THE JOURNAL OF
SEXUAL MEDICINE

ORIGINAL RESEARCH

FEMALE SEXUAL FUNCTION

# One Session of Autogenic Training Increases Acute Subjective Sexual Arousal in Premenopausal Women Reporting Sexual Arousal Problems

Amelia M. Stanton, BA, J. Gregory Hixon, PhD, Lindsey M. Nichols, BA, and Cindy M. Meston, PhD

## ABSTRACT

**Background:** Below average heart rate variability (HRV) has been associated with sexual arousal dysfunction and overall sexual dysfunction in women. Autogenic training, a psychophysiologic relaxation technique, has been shown to increase HRV. In a recent study, sexually healthy women experienced acute increases in physiologic (ie, genital) and subjective sexual arousal after 1 brief session of autogenic training.

**Aim:** To build on these findings by testing the effects of a single session of autogenic training on sexual arousal in a sample of women who reported decreased or absent sexual arousal for at least 6 months.

**Methods:** Genital sexual arousal, subjective sexual arousal, and perceived genital sensations were assessed in 25 women 20 to 44 years old before and after listening to a 22-minute autogenic training recording. HRV was assessed with electrocardiography.

**Outcomes:** Change in genital sexual arousal, subjective sexual arousal, and perceived genital sensations from the pre-manipulation erotic film to the post-manipulation erotic film.

**Results:** Marginally significant increases in discrete subjective sexual arousal ($P = .051$) and significant increases in perceived genital sensations ($P = .018$) were observed. In addition, degree of change in HRV significantly moderated increases in subjective arousal measured continuously over time ($P < .0001$). There were no significant increases in genital arousal after the manipulation.

**Clinical Implications:** The results of this study suggest that autogenic training, and other interventions that aim to increase HRV, could be a useful addition to treatment protocols for women who are reporting a lack of subjective arousal or decreased genital sensations.

**Strengths and Limitations:** There are few treatment options for women with arousal problems. We report on a new psychosocial intervention that could improve arousal. Limitations include a relatively small sample and the lack of a control group.

**Conclusion:** Our findings indicate that autogenic training significantly improves acute subjective arousal and increases perceived genital sensations in premenopausal women with self-reported arousal concerns. **Stanton AM, Hixon JG, Nichols LM, Meston CM. One Session of Autogenic Training Increases Acute Subjective Sexual Arousal in Premenopausal Women Reporting Sexual Arousal Problems. J Sex Med 2018;15:64–76.**



**Key Words:** Female Sexual Arousal; Autogenic Training; Heart Rate Variability; Subjective Sexual Arousal; Genital Sensations

## INTRODUCTION

Low resting-state heart rate variability (HRV) has been associated with decreased sexual arousal and poor overall sexual function.[1] An index of the modification of heart rate over time, HRV has become a widely used measure of autonomic control of the heart and the relative contributions of the sympathetic and parasympathetic nervous systems to that process. In addition to sexual arousal dysfunction, other psychological conditions have been associated with below average resting HRV; these include depression,[2] anxiety,[3] and post-traumatic stress disorder.[4] More broadly, decreases in resting state HRV reflect cardiac autonomic dysfunction, which plays a critical role in the development of cardiovascular disease and the maintenance of psychological states that are characterized by poor self-regulation.[5]

Received July 20, 2017. Accepted November 20, 2017.

Department of Psychology, The University of Texas–Austin, Austin, TX, USA



https://doi.org/10.1016/j.jsxm.2017.11.012

An expanding literature has documented the positive effects of experimentally increasing HRV on psychological health. Several interventions have led to significant increases in HRV and improvements in various psychological measures. HRV biofeedback is one such intervention; a number of HRV biofeedback studies have documented that biofeedback training to increase HRV produces acute and long-term gains.[6,7] These biofeedback protocols have led to significant decreases in symptoms of perinatal depression,[8] chronic fatigue,[9] and post-traumatic stress disorder,[10] among others. A recent meta-analysis found that HRV biofeedback training is associated with decreases in self-reported stress and anxiety.[11]

Although HRV biofeedback effectively decreases symptoms of disorders that are characterized by autonomic imbalance, the intervention requires a considerable time investment. Biofeedback protocols typically involve 4 to 5 visits with an experienced provider,[12] which might not be feasible for certain patients or clinical populations. An intervention that has led to significant increases in HRV, but over a shorter period, is autogenic training. Autogenic training is a psychophysiologic relaxation technique that is believed to improve self-regulatory functions and increase bodily resistance to stress.[13] The rationale for autogenic training is centered on the maintenance of autonomic balance or homeostasis, and the practice is designed to promote the recuperative processes that oppose the physiologic changes that are typically induced by stress.[14] Developed by Schultz and Luthe,[14] autogenic training consists of 6 standard exercises that use verbal instructions to achieve specific goals. The 1st exercise targets muscular relaxation, which the practitioner achieves by repeating a specific phrase that focuses on heaviness ("My right arm is heavy"); the 2nd exercise targets feelings of warmth ("My right arm is warm"). The 3rd exercise isolates cardiac activity ("My heartbeat is calm and regular"), and the 4th emphasizes steady respiration ("It breathes me"). The 5th and 6th exercises focus on warmth in the abdomen ("My solar plexus is warm") and coolness in the cranial region ("My forehead is cool"), respectively. Autogenic training is based on 3 core principles: (i) decreasing internal and external stimulation; (ii) mental repetition of specific verbal instructions; and (iii) "passive concentration," or total, effortless immersion in the task.[15] This training has been associated with increases in HRV after long-term practice[16] and after a single session.[17,18]

In light of the established relationship between depressed HRV and low sexual arousal,[1] a recent study attempted to experimentally increase HRV to facilitate increases in physiologic sexual arousal (ie, genital arousal, measured by vaginal photoplethysmography) and subjective sexual arousal (ie, the degree to which one feels mentally "turned on") in a sample of sexually healthy women.[18] In this study, women's physiologic sexual arousal and subjective sexual arousal were measured before and after they listened to a 14-minute autogenic training recording. There were significant increases in resting HRV after autogenic training compared with before autogenic training, indicating that the manipulation might have effectively targeted the mechanism of interest. Most importantly, physiologic and subjective sexual arousal were significantly higher when measured after the autogenic training recording than when measured before the manipulation, which suggests that increasing HRV can lead to acute increases in these 2 types of arousal in women without sexual arousal concerns. Moreover, change in HRV from pre- to post-manipulation significantly moderated changes in subjective sexual arousal; greater change in HRV was associated with larger increases in subjective sexual arousal. These findings suggest that interventions that increase HRV could be promising therapeutic options not only for sexually functional women but also for women who are having trouble feeling mentally "turned on" at the prospect of sexual activity or during sexual activity.

The present study extends the findings of Stanton and Meston[18] to a population of women who met *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision* (DSM-IV-TR) criteria for female sexual arousal disorder (FSAD; ie, recurrent or persistent inability to attain or maintain an adequate physiologic genital response [eg, vasocongestion, lubrication, swelling of the genitalia] during or until completion of sexual activity). Based on previous studies,[1,19] we had reason to believe that increasing HRV would specifically target arousal and not desire mechanisms. For this reason, we chose to use DSM-IV-TR[20] criteria as opposed to the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* (DSM-5)[21] diagnostic criteria for female sexual interest/arousal disorder, which encompass symptoms of arousal and desire. Although the inclusion criteria for this study focused only on physiologic arousal of the genitals, women with low or absent genital arousal also can have difficulties feeling mentally "turned on" during sexual activity, possibly due to problems with recognizing, processing, or appraising genital responses or because of a general lack of subjective excitement. Recently, researchers have argued for the return of separate diagnostic categories for inadequate genital arousal and low desire,[22] and they have suggested that FSAD should include a genital arousal subtype and a subjective arousal subtype.[23]

Prevalence estimates for sexual arousal concerns vary. When arousal problems are assessed by scores on the arousal domain of the Female Sexual Function Index (FSFI),[24] a well-validated and widely used self-report index that assesses 6 domains of female sexual function (arousal, lubrication, desire, pain, orgasm, and satisfaction), rates range from 11.4% in the United Kingdom[25] to 44.9% in Turkey.[26] Although arousal concerns are relatively common in women, there are no pharmacologic treatments approved by the US Food and Drug Administration that specifically target female sexual arousal, and only a few psychological interventions have led to clinically meaningful increases in arousal.

We hypothesized that, in women with sexual arousal problems, an acute increase in HRV through autogenic training would lead to acute increases in subjective sexual arousal but not in physiologic sexual arousal. Given the brief length of the intervention, we did not believe that there would be a significant

increase in genital arousal in women with clinically low levels of arousal. In addition, based on the results of previous research,[18] we hypothesized that the degree of change in baseline HRV from before to after the intervention would significantly moderate changes in subjective sexual arousal. If the findings of Stanton and Meston[18] are replicated in this sample of premenopausal women with decreased sexual arousal, then the results of this study could have implications for the treatment of sexual arousal problems. Furthermore, this study could help clarify the relation between HRV and female sexual arousal, offering insight that could shape the development of HRV-targeted interventions for the treatment of sexual arousal concerns.

## METHODS

### Participants

Participants were recruited from the community using flyers and print and online advertisements. These advertisements instructed potential participants to call the laboratory and complete a phone screen with a trained research assistant to determine eligibility. All potential participants completed a thorough phone screen, which included the FSFI,[24] a 19-item self-report questionnaire that assesses desire, arousal, lubrication, pain, orgasm, satisfaction, and overall sexual function. Total scores range from 2 to 36, with higher scores indicating greater sexual function. The FSFI has good internal reliability ($r = 0.89-0.97$), test-retest reliabilities ($\alpha = 0.79-0.88$), and has been shown to discriminate between women with and without sexual problems.[24] Only premenopausal women who scored below a 26.55 on the FSFI, the clinical cutoff for sexual function,[27] were included in the study.

In addition to meeting the FSFI cutoff score, participants were required to report decreased or diminished genital sensations and to respond in the affirmative to the following question: "Do you think that you have an arousal problem?" An adapted version of the Female Sexual Dysfunction Diagnosis (FSDD) questionnaire, which was originally developed to identify women who have acquired sexual arousal problems, also was used to determine eligibility with respect to the genital sensations criterion. The adapted FSDD assesses participants' past and current levels of genital sexual arousal by referencing 5 specific genital sensations (pleasurable sexual feeling in your genitals; genital pulsing or throbbing; genital or clitoral fullness, pressure, or engorgement; genital warmth; genital wetness or lubrication) that are typically experienced during sexual activity. Women who identified experiencing at least 2 of these genital sensations in the past and stated that these sensations were currently diminished or absent (for $\geq$6 months) were eligible to participate. In addition, the adapted FSDD evaluates the level of importance associated with these specific genital sensations during sexual activity with a 7-point Likert scale, which ranges from 1 (not at all important) to 7 (extremely important). However, the level of importance attributed to the sensations was not used to determine eligibility for this study.

Other inclusion criteria were generalized low arousal; currently sexually active with a partner; heterosexual or bisexual sexual orientation; and fluency in English. Generalized low arousal was defined as arousal concerns that are *not* situational (ie, low arousal regardless of context, no matter the setting, the partner, the time of day, etc). Exclusion criteria included a history of sexually transmitted diseases; current active or untreated pelvic, vaginal, or urinary tract infections; history of sexual abuse; history of major pelvic surgery; history of neurologic impairment; history of or current psychotic disorder; and currently taking medications that are known to affect genital sexual arousal in women, such as antidepressants, $\beta$-blockers, or benzodiazepines. Participants were compensated $50 for their time.

### Participant Characteristics

The final sample included 25 women who were 20 to 44 years old (average = 31.16, SD = 6.71). The sample was mostly Caucasian (64%), 28% were African American, 4% were Asian American, and 4% were Pacific Islander. Most of the sample considered themselves to be exclusively heterosexual (64%); 36% of the sample considered themselves to be predominantly heterosexual. For relationship status, most women in the sample were single and dating (52%), some women were in committed relationships (36%), and a minority was single but not dating (12%). All participants met the clinical cutoff for sexual dysfunction established by the FSFI (mean = 18.25, SD = 5.36). The average domain scores were 2.74 (SD = 1.13) for desire, 2.77 (SD = 0.866) for arousal, 3.10 (SD = 1.04) for lubrication, 3.04 (SD = 1.14) for orgasm, 3.00 (SD = 1.30) for satisfaction, and 4.29 (SD = 1.48) for pain.

### Measures

#### Heart Rate Variability

Heart rate was measured at a rate of 200 samples per second by electrocardiography. The signal from the electrocardiographic leads was detected using an MP100 data acquisition unit that was equipped with AcqKnowledge 3.9.1 software (Biopac Systems, Inc, Santa Barbara, CA, USA). During each experimental session, the research administrator placed disposable electrodes on the participant's upper right chest, lowermost left rib, and inner right ankle to triangulate the signal. Then, after the administrator left the room, the participant attached the leads to the electrodes.

#### Physiologic Sexual Arousal

Physiologic sexual arousal was assessed using a vaginal photoplethysmograph,[28] a photoreceptive diode that produces 2 measurements: vaginal blood volume and vaginal pulse amplitude (VPA). VPA, which reflects short-term changes in the engorgement of blood in the vaginal tissue,[29] is considered the more sensitive of the 2 indices[30] and has been shown to be a reliable index of women's physiologic, or genital, arousal.[31] Sampled at a rate of 200 samples per second throughout the neutral and erotic films, VPA data were recorded in millivolts,

bandpass filtered (0.5—30 Hz), and collected by an MP150 data acquisition unit equipped with AcqKnowledge 3.9.3 software (Biopac Systems).

### Subjective Sexual Arousal

Subjective sexual arousal was measured discretely and continuously. Discrete subjective arousal scores were calculated by summing the scores of the 3 original subjective sexual arousal items from the Heiman and Rowland's Film Scale.[32] This 15-item self-report questionnaire features a 7-point Likert scale with answer choices ranging from 1 (not at all) to 7 (intensely). On 2 occasions during the experiment (immediately after the 1st neutral-erotic film sequence and immediately after the 2nd neutral-erotic film sequence), participants were asked to rate the degree to which they experienced each of the 3 items on a 7-point scale, and then the 3 scores were combined into a single subjective sexual arousal score for each of the 2 time points. Subjective sexual arousal also was measured continuously during the 2 neutral-erotic film sequences with an arousometer,[33] a computer mouse mounted on a lever that is numbered from 0 (no mental sexual arousal) to 7 (maximum mental sexual arousal). The experimenter instructed the participant to move the mouse up or down as she felt her mental sexual arousal increasing or decreasing during the course of the films.

### Perceived Genital Arousal

Perceived genital arousal was calculated by summing the scores of the 5 perceived genital arousal items from the Film Scale.[32] Immediately after the 1st and 2nd neutral-erotic film sequences, participants rated the intensity of their genital sensations (eg, genital pulsing or throbbing; genital or clitoral fullness, pressure, or engorgement) on a 7-point Likert scale.

## Stimulus Materials

2 9-minute audiovisual films were used as stimulus materials. They were presented in counterbalanced order and matched for content. The 2 films included a 3-minute neutral segment followed by a 6-minute erotic segment. The neutral segments of the 2 films depicted different images of natural landscapes accompanied by classical music. The erotic segments of the 2 films featured different heterosexual couples engaging in 2 minutes of foreplay, 2 minutes of cunnilingus, and 2 minutes of penetrative sexual intercourse. These films have been found to be arousing to women in previous studies that have been conducted in our laboratory.

## Procedure

The institutional review board at The University of Texas at Austin approved the study protocol and all study materials. Eligible participants were greeted and oriented to the study procedures before providing informed consent. After the female experimenter left the room, the participants inserted the vaginal photoplethysmograph and attached the electrocardiographic wires. The experiment took place in a private room equipped with an intercom, which the participants used to communicate with the experimenter during the duration of the study session. The experimenter allowed the participant's data to normalize for 1 to 3 minutes. After this brief habituation period, participants watched the 1st 9-minute stimulus film while their VPA and heart rate data were collected. During the film, participants were asked to move the arousometer up as they felt their mental sexual arousal increasing and down as they felt their mental sexual arousal decreasing.

After watching the 1st 9-minute neutral-erotic film sequence, participants were asked to complete the Film Scale and a demographics form, which typically took 5 to 10 minutes. Then, they were instructed to stay seated, close their eyes, remain as still as possible, and listen carefully to a 22-minute autogenic training recording, which was adapted from an autogenic training manual written by Linden.[34] The recording focused on the 1st 2 of the 6 standard autogenic training exercises (ie, inducing sensations of heaviness and warmth). It is worth noting that the length of the autogenic training recording was increased from what was 14 minutes in a prior study conducted with sexually healthy women[18] to 22 minutes in the present study, which assessed women with sexual arousal concerns. This change was made to allow for the possibility that women with sexual concerns might take longer to acclimate to a laboratory testing environment than sexually healthy women. After listening to the recording, participants watched the 2nd neutral-erotic film sequence as their VPA and heart rate data were collected. During the 2nd film, participants were asked to move the arousometer in concert with changes in their mental sexual arousal.

## Data Analysis

### Data Reduction

Heart rate data were collected using electrocardiography with AcqKnowledge 3.9.3. The AcqKnowledge peak finder function isolated the inter-beat intervals (N-N intervals), which were exported to Excel (Microsoft, Redmond, WA, USA) for artifact removal. Artifacts were visually detected, and then a correction level was applied. The resulting Excel files from the data processing phase were converted to text files and then imported into Kubios HRV Analysis Software (Biosignal Analysis and Medical Imaging Group, University of Kuopio, Kuopio, Finland), a program that offers time-domain and frequency-domain analyses for HRV data.

2 indices of HRV were used in this study: a time-domain measurement, the SD of normal heart beat interval lengths (SDNN), and a frequency-domain measurement, the natural log of the high-frequency (HF) band. The Kubios software calculates time-domain measurements, including SDNN, and isolates the various components of the HRV signal for frequency-domain analyses. SDNN is considered one of the most widely used measures of HRV[35]; it provides information about all

components contributing to HRV during the recording period and thus is a more global measure than the HF band and the other power-spectrum–derived measures. When measured across shorter time spans, SDNN values can be compared only with other SDNN measures derived from recordings of a similar length, because SDNN tends to increase (up to a point) with longer recording periods.[36] For the analysis of parameters in the frequency domain, Kubios derives the intervals between successive normal QRS complexes (ie, N-N intervals) using a fast Fourier transform and then generates a power distribution as a function of frequency. The 2 spectral components that can be isolated from brief recordings are the low-frequency band (0.04–0.15 Hz) and the HF band (0.15–0.4 Hz). It has been suggested that the low-frequency band reflects sympathetic activity,[37,38] but a number of researchers have challenged this view, arguing that this band actually indexes baroreflex activity.[39,40] The HF band is believed to reflect the magnitude of the parasympathetic (ie, vagal) influence on the heart[41]; it is frequently referred to as the "respiratory band" because it corresponds to heart rate variations that are related to respiration (ie, respiratory sinus arrhythmia).[42] The power of the HF band is a commonly used measure of vagally influenced HRV. In short-term recordings, the primary source of variation that contributes to HRV is mediated by the parasympathetic nervous system; therefore, SDNN is correlated with higher frequency rhythms.[42]

VPA data were continuously measured during the pre-manipulation film and the post-manipulation film using AcqKnowledge 3.9.3. These data were exported to Excel for processing. Movement artifacts were removed from the data using an automated processing procedure[43] that was built in the R software environment[44] using the MGCV package for generalized additive modeling.[45] Pulverman et al[43] provide a more comprehensive explanation of this data processing procedure. VPA data were binned into 5-second segments, representing mean peak-to-peak VPA response, and the data within each segment were averaged into a single value. After the data were binned, each participant had a total of 120 VPA data points, which were analyzed with hierarchical linear modeling (HLM).

### Resting HRV

A paired-samples t-test was used to determine the effect of autogenic training on resting HRV. Measurements of baseline (resting) SDNN and baseline HF-HRV, collected during the 4-minute neutral segment before the 1st erotic film, were compared with SDNN and HF-HRV measurements collected during the 4-minute neutral segment immediately after the 22 minutes of autogenic training and before the start of the 2nd erotic film.

### Vaginal Pulse Amplitude

These data were analyzed via percent change scores and with HLM. For percent change score analysis, a mean VPA score was calculated for the pre-manipulation film sequence by averaging VPA during the 1st neutral film and then separately averaging VPA values for the 1st erotic film. Then, a mean VPA difference score ($VPA_{eroticfilm1mean} - VPA_{neutralfilm1mean}$) for the pre-manipulation phase of the experiment was determined for each participant. This difference score was used to calculate a percent change score for the pre-manipulation film sequence: each difference value was divided by the mean VPA during the neutral film and then multiplied by 100. The same procedure was carried out for the post-manipulation phase, resulting in a post-autogenic training mean VPA difference score ($VPA_{eroticfilm2mean} - VPA_{neutralfilm2mean}$) and then in a percent change score for the post-manipulation film sequence. A paired-samples t-test was used to determine the effect of autogenic training on VPA percent change score.

These data also were analyzed using HLM, a sensitive statistical technique that accounts for individual variability by estimating coefficients based on the unique slopes and intercepts of each participant.[46] This technique is particularly well suited for the analysis of continuous, multilevel data, and it has been effectively applied to VPA data in previous research.[47] The VPA analyses using HLM were conducted in R 3.2.3[44] with the NLME package.[48]

### Discrete Subjective Sexual Arousal and Perceived Genital Arousal

A paired-samples t-test was used to determine the effect of autogenic training on discrete subjective, sexual arousal and on perceived genital arousal. Pre-manipulation subjective arousal scores were compared with post-manipulation subjective arousal scores, and pre-manipulation perceived genital arousal scores were compared with post-manipulation perceived genital arousal scores.

### Change in HRV as a Moderator of Change in VPA and in Continuous Subjective Arousal

Several moderation models were tested using HLM in the R software environment[44] with the NLME package.[48] These moderation models examined the degree to which change in HRV from before to after the manipulation influences genital and continuous subjective sexual arousal.

The following 4 models assessed the relation between change in HRV (indexed by SDNN and HF-HRV) and VPA or continuous subjective arousal during the film sequences:

$$Y(VPA)_{ij} = \beta_0 + \beta_1(\Delta SDNN)_{ij} + \beta_2(film)_{ij} + \beta_3(time)_{ij} + \beta_4(\Delta SDNN \times time)_{ij} + \beta_5(\Delta SDNN \times film)_{ij} + \beta_6(film \times time)_{ij} + \beta_7(\Delta SDNN \times film \times time)_{ij} + r_{ij}$$

$$Y(VPA)_{ij} = \beta_0 + \beta_1(\Delta HF - HRV)_{ij} + \beta_2(film)_{ij} + \beta_3(time)_{ij} + \beta_4(\Delta HF - HRV \times time)_{ij} + \beta_5(\Delta HF - HRV \times film)_{ij} + \beta_6(film \times time)_{ij} + \beta_7(\Delta HF - HRV \times film \times time)_{ij} + r_{ij}$$

$$Y(arousometer)_{ij} = \beta_0 + \beta_1(\Delta SDNN)_{ij} + \beta_2(film)_{ij} + \beta_3(time)_{ij} + \beta_4(\Delta SDNN \times time)_{ij} + \beta_5(\Delta SDNN \times film)_{ij} + \beta_6(film \times time)_{ij} + \beta_7(\Delta SDNN \times film \times time)_{ij} + r_{ij}$$

$$Y(arousometer)_{ij} = \beta_0 + \beta_1(\Delta HF - HRV)_{ij} + \beta_2(film)_{ij} + \beta_3(time)_{ij} + \beta_4(\Delta HF - HRV \times time)_{ij} + \beta_5(\Delta HF - HRV \times film)_{ij} + \beta_6(film \times time)_{ij} + \beta_7(\Delta HF - HRV \times film \times time)_{ij} + r_{ij}$$

$Y(VPA)_{ij}$ and $Y(arousometer)_{ij}$ are the *i*th participant's VPA and continuous subjective arousal, respectively, at the *j*th time point. In these models, $\beta_1$ represents the main effect of change in HRV from before to after the manipulation on genital or subjective sexual arousal, $\beta_2$ represents the main effect of the film (0 = pre-manipulation film, 1 = post-manipulation film), $\beta_3$ highlights the main effect of time, and $\beta_{5-7}$ represent the various 2-way interactions effects and the 3-way interaction effect among HRV, film, and time. The time variable refers to the course of each of the neutral-erotic film segments; the pre-manipulation film and the post-manipulation film were 540 seconds in duration. $\beta_0$ represents the participant-specific intercept and $r_{ij}$ are the individual error terms.

To account for the clustering of observations within each subject, these analyses included a random intercept on subject. All other variables were treated as fixed effects. For all analyses, we used an $\alpha$ level of 0.05 to determine statistical significance.

## RESULTS

### Heart Rate Variability

Changes in resting HRV were analyzed using a paired-samples t-test. Resting HRV, measured by SDNN and HF-HRV, differed significantly from pre- to post-autogenic training. SDNN and HF-HRV were significantly greater during the neutral film that followed the manipulation ($t_{24} = -2.93$, $P = .007$; $t_{24} = -3.56$, $P = .002$) than during the pre-manipulation neutral film segment, which indicates that the autogenic training likely contributed to increased HRV, as was expected. These differences had medium effect sizes (d = 0.59 and d = 0.71, respectively). Tables 1 and 2 present a comparison of means and paired-samples t-test results.

### Vaginal Pulse Amplitude

Percent change in VPA was assessed with a paired-samples t-test. The intervention did not lead to significant increases in VPA percent change ($t_{24} = 0.188$, $P =$ not significant). Similarly, HLM analyses did not show a significant main effect for film, indicating that VPA did not significantly change from film 1, which was shown before the presentation of the autogenic training recording, to film 2, which followed the autogenic training intervention. That is, in line with our hypothesis, listening to the brief autogenic training recording was not associated with increased physiologic sexual arousal during the film that followed the recording. Tables 1 and 2 present a comparison of means and results.

### Discrete Subjective Sexual Arousal

Changes in discrete subjective arousal were analyzed with a paired-samples t-test. There was a marginally significant difference in mean discrete subjective arousal, measured by the Film Scale, after the autogenic training manipulation ($t_{24} = -2.05$, $P = .051$). Results are presented in Tables 1 and 2. Participants experienced an increase in subjective sexual arousal after listening

**Table 1.** Mean values before and after autogenic training

| | $M_1$ | SD ($M_1$) | SE ($M_1$) | $M_2$ | SD ($M_2$) | SE ($M_2$) | n |
|---|---|---|---|---|---|---|---|
| HRV (SDNN) | 46.56 | 19.29 | 3.86 | 58.54 | 26.47 | 5.29 | 25 |
| HRV (LnHF) | 5.94 | 1.42 | 0.28 | 6.50 | 1.38 | 0.28 | 25 |
| VPA change (%) | 30.31 | 33.16 | 6.63 | 29.15 | 32.11 | 6.41 | 25 |
| Discrete subjective sexual arousal | 12.64 | 4.45 | 0.89 | 13.80 | 5.10 | 1.02 | 25 |
| Perceived genital sensations | 15.68 | 7.25 | 1.47 | 18.20 | 9.30 | 1.86 | 25 |

HRV = heart rate variability; LnHF = natural log of high-frequency heart rate variability band (0.15–0.40 Hz); $M_1$ = mean before autogenic training; $M_2$ = mean after autogenic training; SDNN = SD of N-N interval; SE = standard error; VPA = vaginal pulse amplitude.

**Table 2.** Results of paired-samples t-tests

| Before to after manipulation | Mean | SD | SE | n | 95% CI for MD | t-ratio | df |
|---|---|---|---|---|---|---|---|
| HRV (SDNN) | −11.98 | 20.37 | 4.07 | 25 | −20.39 to −3.58 | −2.94‡ | 24 |
| HRV (LnHF) | −0.57 | 0.89 | 0.18 | 25 | −0.89 to −0.24 | −3.56‡ | 24 |
| VPA change (%) | 1.16 | 30.81 | 6.16 | 25 | −11.56 to 13.87 | 0.19 | 24 |
| Discrete subjective sexual arousal | −1.16 | 2.82 | 0.565 | 25 | −2.33 to 0.006 | −2.05* | 24 |
| Perceived genital sensations | −2.52 | 4.95 | 0.99 | 25 | −4.56 to −0.48 | −2.55† | 24 |

HRV = heart rate variability; LnHF = natural log of high-frequency heart rate variability band (0.15–0.40 Hz); MD = mean difference; SDNN = SD of N-N interval; VPA = vaginal pulse amplitude.
*$P = .05$; †$P < .05$; ‡$P < .01$.

to the autogenic training recording. This increase had a small to medium effect size (d = 0.41; Figure 1).

## Perceived Genital Arousal

A paired-samples t-test showed that participants experienced a significant acute increase in perceived genital arousal after the 22-minute autogenic training recording ($t_{24} = -2.55$, $P = .018$). That is, participants reported increased genital warmth, wetness or lubrication, pulsing or throbbing, and tenseness or tightness. This difference in perceived genital arousal had a medium effect size (d = 0.51). Tables 1 and 2 present the results and Figure 2 presents a visualization of this increase.

## Change in HRV as a Moderator of Change in VPA

HLM analyses were conducted to determine whether change in resting HRV from before to after the manipulation moderated changes in VPA over time. Regardless of the HRV index used, there was no significant 3-way interaction among change in HRV, film, and time. Therefore, change in HRV was not a significant moderator of changes in VPA (Table 3).

## Change in HRV as a Moderator of Change in Continuous Subjective Sexual Arousal

HLM analyses also were used to determine whether change in resting HRV from before to after the manipulation significantly moderated changes in continuous subjective sexual arousal, measured with the arousometer. When SDNN was used as an index of HRV, there was a significant 3-way interaction among change in SDNN, film, and time ($\beta = 0.0001$, $t_{5,393} = 6.10$, $P < .0001$; Table 3, Figure 3). That is, women who experienced greater changes in resting SDNN from before to after the autogenic training had the greatest increases in continuous subjective sexual arousal from the 1st to the 2nd neutral-erotic film segment. The same significant 3-way interaction emerged when the natural log of the HF band of the HRV signal was used to index HRV ($\beta = 0.0001$, $t_{5,393} = 4.60$, $P < .0001$; Table 4, Figure 4). Therefore, increased change in SDNN and HF-HRV could be driving the increase in subjective sexual arousal from the 1st to the 2nd film sequence. Figures 3 and 4 highlight the differences in subjective sexual arousal from pre- to post-autogenic training at 3 different levels of HRV change (HRV change − 1 SD, mean HRV change, and HRV change + 1 SD).

# DISCUSSION

This study assessed the effect of increasing HRV with autogenic training on genital sexual arousal, subjective sexual arousal, and perceived genital sensations (eg, warmth, fullness, and pressure) in premenopausal women with diminished or absent sexual arousal. The 22-minute autogenic training recording led to marginally significant increases in discrete subjective sexual arousal and significant increases in continuous subjective arousal and perceived genital sensations, but not in genital sexual arousal. Importantly, change in HRV, measured by SDNN and the

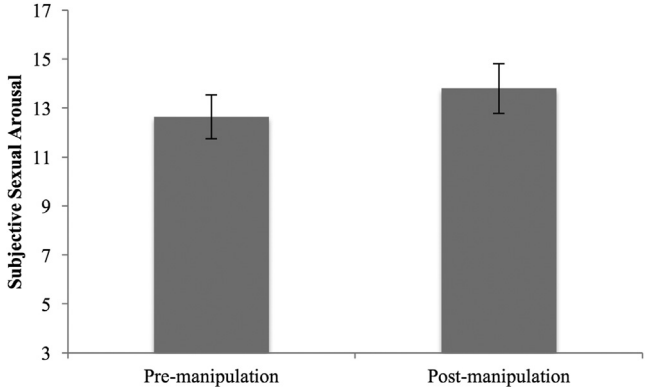


**Figure 1.** Mean subjective sexual arousal, measured with the Film Scale of Heiman and Rowland,[32] before and after manipulation of heart rate variability.

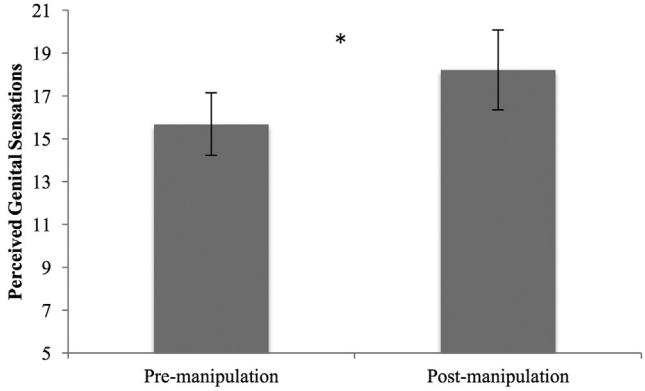


**Figure 2.** Mean perceived genital sensations, measured with the Film Scale of Heiman and Rowland,[32] before and after manipulation of heart rate variability. *$P < .05$.

**Table 3.** Results from hierarchical linear modeling analysis examining the relation among film, time, and ΔSDNN (heart rate variability) from before to after manipulation

| Predictor | $\beta$ | SE | df | t-ratio | *P* value |
|---|---|---|---|---|---|
| (Intercept) | 0.25 | 0.03 | 5369 | 7.56 | .000 |
| ΔSDNN | −0.003 | 0.002 | 23 | −1.56 | .13 |
| Film | 0.07 | 0.005 | 5369 | 13.15 | .000 |
| Time | 0.001 | 0.0002 | 5369 | 61.42 | .000 |
| ΔSDNN × film | 0.001 | 0.0003 | 5369 | 5.51 | .000 |
| ΔSDNN × time | −0.000009 | 0.0000008 | 5369 | −10.37 | .000 |
| Film × time | 0.0001 | 0.00003 | 5369 | 3.06 | .002 |
| ΔSDNN × film × time | 0.00001 | 0.000002 | 5369 | 6.10 | .000 |

df = degrees of freedom; film = before or after manipulation (0 = before, 1 = after); SE = standard error; ΔSDNN = change in resting SD of N-N interval (heart rate variability) during neutral film from before to after manipulation.

natural log of the HF band, moderated changes in subjective sexual arousal when it was measured continuously throughout the films. In other words, larger increases (ie, increases > 1 SD above the mean) in resting HRV from the pre- to the post-manipulation neutral film segment were associated with the greatest increases in subjective sexual arousal over time.

HRV could be related to subjective sexual arousal because of its role in the processing of emotional cues and regulation of emotional responses. Adaptive emotional processing and regulation involve the flexible modulation of emotional experiences, expressions, and physiologic responses.[49] Emotional responses that are consistent with a given situation (eg, sexual activity) are indicative of adaptive emotional regulation, which is critical for overall functioning and well-being.[50] It has been suggested that HRV can be considered an index of emotional regulation because of its relation to the inhibitory pathways that are regulated by the parasympathetic branch of the autonomic nervous system.[51] The heart and other peripheral organs are under the control of the autonomic nervous system, and this process is characterized by the relative dominance of the parasympathetic system over the sympathetic system.[52,53] The regulation of physiologic functions through parasympathetic pathways is influenced by psychological processes such as emotion regulation,[54,55] and emotion regulation is likely relevant for adaptive "mental" sexual arousal function.[56] When women are in a sexual situation, effective regulation of emotional responses "in the moment" will at least in part contribute to the success of the overall experience. That is, feeling mentally turned on during sexual activity could be considered adaptive emotion regulation, because this feeling integrates psychological and physiologic responses to meet the demands of a sexual situation.

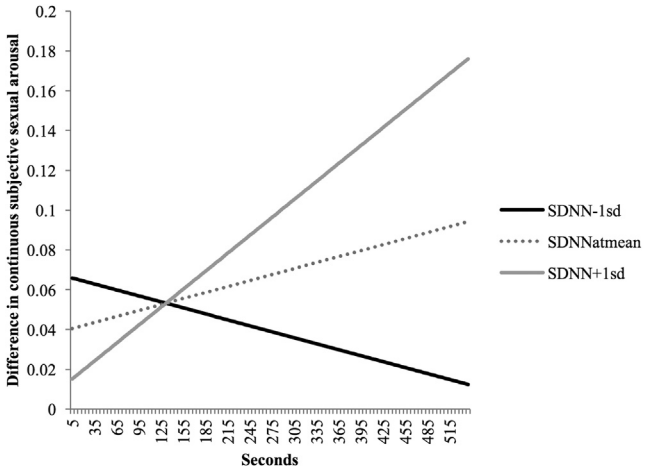


**Figure 3.** Difference in continuous subjective sexual arousal from before to after autogenic training at 3 levels of change in SDNN. All 3 groups experienced increases in subjective sexual arousal after the intervention. However, the women who had the lowest increases in heart rate variability (SDNN) after the intervention experienced the smallest gains in subjective sexual arousal. SDNN = SD of N-N interval.

Barlow's model of sexual dysfunction[57] provides another possible explanation for these findings. According to this model, the locus and quality of the individual's attention and, relatedly, the degree of one's cognitive distraction are maintaining factors of sexual problems. During sexual activity, individuals with sexual dysfunction tend to show decreased focus on erotic cues and increased focus on non-erotic cues,[58] such as negative thoughts about their own level of physical attractiveness. Similarly, spectatoring, which occurs when individuals focus on their arousal only to judge its adequacy during sexual intercourse, has been associated with sexual problems.[59] The autogenic training intervention draws the practitioner's attention to specific areas of the body for extended periods, potentially decreasing cognitive distraction. Spending more than 20 minutes channeling feelings of warmth and heaviness in different limbs could help women direct their attention to similar sensations in their genitals and to other erotic cues during the sexual film, ultimately leading to increases in subjective sexual arousal and perceived genital sensations.

It is notable that the autogenic training manipulation failed to result in significant VPA increases over time. In a previous study, the same protocol was applied to a group of premenopausal women *without* sexual arousal concerns, and there was a significant increase in VPA from pre- to post-manipulation.[18] Given this

**Table 4.** Results from hierarchical linear modeling analysis examining the relation among film, time, and ΔLnHF (heart rate variability) from before to after manipulation

| Predictor | β | SE | df | t-ratio | *P* value |
|---|---|---|---|---|---|
| (Intercept) | 0.82 | 2.13 | 5369 | 0.39 | .69 |
| ΔLnHF | 0.99 | 0.35 | 23 | 2.86 | .009 |
| Film | 2.23 | 0.23 | 5369 | 9.51 | .000 |
| Time | 0.002 | 0.0008 | 5369 | 2.09 | .04 |
| ΔLnHF × film | −0.37 | 0.04 | 5369 | −9.63 | .000 |
| ΔLnHF × time | 0.0005 | 0.0001 | 5369 | 4.18 | .000 |
| Film × time | 0.008 | 0.002 | 5369 | 5.47 | .000 |
| ΔLnHF × film × time | −0.001 | 0.0002 | 5369 | −5.76 | .000 |

df = degrees of freedom; film = before or after manipulation (0 = before, 1 = after); SE = standard error; ΔLnHF = change in natural log of high-frequency heart rate variability band (0.15–0.40 Hz) during the neutral film from before to after manipulation.

discrepancy, it is possible that the intervention might not be applicable to women with a clear genital arousal disorder, such as women with medical comorbidities that affect the neurovascular system. Alternatively, a 22-minute single-session intervention might not be long enough or intense enough to translate into statistically meaningful gains in genital arousal in women who have had low arousal for at least 6 months. To test this hypothesis, researchers could consider titrating the dose of the intervention in future studies (ie, longer sessions or multiple sessions) or altering the protocol to enable women to listen to the autogenic training recording from the privacy of their own homes. The contradictory VPA findings are not unusual, because the literature documenting the effects of psychosocial and pharmacologic interventions on genital sexual arousal is mixed. Some pharmacologic agents have been shown to increase VPA response relative to placebo; these include combined L-arginine glutamine plus yohimbine,[60] ginkgo biloba,[61] tibolone,[62] and sildenafil.[63,64] Other studies, particularly those examining the effects of sildenafil and other phosphodiesterase inhibitors on genital arousal in women, have *not* shown promising results with respect to VPA and/or similar physiologic indices of female genital arousal.[65,66]

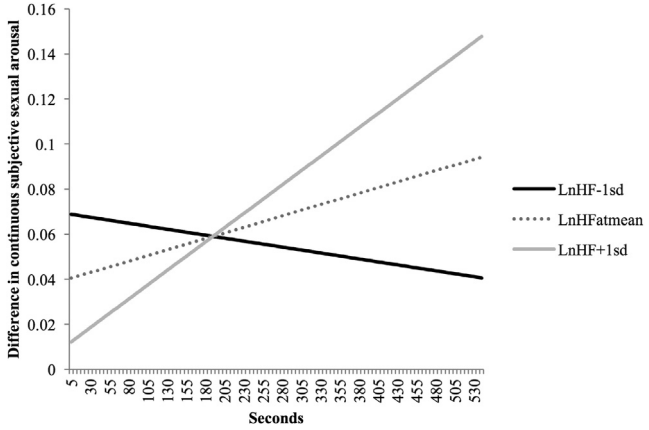


**Figure 4.** Difference in continuous subjective sexual arousal from before to after manipulation at 3 levels of change in LnHF. All 3 groups experienced increases in subjective sexual arousal after the intervention. However, the women who had the lowest increases in heart rate variability (LnHF) after the intervention experienced the smallest gains in subjective sexual arousal. LnHF = natural log of high-frequency heart rate variability band.

Likewise, there is no clear consensus on the effect of psychosocial interventions, such as traditional cognitive-behavioral therapy (CBT) and mindfulness, on genital arousal in women with self-reported arousal concerns. As of 2013, when a meta-analysis of psychosocial interventions for female sexual dysfunction was published, there were no randomized-controlled studies on FSAD to include in the analysis.[67] Some studies have examined the efficacy of CBT for improving other domains of sexual function, such as desire[68] and pain,[69] but increases in female sexual arousal (and VPA, in particular) have yet to be the target of a randomized-controlled CBT trial. This lack of CBT-based options for women with arousal concerns has been somewhat remedied in recent years with the increase in studies examining mindfulness-based interventions for female sexual dysfunction. In 2003, Brotto[70] began a series of experiments that applied mindfulness-based approaches to the treatment of female sexual dysfunction. One of these studies was a 3-session mindfulness-based group psychoeducational intervention for women with FSAD.[71] Although the intervention had a significant positive effect on self-assessed genital wetness and led to marginally significant increases in subjective and self-reported physical arousal, it did not result in any changes in physiologic arousal measured by VPA. Similarly, when they tested their intervention in a sample of women with gynecologic cancer, Brotto et al[72] found a significant effect of the program on self-reported arousal (and on other sexual function domains, including desire, orgasm, satisfaction, and sexual distress), but only a trend toward significantly improved physiologic genital arousal (VPA) and perceived genital arousal. Therefore, the lack of significant VPA findings in the present study is consistent with previous research on mindfulness-based interventions.

Autogenic training, the intervention used in this study to increase HRV, and mindfulness have some similarities but also a few critical differences. Mindfulness has been defined as a purposeful, non-judgmental awareness of the present moment or one's present experience.[73] In mindfulness-based practices, patients are invited to attend to and accept their present experience without judging it or attempting to change it in any way.

Autogenic training also emphasizes non-judgmental attention to the present moment, but it directs practitioners to conjure sensations throughout the body through verbal self-suggestions.[14,74] Mindfulness discourages any attempts to alter one's physical sensations, whereas autogenic training facilitates autonomic self-regulation through these self-suggestions. Some women might find it difficult or frustrating to sit with their sensations without attempting to alter them; for these women, autogenic training could be a viable option. Other women might be attracted to mindfulness-based protocols because they build curiosity, openness, and, perhaps most importantly, acceptance of the body and the environment. If a future randomized-controlled study demonstrates that autogenic training (and/or other interventions that increase HRV) is associated with significant increases in sexual arousal in women with sexual arousal problems, then this protocol could be considered an alternative or a complement to mindfulness-based practices.

The main contribution of the present study is the demonstrated effect of the autogenic training recording on acute subjective sexual arousal and perceived genital arousal. It is arguable that subjective sexual arousal and perceived genital arousal are more important to a woman's individual experience of sexual activity than her VPA. Laboratory studies have shown low correlations between subjective (ie, self-report) and physiologic (ie, VPA) measurements of sexual arousal in women.[75] That is, women are less likely than men to report feeling aroused when their physiologic arousal is elevated. Therefore, it might be more clinically meaningful to look for changes in *perceived* genital sensations and subjective sexual arousal, rather than changes in VPA, when testing psychosocial interventions aimed to increase arousal.

Importantly, we do not believe that these increases are a result of repeated presentation of erotic material. The 2 films were separated by a 22-minute recording, which is likely a long enough period for participants to return to their baseline levels of VPA and subjective arousal. Indeed, the results of a paired-samples t-test comparing average VPA during the 1st 3-minute neutral film and average VPA during the 2nd 3-minute neutral film did not reach statistical significance, which indicates that participants returned to their baseline VPA before the presentation of the 2nd erotic film. Other studies have demonstrated that presenting multiple erotic films in a single session, with sufficient time between the films for the participants to return to their baseline levels of arousal, does not lead to increased subjective sexual arousal. For example, Pulverman et al[76] showed heterosexual and lesbian women 3 different films (featuring heterosexual, lesbian, and gay male couples, respectively), in counterbalanced order, during a single laboratory visit. Subjective sexual arousal differed significantly based on the content of the films, not on the order in which the films were presented. That is, subjective sexual arousal was not significantly greater during the 3rd film compared with the 1st or 2nd film.

There are several limitations of this study that warrant explanation. (i) The study had a small sample and did not include a control group or a "sham" intervention. To more accurately determine the efficacy of autogenic training and other similar interventions in improving sexual arousal in women who meet *International Classification of Diseases, 10th Revision* (ICD-10) criteria for FSAD or DSM-5 criteria for female sexual interest/arousal disorder, it will be crucial to conduct a randomized-controlled trial on a larger sample that includes a placebo or a waitlist control group. Before such a study is conducted, the generalizability of our results to clinical populations is limited. (ii) To minimize the number of visits required to complete the study, we did not randomize the order of the autogenic training recording. Rather than having each participant come to the laboratory twice, once to establish a baseline VPA reading and once to play the autogenic training recording before presenting the neutral-erotic film sequence, we included the pre-manipulation VPA recording and the post-manipulation VPA recording in a single visit. Although we recognize that a multi-visit study design might have more effectively isolated the relative contribution of the autogenic training recording to the arousal response, separating the study into 2 visits could have led to decreased compliance, increased dropout rates, and/or variability in the placement of the vaginal photoplethysmograph. (iii) Lesbian women were excluded from participation. The stimulus films that were used in this study have been demonstrated to increase arousal in heterosexual and bisexual women in previous studies. We chose to limit potential sources of variability by using (i) only 2 films and (ii) films that that had been piloted on other women. Because there were no lesbian women in our sample, we cannot claim that these results generalize to that population. It is critical that future research test the efficacy of this intervention and other sexual arousal interventions on women who are sexually attracted to women.

In addition, we did not use a validated instrument to assess arousal-specific concerns, and we did not inquire about deficits in subjective sexual arousal during the screening phase. The FSDD was originally developed from the ICD-10 and DSM-IV-TR diagnostic criteria for FSAD to highlight potential deficits in genital arousal. It was adapted for this study to assess specific genital sensations that are typically believed to be associated with physiologic sexual arousal (eg, genital warmth, clitoral fullness, etc). We do not have information on the validity and reliability of the adapted FSDD. Women were allowed to participate only if they reported decreased or absent genital arousal; they were not asked about subjective arousal during the phone screen. A broad understanding of arousal incorporates physical and mental readiness for sexual activity. Indeed, Janssen et al[77] and Spiering et al[78] proposed that arousal can be broken down into 2 key components: (i) conscious and unconscious mental processing leading to an automatic genital response (ie, physiologic arousal) and (ii) a cognitive process appraising the sexual content of the stimulus (ie, subjective sexual arousal). There are women who report diminished subjective sexual arousal but no decrements in physiologic arousal,[47] and these women could benefit from the autogenic training intervention.

Based on these results, we cannot definitively conclude that change in HRV has a causal effect on sexual arousal or that autogenic training uniquely targets HRV. Autogenic training might modulate the entire autonomic nervous system, and a consequence of this modulation could be increased HRV. Another consequence of this modulation might be increased arousal, such that increased HRV and increased arousal occur in parallel. The timing and nature of these effects should be further investigated in future research.

Overall, our finding that autogenic training increased acute subjective sexual arousal and perceived genital arousal in women with sexual arousal concerns suggests that this intervention could be a welcome addition to the small group of psychosocial interventions that have shown efficacy in treating sexual arousal concerns. The fact that women who experienced the greatest changes in resting HRV had the greatest increases in subjective sexual arousal indicates that HRV level might be an important mechanism fueling the effects of the autogenic training intervention. Autogenic training is easy to learn, and recordings can be downloaded from the internet at little to no cost. Given the wide-scale availability of the intervention and its potential to improve sexual arousal in women with and without arousal concerns,[18] it is worthwhile to continue investigating the effects of HRV interventions on sexual arousal.

**Corresponding Author:** Cindy M. Meston, PhD, Department of Psychology, The University of Texas—Austin, 108 E Dean Keeton, Stop A8000, Austin, TX, 78712, USA. E-mail: mestoncm@gmail.com

*Conflicts of Interest:* The authors report no conflicts of interest.

Funding: None.

## STATEMENT OF AUTHORSHIP

### Category 1

(a) Conception and Design
Amelia M. Stanton; Cindy M. Meston
(b) Acquisition of Data
Amelia M. Stanton; Lindsey M. Nichols
(c) Analysis and Interpretation of Data
Amelia M. Stanton; J. Gregory Hixon; Cindy M. Meston

### Category 2

(a) Drafting the Article
Amelia M. Stanton; Cindy M. Meston
(b) Revising It for Intellectual Content
Amelia M. Stanton; J. Gregory Hixon; Lindsey M. Nichols; Cindy M. Meston

### Category 3

(a) Final Approval of the Completed Article
Amelia M. Stanton; J. Gregory Hixon; Lindsey M. Nichols; Cindy M. Meston

## REFERENCES

1. Stanton AM, Lorenz TA, Pulverman CS, et al. Heart rate variability: a risk factor for female sexual dysfunction. **Appl Psychophysiol Biofeedback 2015;40:229-237.**
2. Kemp AH, Quintana DS, Gray MA, et al. Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. **Biol Psychiatry 2010;67:1067-1074.**
3. Licht CMM, de Geus JC, van Dyck R, et al. Association between anxiety disorders and heart rate variability in The Netherlands Study of Depression and Anxiety (NESDA). **Psychosom Med 2009;71:508-518.**
4. Chalmers JA, Quintana DS, Abbott MJ-A, et al. Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. **Front Psychiatry 2014;5:1-11.**
5. Chalmers JA, Heathers JAJ, Abbott MJ, et al. Worry is associated with robust reductions in heart rate variability: a transdiagnostic study of anxiety psychopathology. **BMC Psychol 2016;4:32.**
6. Lehrer PM, Vaschillo E, Vaschillo B, et al. Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow. **Psychosom Med 2003;65:796-805.**
7. Nolan RP, Kamath MV, Floras JS, et al. Heart rate variability biofeedback as a behavioral neurocardiac intervention to enhance vagal heart rate control. **Am Heart J 2005;149:1137.**
8. Beckham AJ, Greene TB, Meltzer-Brody S. A pilot study of heart rate variability biofeedback therapy in the treatment of perinatal depression on a specialized perinatal psychiatry inpatient unit. **Arch Womens Ment Health 2013;16:59-65.**
9. Windthorst P, Mazurak N, Kuske M, et al. Heart rate variability biofeedback therapy and graded exercise training in management of chronic fatigue syndrome: an exploratory pilot study. **J Psychosom Res 2017;93:6-13.**
10. Zucker TL, Samuelson KW, Muench F, et al. The effects of respiratory sinus arrhythmia biofeedback on heart rate variability and posttraumatic stress disorder symptoms: a pilot study. **Appl Psychophysiol Biofeedback 2009;34:135-143.**
11. Goessl VC, Curtiss JE, Hofmann SG. The effect of heart rate variability biofeedback training on stress and anxiety: a meta-analysis. **Psychol Med 2017;47:2578-2586.**
12. Lehrer P, Vaschillo B, Zucker T, et al. Protocol for heart rate variability biofeedback training. **Biofeedback 2013;41:98-109.**
13. Kanji N, White A, Ernst E. Autogenic training to reduce anxiety in nursing students: randomized controlled trial. **Issues Innov Nurs Educ 2006;53:729-736.**
14. Schultz JH, Luthe W. Autogenic training: a psychophysiological approach in psychotherapy. New York: Grune and Statton; 1959.
15. Kanji N. Autogenic training. **Complement Ther Med 1997;5:162-167.**
16. Mishima N, Kubota S, Nagata S. Psychophysiological correlates of relaxation induced by standard autogenic training. **Psychother Psychosom 1999;68:207-213.**
17. Miu AC, Heilman RM, Miclea M. Reduced heart rate variability and vagal tone in anxiety: trait versus state, and the effects of autogenic training. **Auton Neurosci 2009;145:99-103.**

18. Stanton A, Meston C. A single session of autogenic training increases acute subjective and physiological sexual arousal in sexually functional women. **J Sex Marital Ther 2016;43:601-617.**
19. Stanton AM, Meston CM. A single session of autogenic training increases acute subjective and physiological sexual arousal in sexually functional women. **J Sex Marital Ther** https://doi.org/10.1080/0092623x.2016.1211206. E-pub ahead of print.
20. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed text rev. Washington, DC: American Psychiatric Press; 2000.
21. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Publications; 2013.
22. Parish SJ, Goldstein AT, Goldstein SW, et al. Toward a more evidence-based nosology and nomenclature for female sexual dysfunctions—part II. **J Sex Med 2016;13:1888-1906.**
23. Althof SE, Meston CM, Perelman MA, et al. Opinion paper: on the diagnosis/classification of sexual arousal concerns in women. **J Sex Med 2017;14:1365-1371.**
24. Rosen R, Brown C, Heiman J, et al. The Female Sexual Function Index (FSFI): a multidimensional self-report instrument for the assessment of female sexual function. **J Sex Martial Ther 2000;26:191-208.**
25. Burri A, Spector T. Recent and lifelong sexual dysfunction in a female UK population sample: prevalence and risk factors. **J Sex Med 2011;8:2420-2430.**
26. Çayan S, Yaman Ö, Orhan İ, et al. Prevalence of sexual dysfunction and urinary incontinence and associated risk factors in Turkish women. **Eur J Obstet Gynecol Reprod Biol 2016;203:303-308.**
27. Wiegel M, Meston C, Rosen R. The Female Sexual Function Index (FSFI): cross-validation and development of clinical cutoff scores. **J Sex Marital Ther 2005;31:1-20.**
28. Sintchak G, Geer JH. A vaginal plethysmograph system. **Psychophysiology 1975;12:113-115.**
29. Rosen RC, Beck JG. Patterns of sexual arousal: psychophysiological processes and clinical applications. New York: Guilford Press; 1988.
30. Heiman JR. A psychophysiological exploration of sexual arousal patterns in females and males. **Psychophysiology 1977;14:266-274.**
31. Laan E, Everaerd W, Evers A. Assessment of female sexual arousal: response specificity and construct validity. **Psychophysiology 1995;32:476-485.**
32. Heiman JR, Rowland DL. Affective and physiological sexual response patterns: the effects of instructions on sexually functional and dysfunctional men. **J Psychosom Res 1983;27:105-116.**
33. Rellini AH, McCall KM, Randall PK, et al. The relationship between women's subjective and physiological sexual arousal. **Psychophysiology 2005;42:116-124.**
34. Linden W. Autogenic training: a clinical guide. New York: Guilford Press; 1990.
35. Xhyheri B, Manfrini O, Mazzolini M, et al. Heart rate variability today. **Prog Cardiovasc Dis 2012;55:321-331.**
36. Ponnusamy A, Marques JLB, Reuber M. Comparison of heart rate variability parameters during complex partial seizures and psychogenic nonepileptic seizures. **Epilepsia 2012;53:1314-1321.**
37. Pal GK, Adithan C, Ananthanarayanan PH, et al. Sympathovagal imbalance contributes to prehypertension status and cardiovascular risks attributed by insulin resistance, inflammation, dyslipidemia and oxidative stress in first degree relatives of type 2 diabetics. **PLoS One 2013;8:e78072.**
38. Pagani M, Lombardi F, Guzzetti S, et al. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. **Circ Res 1986;59:178-193.**
39. MacKinnon S, Gevirtz R, McCraty R, et al. Utilizing heartbeat evoked potentials to identify cardiac regulation of vagal afferents during emotion and resonant breathing. **Appl Psychophysiol Biofeedback 2013;38:241-255.**
40. Porges SW. The polyvagal perspective. **Biol Psychol 2007;74:116-143.**
41. Malliani A, Lombardi F, Pagani M. Power spectrum analysis of heart rate variability: a tool to explore neural regulatory mechanisms. **Br Heart J 1994;71:1-2.**
42. McCraty R, Shaffer F. Heart rate variability: new perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. **Glob Adv Heal Med 2015;4:45-61.**
43. Pulverman CS, Meston CM, Hixon JG. Automated artifact detection procedure for vaginal photoplethysmography data; Presented at: Annual Meeting of the International Society for the Study of Women's Sexual Health; 2015.
44. R Foundation. R: a language and environment for statistical computing; 2014. Available at: https://www.r-project.org. Accessed November 1, 2017.
45. Wood SN. Fast stable restricted maximum likelihood estimation of semiparametric generalized linear models. **J R Stat Soc Series B Stat Methodol 2011;73:3-36.**
46. Snijders TAB, Bosker RJ. Multilevel analysis: an introduction to basic and advanced multilevel modeling. 2nd ed. London: Sage Publishers; 2012.
47. Meston CM, Rellini AH, McCall K. The sensitivity of continuous laboratory measures of physiological and subjective sexual arousal for diagnosing women with sexual arousal disorder. **J Sex Med 2010;7:938-950.**
48. Pinheiro J, Bates D, DebRoy S, et al; Team RC. NLME: linear and nonlinear mixed effects models; 2017.
49. Aldao A. The future of emotion regulation research. **Perspect Psychol Sci 2013;8:155-172.**
50. Baumeister RF, Vohs KD. Handbook of self-regulation: research, theory, and application. New York: Guilford Press; 2004.
51. Williams DP, Cash C, Rankin C, et al. Resting heart rate variability predicts self-reported difficulties in emotion regulation: a focus on different facets of emotion regulation. **Front Psychol 2015;6:261.**

52. Thayer JF, Lane RD. Claude Bernard and the heart-brain connection: further elaboration of a model of neurovisceral integration. **Neurosci Biobehav Rev 2009;33:81-88.**
53. Thayer JF, Åhs F, Fredrikson M, et al. A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. **Neurosci Biobehav Rev 2012;36:747-756.**
54. Thayer JF, Lane RD. A model of neurovisceral integration in ER and dysregulation. **J Affect Disord 2000;61:201-216.**
55. Thayer JF, Sternberg EM. Neural aspects of immunomodulation: focus on the vagus nerve. **Brain Behav Immun 2010; 24:1223-1228.**
56. Heiman JR. Female sexual response patterns: interactions of physiological, affective, and contextual cues. **Arch Gen Psychiatry 1980;37:1311-1316.**
57. Barlow DH. Causes of sexual dysfunction: the role of anxiety and cognitive interference. **J Consult Clin Psychol 1986; 54:140-148.**
58. Purdon C, Holdaway L. Non-erotic thoughts: content and relation to sexual functioning and sexual satisfaction. **J Sex Res 2006;43:154-162.**
59. Meston CM. The effects of state and trait self-focused attention on sexual arousal in sexually functional and dysfunctional women. **Behav Res Ther 2006;44:515-532.**
60. Meston CM, Worcel M. The effects of yohimbine plus L-arginine glutamate on sexual arousal in postmenopausal women with sexual arousal disorder. **Arch Sex Behav 2002;31:323-332.**
61. Meston CM, Rellini AH, Telch MJ. Short- and long-term effects of ginkgo biloba extract on sexual dysfunction in women. **Arch Sex Behav 2008;37:530-547.**
62. Laan E, van Lunsen RHW, Everaerd W. Postmenopausal sexuality and the effects of tibolone: The effects of tibolone on vaginal blood flow, sexual desire and arousability in postmenopausal women. **Climacteric 2001;4:28-41.**
63. Laan E, van Lunsen RHW, Everaerd W, et al. The enhancement of vaginal vasocongestion by sildenafil in healthy premenopausal women. **J Womens Health Gend Based Med 2002; 11:357-365.**
64. Sipski ML, Rosen RC, Alexander CJ, et al. Sildenafil effects on sexual and cardiovascular responses in women with spinal cord injury. **Urology 2000;55:812-815.**
65. Chivers ML, Rosen RC. Phosphodiesterase type 5 inhibitors and female sexual response: faulty protocols or paradigms? **J Sex Med 2010;7:858-872.**
66. Foster R, Mears A, Goldmeier D. A literature review and case reprots on the use of phosphodiesterase inhibitors in the treatment of female sexual dysfunction. **Int J STD AIDS 2009;20:152-157.**
67. Frühauf S, Gerger H, Schmidt HM, et al. Efficacy of psychological interventions for sexual dysfunction: a systematic review and meta-analysis. **Arch Sex Behav 2013;42:915-933.**
68. Ravart M, Marchand A, Trudel G. The efficacy of a cognitive behavioural treatment model for hypoactive sexual desire disorder: an outcome study. **Can J Hum Sex 1996;5:279-294.**
69. Van Lankveld JJ, ter Kuile MM, de Groot HE, et al. Cognitive-behavioral therapy for women with lifelong vaginismus: a randomized waiting-list controlled trial of efficacy. **J Consult Clin Psychol 2006;74:168-178.**
70. Brotto LA. Mindful sex. **Can J Hum Sex 2013;22:63-68.**
71. Brotto LA, Basson R, Luria M. A mindfulness-based group psychoeducational intervention targeting sexual arousal disorder in women. **J Sex Med 2008;5:1646-1659.**
72. Brotto LA, Heiman JR, Goff B, et al. A psychoeducational intervention for sexual dysfunction in women with gynecologic cancer. **Arch Sex Behav 2008;37:317-329.**
73. Kabat-Zinn J. Full catastrophe living: using the wisdom of your body and mind to face stress, pain, and illness. 15th ed. New York: Delta/Bantam; 2005.
74. Stetter F, Kupper S. Autogenic training: a meta-analysis of clinical outcome studies. **Appl Psychophysiol Biofeedback 2002;27:45-98.**
75. Chivers ML, Seto MC, Lalumière ML, et al. Agreement of self-reported and genital measures of sexual arousal in men and women: a meta-analysis. **Arch Sex Behav 2010;39:5-56.**
76. Pulverman CS, Hixon JG, Meston CM. Uncovering category specificity of genital sexual arousal in women: the critical role of analytic technique. **Psychophysiology 2015;52:1396-1408.**
77. Janssen E, Everaerd W, Spiering M, et al. Automatic processes and the appraisal of sexual stimuli: toward an information processing model of sexual arousal. **J Sex Res 2000;37:8-23.**
78. Spiering M, Everaerd W, Janssen E. Priming the sexual system: implicit versus explicit activation. **J Sex Res 2003; 40:134-145.**