Routledge
Taylor & Francis Group

A Single Session of Autogenic Training Increases Acute Subjective and Physiological Sexual Arousal in Sexually Functional Women

Amelia Stanton and Cindy Meston

Department of Psychology, The University of Texas at Austin, Austin, Texas, USA

Heart rate variability (HRV) has recently been associated with female sexual function (Stanton, Lorenz, Pulverman, & Meston, 2015). Below-average HRV was identified as a possible risk factor for sexual arousal dysfunction and overall sexual dysfunction in women. Based on this newly established relationship between HRV and female sexual function, the present study examined the effect of autogenic training to increase HRV on acute physiological and subjective sexual arousal in women. Specifically, vaginal pulse amplitude (VPA), an index of genital sexual arousal, and subjective sexual arousal were assessed in 33 sexually functional women, aged 18 to 27, before and after a short session of autogenic training. Autogenic training, a relaxation technique that restores the balance between the activity of the sympathetic and the parasympathetic branches of the autonomic nervous system, has been shown to significantly increase HRV (Miu, Heilman, & Miclea, 2009). After autogenic training, significant increases in both VPA ($p < .05$) and subjective sexual arousal ($p < .005$) were observed. Moreover, change in HRV from pre- to post-manipulation significantly moderated changes in subjective sexual arousal ($p < .05$) when it was measured continuously during the presentation of the erotic stimulus. This cost-effective, easy-to-administer behavioral intervention may have important implications for increasing sexual arousal in women.

INTRODUCTION

Below-average resting heart-rate variability (HRV) has recently been associated with sexual arousal dysfunction and overall sexual dysfunction in women (Stanton, Lorenz, Pulverman, & Meston, 2015). Low resting HRV has also been linked with a variety of negative physical outcomes, namely poor cardiovascular health and negative psychophysiological health outcomes beyond female sexual dysfunction, including, but not limited to, depression (Kemp et al., 2010) and anxiety (Kemp, Quintana, Felmingham, Matthews, & Jelinek, 2012). Given these established relationships between low HRV and poor mental health outcomes, researchers have attempted to manipulate HRV, typically through biofeedback, in order to decrease symptoms (e.g., Karavidas et al., 2007; Kudo, Shinohara, & Kodama, 2014; Nolan et al., 2005). Another type of manipulation, known as autogenic training, has been shown to significantly increase

Address correspondence to Amelia Stanton, The University of Texas at Austin, 108 E. Dean Keeton Stop A8000, Austin, TX 78712-1043, USA. E-mail: amelia.stanton@utexas.edu

HRV (Mishima, Kubota, & Nagata, 1999; Miu et al., 2009) and to reduce symptoms associated with a wide variety of psychophysiological conditions, including depression and anxiety (for a review, see Stetter & Kupper, 2002). The present study is the first to assess changes in genital sexual arousal and subjective sexual arousal due to HRV manipulation via autogenic training.

In general, HRV is a useful signal for understanding the state of the autonomic nervous system (ANS). Normal variability in heart rate is governed by autonomic neural regulation of the heart (Saul, 1990). Specifically, the balancing action of the two branches of the ANS—the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS)—controls heart rate. The SNS is typically associated with energy mobilization, while the PNS controls the body's restorative functions. When SNS activity increases, PNS activity decreases, leading to cardioacceleration. Therefore, when SNS activity decreases, PNS activity increases, resulting in cardiodeceleration. The degree of variability in the lengths of time between successive heartbeats (HRV) provides important information about the regulation of these systems and about the heart's ability to respond. Higher HRV is reflective of organized variability, rather than static levels of SNS or PNS input, which enables the body to adapt to constantly changing environmental demands (Thayer, Yamamoto, & Brosschot, 2010).

In the past 30 years, numerous studies have pointed to the significant relationship between autonomic imbalance and physiological, specifically cardiovascular, health. When one branch of the ANS dominates the other, researchers believe that dynamic flexibility and physiological health are compromised. This most commonly occurs when the sympathetic system is hyperactive and the parasympathetic system is hypoactive (Thayer et al., 2010). When the SNS dominates for long periods of time, the body cannot keep up with the branch's energy demands. The inability to meet these demands increases the risk of mortality from a variety of conditions and diseases, most notably cardiovascular disease (CVD). Measures of HRV, including the standard deviation of the normal heartbeat interval (SDNN, which is used in the present study), can be analyzed to assess autonomic imbalance and the activity of the vagus nerve, which is a key component of the PNS. The vagus nerve regulates organ systems that do not involve conscious operation, including the heart. Findings from several epidemiological studies (e.g., Schroeder et al., 2003; Singh et al., 2000) provide strong evidence that vagal activity, indexed by HRV, is lower in individuals with cardiometabolic disease.

There is also a significant relationship between autonomic imbalance and female sexual dysfunction. A series of studies by Meston and colleagues has indicated that moderate SNS dominance (relative to parasympathetic dominance) predicts women's level of genital arousal in the laboratory (Meston & Gorzalka, 1995, 1996; Meston & Heiman, 1998). Specifically, there is evidence for a curvilinear relationship between sympathetic nervous-system activation and women's physiological sexual arousal (Lorenz, Harte, Hamilton, & Meston, 2012). Using decreases in HRV as a specific marker of increased SNS activity, Lorenz and colleagues (2012) found that moderate increases in SNS activity were associated with higher genital arousal, while very low or very high SNS activation was associated with lower genital arousal. Based on these results, they concluded that there is likely an optimal level of SNS activation for women's physiological sexual arousal.

These laboratory studies examined the relationship between women's genital sexual arousal and different levels of experimentally induced SNS activation, but they did not investigate the effect of variations in resting-state autonomic balance on sexual arousal function and overall

sexual function. To investigate the possibility that resting-state autonomic balance may also be related to sexual arousal function and overall sexual function, Stanton and colleagues (2015) measured resting-state HRV and sexual function using the Female Sexual Function Index (FSFI; Rosen et al., 2000) in 72 women aged 18 to 39. In this study, women with below-average resting HRV were more likely to report sexual arousal problems and overall sexual problems than women with average and above-average resting HRV. In addition, HRV measured during an erotic film clip (rather than during a neutral film clip, when resting HRV was measured) was also a significant predictor of sexual function. Based on these findings, the authors concluded that low-resting HRV could be a risk factor for female sexual dysfunction. As there are currently no established biological risk factors for female sexual dysfunction, this relationship between low resting-state HRV and sexual dysfunction is particularly notable.

Given that low resting-state HRV is now associated with sexual arousal concerns and overall sexual dysfunction in women, it is logical to consider the manipulation of resting HRV as a means to increase arousal. Research has indicated that autogenic training is an effective way to manipulate HRV. Autogenic training is a psychophysiological relaxation technique that is based on passive concentration of bodily perceptions, such as heaviness and warmth in the legs or arms, that are conjured through verbal self-suggestions (Stetter & Kupper, 2002). Established by Schultz and Luthe (1959), the theory underlying autogenic training suggests that the modulation of physiological functions can be achieved through changes in mental processes (Mitani, Fujita, Sakamoto, & Shirakawa, 2006). There is a recognized association between autogenic training and changes in HRV. One study reported that healthy volunteers who were taught how to engage in autogenic training over a period of three months showed increased variability in interbeat interval lengths (Mishima et al., 1999). Another study indicated that, in comparison to mental stress, brief autogenic training facilitated increases in HRV (Miu et al., 2009). Miu and colleagues (2009) developed an autogenic training protocol that fit into a single session, and all of their subjects were naïve to the training procedure. The present study builds upon the work of Miu and colleagues (2009) by investigating the effect of a single session of autogenic training on different outcome variables: acute VPA and subjective sexual arousal.

A large body of research has shown that autogenic training improves physiological *and* psychophysiologcal health, not just HRV. Physiological health generally refers to the health and vitality of the body, which includes both individual organs and body systems. A meta-analysis revealed that autogenic training has a medium-size effect on clinical outcomes in patients who have been diagnosed with coronary heart disease (Stetter & Kupper, 2002). Autogenic training has also led to improvements in symptoms associated with irritable bowel disease (Shinozaki et al., 2010) and in motor performance among individuals with Parkinson's disease (Ajimsha, Majeed, Chinnavan, & Thulasyammal, 2014). Psychophysiological health is characterized by an interaction of physiological (body systems) and psychological (mental) processes. Female sexual arousal, for example, is a psychophysiological phenomenon, as it involves both physical responsiveness (e.g., vasocongestion, vaginal lubrication and expansion, swelling of the genitalia) and subjective ratings (e.g., being mentally "turned on") of a stimulus. Other psychophysiological processes include emotions such as joy or anxiety, or states of mental arousal that are associated with stress or pain. At an extreme, psychophysiological processes also contribute to psychopathologies such as depression via chronic recruitment of the autonomic nervous system. Autogenic training can influence these psychopathologies, increasing positive aspects of arousal and decreasing negative aspects. For example, research has demonstrated that autogenic training is associated

with reductions in anxiety (e.g., Kanji, White, & Ernst, 2006), pain (e.g., Kanji, 2000), and depression (Krampen, 1999).

To our knowledge, there have been no investigations of the effect of HRV manipulation on acute genital and subjective sexual arousal in women. Based both on evidence that low resting-state HRV predicts female sexual dysfunction (Stanton et al., 2015) and that autogenic training can significantly increase HRV (Miu et al., 2009), we predict that raising resting-state HRV via autogenic training will increase both acute genital sexual arousal and acute subjective sexual arousal in sexually functional women.

METHOD

Participants

Participants were female undergraduate students recruited from the psychology student subject pool of a large university through flyers and print advertisements that explained the sexual nature of the experiment. Prospective participants were instructed to read about the study's inclusion/exclusion criteria online and then sign up for an available time slot if they met all of the inclusion criteria and none of the exclusion criteria. Once they arrived in the lab for their scheduled study sessions, prospective participants were given an eligibility screener to ensure that they met the inclusion criteria. Only women aged 18 to 30 who scored above a 26.55 on the FSFI (Rosen et al., 2000), the clinical cutoff for healthy sexual function in women, were included in the study. Exclusion criteria included history of sexually transmitted diseases; current active or untreated pelvic, vaginal, or urinary tract infections; history of major pelvic surgery; history of neurological 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.

In exchange for university credit, 40 women signed up via the online portal to participate in the study. Of these 40 women, seven were excluded from participation based on the results of the eligibility screener. Four women were excluded because they had not been sexually active over the past month (which was necessary in order to obtain a valid FSFI score); two women were excluded because they were taking antidepressant medications; and one woman was excluded because her FSFI score fell below the clinical cutoff. The final sample included 33 women aged 18 to 27, with an average age of 19.3 ($SD = 1.7$). For full demographics, see Table 1.

Measures

Heart Rate Variability

Heart rate was measured at a rate of 200 samples/sec via electrocardiography (ECG). This sampling rate is adequate to produce a minimally biased estimate of time domain measures of HRV used in the present study (Hejjel & Roth, 2004; Ziemssen, Gasch, & Ruediger, 2008). The research administrator positioned disposable electrodes on the participant's upper right chest,

TABLE 1
Participant Characteristics ($N = 33$)

	M	SD
Age (years)	19.3	1.7
FSFI (total score)	30.9	2.3
	n	%
Race[a]		
African American/Black	3	9.1
Caucasian/White	24	72.7
Asian American	4	12.1
Other	1	3.0
Relationship status		
Single, not dating	5	15.2
Single, dating	8	24.2
In a committed relationship	20	60.6
Sexual orientation		
Exclusively heterosexual	19	57.6
Predominantly heterosexual	13	39.4
Bisexual	1	3.0

Note. FSFI = Female Sexual Function Index (Rosen et al., 2000). [a]Participants could identify as belonging to more than one race or choose to not identify any race.

lowermost left rib, and inner right ankle, and then the participant attached the leads after the administrator left the room. The signal from the ECG leads was detected using an MP100 data-acquisition unit that was equipped with AcqKnowledge 3.9.1 software (Biopac Systems, Inc., Santa Barbara, CA).

Genital Sexual Arousal

Genital sexual arousal was assessed with vaginal photoplethysmography (Sintchak & Geer, 1975), which consists of a tampon-shaped device, known as the vaginal plethysmograph, that is inserted into the vagina. The vaginal plethysmograph transmits a beam of light into the vaginal canal, which is then detected by photocells in the vaginal wall. This process produces two physiological measurements: vaginal blood volume (VBV) and vaginal pulse amplitude (VPA). Vaginal blood volume, the direct signal, highlights slow changes in the pooling of blood in the vaginal tissue (Hatch, 1979). Vaginal pulse amplitude, the indirect signal, reflects short-term changes in the engorgement of blood in the vaginal tissue (Rosen & Beck, 1988). Considered to be the more sensitive of the two indices of change in vaginal blood volume (Heiman, 1977), VPA has been shown to be a reliable index of women's physiological, or genital, arousal (Laan, Everaerd, Van Aanhold, & Rebel, 1993) and was therefore used in the present study. Sampled at a rate of 200 samples/sec throughout both of the erotic films, VPA data were recorded in millivolts and collected by an MP100 data-acquisition unit equipped with AcqKnowledge 3.9.3 software (Biopac Systems, Inc., Santa Barbara, CA).

Subjective Sexual Arousal

Subjective sexual arousal was measured both discretely and continuously. Discrete measurement of the construct was calculated by summing the scores of the three original subjective sexual arousal items from Heiman and Rowland's (1983) Film Scale, which assesses sexual arousal as well as positive and negative affect in response to an erotic film. These three items include an assessment of overall "sexual arousal," a sense of "mental sexual arousal," and one reverse-scored item on feeling "sexually turned off." On two occasions during the experiment (immediately following the first neutral-erotic film sequence and immediately following the second neutral-erotic film sequence), participants rated the degree to which they experienced each of the three items on a 7-point Likert scale, and then the three scores were combined into a single subjective sexual arousal score for each of the two time points. Subjective sexual arousal was also measured continuously during the film presentation with an arousometer (Rellini, McCall, Randall, & Meston, 2005). The arousometer is a computer mouse attached to a lever, which is numbered from 0 to 7. During the neutral-erotic film sequences, the participant is instructed to move the mouse up or down as she feels her mental sexual arousal changing. The device is placed on a small table to the right of the participant's chair for ease of access during the film sequences.

Stimulus Materials

Two 8-minute audiovisual films were used as stimulus materials in this study. Both films included a two-minute neutral segment followed by a six-minute erotic segment. The neutral segments of both films depicted different images of landscapes accompanied by classical music. The erotic segments of both films featured different heterosexual couples engaging in two minutes each of foreplay, cunnilingus, and penetrative sexual intercourse. Both films have been found to be arousing to women in past studies conducted in our laboratory. The films were presented in counterbalanced order and matched for content.

Procedure

A female research administrator oriented participants to the study procedures and obtained informed consent. Testing sessions took place in a private room equipped with an intercom that participants used to communicate with the researcher. Participants were first instructed in how to use the vaginal photoplethysmograph and how to attach the ECG wires before the testing session began. Then, after the researcher applied the electropads and left the room, the participants inserted the vaginal probe and attached the ECG wires. After a two- to four-minute habituation period when no physiological measurements were taken, participants viewed the first eight-minute stimulus film, during which their VPA and HRV were measured.

After the first film, the participants completed a few questionnaires, which typically took them five to 10 minutes, and then listened to a 14-minute autogenic training recording. The recording was adapted from an autogenic training manual developed by Linden (1990). Participants were instructed to close their eyes and listen to a relaxation exercise. They were also asked to follow along with the recording and to try to remain as still as possible. The recording focused specifically on repeatedly inducing sensations of heaviness and heat in the arms and legs. Following the autogenic training, participants watched the second eight-minute film while their VPA and HRV

were measured. After the second film, participants completed measures on subjective sexual arousal and provided demographic information. Participants were compensated for their time in university course credit.

Data Analysis

Data Reduction

Heart-rate variability data were collected using AcqKnowledge 3.9.3 software. The Acq-Knowledge peak finder function was used to isolate the interbeat (NN) intervals, which were then exported to Microsoft Excel for processing. The Excel documents were converted to Text files, which were then imported to the Kubios HRV Analysis Software (Biosignal Analysis and Medical Imaging Group, University of Kuopio, Kuopio, Finland). This software calculates the standard deviation of the normal heartbeat intervals (SDNN), which was used as an index of HRV in the present study. SDNN has been shown to be an accurate marker of HRV, and, as such, it is one of the most widely used measures of HRV (Xhyheri, Manfrini, Mazzolini, Pizzi, & Bugiardini, 2012).

Vaginal pulse amplitude data were exported from AcqKnowledge 3.9.3 to Microsoft Excel for processing. Movement artifacts in the data were identified and removed by an automatic processing procedure (Pulverman, Meston, & Hixon, 2015) that has been shown to effectively remove outliers more accurately than visual inspection. This automatic processing procedure uses the R software environment (Team, 2010). For a more comprehensive explanation of this data-reduction procedure, see Pulverman et al. (2015).

Resting Heart Rate Variability

A paired-samples *t* test was used to determine the effect of autogenic training on resting HRV. Measurements of baseline (resting) SDNN, collected during the two-minute neutral segment before the first erotic film, were compared to SDNN measurements collected during the two-minute neutral segment immediately following the 14 minutes of autogenic training, prior to the start of the second erotic film.

Vaginal Pulse Amplitude

After movement artifacts were removed from the VPA data via the automatic processing procedure, a paired-samples *t* test was used to determine the effect of autogenic training on VPA. Premanipulation VPA was measured during the two-minute neutral segment and the six-minute erotic segment of the first film, and postmanipulation VPA was measured during the two-minute neutral segment and the six-minute erotic segment of the second film, which followed the 14 minutes of autogenic training. For each participant, the mean VPA level during the premanipulation neutral film segment was subtracted from the mean VPA level during the premanipulation erotic film segment to obtain a mean VPA difference score ($VPA_{eroticfilm1mean} - VPA_{neutralfilm1mean}$) for the premanipulation phase of the experiment. This difference score was divided by mean VPA during the first neutral film and then multiplied by 100 to yield a percent change score for

the premanipulation film sequence. The same procedure was carried out for the postmanipulation phase, resulting in a postautogenic training mean VPA difference score ($VPA_{eroticfilm2mean} - VPA_{neutralfilm2mean}$) and then finally in a percent change score for the postmanipulation film sequence.

Subjective Sexual Arousal

A paired-samples t test was used to determine the effect of autogenic training on subjective, or psychological, sexual arousal. Immediately following the presentation of the first neutral-erotic film sequence, participants completed the subjective sexual arousal items from Heiman and Rowland's (1983) Film Scale. Participants then listened to the autogenic training recording, after which they watched another neutral-erotic film sequence. Then, participants answered the same subjective arousal items from the Heiman and Rowland (1983) Film Scale, and the two subject arousal scores were compared.

Change in HRV as a Moderator of Change in Vaginal Pulse Amplitude

Hierarchical linear modeling software (HLM7; Raudenbush, Bryk, Cheong, Congdon, & Du Toit, 2011) was used to examine HRV as a moderator of change in continuous vaginal pulse amplitude from pre- to postmanipulation. A statistical technique that models parameters that vary at more than one level, hierarchical linear modeling (HLM) is particularly useful when examining VPA because baseline VPA differs by individual. This technique allows for each participant to act as her own control, as individual slopes and intercepts at Level 1 become outcome variables at Level 2.

A Level 1 analysis was conducted to evaluate the relationship between continuous VPA and film (where $0 =$ premanipulation, $1 =$ postmanipulation). Here, film is treated as a simple Level 1 fixed effect. The equation for this analysis is listed below:

$$VPA = \beta_{0j} + \beta_{1j}(FILM) + r_{ij} \quad \text{(Level 1)}$$

In the above equation, β_{0j} represents the intercept, or the expected VPA, for participant j when film equals 0; β_{1j} represents the slope, or the expected change in VPA, that is associated with change in film from pre- to postmanipulation for participant j, and r_{ij} is an error term.

A Level 2 moderation analysis was conducted to determine if raw change in HRV from pre- to postmanipulation accounted for changes in the relationship between film and VPA. Raw change in HRV was centered around the grand mean. The equation for this analysis is listed below:

$$VPA = \beta_{0j} + \beta_{1j}(FILM) + r_{ij} \quad \text{(Level 1)}$$

$$\beta_{0j} = \gamma_{00} + \gamma_{01j} + u_{0j} \quad \text{(Level 2)}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11j}(RAW\ CHANGE\ IN\ HRV) + u_{1j}$$

In the above equations, γ_{00} and γ_{10} are the mean intercepts for all participants adjusted for film; γ_{01j} represents the association between film and VPA for participant j; γ_{11j} is the association between film and VPA moderated by change in HRV, and u_{0j} and u_{1j} are the error terms.

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

Similarly, hierarchical linear modeling software (HLM7; Raudenbush et al., 2011) was used to examine HRV as a moderator of change in continuous subjective sexual arousal from pre- to postmanipulation.

A Level 1 analysis was conducted to evaluate the relationship between arousometer scores, or continuous subjective arousal, and film (where 0 = premanipulation, 1 = postmanipulation). Film was treated as a simple Level 1 fixed effect. The equation for this analysis is listed below:

$$\text{Arousometer} = \beta_{0j} + \beta_{1j}(FILM) + r_{ij}, \quad \text{(Level 1)}$$

In the above equation, β_{0j} represents the intercept, or the expected subjective arousal for participant j when film equals 0; β_{1j} represents the slope, or the expected change in subjective arousal, that is associated with change in film from pre- to postmanipulation for participant j, and r_{ij} is an error term.

A Level 2 mediation analysis was conducted to determine if raw change in HRV from pre- to postmanipulation accounted for change in the relationship between film and subjective sexual arousal. Raw change in HRV was centered around the grand mean. The equation for this analysis is listed below:

$$\text{Arousometer} = \beta_{0j} + \beta_{1j}(FILM) + r_{ij}, \quad \text{(Level 1)}$$

$$\beta_{0j} = \gamma_{00} + \gamma_{01j} + u_{0j} \quad \text{(Level 2)}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11j}(RAW\ CHANGE\ IN\ HRV) + u_{1j},$$

In the above equations, γ_{00} and γ_{10} are the mean intercepts for all participants adjusted for film; γ_{01j} represents the association between film and subjective sexual arousal for participant j; γ_{11j} is the association between film and subjective sexual arousal mediated by change in HRV, and u_{0j} and u_{1j} are the error terms.

RESULTS

Heart Rate Variability

Average resting HRV (i.e., HRV during the neutral film segments) across participants differed significantly from preautogenic training to postautogenic training, $t = -4.83, p < .0001$ (Table 2). Specifically, SDNN levels measured during the neutral film that followed the autogenic training intervention were significantly greater ($M = 64.13, SE = 4.04$) than SDNN levels measured at baseline ($M = 52.32, SE = 3.61$), indicating that the manipulation may have targeted and increased HRV. This difference had a large effect size ($d = 0.840$).

Vaginal Pulse Amplitude

There was a significant difference between percent change in VPA before the autogenic training manipulation and percent change in VPA following the manipulation, $t = -2.06, p < .05$ (Figure 1).

TABLE 2
Results of Paired Samples *t* Tests

	M	*SD*	*SE*	*n*	95% CI for Mean Difference	*t*	*df*
Premanipulation HRV to postmanipulation HRV	−11.80	14.05	2.45	33	[−16.78, −6.82]	−4.83***	32
Premanipulation percent change in VPA to postmanipulation percent change in VPA	−13.57	35.36	6.16	33	[−26.11, −1.03]	−2.21*	32
Premanipulation subjective sexual arousal to postmanipulation subjective sexual arousal	−1.61	2.41	0.42	33	[−2.46, −0.75]	−3.83**	32

Note. CI = confidence interval; HRV = heart rate variability; VPA = vaginal pulse amplitude.
*$p < .05$. **$p < .01$. ***$p < .0001$.

In other words, increasing HRV may have facilitated a significant increase in genital sexual arousal. This difference in VPA pre- to postmanipulation had a small effect size ($d = 0.37$).

Subjective Sexual Arousal

There was also a significant difference between mean subjective arousal score before the manipulation and mean subjective arousal score after the manipulation, $t = -3.83$, $p < .001$ (Figure 2). Overall, after listening to the autogenic training recording, participants experienced a significant increase in subjective sexual arousal, as measured by the combined score of the three subjective arousal questions on the Heiman and Rowland (1983) Film Scale. The pre-to-post difference in subjective sexual arousal scores had a medium to large effect size ($d = 0.67$).
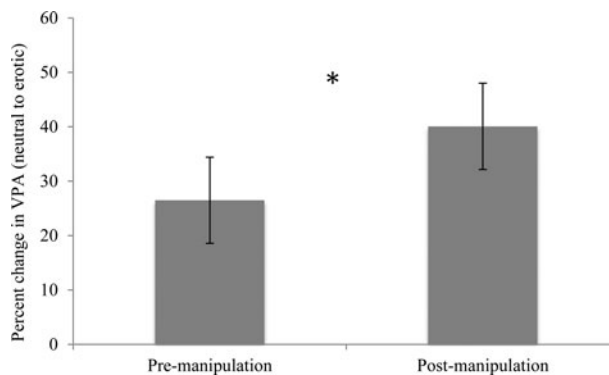

FIGURE 1 Percent change in VPA before and after HRV manipulation.
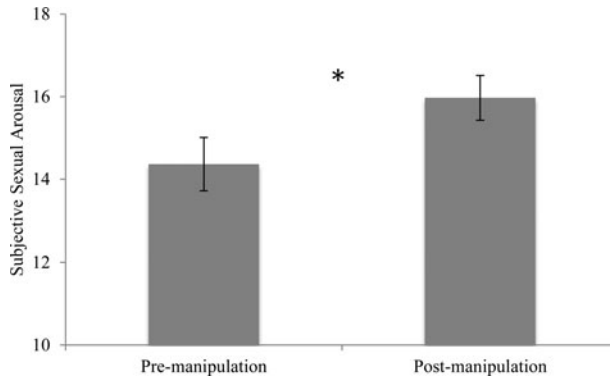
FIGURE 2 Mean subjective sexual arousal, measured via Heiman and Rowland's (1983) Film Scale, before and after HRV manipulation.

Change in HRV as a Moderator of Change in Vaginal Pulse Amplitude

The null model (model with no predictors) was significant ($X^2 = 9361.12$, $p < .001$), which suggested that multilevel modeling was warranted. Within the multilevel model, film significantly predicted VPA, $\beta = 0.56$, $t = 8.18$, $p < 0.001$. Premanipulation (film = 0), a participant could be expected to have a mean VPA of 7.67 mV. Postmanipulation (film = 1), a participant could be expected to have a mean VPA of 8.23 mV. In other words, there was a 0.56 mV increase in VPA on average from pre- to postmanipulation. When raw change in HRV was added to the model, it did not moderate the relationship between film and VPA, $\beta = 0.0068$, $t = 1.39$, $p = .16$.

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

The null model (model with no predictors) was significant ($X^2 = 2060.80$, $p < .001$), which suggested that multilevel modeling was warranted. Within the multilevel model, film significantly predicted subjective sexual arousal, $\beta = 0.076$, $t = 9.78$, $p < 0.001$. This model shows that the predicted mean of subjective sexual arousal premanipulation (film = 0) was 0.35 units; postmanipulation (film =1), the predicted mean of subjective sexual arousal increased to 0.43 units. That is, there was a 0.076 unit increase in subjective sexual arousal on average from pre- to postmanipulation. When raw change in HRV was added to the model, it significantly moderated the relationship between film and subjective sexual arousal, $\beta = 0.0012$, $t = 2.151$, $p = .031$. Therefore, greater change in HRV from pre- to postmanipulation was associated with larger increases in continuous subjective arousal.

DISCUSSION

This study examined the effect of HRV manipulation on acute genital and subjective sexual arousal in sexually healthy women. Only 14 minutes of autogenic training, a relaxation technique known to restore the balance between the parasympathetic and sympathetic branches of the

autonomic nervous system, led to significant increases in both physiological and subjective indices of sexual arousal. Moreover, change in HRV from premanipulation to postmanipulation significantly moderated changes in continuous subjective arousal from the first neutral-erotic film sequence to the second neutral-erotic film sequence. These findings may have implications for facilitating increases in sexual arousal, particularly subjective sexual arousal, in women.

Heart-rate variability manipulation may increase genital sexual arousal via direct vascular mechanisms. Physiological sexual arousal depends on a specific degree of blood pressure in the clitoris and vaginal walls. In women, smooth muscle relaxation and vasodilation facilitate increased blood flow to the genitals. Diminished pelvic blood flow can lead to decreased inflow to the clitoris and the vagina, potentially resulting in fibrosis of the vaginal wall and clitoral smooth muscle (Park et al., 1997). Although there are other physiological and psychological factors that contribute to female sexual dysfunction, arterial insufficiency and other vascular problems can affect the development and maintenance of sexual problems. Heart rate variability is considered to be a marker of heart health and is thought to reflect the heart's ability to quickly adapt to changing external triggers (Acharya, Joseph, Kannathal, Lim, & Suri, 2006). In other words, HRV can indicate the body's ability to coordinate increased blood flow to certain regions as warranted by environmental demands, which is particularly relevant to sexual situations as the body responds to both internal and external sexual cues.

Heart-rate variability manipulation may also indirectly affect female sexual arousal, specifically subjective sexual arousal, by targeting the processing of emotional cues. Research suggests that HRV level is an index of emotional responding. Higher levels of respiratory sinus arrhythmia, the high-frequency band of HRV, have been linked to greater self-reported emotion regulation in adults (Fabes & Eisenberg, 1997) and to active coping strategies in recently bereaved individuals (O'Connor, Allen, & Kaszniak, 2002). Generally, it is accepted that higher HRV reflects a greater capacity for regulation of emotional responses (Appelhans & Luecken, 2006). The ability to regulate emotional expression is considered to be critical for adaptive functioning (Baumeister & Vohs, 2004), and a lack of adequate control over emotion may be a contributing catalyst to the development of psychopathology (Calkins, 1994). Emotion regulation may be particularly germane to female sexual arousal function (Heiman, 1980), as emotional bonding is considered to be an important cue for sexual activity (McCall & Meston, 2006) and thus perhaps for sexual arousal as well. This conclusion, however, is tentative; future studies should directly examine the relationship among changes in affect, changes in subjective sexual arousal, and changes in HRV.

The present study was conducted with sexually functional women without sexual arousal problems. An important next step is to test the autogenic training protocol on women who are distressed by diminished and/or lacking mental sexual arousal and/or by a reduction in or lack of pleasurable genital sensations. If the findings of this study are replicated among these women, then autogenic training may prove to be a valuable tool for clinicians who work with this population. Women with clinically relevant sexual dysfunction have been shown to report lower subjective sexual arousal during the presentation of an erotic stimulus compared to their sexually functional counterparts (Morokoff & Heiman, 1980). Given that changes in HRV moderated changes in continuous subjective sexual arousal, HRV manipulation may be beneficial for women who are struggling to feel mentally "turned on." Though vaginal photoplethysmography has not been shown to differentiate between women with and without sexual arousal dysfunction (e.g., Laan, Van Driel, & Van Lunsen, 2008), it is possible that higher levels of vaginal blood flow are

associated with increased vaginal sensations compared to lower levels of vaginal blood flow. Unfortunately, higher levels of sexual arousal, up to and during orgasm, are not often obtained during laboratory experiments (Laan, Everaerd, Van der Velde, & Geer, 1995), which makes it challenging to test the association between increased genital arousal, measured by VPA, and increased self-reported genital sensations.

Currently, there are no FDA-approved drugs for genital arousal problems in women. When women report having diminished genital sensations, their providers typically offer them topical lubricants, which help mask impairments in vaginal lubrication, even when lubrication is not their primary concern. Anecdotally, many women do report an increase in genital sensations with lubricant use; however, lubricants may not enhance all genital sensations, which, in addition to genital wetness, include genital pulsing or throbbing, genital or clitoral fullness, and genital warmth. There is some evidence from limited placebo-controlled studies that indicates that Viagra increases genital engorgement in healthy, premenopausal women (Laan, Smith, Boolell, & Quirk, 2002) and in postmenopausal women with genital arousal problems (Basson & Brotto, 2003); however, studies in general have not shown that this drug increases psychological sexual arousal. The EROS clitoral therapy device (Urometrics, St. Paul, MN), approved by the FDA in 2000 to treat sexual arousal and orgasmic disorders, increases vasocongestion in the clitoral and labial region via a suction mechanism, which has been shown to increase genital sensations (Billups et al., 2001) but not subjective sexual arousal.

Psychological treatments of sexual arousal problems generally include elements from traditional sex therapy, such as sensate focus and masturbation training, as well as mindfulness-based exercises (for a review, see Brotto, Bitzer, Laan, Leiblum, & Luria, 2010). However, there is a lack of clear treatment protocols for this population, and, according to Brotto and colleagues (2010), there is also great need for controlled efficacy studies in this area. The few studies that have examined the effects of certain psychological treatments on arousal function have involved multiple sessions, which involve a relatively large time commitment for both the provider and the patient, and have generally shown increases in subjective but not physiological sexual arousal (e.g., Brotto, Basson, & Luria, 2008; Brotto et al., 2012).

It is important to note some limitations of the present study. Given that we did not include a control group, we cannot definitively conclude that the autogenic training, rather than repeated testing (i.e., repeated exposure to erotic material), led to increases in acute physiological and subjective sexual arousal. However, participants' mean VPA during the neutral segment of the second film sequence, which followed the manipulation, did not significantly differ from their mean VPA during the neutral segment of the first film sequence. This suggests both that there was enough time between the presentations of the two erotic stimuli for women to return to baseline levels of genital responsiveness and that there was no carryover of increased genital blood flow from the first film sequence to the second film sequence. In addition, participants in this study were undergraduate women who were enrolled in a psychology course at a large university. Therefore, the findings of this study may not generalize to the larger population of sexually functional women. Finally, we did not measure indices of compliance or attentiveness to the autogenic training recording; therefore, we must assume that participants followed the instructions provided to them by the experimenter. The assumption that participants are listening to and following an experimenter's instructions is not uncommon in psychological research. In future studies, it may be beneficial to assess level of attentiveness to autogenic training recordings, as this variable may act as a treatment moderator.

Another limitation of the current study was the timing of the autogenic training recording in reference to the two neutral-erotic film sequences. The design of the study situated the autogenic training recording between the two neutral-erotic film sequences, such that the first film sequence was always the "premanipulation" sequence and the second sequence was always the "postmanipulation" sequence. Based on this design, we cannot definitively determine that the autogenic training recording, rather than the effect of time, facilitated the observed changes in acute physiological and subjective sexual arousal. If the mechanism of action driving the effects of the autogenic training is indeed its ability to manipulate autonomic balance, then the timing of the autogenic training should not impact study findings. To conclusively rule out the effect of timing as a causal factor, future research may randomize the order of the autogenic training recording, such that participants would first listen to the autogenic training recording, watch a neutral-erotic film sequence (which, in this case, would be the "postmanipulation" sequence), return to baseline arousal, and then watch another neutral-erotic film sequence (which would be the equivalent of the "premanipulation" sequence).

Though one session of autogenic training has been shown to increase HRV (Miu et al., 2009), autogenic training protocols typically involve 1-2 sessions per week with a clinician for 4-8 weeks (e.g., Jain et al., 2007; Mitani et al., 2006). Our brief 14-minute intervention cannot be considered a complete, comprehensive round of autogenic training. Rather, it was an introduction to the procedure that was intended to produce acute increases in resting HRV. It may be useful to increase the number of autogenic training sessions in future research. It is likely that additional sessions of training will be necessary in order for women to experience long-term rather than acute gains in subjective and physiological sexual arousal. In addition, it may also be worthwhile to compare HRV-related changes in sexual arousal due to prolonged autogenic training with HRV-related changes in sexual arousal due to HRV biofeedback, another form of HRV manipulation. Briefly, HRV biofeedback involves slowing down the breath to a frequency of about 5-6 breaths per minute, which, in most people, maximizes respiratory sinus arrhythmia. Generally, HRV biofeedback protocols range from five sessions (e.g., Lehrer, Vaschillo, & Vaschillo, 2000) to 10 sessions (Lehrer et al., 2013), although some clinicians may choose to extend the length of the protocols depending on the severity of the symptoms. Regular practice of this technique has resulted in clinically significant improvement for a variety of disorders, and, given the findings of the present study, it is possible that HRV biofeedback may also increase sexual arousal, particularly mental sexual arousal.

In summary, our finding that autogenic training acutely increased both subjective and physiological components of sexual arousal is notable in that it differs from past research examining drug or psychotherapy treatments that generally increase only one or the other of these components of sexual arousal. In addition, the finding that change in resting HRV from pre to postautogenic training moderated changes in continuous subjective arousal suggests that increasing HRV may help facilitate increased mental sexual arousal. Autogenic training is easy to learn in a single session with a psychologist or another behavioral health provider, and it has the advantage of being free or very cheap. Recordings of the procedure can be downloaded from online platforms, which provide easy access to patients who cannot afford therapy. In addition, patients who are reluctant to seek out therapy for their sexual concerns due to feelings of shame or discomfort in discussing sexual issues will be able to listen to the autogenic training protocol from the privacy of their own homes. Though preliminary, the present study highlights the importance of continued examinations of the effects of HRV manipulation on sexual arousal in women.

REFERENCES

Acharya, U. R., Joseph, K. P., Kannathal, N., Lim, C. M., & Suri, J. S. (2006). Heart rate variability: A review. *Medical & Biological Engineering & Computing*, *44*(12), 1031–1051. doi:10.1007/s11517-006-0119-0

Ajimsha, M. S., Majeed, N. A., Chinnavan, E., & Thulasyammal, R. P. (2014). Effectiveness of autogenic training in improving motor performances in Parkinson's disease. *Complementary Therapies in Medicine*, *22*(3), 419–425. doi:10.1016/j.ctim.2014.03.013

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*(3), 229–240. doi:10.1037/1089-2680.10.3.229

Basson, R., & Brotto, L. A. (2003). Sexual psychophysiology and effects of sildenafil citrate in oestrogenised women with acquired genital arousal disorder and impaired orgasm: A randomised controlled trial. *BJOG: An International Journal of Obstetrics and Gynaecology*, *110*(11), 1014–1024. doi:10.1111/j.1471-0528.2003.02438.x

Baumeister, R. F., & Vohs, K. D. (2004). *Handbook of self-regulation: Research, theory, and applications*. New York, NY: Guilford Press.

Billups, K. L., Berman, L., Berman, J., Metz, M. E., Glennon, M. E., & Goldstein, I. (2001). A new non-pharmacological vacuum therapy for female sexual dysfunction. *Journal of Sex & Marital Therapy*, *27*(5), 435–441. doi:10.1080/713846826

Brotto, L. A., Basson, R., & Luria, M. (2008). A mindfulness-based group psychoeducational intervention targeting sexual arousal disorder in women. *The Journal of Sexual Medicine*, *5*(7), 1646–1659. doi:10.1111/j.1743-6109.2008.00850.x

Brotto, L. A., Bitzer, J., Laan, E., Leiblum, S. R., & Luria, M. (2010). Women's sexual desire and arousal disorders. *Journal of Sexual Medicine*, *7*(1, Pt 2), 586–614.

Brotto, L. A., Erskine, Y., Carey, M., Ehlen, T., Finlayson, S., Heywood, M., . . . Miller, D. (2012). A brief mindfulness-based cognitive behavioral intervention improves sexual functioning versus wait-list control in women treated for gynecologic cancer. *Gynecologic Oncology*, *125*(2), 320–325. doi:10.1097/PEC.0b013e3181fe9211.A

Calkins, S. D. (1994). Origins and outcomes of individual differences in emotion regulation. In N. A. Fox (Ed.), *The Development of emotion regulation: Biological and behavioral considerations (*pp. *53–72). Monographs of the Society for Research in Child Development Chicago*, IL: University of Chicago Press

Fabes, R. A., & Eisenberg, N. (1997). Regulatory control and adults' stress-related responses to daily life events. *Journal of Personality and Social Psychology*, *73*(5), 1107–1117.

Hatch, J. P. (1979). Vaginal photoplethysmography: Methodological considerations. *Archives of Sexual Behavior*, *8*(4), 357–374.

Heiman, J. R. (1977). A psychophysiological exploration of sexual arousal patterns in females and males. *Psychophysiology*, *14*(2), 266–274.

Heiman, J. R. (1980). Female sexual response patterns: Interactions of physiological, affective, and contextual cues. *Archives of General Psychiatry*, *37*(11), 1311–1316.

Heiman, J. R., & Rowland, D. L. (1983). Affective and physiological sexual response patterns: The effects of instructions on sexually functional and dysfunctional men. *Journal of Psychosomatic Research*, *27*(2), 105–116.

Hejjel, L., & Roth, E. (2004). What is the adequate sampling interval of the ECG signal for heart rate variability analysis in the time domain? *Physiological Measurement*, *25*(6), 1405–1411. doi:10.1088/0967-3334/25/6/006

Jain, S., Shapiro, S. L., Swanick, S., Roesch, S. C., Mills, P. J., Bell, I., & Schwartz, G. E. R. (2007). A randomized controlled trial of mindfulness meditation versus relaxation training: effects on distress, positive states of mind, rumination, and distraction. *Annals of Behavioral Medicine: A Publication of the Society of Behavioral Medicine*, *33*(1), 11–21. doi:10.1207/s15324796abm3301_2

Kanji, N. (2000). Management of pain through autogenic training. *Complementary Therapies in Nursing & Midwifery*, *6*(3), 143–148. doi:10.1054/ctnm.2000.0473

Kanji, N., White, A., & Ernst, E. (2006). Autogenic training to reduce anxiety in nursing students: Randomized controlled trial. *Issues and Innovations in Nursing Education*, *53*(6), 729–736. doi:10.1111/j.1365-2648.2006.03779.x

Karavidas, M. K., Lehrer, P. M., Vaschillo, E., Vaschillo, B., Marin, H., Buyske, S., . . . Hassett, A. (2007). Preliminary results of an open label study of heart rate variability biofeedback for the treatment of major depression. *Applied Psychophysiology and Biofeedback*, *32*(1), 19–30. doi:10.1007/s10484-006-9029-z

Kemp, A. H., Quintana, D. S., Felmingham, K. L., Matthews, S., & Jelinek, H. F. (2012). Depression, comorbid anxiety disorders, and heart rate variability in physically healthy, unmedicated patients: Implications for cardiovascular risk. *PloS One*, *7*(2), e30777. doi:10.1371/journal.pone.0030777

Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. *Biological Psychiatry*, *67*(11), 1067–1074. doi:10.1016/j.biopsych.2009.12.012

Krampen, G. (1999). Long-term evaluation of the effectiveness of additional autogenic training in the psychotherapy of depressive disorders. *European Psychologist*, *4*(1), 11–18.

Kudo, N., Shinohara, H., & Kodama, H. (2014). Heart rate variability biofeedback intervention for reduction of psychological stress during the early postpartum period. *Applied Psychophysiology and Biofeedback*, *39*, 203–211. doi:10.1007/s10484-014-9259-4

Laan, E., Everaerd, W., Van Aanhold, M.-T., & Rebel, M. (1993). Performance demand and sexual arousal in women. *Behaviour Research and Therapy*, *31*(1), 25–35.

Laan, E., Everaerd, W., Van der Velde, J., & Geer, J. H. (1995). Determinants of subjective experience of sexual arousal in women: Feedback from genital arousal and erotic stimulus content. *Psychophysiology*, *32*(5), 444–451. doi:10.1111/j.1469-8986.1995.tb02095.x

Laan, E., Smith, M. D., Boolell, M., & Quirk, F. (2002). Development of a sexual function questionnaire for clinical trials of female sexual dysfunction. *Journal of Women's Health & Gender-Based Medicine*, *11*(3), 277–289.

Laan, E., Van Driel, E. M., & Van Lunsen, R. H. W. (2008). Genital responsiveness in healthy women with and without sexual arousal disorder. *Journal of Sexual Medicine*, *5*(6), 1424–1435. doi:10.1111/j.1743-6109.2008.00827.x

Lehrer, P. M., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: Rationale and manual for training. *Applied Psychophysiology and Biofeedback*, *25*(3), 177–191.

Lehrer, P., Vaschillo, B., Zucker, T., Graves, J., Katsamanis, M., Aviles, M., & Wamboldt, F. (2013). Protocol for heart rate variability biofeedback training. *Biofeedback*, *41*(3), 98–109. doi:10.5298/1081-5937-41.3.08

Linden, W. (1990). *Autogenic training: A clinical guide*. New York, NY: Guilford Press.

Lorenz, T. A., Harte, C. B., Hamilton, L. D., & Meston, C. M. (2012). Evidence for a curvilinear relationship between sympathetic nervous system activation and women's physiological sexual arousal. *Psychophysiology*, *49*(1), 111–117. doi:10.1111/j.1469-8986.2011.01285.x.Evidence

McCall, K. M., & Meston, C. M. (2006). Cues resulting in desire for sexual activity in women. *Journal of Sexual Medicine*, *3*(5), 838–852. doi:10.1111/j.1743-6109.2006.00301.x

Meston, C. M., & Gorzalka, B. B. (1995). The effects of sympathetic activation on physiological and subjective sexual arousal in women. *Behaviour Research and Therapy*, *33*(6), 651–664.

Meston, C. M., & Gorzalka, B. B. (1996). The effects of immediate, delayed, and residual sympathetic activation on sexual arousal in women. *Behavior Research and Therapy*, *34*(2), 143–148. doi:10.1016/0005-7967(95)00050-X

Meston, C. M., & Heiman, J. R. (1998). Ephedrine-activated physiological sexual arousal in women. *Archives of General Psychiatry*, *55*(7), 652–656.

Mishima, N., Kubota, S., & Nagata, S. (1999). Psychophysiological correlates of relaxation induced by standard autogenic training. *Psychotherapy and Psychosomatics*, *68*(4), 207–213.

Mitani, S., Fujita, M., Sakamoto, S., & Shirakawa, T. (2006). Effect of autogenic training on cardiac autonomic nervous activity in high-risk fire service workers for posttraumatic stress disorder. *Journal of Psychosomatic Research*, *60*(5), 439–444. doi:10.1016/j.jpsychores.2005.09.005

Miu, A. C., Heilman, R. M., & Miclea, M. (2009). Reduced heart rate variability and vagal tone in anxiety: Trait versus state, and the effects of autogenic training. *Autonomic Neuroscience: Basic & Clinical*, *145*(1–2), 99–103. doi:10.1016/j.autneu.2008.11.010

Morokoff, P. J., & Heiman, J. R. (1980). Effects of erotic stimuli on sexually functional and dysfunctional women: Multiple measures before and after sex therapy. *Behavior Research and Therapy*, *18*, 127–137.

Nolan, R. P., Kamath, M. V., Floras, J. S., Stanley, J., Pang, C., Picton, P., & Young, Q. R. (2005). Heart rate variability biofeedback as a behavioral neurocardiac intervention to enhance vagal heart rate control. *American Heart Journal*, *149*(6), 1137. doi:10.1016/j.ahj.2005.03.015

O'Connor, M.-F., Allen, J. J. B., & Kaszniak, A. W. (2002). Autonomic and emotion regulation in bereavement: A longitudinal study. *Journal of Psychosomatic Research*, *52*, 183–185.

Park, K., Goldstein, I., Andry, C., Siroky, M. B., Krane, R. J., & Azadzoi, K. M. (1997). Vasculogenic female sexual dysfunction: The hemodynamic basis for vaginal engorgement insufficiency and clitoral erectile insufficiency. *International Journal of Impotence Research*, *9*(1), 27–37. doi:10.1038/sj.ijir.3900258

Pulverman, C. S., Meston, C. M., & Hixon, J. G. (2015). Automated artifact detection procedure for vaginal photoplethysmography data. Paper presented at the annual meeting of the International Society for the Study of Women's Sexual Health (ISSWSH), Austin, TX.

Raudenbush, S., Bryk, A., Cheong, Y., Congdon, R., & Du Toit, M. (2011). *HLM 7* [Computer Software]. Lincolnwood, IL: Scientific Software International Inc.

Rellini, A. H., McCall, K. M., Randall, P. K., & Meston, C. M. (2005). The relationship between women's subjective and physiological sexual arousal. *Psychophysiology*, *42*(1), 116–124. doi:10.1111/j.1469-8986.2005.00259.x

Rosen, R. C., & Beck, J. G. (1988). *Patterns of sexual arousal: Psychophysiological processes and clinical applications*. Guilford Press.

Rosen, R., Brown, C., Heiman, J., Leiblum, S., Meston, C., Shabsigh, R., . .. D'Agostino, R. (2000). The Female Sexual Function Index (FSFI): A multidimensional self-report instrument for the assessment of female sexual function. *Journal of Sex & Marital Therapy*, *26*(2), 191–208. doi:10.1080/009262300278597

Saul, J. P. (1990). Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow. *Physiology*, *5*(1), 32–37.

Schroeder, E. B., Liao, D., Chambless, L. E., Prineas, R. J., Evans, G. W., & Heiss, G. (2003). Hypertension, blood pressure, and heart rate variability: The Atherosclerosis Risk in Communities (ARIC) study. *Hypertension*, *42*(6), 1106–1111. doi:10.1161/01.HYP.0000100444.71069.73

Schultz, J. H., & Luthe, W. (1959). *Autogenic training: A psycho-physiological approach in psychotherapy*. New York, NY: Grune and Stratton.

Shinozaki, M., Kanazawa, M., Kano, M., Endo, Y., Nakaya, N., Hongo, M., & Fukudo, S. (2010). Effect of autogenic training on general improvement in patients with irritable bowel syndrome: A randomized controlled trial. *Applied Psychophysiology Biofeedback*, *35*(3), 189–198. doi:10.1007/s10484-009-9125-y

Singh, J. P., Larson, M. G., O'Donnell, C. J., Wilson, P. F., Tsuji, H., Lloyd-Jones, D. M., & Levy, D. (2000). Association of hyperglycemia with reduced heart rate variability (The Framingham Heart Study). *American Journal of Cardiology*, *86*(1), 309–312.

Sintchak, G., & Geer, J. H. (1975). A vaginal plethysmograph system. *Psychophysiology*, *12*(1), 113–115.

Stanton, A. M., Lorenz, T. A., Pulverman, C. S., & Meston, C. M. (2015). Heart rate variability: A risk factor for female sexual dysfunction. *Applied Psychophysiology and Biofeedback*, *40*(3), 229–237. doi:10.1007/s10484-015-9286-9

Stetter, F., & Kupper, S. (2002). Autogenic training: A meta-analysis of clinical outcome studies. *Applied Psychophysiology and Biofeedback*, *27*(1), 45–98. doi:10.1023/A:1014576505223

Team, R.D.C. (2010). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.R-project.org

Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology*, *141*(2), 122–131. doi:10.1016/j.ijcard.2009.09.543

Xhyheri, B., Manfrini, O., Mazzolini, M., Pizzi, C., & Bugiardini, R. (2012). Heart rate variability today. *Progress in Cardiovascular Diseases*, *55*(3), 321–331. doi:10.1016/j.pcad.2012.09.001

Ziemssen, T., Gasch, J., & Ruediger, H. (2008). Influence of ECG sampling frequency on spectral analysis of RR intervals and baroreflex sensitivity using the EUROBAVAR data set. *Journal of Clinical Monitoring and Computing*, *22*(2), 159–168. doi:10.1007/s10877-008-9117-0