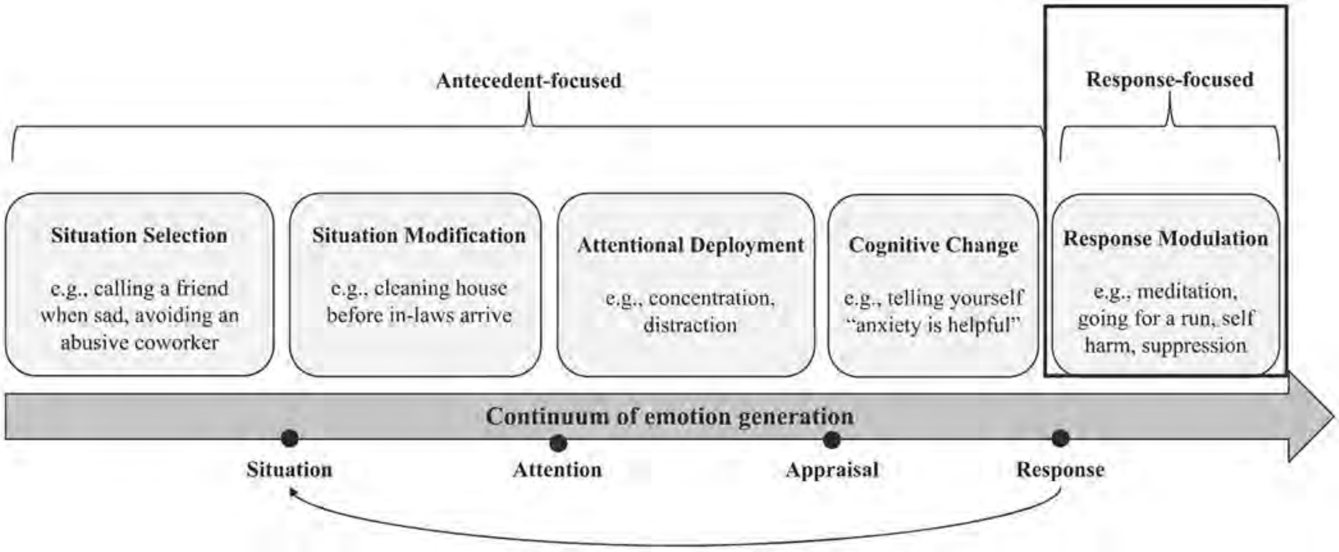

Figure 1.

Suppression in Relation to The Process Model of Emotion Regulation. *Note*. Adapted from 'Gross, J. J. (2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York, NY: Guilford Press.'

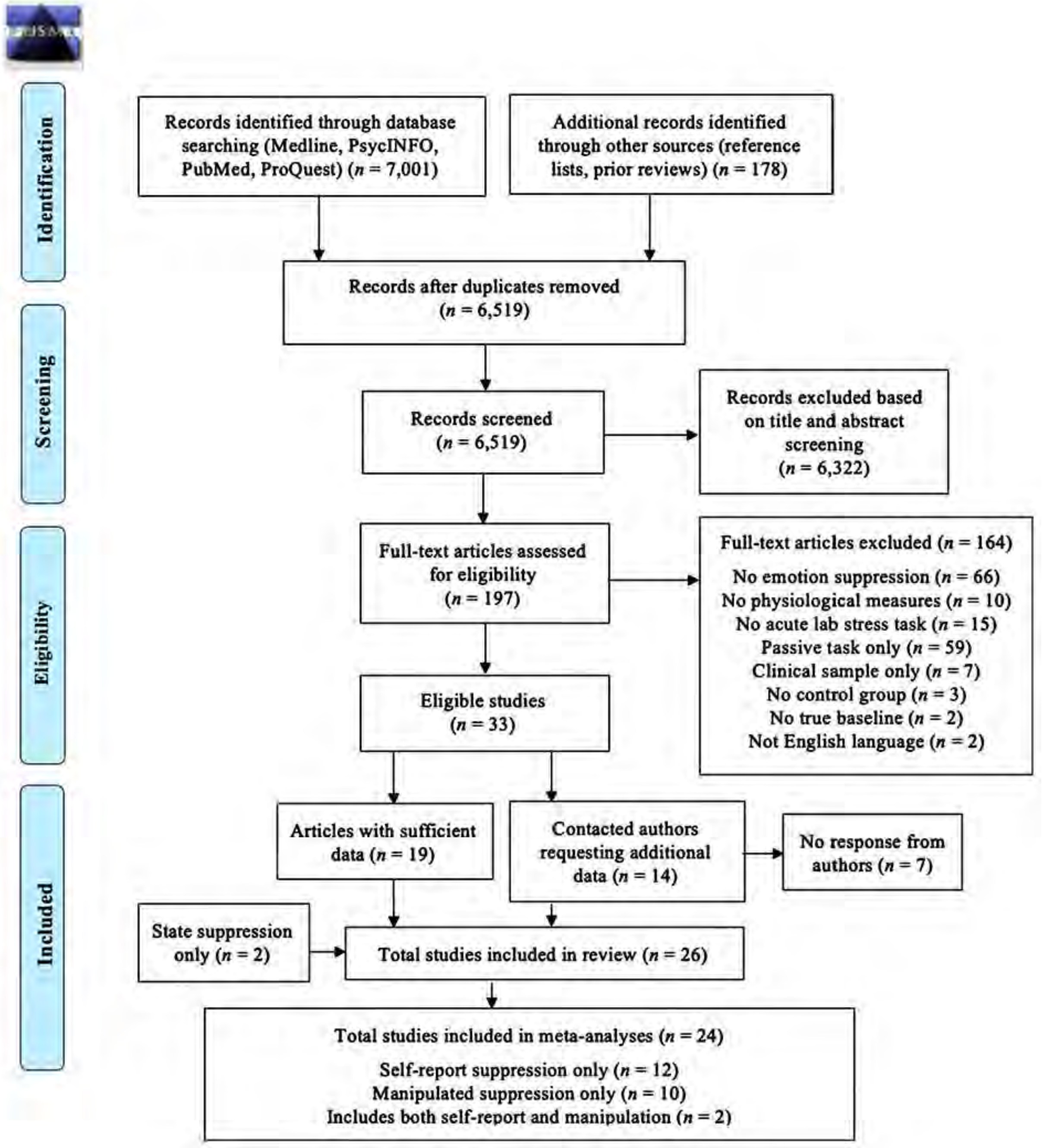

Figure 2.

Prisma Flow Diagram. *Note*. Adapted from:Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & The PRISMA Group (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *PLoS Medicine*, 6, e1000097. https://doi.org/10.1371/journal.pmed1000097. For more information, visit www.prisma-statement.org.

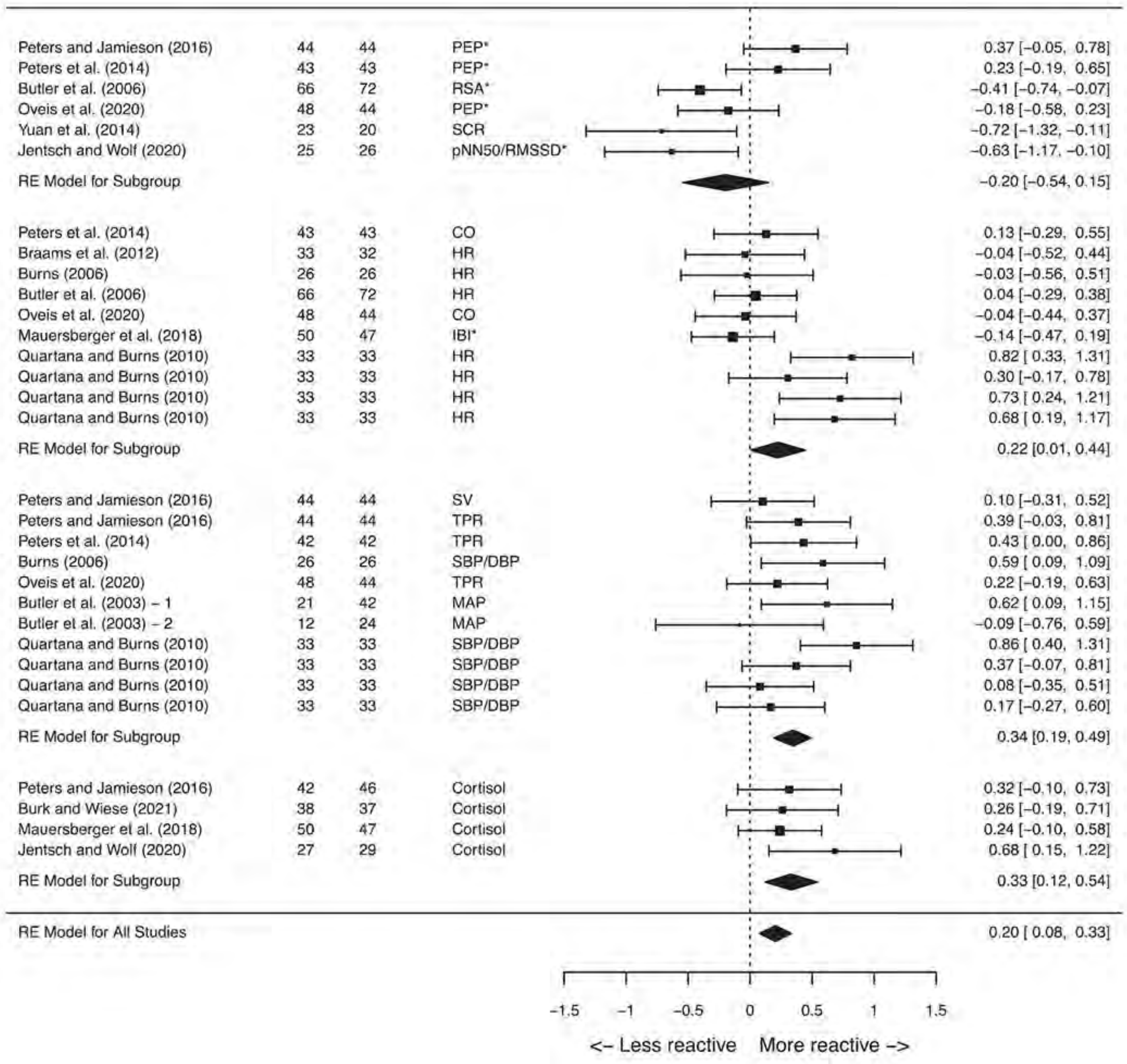

Figure 3.

Manipulated Suppression Versus Control on Physiological Stress Reactivity Outcomes.

Note. *denotes that the effect sizes of these physiological parameters were reverse coded to maintain consistency of interpretation; RSA = respiratory sinus arrhythmia; PE*P* = pre-ejection period; SCR = skin conductance response; pNN50 = proportion of NN50 divided by total number of NN (R-R) intervals; RMSSD = root mean square of successive differences; HR = heart rate; CO = cardiac output; IBI = interbeat interval; SV = stroke volume; SB*P* = systolic blood pressure; DB*P* = diastolic blood pressure; TPR = total peripheral resistance; MA*P* = mean arterial pressure.

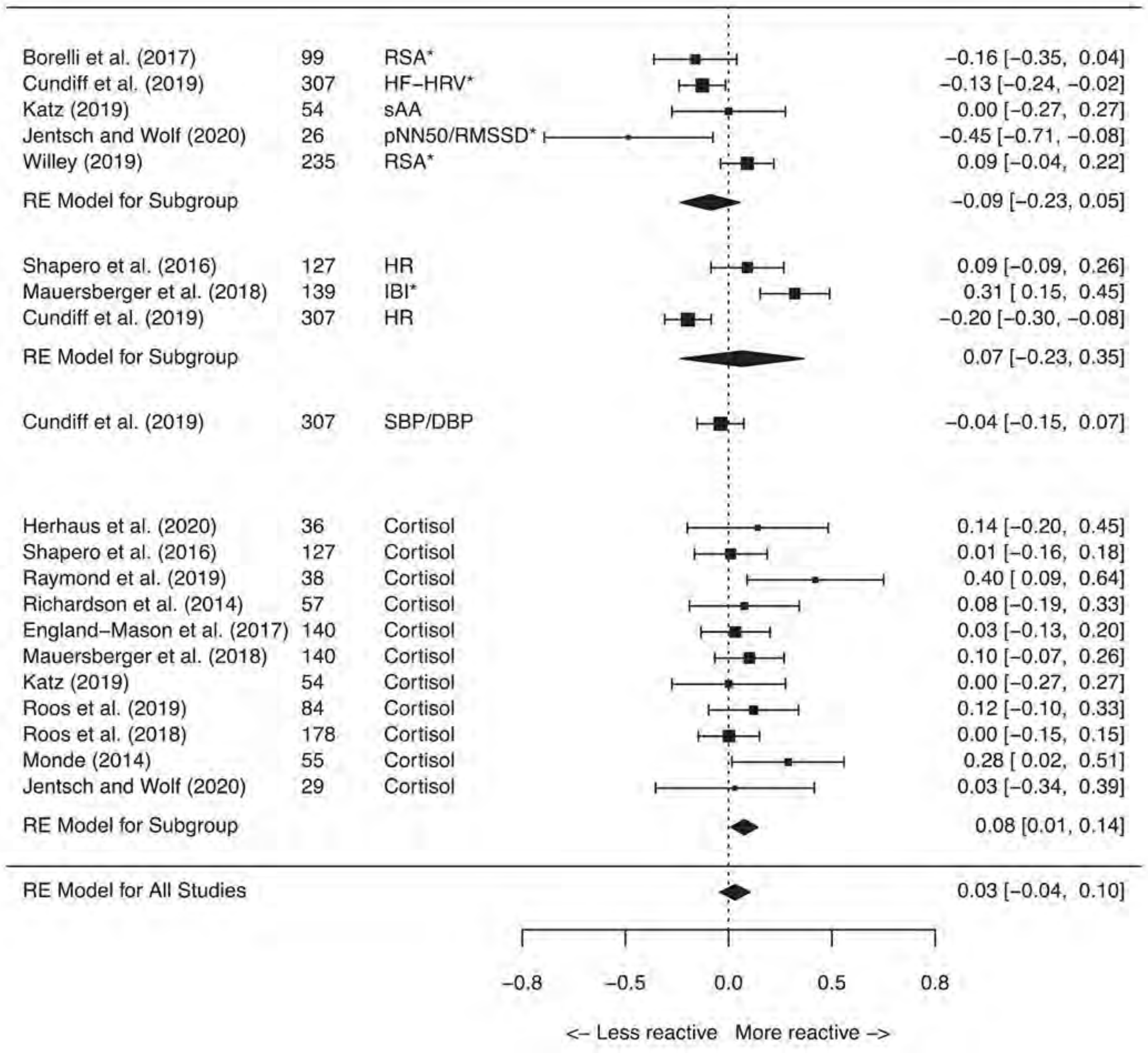

Figure 4.

Self-Report Trait Suppression on Physiological Stress Reactivity Outcomes. *Note*. *denotes that the effect sizes of these physiological parameters were reverse coded to maintain consistency of interpretation; RSA = respiratory sinus arrhythmia; HF-HRV = high frequency heart rate variability; sAA = salivary alpha-amylase; pNN50 = proportion of NN50 divided by total number of NN (R-R) intervals; RMSSD = root mean square of successive differences; RSA = respiratory sinus arrythmia; HR = heart rate; IBI = interbeat interval; SB*P* = systolic blood pressure; DB*P* = diastolic blood pressure.

Table 1.

Characteristics of samples included in meta-analyses.

	Manipulated Suppression (*k* studies = 12)		**Self-Report Suppression (*k* studies = 14)**	
	***k* samples**	***M (SD)***	***k* samples**	***M (SD)***
Study Characteristics				
Overall Sample Size	40	174.85 (58.44)	33	145.33 (104.24)
Suppression Group Size	40	37.72 (11.27)	–	–
Control Group Size	40	38.12 (10.77)	–	–
Task Duration (minutes)	40	8.61 (7.98)	33	11.09 (9.39)
Demographics				
Mean Age	40	21.77 (3.54)	33	24.27 (7.20)
% Women	40	66.00 (16.84)	33	51.32 (33.69)
% Caucasian	32	66.07 (22.23)	13	70.99 (21.91)
	***k* samples (%)**		***k* samples (%)**	
Type of Control Group				
No instruction	15 (37.5)		-	
Express or enhance emotions	7 (17.5)		-	
Experience naturally	6 (15.0)		-	
Control-mixed	12 (30.0)		-	
Type of Self-Report Suppression				
Trait Suppression	–		22 (66.7)	
State Suppression	–		11 (33.3)	
Type of Physiology				
Heart rate	8 (20.0)		5 (15.2)	
Systolic blood pressure	5 (12.5)		2 (6.1)	
Diastolic blood pressure	5 (12.5)		2 (6.1)	
Cortisol	4 (10.0)		13 (39.4)	
Heart rate variability	3 (7.5)		6 (18.2)	
Cardiac output	3 (7.5)		–	
Stroke volume	1 (2.5)		–	
Pre-ejection period	4 (10.0)		–	
Total peripheral resistance	4 (10.0)		–	
Skin conductance	2 (3.5)		1 (3.0)	
Mean arterial pressure	1 (2.5)		–	
Finger pulse/temperature	–		2 (6.1)	
Salivary alpha-amylase	–		2 (6.1)	
Stress Task Type				
Dyadic interaction	17 (42.5)		2 (6.1)	
Trier Social Stress Test	3 (7.5)		13 (39.4)	
Speech	3 (7.5)		8 (24.2)	
Mental arithmetic	12 (30.0)		–	
Cold pressor	3 (7.5)		–	

	Manipulated Suppression (*k* studies = 12)		**Self-Report Suppression (*k* studies = 14)**	
	***k* samples**	***M (SD)***	***k* samples**	***M (SD)***
Threat of shock	1 (2.5)		–	
Frustrating computer arithmetic	1 (2.5)		–	
Anger recall	–		8 (24.2)	
Performance challenge task	–		1 (3.0)	
Stroop	–		1 (3.0)	

Note. Means and standard deviations are across samples.

Table 2.

Tests of Heterogeneity.

	Q	df	*p*-value	I^2
Experimental Manipulation of Suppression				
Overall	102.90	39	**< .001**	62.1
Autonomic	20.52	7	**0.005**	65.89
Cardiac	23.64	10	**0.009**	57.7
Hemodynamic	23.44	16	0.103	31.73
Neuroendocrine	2.06	3	0.559	0.00
Self-Report Trait Suppression				
Overall	63.15	21	**< .001**	66.96
Autonomic	16.26	5	**0.01**	72.78
Cardiac	26.80	2	**< .001**	91.49
Neuroendocrine	8.98	10	0.534	0.00

Note. Significant *p*-values (≤ .05) reveal heterogeneity in the true effect size across studies.

Table 3.

Tests of Publication Bias.

	Egger's *p*-value	**Rosenthal Fail-Safe N**
Manipulated Suppression vs Control		
Cardiac	**0.041**	26
Hemodynamic	0.971	86
Neuroendocrine	0.244	12
Self-Report Trait Suppression		
Neuroendocrine	0.100	18