

The effect of heart rate variability biofeedback training on stress and anxiety: a meta-analysis

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Background. Some evidence suggests that heart rate variability (HRV) biofeedback might be an effective way to treat anxiety and stress symptoms. To examine the effect of HRV biofeedback on symptoms of anxiety and stress, we conducted a meta-analysis of studies extracted from PubMed, PsycINFO and the Cochrane Library.

Methods. The search identified 24 studies totaling 484 participants who received HRV biofeedback training for stress and anxiety. We conducted a random-effects meta-analysis.

Results. The pre-post within-group effect size (Hedges' g) was 0.81. The between-groups analysis comparing biofeedback to a control condition yielded Hedges' $g = 0.83$. Moderator analyses revealed that treatment efficacy was not moderated by study year, risk of study bias, percentage of females, number of sessions, or presence of an anxiety disorder.

Conclusions. HRV biofeedback training is associated with a large reduction in self-reported stress and anxiety. Although more well-controlled studies are needed, this intervention offers a promising approach for treating stress and anxiety with wearable devices.

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Introduction

Individuals with elevated levels of anxiety and stress often report using complementary and alternative therapies (Kessler *et al.* 2001). One of these interventions, heart rate variability (HRV) biofeedback training, has received increasing attention as a potential treatment for a variety of disorders, including anxiety and stress (Lehrer & Gevirtz, 2014).

It has been suggested that stress and negative affect can be improved through adaptive emotion regulation (Gross, 2002; Hofmann, 2014), which is a form of self-regulation that is expressed through certain physiological measures, especially HRV. HRV is a measure of cardiac vagal tone that can be quantified through the application of spectral analysis of the beat-to-beat (R-R) intervals (e.g. Porges, 2007). More specifically, this measure can be derived by integrating over the high frequency (HF) spectral component of R-R intervals at 0.15–0.40 Hz (in ms^2 ; see Camm *et al.* 1996; Berntson *et al.* 1997). This high-frequency peak is thought to reflect the magnitude of respiratory sinus

arrhythmia without requiring the assessment of respiratory rate. Low HRV has been associated with a number of psychopathological states, including anxiety (e.g. Hofmann *et al.* 2005; Friedman, 2007).

High resting HRV has been shown to predict self-regulatory strength and reduced negative emotion during acute stress (Khodik, 2013). Some research indicated that HRV might be an index of self-regulatory strength (Segerstrom & Nes, 2007). In addition to cultivating enhanced self-awareness (Kim *et al.* 2015), HRV biofeedback might enable individuals to regulate their physiological functioning for example through breathing training, which thereby contributes to relaxation (for review see Khazan, 2013). This approach is in line with mindfulness meditation exercises (Lehrer & Gevirtz, 2014) and may enhance self-regulation capacities (Vago & Silbersweig, 2012). Indeed, several studies suggest that HRV biofeedback may be an effective treatment for generalized anxiety disorder and post-traumatic stress disorder (i.e. Zucker *et al.* 2009; Kemp *et al.* 2012).

A number of qualitative reviews (Futterman & Shapiro, 1986; Gevirtz, 2013; Tabachnick, 2015) supported the notion that HRV biofeedback is effective for improving stress and anxiety. However, to our knowledge, there is no quantitative (meta-analytic) review examining the efficacy of this intervention. We hypothesized that HRV biofeedback is an effective intervention for anxiety and stress. Although

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biofeedback has a relatively long history, it is not a commonly-used intervention, partly because of the cost of earlier devices. If HRV biofeedback shows promise, then this might provide impetus for the further development of wearable devices, such as fitness trackers and smartwatches.

Methods

Identification and selection of studies

To identify eligible studies, a literature search was conducted in PubMed, PsycINFO and Cochrane Library databases. The following search terms were used: (*heart rate variability biofeedback OR HRVB OR respiratory sinus arrhythmia biofeedback OR RSA biofeedback OR resonance frequency feedback OR RFF OR biofeedback**) AND (*Anxiety OR anxiety disorders OR anxiety disorder OR anxious OR panic OR panic disorder OR agoraphobia OR social phobia OR social anxiety OR social anxiety disorder OR sad OR generalized anxiety OR gad OR general anxiety disorder OR obsessive compulsive OR obsessive-compulsive OR ocd OR obsessive compulsive disorder OR obsessive-compulsive disorder OR specific phobia OR simple phobia OR phob** OR *post-traumatic stress OR posttraumatic stress OR ptsd OR acute stress OR posttraumatic stress OR post-traumatic stress disorder OR posttraumatic stress disorder OR post traumatic stress disorder OR asd*).

The initial search produced 2297 results, with 1801 publications remaining after duplicates were excluded (see Fig. 1). Furthermore, we examined the references of the eligible papers. No language restrictions were applied.

Studies were included in the present meta-analysis if (1) at least one treatment condition was HRV biofeedback; (2) a psychometrically adequate measure of self-reported stress or anxiety was used; (3) the sample included individuals 18 years or older; and (4) sufficient descriptive statistics were provided to compute effect sizes.

Studies were excluded if (1) the paper was a review, a meta-analysis, a survey, a manual, or a conference abstract; if (2) they used other methods of biofeedback like electromyography (EMG) or electroencephalography (EEG); or if (3) HRV biofeedback was combined with another active treatment (e.g. cognitive behavioral therapy, mindfulness meditation, progressive muscle relaxation, motivational interviewing). However, studies were permitted if they combined HRV biofeedback with some aspects of common factors (e.g. initial education about biofeedback). If a study met all inclusion criteria, but the published paper lacked the necessary data to calculate an effect size, we emailed the corresponding author to request the data to conduct the analyses. For each selected study,

the authors extracted data on self-reported stress and anxiety measures at pre- and post-treatment for the HRV Biofeedback intervention, as well as data from control and comparison conditions if included. In addition, we extracted data for a number of sample and study characteristics, including sample size, treatment duration, gender, clinical status of the participants and the study year.

Quality assessment

For assessing the study quality, we used the Cochrane Handbook for assessing the risk of bias (Higgins *et al.* 2011). Using this tool, each study was classified as having a high, low or unclear level of bias risk for a number of domains using pre-specified criteria. The domains used in this assessment were: (1) *Sequence Generation*, which assesses whether all participants are adequately randomized to the different treatment conditions; (2) *Allocation Concealment*, which assesses whether investigators and participants are blind for the treatment assignment prior to randomization; (3) *Incomplete Outcome Data*, which assesses whether the studies reported missing data and whether appropriate methods were used for calculation (e.g. multiple imputation, full-information maximum likelihood estimation, etc.); and (4) *Selective Outcome Reporting*, which assesses whether all measurements of interest were adequately and completely reported. For each study a total bias assessment was created. Following the recommendations from the Cochrane guidelines, studies were rated as 'unclear risk', when at least one of the four categories showed an 'unclear' rating. If one of the four categories were rated with a 'high' risk, the study received a 'high risk' overall rating. 'Low risk' studies had to be rated as 'low' risk in all four categories.

Meta-analysis

We collected data on study characteristics including study year, number of biofeedback sessions, percentage of female participants, clinical diagnosis of an anxiety disorder, and risk of study bias.

We estimated the effect size by using Hedges' *g*, which corrects for parameter bias due to small sample size (Rosenthal, 1991). Both within and between pre-post effect sizes were calculated. To compute Hedges' *g*, we extracted means and standard deviations, as well as information from significance tests (e.g. *t*, *F*) (Rosenthal, 1991). The pooled effect sizes were estimated using random effects models, which assume significant heterogeneity of the included studies. Following Rosenthal (1991), we estimated the pre-post correlation to be $r=0.70$. All analyses were completed with Comprehensive Meta-Analysis (Borenstein & Rothstein, 2014). The magnitude of Hedges' *g* may be

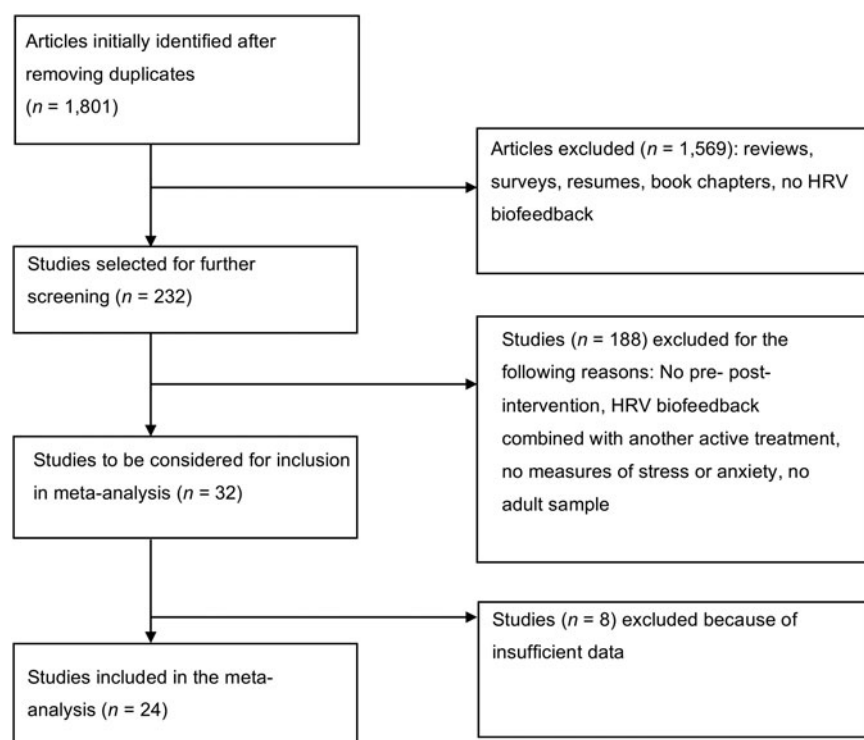


Fig. 1. Flow diagram of study selection process.

interpreted using Cohen's (1988) convention as small (0.2), medium (0.5), and large (0.8).

To investigate the influence of potential moderator variables on the effect of HRV biofeedback, we employed the between-group heterogeneity statistic (Q_B) recommended by Hedges & Olkin (1985) and meta-regression procedures for categorical and continuous moderators, respectively. Moderators of interest included both treatment characteristics (i.e. study year, number of biofeedback sessions) and sample characteristics (i.e. percentage of females per study, clinical diagnosis of an anxiety disorder, and risk of study bias).

To examine the presence of publication bias, we inspected the funnel plot. In addition, we used the fail-safe N method to determine the number of additional studies with a null result needed to reduce the overall effect size to non-significance (Rosenthal, 1991). If the fail-safe N exceeds 5 multiplied by K (i.e. the number of studies in the meta-analysis) +10, then the results may be considered statistically robust. Although a commonly used method, the fail-safe N approach tends to overestimate the number of studies needed to make moderate effect sizes non-significant. Therefore, we also examined the funnel plot to evaluate symmetry relative to the mean effect size, with greater symmetry corresponding to decreased likelihood of publication bias. To complement the funnel plot inspection, the trim and fill method (Duval &

Tweedie, 2000) was utilized to determine the nature of potential publication bias and to compute an imputed effect size that accounts for it. Finally, we examined Egger's regression intercept to determine whether results might be biased as a consequence of study number.

Results

Study characteristics

A total of 232 articles with HRV biofeedback treatment were found. Of those, 188 articles were excluded because they did not satisfy the inclusion criteria. Eight articles were excluded because of insufficient data. A total of 24 studies totaling 484 subjects that met inclusion criteria were included in this meta-analysis (Fig. 1). Characteristics of these 24 studies are described in Table 1. Subjects were recruited from both community ($n=14$ studies) and clinical settings ($n=10$ studies). The number of sessions varied between 1 and 50. Participants were told to train at home with a portable biofeedback device or were treated with a fixed number of sessions from a biofeedback trainer. There were 13 studies that included a comparison group (i.e. six were waitlist, one was standard care, two were treatment as usual, one was a daily thought record, one was progressive muscle relaxation, and two were sham biofeedback).

Table 1. Characteristics of randomized controlled studies examining the effect of HRV Biofeedback on self-reported stress and anxiety symptoms

Study	Country	Mean age of sample	Population	Sample size (n)	Percent of female	Instrument	Number of sessions	Type of symptom	Risk of study bias				
									R	A	I	S	T
Beckham <i>et al.</i> (2013)	USA	31	Clinical	14	100	STAI-S	2	Perinatal depression	1	1	1	2	1
Browne (2001)	USA	39	Clinical	10	50	DSP	10	Stress symptoms	2	2	1	2	1
Gatchel & Proctor (1976)	USA	–	Normal	36	–	Self-Report	3	Speech anxiety	1	1	1	2	1
Giardino <i>et al.</i> (2004)	USA	63	Clinical	20	50	HADS	9	COPD	1	1	1	2	1
Henriques <i>et al.</i> (2011)	USA	–	Normal	9	–	STAI-T	20	Performance anxiety	1	1	3	2	3
Keeney (2009)	USA	28	Normal	7	100	STAI-T	5	Stress symptoms	1	1	3	2	3
Lee <i>et al.</i> (2015)	Korea	27	Normal	5	20	STAI-T	4	Trait anxiety	1	1	1	2	1
Mikosch <i>et al.</i> (2010)	EU	66	Clinical	106	50	STAI-S	1	CA	3	3	1	2	3
Munafò <i>et al.</i> (2016)	EU	50	Normal	16	0	STAI-T	5	Stress symptoms	1	1	1	2	1
Nance (2015)	USA	37	Clinical	13	100	BAI	10	BPD	1	1	2	2	1
Patron <i>et al.</i> (2013)	EU	61	Clinical	13	15	STAI-T	5	Cardiac surgery	1	1	1	2	1
Paul & Garg (2012)	India	21	Normal	10	44.33	STAI-T	10	Trait anxiety	1	1	1	2	1
Prinsloo <i>et al.</i> (2011)	Africa	33	Normal	9	0	STAI-S	1	Stress symptoms	2	1	3	2	3
Prinsloo <i>et al.</i> (2013)	Africa	33	Normal	9	0	STAI-S	1	Stress symptoms	2	1	1	2	1
Ratanasiripong <i>et al.</i> (2012)	USA	19	Normal	30	100	STAI-S	35	Stress symptoms	1	1	1	2	1
Reiner (2008)	USA	–	Clinical	19	50	STAI-T	21	Sympathetic over-arousal	1	1	3	2	3
Sherlin <i>et al.</i> (2009)	USA	33	Normal	43	48.8	STAI-S	1	Stress symptoms	1	1	3	2	3
Sutarto <i>et al.</i> (2012)	Indonesia	36	Normal	19	100	DASS	5	Anxiety	1	1	3	2	3
Tan <i>et al.</i> (2011)	USA	36	Clinical	20	0	PCL-S	8	PTSD	1	1	1	2	1
Thurber (2007)	USA	23	Normal	7	42.86	STAI-T	2	Performance anxiety	3	1	3	2	3
Wells <i>et al.</i> (2012)	Australia	30	Normal	14	52.17	STAI-S	1	Performance anxiety	2	1	3	2	3
White (2008)	USA	45	Clinical	13	10	DAPS	50	Substance abuse	3	1	3	2	3
Zucker <i>et al.</i> (2009)	USA	–	Clinical	19	44.7	DAPS	20	PTSD	2	2	1	2	1
van der Zwan <i>et al.</i> (2015)	EU	27	Normal	23	68	DASS	35	Stress symptoms	2	2	2	2	2

Note. Outcome measures: BPD, borderline personality disorder; COPD, chronic obstructive pulmonary disease; CA, coronary angiography; PTSD, posttraumatic stress disorder; Instrument: BAI, Becks Anxiety Inventory; HADS, Hospital Anxiety and Depression Scale; STAI-S, State-Trait Anxiety Inventory, State Version; STAI-T, State-Trait Anxiety Inventory, Trait Version; PCL-S, PTSD Checklist; SCL-90-R, Symptom Checklist 90 Revised; PSS, Perceived Stress Scale; DAPS, Detailed Assessment of Posttraumatic Stress; DSP, Derogatis Stress Profile; DASS, Depression Anxiety Stress Scale. Risk of study bias: R, randomization; A, allocation concealment; I, incomplete data; S, selective outcome reporting; unclear risk = 1, low risk = 2, high risk = 3.

We observed heterogeneity in the quality ratings of the studies. In only two studies, allocation concealment to conditions was conducted by an independent party. In 15 studies, the randomization procedures were not adequately described and had an unclear risk. In three studies, improper randomization procedures were used. In 20 studies, the authors did not report the concealment of random allocation to respondents. In one study, allocation concealment procedures were explicitly described. One study had a high risk in allocation concealment. The handling of missing data was adequately addressed in two studies. Risk of bias due to missing data remained unclear for 13 studies, and nine studies employed procedures that did not adequately address missing data. In all studies, the measurements of interest were adequately and completely reported.

Efficacy of biofeedback

Pre-post within-group effects

The random effects meta-analysis yielded an overall within-group effect size on anxiety of Hedges' $g = 0.81$ [95% confidence interval (CI) 0.55–1.06, $z = 6.23$, $p < 0.001$] (Table 2). The fail-safe N analysis for the

within-group effect size was robust with $N = 1858$ ($z = 17.35$). Inspection of the funnel plot revealed a distribution of effect sizes concentrated to the left of the mean effect size, which indicates a decreased likelihood of publication bias from small studies with disproportionately large effect sizes (Fig. 2). The Trim and Fill method was used to further examine potential bias as determined by the funnel plot. This analysis showed that zero studies would need to fall to the left of the mean (i.e. have an effect size smaller than the mean) and three studies would need to fall to the right of the mean (i.e. have an effect size larger than the mean) to make the plot symmetrical, suggesting that the computed effect size is a conservative estimate. The random-effects model for the new imputed mean effect size revealed a Hedges' $g = 0.88$ (95% CI 0.81–0.96). Furthermore, the Egger's regression intercept was not significant (intercept = 0.64, $p = 0.74$), suggesting that the parameter estimates were not influenced by the number of studies.

Pre-post between-group effect sizes

For the between-groups analysis comparing biofeedback to another condition (i.e. standard care, waitlist,

Table 2. Within-group effect sizes of HRV biofeedback

Study (year)	Outcome	Hedges' g	Standard Error	Variance	Lower limit	Upper limit	Z-value	p value
Beckham et al. (2013)	STAI-S	0.24	0.20	0.04	-0.15	0.63	1.22	0.22
Browne (2001)	DSP	0.79	0.26	0.07	0.28	1.31	3.02	0.003
Gatchel & Proctor (1976)	Self-Report	0.59	0.18	0.03	0.24	0.93	3.31	0.001
Giardino et al. (2004)	HADS	0.17	0.17	0.03	-0.16	0.50	1.04	0.30
Henriques et al. (2011)	STAI-T	0.74	0.27	0.07	0.22	1.28	2.77	0.006
Keeney (2009)	STAI-T	0.28	0.26	0.07	-0.23	1.23	0.79	0.29
Lee et al. (2015)	STAI-T	2.44	0.66	0.44	1.15	3.74	3.70	0.001
Mikosch et al. (2010)	STAI-S	2.09	0.13	0.02	1.83	2.35	15.6	0.001
Munafò et al. (2016)	STAI-T	0.81	0.28	0.08	0.26	1.35	2.91	0.004
Nance (2015)	BAI	0.62	0.22	0.05	0.19	1.06	2.80	0.005
Patron et al. (2013)	STAI-T	0.05	0.20	0.04	-0.35	0.44	0.24	0.81
Paul & Garg (2012)	STAI-T	3.09	0.58	0.34	1.95	24.23	5.33	0.001
Prinsloo et al. (2011)	STAI-S	1.19	0.32	0.10	0.57	1.82	3.73	0.001
Prinsloo et al. (2013)	STAI-S	1.19	0.32	0.10	0.57	1.82	3.73	0.001
Ratanasiripong et al. (2012)	STAI-S	0.50	0.15	0.02	0.22	0.79	3.44	0.001
Reiner (2008)	STAI-T	0.56	0.18	0.03	0.20	0.92	3.03	0.002
Sherlin et al. (2009)	STAI-S	1.19	0.15	0.02	0.89	1.49	7.78	0.001
Sutarto et al. (2012)	DASS	0.86	0.26	0.07	0.35	1.37	3.31	0.001
Tan et al. (2011)	PCL-S	0.94	0.20	0.04	0.54	1.33	4.63	0.001
Thurber (2007)	STAI-T	0.15	0.26	0.07	-0.35	0.66	0.58	0.56
van der Zwan et al. (2015)	DASS	0.29	0.16	0.03	-0.02	0.60	1.81	0.07
Wells et al. (2012)	STAI-S	1.06	0.32	0.10	0.43	1.69	3.30	0.001
White (2008)	DAPS	0.36	0.21	0.04	-0.05	0.76	1.71	0.09
Zucker et al. (2009)	DAPS	1.08	0.22	0.05	0.65	1.51	4.96	0.001
Average effect size		0.81	0.13	0.02	0.55	1.06	6.23	0.001

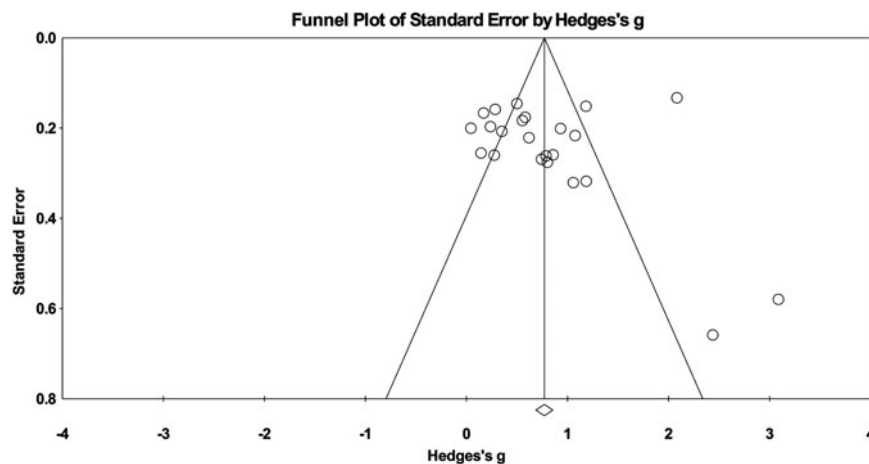


Fig. 2. Funnel Plot of standard error by Hedges' g .

daily record, progressive muscle relaxation, treatment as usual, meditation-based, or sham biofeedback), the random-effects analyses yielded an overall effect size of Hedges' $g = 0.83$ (95% CI 0.34–1.33, $z = 3.34$, $p < 0.001$) (Table 3). The fail-safe N for this analysis was robust with $N = 243$ ($z = 8.69$). The Trim and Fill analysis revealed that no studies would need to fall to the right or left of the mean to make the plot symmetrical, suggesting a conservative effect size estimate. The random-effects model for the new imputed mean effect size revealed a Hedges' $g = 0.83$ (95% CI 0.34–1.33). Furthermore, the Egger's regression intercept was not significant (intercept = -1.72 , $p = 0.35$).

Moderator analyses

Moderator analyses were conducted to determine whether within-group treatment efficacy varied as a function of participant and study characteristics. Specifically, the following five moderator variables were examined: study year, number of biofeedback sessions, percentage of females per study, clinical diagnosis of an anxiety disorder, and risk of study bias.

The results suggested that the effect of risk of study bias on treatment efficacy was not statistically significant ($Q_B = 0.12$, $df = 1$, $p = 0.73$). Because only one study exhibited low risk of study bias, this moderation analysis was conducted with uncertain and high risk studies. Effect sizes were not significantly related to study year ($B = 0.03$, $s.e. = 0.04$, $p = 0.48$), percentage of females ($B = -0.004$, $s.e. = 0.004$, $p = 0.30$), or number of sessions ($B = -0.01$, $s.e. = 0.01$, $p = 0.21$). The efficacy of HRV biofeedback on trait anxiety was not significantly different from that on state anxiety ($Q_B = 2.92$, $df = 1$, $p = 0.09$). Furthermore, treatment efficacy was not significantly related to the presence of an anxiety disorder ($Q_B = 0.36$, $df = 1$, $p = 0.55$).

Discussion

The results of this meta-analysis support the findings of earlier qualitative reviews (i.e. Futterman & Shapiro, 1986; Gevirtz, 2013; Tabachnick, 2015), suggesting that HRV biofeedback is an effective treatment for anxiety. The within-group analysis revealed an effect size of Hedges' $g = 0.81$, which was robust with a low likelihood of a publication bias. For the between-group analysis comparing HRV biofeedback with a comparison condition, the random-effects analyses yielded an overall effect size of Hedges' $g = 0.83$. These results suggest that HRV biofeedback is a beneficial treatment for people with anxiety and stress.

The moderator analyses revealed that treatment efficacy was not significantly related to study year, risk of study bias, percentage of females, number of sessions, outcome measure (i.e. trait *v.* state anxiety), or presence of an anxiety disorder. It could be the case that the efficacy of HRV biofeedback is robust across a variety of treatment conditions and patient characteristics; however, it is impossible for the current meta-analysis to definitively address this question because a lack of statistical significance cannot be interpreted as evidence in favor of a null-hypothesis. It will be important for future research to identify whether certain patient characteristics predict differential treatment response to HRV biofeedback, which is consistent with precision medicine.

Although there is good evidence to suggest that this intervention appears to be effective for anxiety and stress, the true size of the effect can only be determined after more rigorous clinical trials are completed in the future. In addition, several other limitations should be noted. First, although our meta-analysis included a relatively large number of studies, there were few studies with a clinical population. Because most of the

Table 3. Between-group effect sizes of HRV Biofeedback

Study	Outcome	Comparison condition	Hedges' g	Standard error	Variance	Lower limit	Upper limit	Z-value	p value
Browne (2001)	DSP	WL	1.32	0.48	0.23	0.39	2.26	2.78	0.01
Lee et al. (2015)	STAI-T	WL	2.68	0.83	0.69	1.06	4.30	3.24	0.001
Mikosch et al. (2010)	STAI-S	SC	1.67	0.16	0.03	1.36	1.98	10.49	0.001
Munafò et al. (2016)	STAI-T	DR	0.08	0.35	0.12	-0.61	0.77	0.22	0.82
Patron et al. (2013)	STAI-T	TAU	-0.68	0.39	0.15	-0.08	1.45	-1.75	0.08
Paul & Garg (2012)	STAI-T	WL	3.21	0.66	0.44	1.91	4.51	4.84	0.001
Prinsloo et al. (2013)	STAI-S	SB	0.23	0.45	0.20	-0.66	1.11	0.50	0.62
Ratanasiripong et al. (2012)	STAI-S	WL	1.06	0.27	0.07	0.53	1.60	3.89	0.001
Sherlin et al. (2009)	STAI-S	SB	0.87	0.32	0.10	0.24	1.49	2.73	0.01
Tan et al. (2011)	PCL-S	TAU	1.06	0.40	0.16	0.28	1.85	2.65	0.01
Thurber (2007)	STAI-T	WL	-0.15	0.50	0.25	-1.14	0.83	-0.31	0.76
Wells et al. (2012)	STAI-S	WL	0.56	0.37	0.14	-0.16	1.29	1.53	0.13
Zucker et al. (2009)	DAPS	PMR	0.17	0.32	0.10	-0.45	0.79	0.53	0.59
Average effect size			0.83	0.25	0.06	0.34	1.33	3.34	0.001

Note. Types of comparison conditions: SC, standard care; TAU, treatment as usual; DR, daily record; SB, sham biofeedback; PMR, progressive muscle relaxation; WL, waitlist.

studies did not report on specific anxiety disorder diagnoses, we were not able to calculate meaningful sub-analyses to examine whether HRV biofeedback is particularly effective for any specific anxiety disorder (and why). Second, due to the lack of studies with follow-up analyses, the long-term efficacy of HRV biofeedback remains uncertain. Third, prior research has suggested that outcome measure format (i.e. clinician rated *v.* self-report) can influence effect size estimates (Cuijpers et al. 2010). All the outcome measures of the studies in the current meta-analysis were self-report, which may bias effect size estimates. Fourth, it could be the case that non-specific factors (e.g. patient expectancies, patient-therapist interactions, etc.) contributed to the effect sizes of HRV biofeedback. To better determine the efficacy of this intervention, adequate comparison conditions need to be developed to examine the mechanism of HRV biofeedback. Fifth, the included studies did not provide adequate detail to quantify the amount of time with therapists. Thus, we were not able to include this as a moderator. Sixth, it is difficult to account for the lack of moderator effect for number of sessions, which may raise some concern with regard to the assumed mechanism of the intervention. The absence of a dose-response relationship in HRV biofeedback has also been observed in individual studies (Zucker et al. 2009). Finally, the studies included in the current meta-analysis varied in the biofeedback protocols, which introduced a methodological confound. In the current study, the number of studies using any given protocol was small, which

precluded moderator analyses to examine differences in efficacy across separate biofeedback protocols.

Despite these limitations, the results suggest that HRV biofeedback is a highly promising intervention for reducing anxiety and stress. The overall results could provide a compelling rationale to examine HRV biofeedback as an adjunct intervention in combination with other empirically supported treatments (e.g. cognitive behavioral therapy). This intervention is becoming increasingly more attractive as a treatment aid with the rapid improvements and affordability of wearable devices (such as fitness trackers and smartwatches).

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None.

Declaration of Interest

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also receives royalties and payments for his editorial work from various publishers.

Author Contributions

V.C.G. conceptualized the study, extracted the data, and wrote and revised the original draft. J.E.C. performed the statistical analysis, wrote the corresponding results sections and reviewed and edited the draft. S.G.H. conceptualized the study, reviewed and edited the draft.

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