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Vagal Activity During Physiological Sexual Arousal in Women With and Without Sexual Dysfunction

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Recently, heart rate variability (HRV) level has been found to be a risk factor for female sexual dysfunction. Low HRV was a significant predictor of female sexual arousal dysfunction and overall sexual dysfunction. Building upon this finding, the present study examined whether differences in vagal activity between sexually functional and sexually dysfunctional women may be driving the association between low HRV and female sexual dysfunction. Specifically, respiratory sinus arrhythmia (RSA) was assessed before, during, and after physiological sexual arousal in 84 women, aged 18 to 47, to examine potential differences in vagal activity between sexually functional and sexually dysfunctional women. Significant differences in vagal activity between these two groups were observed ($p = .02$). These findings provide additional specificity to the recently established relationship between HRV and female sexual function while also proposing a mechanism to target during treatments for sexual dysfunction.

INTRODUCTION

Heart rate variability (HRV) has recently been associated with female sexual function (Stanton, Lorenz, Pulverman, & Meston, 2015). In that study, HRV level, indexed by the standard deviation of the inter-beat interval lengths (SDNN; a time-domain measure of HRV), was a significant predictor of sexual function status. Women with below-average HRV were significantly more likely to report sexual arousal dysfunction and overall sexual dysfunction than women with average HRV and women with above-average HRV. In other words, low HRV may be a risk factor for female sexual dysfunction, as it is for depression (Kemp et al., 2010) and anxiety (Kemp, Quintana, Felmingham, Matthews, & Jelinek, 2012), disorders all characterized by autonomic imbalance.

Fluctuation in HRV that occurs in the high frequency range, most often defined as 0.15 to 0.4 hertz (Hz), is a result of respiratory sinus arrhythmia (RSA), the increase and decrease in heart rate that occurs with respiration (Porges, Doussard-Roosevelt, & Maiti, 1994). RSA is primarily associated with parasympathetic nervous system (PNS) activity via the vagus (10th cranial) nerve (Berntson, Cacioppo, & Quigley, 1993). High-frequency HRV, indexed by RSA, has been shown

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to be a reliable measure of both overall PNS activity and the strength of vagal influence on the heart (Berntson et al., 1997; Malik et al., 1996).

There is a large body of evidence indicating that the PNS modulates psychophysiological arousal. Much of the variation in heart rate derives from the PNS, which controls the brain stem regions that connect to the heart through the vagus nerve. During inhalation, heart rate increases as vagal, or PNS, influence decreases. During exhalation, heart rate decreases as vagal influence increases. This process of inhalation and exhalation, increasing and decreasing PNS influence, has regular period cycles.

The quantification of RSA provides an opportunity to dynamically monitor vagal regulation of the heart in a variety of conditions. The vagus responds rapidly to changing metabolic demands due to emotional arousal, environmental triggers, or even simple changes in posture (Salata & Zipes, 1991). Optimally, during physical and emotional challenges that demand physiological mobilization, there is an acute withdrawal of vagal inhibition of the heart, which leads to increased heart rate. During these moments of physiological and psychological stress, sympathetic nervous system (SNS) activity becomes dominant relative to parasympathetic nervous system (PNS) activity. The dominance of the SNS allows for increased physiological arousal to help the body respond to challenges (Appelhans & Luecken, 2006).

The SNS has been shown to play an important role in female sexual arousal. Moderate SNS dominance (relative to PNS dominance) has been shown to predict women’s level of genital arousal in the laboratory (Lorenz & Meston, 2012; Meston & Gorzalka, 1995, 1996a, 1996b; Meston & Heiman, 1998). Research has consistently indicated that moderate SNS activation facilitates genital sexual arousal in women (Meston & Gorzalka, 1995, 1996a, 1996b). By contrast, there has been little direct examination of the role of the PNS in female sexual function. As differences in vagal activity have been shown to discriminate between individuals with disorders characterized by autonomic imbalance and individuals without those disorders, it is possible that differences in vagal activity between sexually functional and sexually dysfunctional women may help account for the association between low HRV and female sexual dysfunction.

With regard to the PNS, there are two commonly used methods of assessing vagal activity—measuring resting RSA and measuring acute changes in RSA due to stressful situations or demanding tasks. Most relevant here, acute changes in RSA have been linked to positive psychological phenomena. In healthy individuals, rapid withdrawal of vagal activity, or rapid decrease in RSA, occurs in response to stressful situations (Berntson et al., 1994). Rapid withdrawal of vagal activity leads to quick increases in heart rate (Berntson et al., 1993) and enables the body to respond effectively to environmental triggers. In other words, rapid vagal withdrawal in demanding situations is healthy and adaptive. Smaller acute differences, or slower vagal withdrawal, in response to challenging psychological and physiological tasks have been associated with disorders characterized by autonomic imbalance, including major depressive disorder (Rottenberg, Salomon, Gross, & Gotlib, 2005), generalized anxiety disorder (McLeod, Hoehn-Saric, Porges, Kowalski, & Clark, 2000), and posttraumatic stress disorder (Sack, Hopper, & Lamprecht, 2004). Following the stressful situation, rapid vagal rebound is also typical of healthy populations (Mezzacappa, Kelsey, Katkin, & Sloan, 2001). Poor vagal rebound has been shown to predict autonomic imbalance, specifically poor response to physiological stress (Cole, Blackstone, Pashkov, Snader, & Lauer, 1999) and risk for cardiovascular disease (La Rovere, Bigger, Marcus, Mortara, & Schwartz, 1998).
In the field of sexual medicine, researchers have examined the relationship between vagal activity and orgasm. Komisaruk, Gerdes, and Whipple (1997) studied vaginal and cervical self-stimulation in a sample of women with traumatic spinal cord injury at various levels of the spinal column and determined that vagal pathways can convey sensory activity from the cervix, independent of the spinal cord. Studies using both positron emission tomography (PET; Whipple & Komisaruk, 2002) and functional magnetic resonance imaging (fMRI; Komisaruk & Whipple, 2005) confirmed that the vagus nerves convey genital sensory activity from the cervix to the brain in women with and without spinal cord injury. Moreover, Frangos, Ellrich, and Komisaruk (2015) recently indicated that stimulation of vagal afferents in the external ear activates genital sensations. This body of evidence suggests an important role for vagal pathways in the orgasm domain of female sexual function.

Even though vagal pathways have been shown to facilitate the communication of sensory activity from the vagina to the brain, to our knowledge there have been no investigations of the relationship between vagal activity and sexual dysfunction in women beyond the orgasm domain. The present study is the first to examine vagal activity before, during, and after physiological sexual arousal in women with and without clinically relevant sexual dysfunction. Based both on evidence that SNS activation, which is inversely correlated with PNS activation, facilitates female sexual arousal (Lorenz & Meston, 2012; Meston & Gorzalka, 1995, 1996a, 1996b; Meston & Heiman, 1998) and that HRV (SDNN) is associated with female sexual function (Stanton et al., 2015), we predict that women with sexual dysfunction would show higher RSA during sexual arousal, slower vagal withdrawal, and slower vagal recovery, than sexually functional women.

METHOD

Participants

Participants with and without sexual dysfunction were recruited from the Austin, Texas area using flyers, online advertisements, and print advertisements that highlighted the sexual nature of the experiment. Potential participants were screened over the phone to ensure that they met the inclusion criteria. Inclusion criteria were as follows: at least 18 years of age, currently sexually active, fluent in English, heterosexual or bisexual, and current sexual dysfunction or no current sexual dysfunction. Exclusion criteria included currently breastfeeding or pregnant; current diagnosis of posttraumatic stress disorder, major depressive disorder, or generalized anxiety disorder; history or current diagnosis of sexually transmitted disease; history of major pelvic surgery; currently taking medications likely to affect sexual arousal, such as anxiolytics or beta blockers; and current diagnosis of psychosis (e.g., bipolar disorder or schizophrenia). Women currently taking alprazolam (Xanax) were permitted to participate in the study if they agreed to refrain from taking it the day of their study session.

Procedure

The experimenter greeted the participants and invited them to read and sign a consent form. Testing sessions took place in a private room with an intercom that participants used to communicate with the researcher. Vaginal photoplethysmography was used to assess physiological sexual
arousal, but these data are not considered in the present article. An electrocardiogram (ECG) was used to isolate HRV, specifically high frequency HRV, during the session. Participants were instructed in how to use the vaginal photoplethysmograph and attach the ECG wires before the session began. Once the participants had the equipment in place, they underwent a three- to five-minute habituation period where no physiological measurements were taken. They then viewed a nine-minute film composed of neutral (three minutes) and erotic (six minutes) content while their genital sexual arousal and HRV were measured. The erotic film featured a heterosexual couple engaging in foreplay, cunnilingus, and vaginal intercourse. Baseline HRV measurements were collected during the neutral film segment, arousal HRV measurements were collected during the erotic film segment, and recovery HRV measurements were collected in the three minutes following the erotic film segment. All participants completed measures on demographics and sexual function (see below). Participants were compensated $50 cash for their time. This procedure was approved by the Institutional Review Board at The University of Texas at Austin.

Measures

**High-Frequency Heart Rate Variability (Respiratory Sinus Arrhythmia)**

Heart rate was measured at a rate of 200 samples/sec. This sampling rate was adequate to produce a minimally biased estimate of frequency domain measures of HRV, such as those presented here (Heijel & Roth, 2004; Ziemssen, Gasch, & Ruediger, 2008). The three leads of the ECG were placed under the participant’s right collarbone, below the lowermost left rib, and on the right ankle. The signals from the leads were collected with AcqKnowledge software, and movement artifacts were removed manually. The AcqKnowledge peak-finder function was used to extract the beat-to-beat (NN) intervals.

Power spectral densities of the NN-interval variability were determined via the Fast Fourier Transform using Kubios HRV Analysis Software (Biosignal Analysis and Medical Imagine Group, University of Kuopio, Kuopio, Finland). Following the guidelines established by the North American Society of Pacing and Electrophysiology (Malik et al., 1996), this software isolates spectral power into low frequency (LF; 0.04–0.15 Hz) and high frequency (HF; 0.15–0.4 Hz) bands. For the present study, only HF HRV was examined, as it is associated exclusively with PNS activity (Bernston et al., 1993). Respiratory sinus arrhythmia, defined as HF HRV, was transformed via the natural logarithm to reduce the skewness of its distribution. This natural logarithm-transformed measure of RSA was then used as an index of vagal activity.

**Sexual Function**

Sexual function was assessed with the Female Sexual Function Index (FSFI; Rosen et al., 2000), an empirically validated, 19-item questionnaire. The FSFI is a widely used measure of female sexual function. The clinical cutoff that reliably discriminates between women with and without a sexual dysfunction diagnosis is 26.55 (Rosen et al., 2000). In the present study, women whose scores were above that cutoff were considered sexually functional, and women whose scores fell below that cutoff were considered sexually dysfunctional. The FSFI assesses six domains of sexual function: desire (two items), arousal (four items), vaginal lubrication (four
items), orgasm (three items), satisfaction (three items), and sexual pain (three items). However, only FSFI total scores were used in the present study.

**Subjective sexual arousal**

Subjective sexual arousal was assessed with 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 “sexual arousal,” a sense of “mental sexual arousal,” and one reverse-scored item on feeling “sexually turned off.” Participants rated the degree to which they experienced each of the three items on a 7-point Likert scale. Subjective sexual arousal was used to ensure that the sexually functional group and the sexually dysfunctional group found the erotic film to be comparably arousing.

**Data Analysis**

To determine the overall trajectory of RSA before, during, and after physiological sexual arousal, we applied a linear mixed-effects model, a form of hierarchical linear modeling, to the data. The term “mixed effects” refers to the use of both fixed and random effects in the same analysis. Mixed effects models are essential tools for the analysis of longitudinal data, as they provide a flexible approach to the analysis of repeated measurements on each subject over time (Peng & Lu, 2012). Although these data are not longitudinal in the traditional sense, they do map three distinct time points (before, during, and after the erotic film) across a 12-minute period.

To examine potential differences in vagal withdrawal and recovery between sexually functional and sexually dysfunctional women, RSA difference scores (between baseline and erotic, and erotic and recovery) were calculated for each participant. Welch’s $t$ tests were applied to these difference scores.

**RESULTS**

**Sample Characteristics**

The final sample included 84 women, aged 18 to 47 ($M = 26.9$, $SD = 6.8$). With respect to relationship status, 51.2% of the sample were in a committed relationship. The majority of the sample was Caucasian (65.5%), 10.7% were Asian American, 8.3% were African American, 1.2% were American Indian or Native Alaskan, 1.2% were Pacific Islanders, and 10.7% listed “other.” In terms of sexual function status, 42.9% of the participants were categorized as sexually functional based on the FSFI (see Table 1 for full demographics). Between the sexually functional and sexually dysfunctional groups, there were no significant differences with respect to age, relationship status, or race.

**Respiratory Sinus Arrhythmia and Sexual Function**

The fixed effects considered in these analyses were time and sexual function group (functional or dysfunctional), and the random effect was an intercept value that applied to the subject
TABLE 1
Participant Characteristics (N = 84)

<table>
<thead>
<tr>
<th></th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>26.9</td>
<td>6.8</td>
</tr>
<tr>
<td>FSFI (total score)</td>
<td>24.6</td>
<td>6.3</td>
</tr>
<tr>
<td>Sexually functional group</td>
<td>30.2</td>
<td>2.3</td>
</tr>
<tr>
<td>Sexually dysfunctional group</td>
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<td>4.3</td>
</tr>
<tr>
<td>Race</td>
<td>n</td>
<td>%</td>
</tr>
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<td>1.2</td>
</tr>
<tr>
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<td>8.3</td>
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<td>Caucasian/White</td>
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<td>65.5</td>
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<tr>
<td>Asian American</td>
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<td>10.7</td>
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<tr>
<td>Pacific Islander</td>
<td>1</td>
<td>1.2</td>
</tr>
<tr>
<td>Other</td>
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<td>10.7</td>
</tr>
<tr>
<td>Relationship status</td>
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<tr>
<td>Single, not dating</td>
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<td>16.7</td>
</tr>
<tr>
<td>Single, dating</td>
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</tr>
<tr>
<td>In a committed relationship</td>
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<tr>
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<td>Sexually functional</td>
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<tr>
<td>Invalid FSFI score</td>
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<td>9.5</td>
</tr>
</tbody>
</table>

Notes. FSFI = Female Sexual Function Index (Rosen et al., 2000).

*aAs defined by the FSFI.

*bParticipants could identify as belonging to more than one race.

Identification number. The time variable violated the homogeneity of variance assumption, so a correction was applied. There was a trend toward a significant interaction effect between sexual function group and time (before, during, and after the erotic film) on RSA, \( F(2, 140) = 2.76, p = 0.07 \).

With respect to vagal reactivity from the baseline segment to the erotic segment, a Welch’s \( t \) test revealed that the change in RSA from baseline to erotic was significantly different between the two groups (\( t = -2.35, p = .02 \)). As is evident in Figure 1, the dysfunctional group exhibited activation, rather than withdrawal, between baseline measurements and erotic measurements. The functional group had some, though limited, RSA withdrawal. With respect to RSA rebound, the change in RSA from erotic to recovery was not significantly different between the two groups.

Subjective Sexual Arousal

A Welch’s \( t \) test revealed that the level of subjective sexual arousal did not differ significantly between the sexually functional group and the sexually dysfunctional group (\( t = 0.04, p = .97 \)). The two groups of participants found the stimulus film to be comparably arousing.
This study examined the relationship between vagal activity and sexual function in women during physiological sexual arousal in the laboratory. Results revealed a trend toward a statistically significant difference in RSA based on sexual function group across baseline, arousal, and recovery. Most importantly, the shift in RSA from the baseline time segment to the erotic time segment significantly differed between the sexually functional women and the sexually dysfunctional women. Sexually functional women exhibited vagal withdrawal (evidenced by a decrease in RSA), as would be expected, yet sexually dysfunctional women showed vagal activation (evidenced by an increase in RSA). In other words, differences in vagal activity from baseline to the presentation of an erotic stimulus may be contributing to the association between low HRV and female sexual dysfunction. To our knowledge, this is the first study to measure RSA during sexual arousal in women with clinically relevant sexual dysfunction. This study adds additional specificity to our understanding of the relationship between HRV and female sexual function, indicating that vagal pathways may be worth targeting in treatments for sexual dysfunction.

The finding that sexually dysfunctional women showed vagal activation from the baseline to the erotic segment is consistent with research on autonomic function during female genital arousal in sexually healthy women. Laboratory studies have indicated that moderate SNS activation contributes to increased genital sexual arousal in sexually functional women (Meston & Gorzalka, 1995, 1996a). Considering that SNS activation is associated with increases in sexual arousal in
healthy women, it is not surprising that sexually dysfunctional women exhibit vagal activation, which is indicative of PNS dominance over the SNS, during sexual arousal.

The results of this study also align with previous research on vagal activity in both healthy and clinical populations. In healthy individuals, vagal activity is highest when the body is unchallenged or at rest (Rottenberg, 2007). The vagal pathway slows heart rate below its autonomous rhythm, allowing the body to conserve energy until environmental conditions become more demanding. This pattern was evident in the sexually functional group, who showed a slight drop in RSA from baseline to the erotic film segment (see Figure 1). According to polyvagal theory (Porges, 1995), the inability to withdraw the vagal break and to allow for sympathetic activation in the face of environmental demands prevents optimal engagement and the ability to cope with challenging tasks. Diminished vagal reactivity and lack of vagal withdrawal in response to environmental demands has been associated with different forms of psychopathology, including major depressive disorder (Rottenberg et al., 2005), generalized anxiety disorder (McLeod et al., 2000), and posttraumatic stress disorder (Sack et al., 2004). Now, based on this data, we may be able to apply polyvagal theory to female sexual dysfunction, which, like the disorders mentioned above, is associated with autonomic imbalance. In the sexual dysfunction group, there was no withdrawal of the vagal break to allow for sympathetic activation, thus preventing healthy sexual function.

It is worth noting that the observed differences in vagal activity between sexually functional and sexually dysfunctional women cannot be attributed to differences in subjective sexual arousal. In other words, the erotic stimulus was comparably arousing to both groups of women. Using the same measurement instrument as the current study, past studies have also not found significant differences in subjective sexual arousal between sexually functional and sexually dysfunctional women (Meston & Gorzalka, 1996a, 1996b).

This examination of vagal activity, indexed by RSA, during sexual arousal in women with and without sexual dysfunction has a number of important clinical implications. It is possible that lack of vagal withdrawal during sexual arousal, which requires physiological mobilization, contributes to the precipitation and maintenance of female sexual dysfunction. Future research may point to effective methods of facilitating vagal withdrawal during sexual arousal as a potential treatment for female sexual dysfunction. Such treatments may feature physiological awareness training, mindfulness components, and intentional manipulation of RSA. HRV biofeedback may be helpful to increase resting state HRV in women with sexual dysfunction, which may in turn reduce vagal activation during sexual arousal. Although preexisting HRV interventions, which are designed to take place when patients are at rest, may be effective for treating sexual dysfunction, it is also possible that targeted physiological awareness interventions that specifically decrease vagal activation during sexual arousal may need to be developed.

Furthermore, it would be clinically beneficial for the field to clarify the relationship between vagal activity and sexual function in different populations of women, such as women with sexual dysfunction related to childhood sexual abuse (CSA) and women with antidepressant medication-induced sexual dysfunction. Women with CSA histories are known to have unique disruptions in autonomic nervous system activity (Putnam, 2003), and they are less responsive to standardized sex therapy treatments than nonabused women (Berman, Berman, Bruck, Pawar, & Goldstein, 2001; Maltz, 2002, 2012). A better understanding of vagal activity during sexual arousal in women with CSA histories would be an integral first step in developing effective physiological awareness-based treatments for this population. Women with antidepressant-induced sexual
dysfunction may have a different trajectory of vagal activity during sexual arousal than women who are not on antidepressants, as two years of antidepressant medication use has been shown to significantly decrease RSA and cardiac vagal control (Licht et al., 2008). An estimated one in six American women has been prescribed an antidepressant (Rosen, Lane, & Menza, 1999), and all antidepressants are associated with sexual side effects. However, there are few treatments for these sexual side effects that do not interfere with therapeutic efficacy, and the treatments that have shown some promise are entirely pharmacologic in nature (Taylor, Rudkin, & Hawton, 2005). More recent evidence indicates that acute exercise may improve antidepressant-related genital problems (Lorenz & Meston, 2012), therefore it may be worth examining other physiological-based treatments for this vulnerable population. If women who are prescribed antidepressants stop taking their medication due to sexual side effects, they may be at risk of having a major depressive episode. Developing effective nonpharmacologic, physiologically oriented treatments for this subgroup of women may help prevent the onset of a new episode.

This study underscores an important clinical distinction between male sexual function and female sexual function. In men, parasympathetic pathways have been shown to facilitate sexual arousal (Giuliano & Rampin, 2004). An increase in PNS activity during sexual arousal activates endothelial cells in the penis to relax the smooth muscle in the arteries supplying the erectile tissue (Solomon, Man, & Jackson, 2003). Based on the findings of the present study, it seems that, in women, rapid vagal activation is characteristic of sexual dysfunction rather than sexual function. Clinicians, particularly primary care physicians, gynecologists, sex therapists, and other sexual medicine specialists, need to be sensitive to this difference when treating female patients with sexual problems.

It is important to note some limitations of the present study. First, we relied on the FSFI, a self-report measure, to dichotomize participants into either the sexually functional group or the sexually dysfunctional group. Although this index has been extensively validated and translated into over 30 languages worldwide (Sun, Li, Jin, Fan, & Wang, 2011), the FSFI does not differentiate among the different female sexual dysfunction disorders, which may have led to a fairly heterogeneous collection of sexual dysfunctions in the sexually dysfunctional group of the present study. On the other hand, the potential for heterogeneity within the sexual dysfunction group adds strength to our findings, indicating that the observed difference in vagal activity between the two groups is not specific to a certain type of dysfunction. Second, the use of vaginal photoplethysmography may have altered our overall measurement of HRV, especially for those women in the sexual dysfunction group who may have become anxious or concerned about the insertion of the probe. However, vaginal photoplethysmography is generally considered noninvasive (Janssen, Prause, & Geer, 2007), and participants were given a standardized habituation period to adjust to the presence of the plethysmograph prior to the collection of physiological data. Finally, though we did exclude participants who reported a current diagnosis of major depressive disorder or generalized anxiety disorder, we did not collect data on trait or state anxiety or depressive symptoms, which may mediate the relationship between vagal activity and sexual function. Vagal activity is known to be blunted in anxious populations (Thayer, Friedman, & Borkovec, 1996), and there have been mixed reports on the relationship between vagal activity and depression (for review, see Rottenberg, 2007). Future research on the relationship between vagal activity and sexual function should assess these variables.

Despite these limitations, the results of this study provide more specificity to our understanding of the recently established relationship between low HRV and clinically relevant female sexual
dysfunction by pointing to vagal pathways as a potential target for future treatment. This study also lends additional support to the suggestion that HRV and its component frequency bands may play a mechanistic role in female sexual function (Stanton et al., 2015). High frequency HRV, indexed by RSA and mediated entirely by vagal pathways, may be an important internal marker of individual differences in physical responsiveness and healthy adaptation to sexual situations. Given this possibility, interventions that target changes in RSA may be a valuable option for women with sexual dysfunction.

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Vagal Activity During Female Sexual Arousal


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