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Laboratory-Induced Hyperventilation Differentiates Female Sexual Arousal Disorder Subtypes

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Abstract The effects of heightened sympathetic nervous system (SNS) activity via laboratory-induced hyperventilation (LIH) on subjective and physiological sexual arousal were examined in a heterogeneous group of women with Sexual Arousal Disorder (SAD; $n = 60$), as well as across subtypes of SAD, in comparison to a control group of women without sexual difficulties ($n = 42$). Participants took part in 2 min of rapid breathing, a technique previously found to increase SNS activity, immediately prior to viewing erotic stimuli. Physiological arousal (i.e., vaginal pulse amplitude; VPA) was measured via the vaginal photoplethysmograph and subjective arousal was measured via self-report questionnaires. LIH differentiated women with SAD from those in the control group, with LIH increasing VPA in the latter, but having no significant effect in the heterogeneous SAD group. However, among subtypes of SAD, LIH differentiated women with genital ($n = 16$) and subjective ($n = 16$) subtypes of SAD from women with combined SAD ($n = 28$) and women without sexual difficulties. Specifically, women in the control group and those with combined SAD had a significant increase in VPA whereas women with genital or subjective SAD had a significant decrease in VPA following LIH. There was no significant effect of LIH on any self-report measure of sexual arousal following erotic stimuli. Implications of the results for the conceptualization, diagnosis, and treatment of SAD are discussed.

Keywords Female Sexual Arousal Disorder · Sexual dysfunction · Sympathetic nervous system · Photoplethysmograph

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Introduction

Diagnostic Issues in Female Sexual Arousal Disorder

Masters and Johnson's (1966) proposed human sexual response cycle conceptualized the stages of the cycle—excitement (now termed arousal), plateau, orgasm, and resolution—in terms of physiological changes. This conceptualization has carried on to the current classification system of sexual dysfunctions. Both the current versions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000) and the *International Classification of Diseases* (ICD-10; World Health Organization, 1999) define the majority of the sexual dysfunctions solely in terms of physiological difficulties as described by Masters and Johnson.

Female Sexual Arousal Disorder (SAD) is defined according to both the DSM-IV-TR and the ICD-10 as "persistent or recurrent inability to attain, or to maintain until completion of the sexual activity, an adequate lubrication or swelling response of sexual excitement." Sexual arousal complaints are seen in approximately 12–21% of women in American and Swedish samples (Fugl-Meyer & Fugl-Meyer, 1999; Laumann, Paik, & Rosen, 1999), with even higher prevalence rates found in Asian and Southeast Asian women (Laumann et al., 2005). Among women seeking routine care in gynecologic settings, as many as 75% have been suggested

to have problems with sexual arousal (Nusbaum, Gamble, Skinner, & Heiman, 2000).

Criticisms have been raised about the current definition and classification of SAD in that it may not reflect the complaints of sexual arousal among women seen in the clinical setting (Basson, 2001b, 2002a, 2002b). Specifically, the DSM-IV-TR definition lacks any mention or recognition of subjective sexual arousal. Clinically, impairments of genital arousal in women are usually confined to specific subgroups, such as those with diabetes, pelvic surgery, and spinal cord injuries (Schreiner-Engel, Schiavi, Vietorisz, & Smith, 1987; Sipski, Alexander, & Rosen, 2001; West, Vinikoor, & Zolnoun, 2004). In an effort to critically re-evaluate the sexual dysfunction categories, including SAD, there have been two consensus conferences established during which international experts were convened (Basson et al., 2000, 2003). One result of these meetings was a new proposed definition and classification of SAD in which three SAD subtypes were proposed. The first subtype, Genital SAD, corresponded somewhat to the *DSM-IV-TR* and *ICD-10* definitions of SAD, and focuses on complaints of absent or impaired genital sexual arousal, and reduced sexual sensations from caressing the genitalia, however, these individuals have normal subjective arousal. The second subtype, Subjective SAD, referred to an absence or marked decrease in subjective feelings of sexual excitement and pleasure in women with unproblematic genital arousal. The third subtype, Combined SAD, referred to absent or markedly diminished subjective and physiological arousal in response to sexual stimulation. These proposed subtypes have been suggested to represent an improved set of criteria that may have more applicability in the clinical and research settings, particularly as treatments are being developed and tested (Basson et al., 2004). Neither epidemiological nor pathophysiological data on the SAD subtypes exist at present, and one aim of the current study was to explore the pathophysiology involved.

There is suggestive evidence that such a categorization of SAD subtypes may be useful and valid (Brotto, Basson, & Gorzalka, 2004). Specifically, when women with the three subtypes of SAD were combined into a single, heterogeneous SAD group and compared to women with no sexual difficulties, no significant differences in either physiological or subjective arousal emerged when women were tested in a laboratory. However, when SAD subtypes were separated, those with Genital SAD showed impaired vaginal pulse amplitude (VPA), while those with Subjective and Combined SAD showed a significant increase in VPA, with the Subjective SAD group showing a much greater percentage increase in VPA than all other groups. With respect to the groups' self-reported arousal, those with Genital and Combined SAD had significantly lower self-reported genital arousal than women with Subjective SAD and the control group, as might be predicted. That Brotto et al. (2004) found no significant differences between the heterogeneous SAD and the control group, but did find significant differences when the subtypes were compared, suggests that examining women with arousal complaints as a heterogeneous whole may mask interesting and potentially important subgroup differences.

One place where such group heterogeneity has masked a subgroup effect is in the pharmacological literature for female sexual dysfunction. For example, with regard to the effects of sildenafil citrate (Viagra) on SAD, a study by Basson, McInnes, Smith, Hodgson, and Koppiker (2002) found this vasoactive agent to be completely ineffective. However, when women from a more diagnostically homogeneous group with Genital SAD were examined, there was some evidence for the efficacy of sildenafil (Basson & Brotto, 2003).

Sympathetic Nervous System and Female Sexual Arousal Disorder

Research over the past decade has explored the role of the sympathetic nervous system (SNS) in the pathophysiology of sexual complaints. In women with no sexual difficulties, there is an accumulating body of literature supporting the effect of SNS activation on sexual arousal. For example, plasma norepinephrine has been shown to correlate with increases in sexual arousal, reaching a peak during orgasm (Exton et al., 1999). Using vaginal photoplethysmography, there were facilitatory effects on sexual arousal of both pharmacological agents (Meston & Heiman, 1998; Rosen, Phillips, Gendrano, & Ferguson, 1999; Rubio-Aurioles et al., 2002) and non-pharmacological interventions (Brotto & Gorzalka, 2002; Meston & Gorzalka, 1995, 1996a, 1996b; Palace & Gorzalka, 1990), which increase SNS activity. Meston, Gorzalka, and Wright (1997) found that clonidine, a selective α_2-adrenergic agonist which inhibits SNS activity, inhibited genital arousal. The photoplethysmograph has been shown to be sensitive to the vasocongestive effects of these agents, even in cases where subjective sexual arousal is not significantly affected (e.g., Meston & Gorzalka, 1995, 1996a, 1996b; Meston & Heiman, 1998; Meston & Worcel, 2002; Palace & Gorzalka, 1992).

In the few studies which have examined the effects of SNS activation in women with sexual difficulties, the results paralleled the findings when women used sildenafil: namely, SNS studies have involved heterogeneous groups of women with varying sexual difficulties and the results have been largely inconsistent. For example, Palace and Gorzalka (1990) found that vaginal blood volume (VBV), but not VPA, significantly increased after visual sexual stimulation following exposure to an anxiety-eliciting film in a sample of women with mixed sexual dysfunction. They hypothesized that anxiety-eliciting stimuli, by facilitating SNS activity,

may play a role in restoring genital arousal in women with sexual dysfunction. However, their inclusion of women with four different types of sexual dysfunction renders any conclusion about the link between SNS and specific sexual dysfunction pathophysiologies inconclusive.

In contrast, the only study to separate sexual dysfunction subtypes found differential effects of SNS activation. Using physical exercise as a method of enhancing SNS activity, Meston and Gorzalka (1996a) showed that sexually healthy women and those with low sexual desire (but normal orgasmic function) responded with increased VPA after the exercise manipulation whereas anorgasmic women had an impaired VPA. It was speculated that impairments in autonomic nervous system functioning may play an etiological role in female orgasmic disorders (Meston & Gorzalka, 1996a).

Three studies have investigated SNS-enhancing agents in women with SAD. One study found a facilitatory effect of phentolamine mesylate, a non-selective α_1- and α_2-adrenergic antagonist, on VPA in women with SAD diagnosed according to the DSM-IV-TR (Rosen et al., 1999). In another study, Rubio-Aurioles et al. (2002) found a significant facilitatory effect of phentolamine mesylate on VPA in postmenopausal women with DSM-IV-TR defined SAD who were receiving hormone replacement. However, Meston and Worcel (2002) failed to find an effect of the α_2-adrenergic antagonist, yohimbine, on genital or subjective sexual arousal in a sample of women with Genital SAD, but when yohimbine was combined with the vasodilator, L-arginine glutamate, this SNS-enhancing drug significantly facilitated VPA. Given that no group received L-arginine glutamate alone, it is difficult to draw conclusions with respect to the role of the SNS.

Objectives of the Current Study

Based on the literature that (1) heightened SNS activity significantly facilitates genital arousal in sexually healthy premenopausal women (Brotto & Gorzalka, 2002; Meston & Gorzalka, 1995, 1996b) and in premenopausal women with low desire (Meston & Gorzalka, 1996a), (2) pharmacologic agents which enhance SNS activity increase genital arousal in women with SAD diagnosed according to the *DSM-IV-TR* (Rosen et al., 1999; Rubio-Aurioles et al., 2002), and (3) agents which decrease SNS activity result in inhibited genital arousal in sexually healthy women (Meston et al., 1997), it seems reasonable to explore the effects of heightened SNS activity on genital arousal in women with different subtypes of SAD, diagnosed according to the recently proposed definitions (Basson et al., 2003). Laboratory-induced hyperventilation (LIH), a technique that reliably facilitates SNS activity (George et al., 1989; Olsen et al., 1998; St. Croix, Satoh, Morgan, Skatrud, & Dempsey, 1999) and produces SNS predominance for at least 7 min (Achenbach-Ng, Siao, Mavroudakis, Chiappa, & Kiers, 1994), was used to enhance SNS activity in the current study. LIH has previously been found to significantly facilitate genital sexual arousal in sexually healthy premenopausal women (Brotto & Gorzalka, 2002).

We specifically hypothesized that (1) based on the findings of Brotto et al. (2004), women with Genital SAD would show an impaired VPA response compared to all other groups, and women with Subjective SAD would show an increased VPA response compared to all other groups when tested in the no LIH condition; (2) the LIH manipulation would significantly enhance VPA among women in the control group and in women with Genital SAD; and (3) VPA patterns in women with Subjective SAD would be unaffected by the LIH given that they do not have an impairment in physiological arousal. The effects of LIH in the Combined SAD group are unknown and exploratory, and effects of LIH on subjective measures of arousal are also exploratory given our belief that the effects of SNS on genital arousal invoke a physiological mechanism of action.

Method

Participants

We received inquiries from 132 women and 102 women between the ages of 18 and 45 participated in this study. All participants were recruited through two separate advertisements (one recruiting women with, and the other recruiting women without, sexual difficulties) posted throughout a university and the community, and in the local newspapers of Vancouver, Canada.

A telephone screen was used to assess interested participants for eligibility. Specifically, women were assessed either for a complete lack of sexual difficulties (control group, $n = 42$), or for sexual difficulties meeting the SAD subtype criteria proposed and defined by Basson et al. (2003). There were 16 women with Genital SAD (absent or impaired genital sexual arousal with normal subjective sexual excitement to non-genital sexual stimuli), 16 women with Subjective SAD (absence of, or markedly diminished, feelings of sexual arousal from any type of sexual stimulation, with normal vaginal lubrication or other signs of physical response), and 28 women with Combined SAD (absence of, or markedly diminished, feelings of sexual arousal from any type of sexual stimulation in addition to complaints of absent or impaired genital sexual arousal). Specifically, descriptions of the SAD diagnostic groups as described in Basson et al. (2003) were converted into a series of yes/no questions by Basson and the first author (L.A.B). L.A.B., who was also a member of the consensus conference that described these SAD subtypes,

together with two trained Ph.D. level clinical psychology students, categorized participants to SAD subgroups.

Diagnoses initially made during the telephone screen were validated in person during the first session with a semi-structured clinical interview. In rare cases where the information provided during the telephone screen conflicted with information provided during the interview, we used Female Sexual Function Index (FSFI; Rosen et al., 2000) scores (arousal and lubrication domains) to determine SAD group assignment. Only women who reported that their arousal difficulties were acquired (determined by asking women if there had ever been a time when the sexual response was not a problem for them) and generalized (determined by asking women if there was any current situation in which they did not experience the arousal complaint) were included. Many women also indicated sexual desire complaints or orgasmic difficulties due to their low or absent arousal; however, we only included those women if the sexual arousal complaints were more distressing than the desire or orgasm complaints.

Participants were excluded for current use of medications or presence of a major medical condition known to affect sexual functioning ($n = 6$; e.g., antidepressants, anti-hypertensive medications, diabetes, and hypertension). In addition, women were excluded if they currently experienced Major Depressive Disorder or any other major mental illness ($n = 6$), were not exclusively heterosexual ($n = 2$), and never had had sexual intercourse ($n = 2$). We also excluded any woman with a diagnosis of Dyspareunia or Female Orgasmic Disorder alone, or for whom the arousal complaints were situational ($n = 14$).

As shown in Table 1, demographic characteristics were similar across participants in both the control group and the group of women with heterogeneous arousal complaints, and across the SAD subtypes. The ages ranged from 18 to 46, with no significant group differences in age. There were similarly no significant group differences in marital status, ethnicity, and level of education, all ps $> .05$.

Measures

Film Stimuli

Film stimuli consisted of two 7-min films which included a 1-min display of the word "relax" followed by a 3-min clip containing neutral material (either a clip from a documentary about Stonehenge or glaciers), followed by a 3-min clip containing erotic material involving a nude, heterosexual couple engaging in foreplay and sexual intercourse. Two different erotic video clips were used, and the content was derived from female-friendly erotica that was matched on the number, order, type, and duration of sexual activities, and had previously been found to increase genital and subjective sexual arousal in women (Basson & Brotto, 2003). The different segments of the erotic films were professionally spliced together to form one continuous videotape with audio accompaniment.

Vaginal Photoplethysmograph

The vaginal photoplethysmograph (Sintchak & Geer, 1975) was used to measure physiological sexual arousal. The vaginal photoplethysmograph is a small, tampon-shaped device, which is self-inserted into the vagina, and measures vaginal vasocongestion—an indirect measure of sexual arousal. We used VPA rather than VBV, as VPA has been shown to be a more sensitive measure of genital arousal (Laan, Everaerd, van der Velde, & Geer, 1995).

Subjective Measurement of Arousal: Film Scale

The Film Scale, a 33-item self-report questionnaire, was used to assess subjective arousal and affective reactions to the films. This scale was adapted from Heiman and Rowland (1983) and assessed six domains: mental arousal (1 item), perceptions of physical arousal (4 items), autonomic arousal (5 items), anxiety (1 item), and positive and negative affect (11 items each). Items were rated on a 7-point Likert scale from *not at all* (1) to *intensely* (7). The scale has been found to be a valid and sensitive measure of emotional reactions to erotic stimuli (Heiman, 1980; Heiman & Hatch, 1980; Heiman & Rowland, 1983).

Interview

In order to verify the classification of participants into the control and SAD subtype groups made during the telephone screen, a semi-structured interview assessing sexual arousal was conducted during the first session by one of the female researchers. We used criteria from the new definitions (Basson et al., 2003) to verify correct subtyping of women with SAD by having women respond to a series of yes/no questions.

Self-Report Questionnaires

The following self-report measures were administered to provide an index of sexual functioning and mood. Unfortunately, the questionnaires were incorporated into the protocol part-way into the study and, as a result, only a subsample completed all measures (sexually healthy control group $n = 12$; Genital SAD $n = 9$; Subjective SAD $n = 8$; Combined SAD $n = 14$).

Table 1 Demographic characteristics of participants

Variable	Control group ($n = 42$)		Genital SAD ($n = 16$)		Subjective SAD ($n = 16$)		Combined SAD ($n = 28$)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age (in years)	23.7	5.4	24.7	6.1	27.5	8.9	26.6	7.0
Education (in years)	14.6	1.9	15.2	1.5	14.9		14.9	1.4
Relationship status (%)								
Single	85.7		81.3		75.0		75.0	
Married or common-law	14.3		18.7		25.0		25.0	
Ethnicity (%)								
Euro-Canadian	61.9		81.3		68.8		75.0	
Asian	28.6		12.5		18.8		25.0	
African	2.3		6.3		12.5		0	
First Nations	2.3		0		0		0	
Other	4.8		0		0		0	

Female Sexual Functioning Index (FSFI) The FSFI (Rosen et al., 2000) is a 19-item measure assessing desire, subjective arousal, lubrication, orgasm, satisfaction, and pain during sexual activity over the past month. The FSFI has been shown to be a valid measure for differentiating women with SAD from sexually healthy women. Possible scores range from 1.2 to 6.0 for Desire; from 0 to 6 for Arousal, Lubrication, Orgasm, and Pain; from 0.8 to 6 for Satisfaction, and from 2 to 36 for the FSFI total score, with higher scores indicating better levels of sexual functioning.

Beck Anxiety Inventory (BAI) The BAI (Beck, Epstein, Brown, & Steer, 1988) is a commonly used, 21-item self-report measure of the extent to which one has been experiencing symptoms of anxiety over the past week with higher scores indicating more anxiety (scale: 0–63). The measure was included given prior research showing an effect of anxiety on SNS levels and physiological sexual arousal (Palace & Gorzalka, 1990).

Fear of Negative Evaluation (FNE) The FNE (Watson & Friend, 1969) is a 30-item true/false questionnaire that was administered as a measure of social-evaluative anxiety to tap into the extent to which participants may be inclined to respond in a certain fashion out of a fear of negative evaluation. Higher scores indicate more social-evaluative anxiety (scale: 0–30).

Procedure

Participants were informed that the purpose of the study was to explore the effects of deep breathing on sexual response. Each woman participated in two 1-h sessions. A between-within repeated measures design allowed for the comparison between SAD and control groups, between baseline and heightened SNS conditions, and also within groups to explore the effects of the erotic stimulus compared to the neutral stimulus.

The two sessions were conducted by one of the female researchers. The first session began with a researcher restating the procedures, orienting the participant to the laboratory, obtaining written consent, and answering any questions about the study protocol. Following this, the semi-structured interview was conducted to verify the original SAD classification derived from the telephone screen. Finally, participants were left alone to complete the battery of self-report questionnaires.

The second session was booked approximately 1 week later. During this session, women viewed two neutral-erotic film sequences. Prior to one of the film sequences, women took part in the LIH procedure. Upon entering the laboratory, participants were seated comfortably in a reclining chair in the privacy of an internally locked room adjacent to the experimenter's room, and asked to insert the vaginal photoplethysmograph with the aid of diagrammed instructions, after the female researcher had left the room. Participants remained fully clothed and were able to cover themselves with a light blanket at all times. Standard placement of the vaginal probe was used such that it was unnoticed by the participant.

The photoplethysmograph was turned on 30 min prior to use in order to minimize potential light history and temperature sensitivity effects. Once the photoplethysmograph probe was comfortably in place, participants were asked to sit back quietly in order to allow for a 5-min adaptation period. Following this, participants completed the first of four Film Scales. If they were randomized to the LIH first group, women then took part in the LIH protocol as described by Brotto and Gorzalka (2002). This involved 2 min of rapid, deep breathing at a rate of 30 breaths/min. Subjects breathed along with a pre-recorded audiocassette of paced respiration and were asked to breathe in and out as deeply as possible. The female

researcher remained in the room with the participant during the LIH procedure to ensure similar breathing rates and intensities across participants, and left the room once the 2-min LIH segment ended.

Immediately after the researcher left the room, women were shown the first of two neutral-erotic video sequences, presented in a randomized, counterbalanced order on a color television monitor positioned where the woman could sit comfortably in a recliner with full view of the screen. Videos began with the word "relax" presented on the screen for 1 min, followed by 3 min of neutral footage, and then 3 min of erotic material. During presentation of the films, physiological arousal was measured with the photoplethysmograph. Immediately following the erotic film, women completed the second of four Film Scales. There was then a 15-min rest period in order to allow arousal levels to return to baseline. Following the rest period, women took part in the no-LIH segment during which a different neutral-erotic film sequence was shown. Immediately prior to the neutral film and following the erotic film, women completed the third and fourth Film Scales, respectively. The procedure was identical for women randomized to the LIH second condition except that the LIH procedure took place after the first neutral-erotic film sequence was shown. For this segment, the female researcher entered the room to ensure proper adherence to the LIH protocol and participants covered themselves with a light blanket to ensure privacy given that the probe remained inserted. The order of the neutral-erotic film sequences (A or B) and LIH first/LIH second groups was counterbalanced across participants. Apart from when the researcher was in the room with the participants during the LIH, all communication took place over a voice-activated intercom between rooms.

At the completion of the second session, participants were debriefed and informed of the study hypotheses. All participants were paid a $40.00 honorarium and were given a copy of their personal psychophysiological assessment with an accompanying description of their VPA output. All procedures were approved by the university's Clinical Research Ethics Board.

Psychophysiological Recording

Psychophysiological data were continuously recorded during presentation of the film sequences using Acqknowledge III, Version 3.5 (BIOPAC Systems Inc., Santa Barbara, CA), a Model MP100WSW data acquisition unit (BIOPAC Systems Inc.), and an HP Vectra Celeron personal computer. A sampling rate of 200 samples/second was used for VPA throughout the 180 s of neutral and 180 s of erotic film exposure. The signal was band-pass filtered (0.5–30 Hz). One of two vaginal probes (Behavioral Technology Inc., Salt Lake City, UT) was used. Data were analyzed in 30 s segments, then averaged over the neutral and erotic segments separately, resulting in two data points per subject in each of the LIH and no-LIH conditions. Artifact detection following visual inspection of the data permitted the smoothing of artifacts. The vaginal probe was sterilized in a solution of Cidex OPA (ortho-phthalaldehyde 0.55%), a high level disinfectant, immediately following each session. Analyses of VPA in the no-LIH condition were conducted comparing mean VPA during the neutral with mean VPA during the erotic conditions. To explore effects of LIH on VPA, a VPA percent change score was calculated for the no-LIH and LIH conditions separately and then compared. This change score was obtained by the following formula: (mean erotic VPA − mean neutral VPA)/mean neutral VPA.

Results

Sexual and Affective Characteristics of the Samples

Scores from the FSFI, BAI, and FNE were analyzed in order to compare differences between women with and without sexual dysfunction on measures of current sexual functioning and to ensure the absence of significant differences on measures of anxiety and fear of negative evaluation. We also compared women across SAD groups to women in the control group and present these data in Table 2. A Bonferroni correction was applied to FSFI analyses given that the FSFI subscales are significantly correlated (Rosen et al., 2000). Thus, a p value of .007 (.05/7) was necessary in order to reach statistical significance.

Table 2 shows significant group differences on the Desire, $F(3, 41) = 11.30$, $p = .001$, Arousal, $F(3, 41) = 7.93$, $p = .001$, and Orgasm, $F(3, 41) = 9.00$, $p = .001$ subscales of the FSFI, as well as the Full Scale FSFI score, $F(3, 41) = 6.35$, $p = .001$, with a trend towards significance on the Lubrication domain, $F(3, 41) = 3.88$, $p = .016$. Tukey's post-hoc test revealed that women with Subjective and Combined SAD had significantly less desire than those in the control group and the Genital SAD group. Women with Subjective and Combined SAD had significantly less arousal than those in the control group. All three SAD subgroups had significantly poorer orgasmic function than the control group. And, finally, using the FSFI total score as an overall measure of sexual function, the Subjective and Combined SAD groups had significantly lower scores than the control group. There were no significant group differences on the Satisfaction or Pain subscales between the groups.

A one-way analysis of variance (ANOVA) revealed a significant group difference on BAI scores, $F(3, 43) = 3.32$, $p = .029$. Tukey's post-hoc test revealed that women in the Combined SAD group were significantly more anxious than women in the control group. No significant difference was found between the groups on the FNE, $F(3, 43) = 1.16$.

Table 2 Group differences on scores from the Female Sexual Functioning Index (FSFI), Beck Anxiety Inventory (BAI), and Fear of Negative Evaluation (FNE)

Variable	Control group (n = 20)		Genital SAD (n = 10)		Subjective SAD (n = 10)		Combined SAD (n = 15)			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	*p*
FSFI desire[a]	4.57	1.00	4.20	0.90	2.60	0.79	3.00	0.97	11.30	.001
FSFI arousal[b]	4.94	1.58	3.87	1.02	3.37	0.51	2.83	1.05	7.93	.001
FSFI lubrication[b]	5.00	1.62	4.17	0.93	4.53	1.00	3.36	1.28	3.88	.017
FSFI orgasm[b]	4.74	1.94	2.89	1.29	2.62	1.43	1.80	1.16	9.00	.001
FSFI satisfaction[c]	4.12	1.94	4.22	1.43	2.93	1.59	2.89	1.22	2.42	ns
FSFI pain[b]	4.28	2.50	3.42	2.12	4.22	1.95	4.06	2.24	.295	ns
FSFI Full Scale[d]	27.65	8.36	22.77	2.93	20.28	4.22	17.94	5.60	6.35	.001
BAI[e]	4.62	3.20	9.22	6.76	11.25	10.57	13.14	7.97	3.32	.029
FNE[f]	13.31	8.08	14.11	9.62	18.63	6.90	17.71	7.23	1.16	ns

Higher scores denote better sexual function on the FSFI. *Scale ranges*: [a]1.2–6.0, [b]0–6.0, [c]0.8–6.0, [d]2–36

Higher scores denote more anxiety on the BAI and greater fear of negative evaluation on the FNE. *Scale ranges*: [e]0–63, [f]0–30

Effects of Erotic Stimuli and SAD on Physiological Sexual Arousal in the Absence of Heightened Sympathetic Nervous System Activity

A repeated measures ANOVA revealed a significant main effect of film, $F(1, 100) = 30.59, p < .001, d = 0.53$, such that there was a mean increase in VPA from the neutral to the erotic film. The interaction between group and film (neutral versus erotic) was not significant for VPA, $F(1, 100) = 2.99, p > .05$.

Subsequently, a repeated measures ANOVA was conducted using SAD subtypes and control as the between-subjects group factor. There was a significant interaction between group and film, $F(3, 98) = 4.51, p < .01$, such that the mean increase in VPA from neutral to erotic stimuli varied by group. This is displayed graphically in Fig. 1 using percent change scores for each group. Follow up simple effects analyses revealed that women with Subjective SAD had the largest increase in VPA from neutral to erotic stimulus conditions compared to women in the other three groups ($p < .05$, Control Group $d = 0.38$; Genital SAD $d = 0.39$; Subjective SAD $d = 0.85$; Combined SAD $d = 0.69$). A comparison between the groups on baseline VPA (i.e., mean VPA during the neutral film of the no-LIH condition only) failed to reveal a significant main effect, $F(3, 98) = 1.00$.

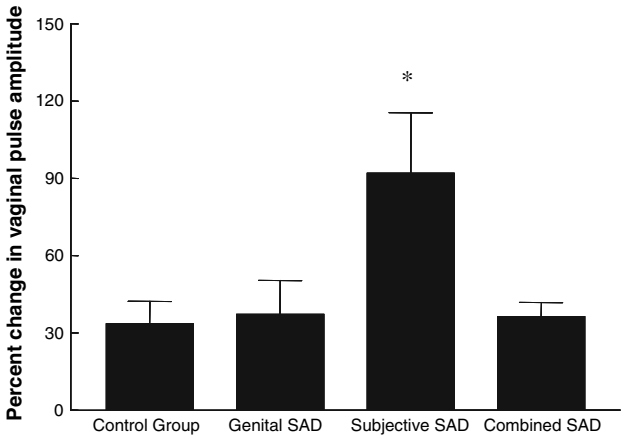

Fig. 1 Effects of an erotic stimulus on vaginal pulse amplitude (VPA) in the absence of heightened sympathetic nervous system activity. Data represent the mean percent increase in VPA from the neutral to the erotic stimulus conditions ± SEM. *$p < .01$, comparing women with Subjective Sexual Arousal Disorder to women in all other groups

Effects of Erotic Stimuli and SAD on Self-Report Measures in the Absence of Heightened Sympathetic Nervous System Activity

A repeated measures ANOVA on the Film Scale measures revealed that the main effect of film was significant for autonomic arousal, $F(1, 42) = 19.58$, $p < .001$, $d = 0.83$; perception of genital arousal, $F(1, 42) = 38.73$, $p < .001$, $d = 1.23$; mental sexual arousal, $F(1, 42) = 32.94, p < .001$, $d = 0.92$; and positive affect, $F(1, 42) = 23.06$, $p < .001$, $d = 0.80$, with each of these self-report scores increasing in response to the erotic stimulus. The main effect of film on anxiety, $F(1, 42) = 1.68$, $d = 0.26$, and negative affect, $F(1, 42) = 1.39$, $d = 0.06$, was not significant. No significant interactions between film and SAD (heterogeneous group) emerged. When analyses were repeated to compare the different SAD subtypes and control women, there was also no significant interaction between film and SAD subtypes.

Effects of Heightened Sympathetic Nervous System Activity on Physiological Sexual Arousal

Percentage change scores in VPA from neutral to erotic film conditions were computed during the no-LIH and LIH

conditions and then compared. There was a significant interaction between LIH condition (no-LIH and LIH) and group (control and heterogeneous SAD) on VPA, $F(1, 100) = 7.57$, $p < .01$, as illustrated in Fig. 2. Planned dependent t-tests for each group revealed that LIH significantly increased VPA in the control group, $t(41) = -2.41$, $p < .05$, $d = 0.45$, but not in the heterogeneous SAD group, $t(59) = 1.00$, $d = 0.15$.

Reanalysis of these data using a repeated measures ANOVA with SAD subtypes as the between-subjects factor also revealed a significant LIH by SAD subtype interaction, $F(3, 98) = 5.60$, $p < .01$, as shown in Fig. 3. Simple effects analyses performed on each SAD subtype revealed significant increases in VPA in the control, $p < .05$, $d = 0.44$, and the Combined SAD groups, $p < .05$, $d = 0.49$, and a significant reduction in VPA among women with Genital, $p < .05$, $d = 0.56$, and Subjective SAD, $p < .05$, $d = 0.47$.

We also compared baseline VPA during the LIH condition (i.e., mean VPA during the neutral film only) but this failed to reveal a significant main effect of group, $F(3, 99) = 2.12$, $p > .05$.

Effects of Heightened Sympathetic Nervous System Activity on Self-Report Measures

There were no significant main effects of group (control group versus heterogeneous SAD group) or LIH, nor a significant LIH by group interaction, for any of the Film Scale measures.

There were also no significant main effects nor an interaction between SAD subtypes and LIH for any of the Film Scale measures.

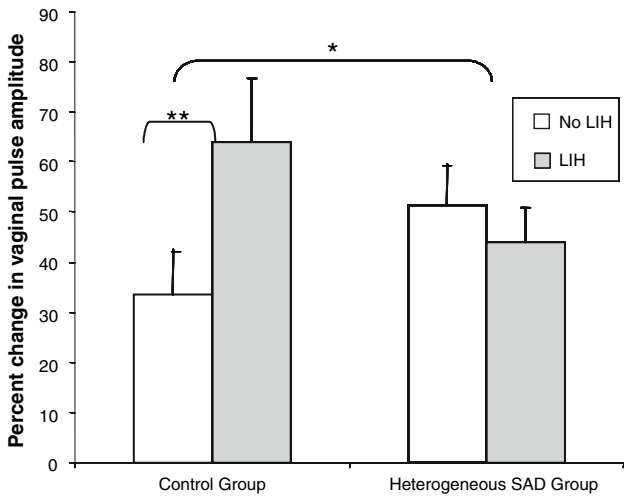

Fig. 2 Effects of laboratory-induced hyperventilation (LIH) on vaginal pulse amplitude (VPA) percent increase scores (neutral to erotic) in women without and with heterogeneous complaints of SAD. Data represent means ± SEM. *$p < .05$, significant interaction between group and sympathetic nervous system activity, **$p < .05$, significant effect of LIH

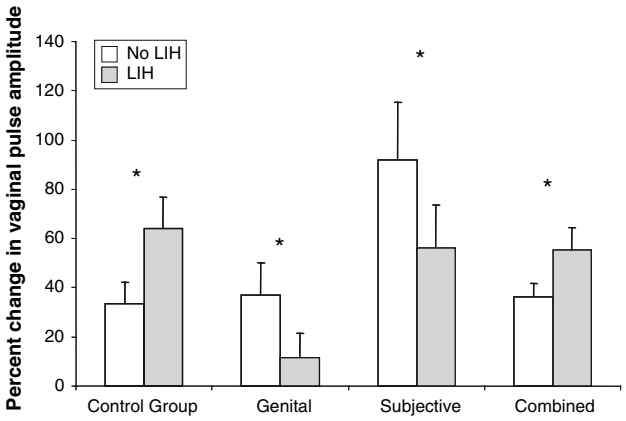

Fig. 3 Effects of laboratory-induced hyperventilation (LIH) on vaginal pulse amplitude (VPA) percent increase scores (neutral to erotic) in subtypes of FSAD and the control group. Data represent means ± SEM. *$p < .05$, significant effect of LIH

Discussion

The erotic film used in this study effectively increased both physiological and self-reported sexual arousal, autonomic activity, and positive affect. The lack of a group by film interaction suggests that the film stimuli were effective at increasing arousal in all participants, independent of group status, and that female-made, female-focused erotic stimuli were viewed positively by women with and without sexual dysfunction.

Significant differences in VPA from neutral to erotic film conditions were found among the different SAD subtypes in the no-LIH condition, but not when the different SAD subtypes were examined as a heterogeneous whole. Specifically, the Subjective SAD group showed the largest increase in VPA compared to all other groups, replicating the findings of Brotto et al. (2004). While the study by Brotto et al. found that women with Genital SAD did not exhibit a significant increase in VPA with erotic film exposure, the present study found that women with Genital SAD showed a normal VPA response. Although these data contrast with those of Brotto et al. (2004), they replicate the findings of van Lunsen and Laan (2004) who did not find any significant VPA differences between older women with and without genital SAD in their studies. Also in the present study, women with Combined SAD showed a normal VPA response indistinguishable from women without sexual complaints. Combined with the finding that women with Genital SAD showed normal VPA responses, the data suggest that although subjectively complaining of decreased genital arousal, the women assigned to the Genital and Combined SAD groups did not show objectively lower levels of genital arousal than women in the control group. There were also no significant group differences in baseline VPA (measured during the neutral film only), suggesting that baseline differences in VPA cannot

account for the increase in VPA. As pointed out by Brotto et al. (2004), a normal VPA response among women complaining of a lack of physical arousal may reflect impairments in their ability to attend to and detect these genital responses.

One must consider the proposition that physiological and subjective arousal in women may represent two separate components of overall sexual arousal. Specifically, based on research in which it was found that women, but not men, showed a physiological arousal response to sexual stimuli involving non-human primates (while not reporting any subjective arousal), Chivers and Bailey (2005) proposed that women's genital vasocongestion is provoked by exposure to non-specific sexual features, independent of what those features are. Further, they proposed that this response may be an evolved adaptation which protects women from physical harm or infection by preparing the vagina for sexual activity. According to this hypothesis, women become physiologically aroused in the presence of non-specific sexual stimuli, but then make a cognitive appraisal of the stimuli as either subjectively arousing or not. It is possible that among women with Genital and Combined SAD, there are impairments in the cognitive appraisal of triggered genital arousal events and it is the latter that is noted as being problematic in these women and which prompts them to seek clinical attention.

With respect to the LIH data, we found a group by LIH interaction such that sexually healthy women showed enhanced VPA to heightened SNS activity, which replicates prior research (Brotto & Gorzalka, 2002; Meston & Gorzalka, 1995, 1996a, 1996b; Meston & Heiman, 1998; Meston & Worcel, 2002; Palace & Gorzalka, 1990, 1992; Rosen et al., 1999; Rubio-Aurioles et al., 2002). In contrast, heightened SNS activity had no significant effect on physiological arousal in women with heterogeneous SAD. These results are not surprising, however, given the lack of conclusive findings and inconsistencies in the literature on the effects of heightened SNS activity in women with heterogeneous sexual complaints (Meston & Gorzalka, 1996a; Meston & Worcel, 2002; Palace & Gorzalka, 1990; Rosen et al., 1999; Rubio-Aurioles et al., 2002). However, significant differences in VPA during the LIH condition emerged when the subtypes of SAD were examined separately. Specifically, women with Combined SAD exhibited similar results as control participants, showing a significant increase in VPA to the erotic stimulus with heightened SNS activity, while women with Genital and Subjective SAD displayed the opposite effect. Rather than enhancing VPA, heightened SNS activity exerted a negative effect on physiological arousal in response to erotic stimuli, thereby significantly decreasing VPA in women with Genital and Subjective SAD.

What are the mechanisms by which the LIH manipulation enhanced VPA in women in the Combined SAD group? Because the LIH manipulation had no effect on subjective arousal in any of the groups of women, this suggests that the physiological effects were not perceived at a cognitive level. Furthermore, because the SNS effect was present only after exposure to the erotic film, this suggests that the SNS-enhancing effect of LIH was dependent on a present state of sexual arousal, and that LIH had no VPA-enhancing effects on its own. This is consistent with the finding that acute, intense exercise, which enhances SNS activity, significantly enhanced VPA only after an erotic film was shown (Meston & Gorzalka, 1995, 1996a, 1996b; Meston et al., 1997; Meston & Heiman, 1998).

The SNS has been found to play a significant role in the female sexual response, with increased SNS activity occurring during intercourse and orgasm, and rapidly declining thereafter (Fox & Fox, 1969; Wiedeking, Ziegler, & Lake, 1979). The mechanism by which heightened SNS activity increases sexual response in women remains to be determined as it is unknown whether physiological arousal in the form of vaginal vasodilation occurs in the veins, arteries, arterioles, capillaries, or venules (Levin, 1992; Schneiden & Rees, 1985). However, previous studies examining the effects of SNS activity on physiological sexual arousal have concluded that enhanced SNS activity may selectively prepare the body for genital arousal as opposed to inducing a more general increase in peripheral resistance (Meston & Heiman, 1998; Palace, 1995; Palace & Gorzalka, 1990). Because only women in the Combined SAD group and the control groups were found to display this beneficial effect, these conclusions are limited to these groups of women.

In contrast, women with Subjective or Genital SAD showed an impaired VPA response after the LIH manipulation, inconsistent with our initial hypothesis. That these two groups differed from the Combined SAD group is reminiscent of the findings of Meston and Gorzalka (1996a), who found that women with orgasmic difficulty experienced a decreased physiological response to exercise-induced increases in SNS activity, whereas orgasmic women showed increased physiological patterns. It was suggested that the physiological events taking place during heightened SNS activity are detrimental to the sexual response in anorgasmic women. Although orgasmic complaints similarly affected each of the SAD subtypes in the present study, the conclusions by Meston and Gorzalka (1996a) may be extended to the present samples in that it is possible that differences in baseline autonomic activity exist between the groups. When the groups are then exposed to an activity, which increases their SNS level, their new overall levels of SNS activity may then interact with a sexual stimulus to alter their physiological arousal. Partial support for this lies in the fact that we found the Combined SAD group to have significantly higher levels of baseline anxiety, as measured by the BAI. Speculating on a potential non-linear effect of SNS activity, it is possible that low levels of SNS activation may be inhibitory (as seen in the non-anxious women with Subjective or Genital SAD) whereas

higher levels of SNS activity may be facilitatory (as found in the anxious women with Combined SAD). However, among the control group, who were also non-anxious, LIH exerted a facilitatory effect. This also suggests that the SNS effects may interact with whether or not the woman has a sexual dysfunction, to influence VPA. Support for this stems from Barlow's theory that autonomic arousal may exert differential effects on sexual complaints depending on whether or not an individual has a sexual dysfunction (Barlow, 1986). As this is entirely speculative, these assumptions must be tested and the current results must be replicated.

Still another possibility, for which there is no empirical evidence as yet, is that the Genital and Subjective SAD subtypes may already be functioning at a higher level of SNS activity than the other SAD groups and that it is possible that the addition of the SNS manipulation functioned to reduce VPA. According to the Yerkes-Dodson Law of arousal and performance (Yerkes & Dodson, 1908), there is an optimal level of arousal below and above which performance is significantly impaired. If the facilitatory effect of heightened SNS activity on VPA indeed follows such an inverted-U pattern, this would provide support for this conjecture. Given that autonomic activity was not assessed and only inferred indirectly, future studies should attempt to test this hypothesis by directly measuring SNS activity at baseline in women with sexual dysfunction.

It is possible that cognitive distraction induced by the LIH procedure may have contributed to the impaired VPA response in the Genital and Subjective SAD groups. Excitation transfer theory posits that individuals experiencing heightened activity followed by a sexual stimulus will report significantly lower levels of sexual arousal if they perceive residual effects from the prior activity (Cantor, Zillmann, & Bryant, 1975), and techniques that draw attention away from genital excitement indeed have been shown to significantly reduce the level of genital sexual arousal (Sakheim, Barlow, Beck, & Abrahamson, 1985). Excitation transfer may therefore have explained SAD subgroup differences in genital arousal but not self-reported arousal given that the groups did not significantly differ in their Film Scale responses to the erotic stimulus.

It is also possible that the negative effect of LIH on VPA in the Genital and Subjective SAD groups was due to a failure of our manipulation to evoke sufficiently high levels of SNS activity. In other words, it may be that the LIH procedure was more effective in the control and Combined SAD groups. Meston and Gorzalka (1996b) employed intense, acute exercise, which has been found to lead to significantly elevated levels of SNS activity for up to 40 min following cessation of exercise and to lead to changes in levels of testosterone, cortisol, and prolactin. In an effort to employ a technique that did not preclude the participation of women who are not physically capable of intense exercise for 20 min, we used LIH, but a state of SNS predominance lasts for only 7 min (Achenbach-Ng et al., 1994). It is possible that the use of exercise instead of hyperventilation would have led to different patterns of VPA response due to more effectively enhancing SNS activity and for a longer duration. It is also possible that a pharmacological intervention (e.g., phentolamine mesylate) would have been more effective at enhancing VPA, though the potential side effects of such a manipulation would have to be considered.

The lack of a significant effect of enhanced SNS activity on subjective arousal may be attributable to a number of factors. It is plausible that increased SNS activity was not registered at the conscious, subjective level—that participants were unaware of the general physiological changes caused by increased SNS activity as well as the specific effects on genital arousal. It is also plausible that the method of measuring subjective arousal was ineffective. Specifically, we used discrete measurements of subjective arousal with the Film Scale, which was administered immediately before and after viewing of the erotic stimuli. Although such discrete measures of arousal are found to be sensitive and simple to use (Steinman, Wincze, Sakheim, Barlow, & Mavissakalian, 1981), Rellini, McCall, Randall, and Meston (2005) found that a continuous measurement of subjective sexual arousal may be more sensitive than discrete measurement.

The finding that women with Combined SAD were more similar to control participants than they were to women with Genital or Subjective SAD, in terms of their VPA responses to heightened SNS activity, despite complaining of genital and subjective impairments, suggests that this subtype of SAD may be qualitatively different. Moreover, Combined SAD may have a more complex pathophysiology given that women in this group do not show an impaired VPA response at baseline, and a heightened SNS manipulation facilitates their genital arousal although this was not detected cognitively. Clearly, this group requires more study in the future with larger sample sizes. In addition, a standardized method of assessing women in this subgroup will ensure correct categorization of women to this group in future research trials.

Clinicians have speculated that women complaining of impaired subjective sexual arousal may not benefit from vasoactive medication designed to increase genital vasocongestion (Basson, 2002a, 2002b), given that the complaints in this group are limited to cognitive and not physiological impairments. Because SNS activation in the current study reduced VPA among women with Subjective SAD, and because vasoactive medications may have similar SNS-enhancing effects, one might conclude that agents designed to increase SNS activity would impair genital arousal in this group. Furthermore, based on the present findings, one might also conclude that vasoactive medications with SNS-enhancing effects might also be detrimental among women with Genital SAD. Until the present study can be replicated in much larger groups of women with Genital and Subjective SAD, these conclusions should be viewed as tentative.

The data from this study have important implications for the search for effective treatments for SAD. The fact that no effect of heightened SNS activity was seen when subtypes of SAD were combined into one, heterogeneous group, but that significant differences emerged when subtypes were examined, may clarify the past inconsistencies in the literature on the effects of various interventions with SAD in which subtypes were not delineated. More specifically, the current results suggest that SNS-enhancing drugs, such as phentolamine mesylate, may be effective for women with Combined SAD but not for women with Genital or Subjective SAD. Given that no self-report measure of sexual arousal or affect was influenced by the SNS manipulation, agents designed to increase SNS activity may prove to be ineffective at the cognitive level. It follows, then, that treatments for Subjective SAD should target arousal in the mind as opposed to the genitals.

The current findings also have implications for the diagnostic category of SAD. Mounting evidence has demonstrated the elusive nature and complexity of SAD (Basson 2000, 2001a, 2001b, 2002a, 2002b) and has argued against the current *DSM-IV-TR* classification, which necessitates lubrication difficulties in order for a diagnosis of SAD to be made. The current findings partially replicated the findings of Brotto et al. (2004), who found the highest VPA increase to an erotic film among women with Subjective SAD. However, we did not replicate their findings in that women with Genital SAD in the present study showed a normal VPA response in the absence of SNS activation. These contradictory findings may be attributable to subtle differences in diagnosing women across the two studies and calls for the validation of instruments used to diagnose SAD subtypes. It is also possible that small sample sizes in the Brotto et al. (2004) study accounted for the absence of a significant increase in VPA. The modest effect size for an effect of the erotic film on VPA found in this group in the current study lends support to this conclusion. One must also consider the limitations inherent to the use of vaginal photoplethysmography in interpreting the contradictory findings. Because of the lack of an absolute metric, and because direct group comparisons are meaningless, we are limited to exploring the degree of change from neutral to erotic stimulus conditions between groups. It is possible that other methods of assessing genital congestion in women (e.g., thermal imaging; Kukkonen, Binik, Amsel, & Carrier, 2007) would be more reliable for examining SAD subtype differences.

Overall, the current findings lend further support to the classification of women with arousal complaints into discrete SAD subtypes. Future studies should aim to elucidate the precise role of the SNS in the development and maintenance of SAD and its subtypes in larger studies with more standardized methods of diagnosing SAD and using different methods of assessing genital arousal.

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