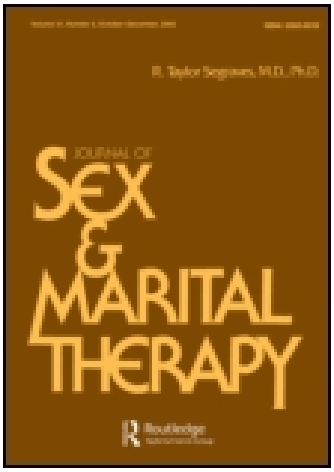

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Anxiety and Female Sexual Functioning: An Empirical Study

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Anxiety and Female Sexual Functioning: An Empirical Study

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*Previous research regarding anxiety and female sexual functioning has yielded conflicting conclusions. This study examined the effect of state/trait anxiety and anxiety sensitivity on sexual responding and the propensity toward sexual inhibition/excitation in women without an anxiety disorder (*n = *100,* M *age = 28.8 years) compared with women with an anxiety disorder (panic disorder or generalized anxiety disorder,* n = *30,* M *age = 30.2 years). Participants completed self-report measures of state and trait anxiety, anxiety sensitivity, sexual functioning, and sexual inhibition/excitation. Women with an anxiety disorder reported worse sexual functioning compared with those without an anxiety disorder (except for desire, lubrication, and pain) and a greater propensity toward sexual inhibition, because of the threat of performance failure and its consequences. Dispositional anxiety and related worries significantly predicted various types of sexual dysfunctions. Findings suggested the importance of considering the relation between anxiety and sexual functioning to design optimal prevention and therapeutic interventions for women with anxiety disorders.*

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INTRODUCTION

According to several studies conducted in the past few years, the relation between anxiety and sexual functioning is complex (van Minnen & Kampman, 2000), and there are still many unexplored questions regarding the comorbidity of sexual dysfunction and anxiety disorders (Figueira, Possidente, Marques, & Hayes, 2001; Laurent & Simons, 2009). Clinicians and researchers, whether adopting a psychoanalytic or a behavioral perspective, have noted the relation between sexual dysfunction and anxiety (Cooper, 1969; Rachman, 1961; Stekel, 1927; van den Hout & Barlow, 2000; Wolpe, 1958).

The research literature regarding the effect of anxiety on sexual arousal has focused mainly on anxiety related to sexual performance concerns (Cranston-Cuebas & Barlow, 1990). In contrast, this study considered the effect of general anxiety levels on sexual functioning on sexual arousal specifically, rather than the effects of anxiety focused on sexual performance.

Many studies have focused their attention on female sexuality and anxiety (Aksaray, Yelken, Kaptanoglu, Oflu, & Ozaltin, 2001; Beggs, Calhoun, & Wolchik, 1987; Hoon, Wincze, & Hoon, 1977; Laan, Everaerd, van Aanhold, & Rebel, 1993; Palace & Gorzalka, 1990). Research on sexual functioning has confirmed the complexity of sexual responding and its wide variability in the female population (Boncinelli, Valentino, Simi, & Genazzani, 2008; Dennerstein & Lehert, 2004; Graziottin, Serafini, & Palacios, 2009; Woodard & Diamond, 2009; Wylie & Mimoun, 2009).

Previous research has found that anxiety has a twofold and conflicting effect on sexual arousal itself (Laurent & Simons, 2009): Clinical studies based on self-reports (Bradford & Meston, 2006) generally report a negative link between anxiety and sexual arousal (in that anxiety weakens sexual arousal), whereas many laboratory studies (based on vaginal pulse amplitude) suggest that anxiety, under certain conditions, can facilitate sexual arousal (Meston & Gorzalka, 1995; Palace, 1995; Palace & Gorzalka, 1990). Therefore, in women, it is particularly important to distinguish between subjective reports of sexual excitation and genital or other somatic responses, rather than considering sexual arousal in an absolute and generic sense (Basson, 2002; Everaerd, Laan, Both, & van der Velde, 2000; Woodard & Diamond, 2009).

Meston and Gorzalka (1995), Palace (1995), and Palace and Gorzalka (1990) concluded that experimentally induced anxiety in women increases the vascular-congestive response (physiological sexual arousal) to sexually arousing stimuli in the laboratory; however, these data were inconsistent with findings cited earlier regarding subjective sexual arousal that show an opposite pattern. The facilitating effect of anxiety on sexual functioning in women can, therefore, be explained by a process that involves the activation of the sympathetic nervous system, that is, via the physiological component of anxiety that results in a condition of provoked anxiety that in itself has a

facilitating effect. These data demonstrate in women desynchronous patterns of sexual physiological and subjective arousal; this was also recently confirmed by Suschinsky, Lalumière, and Chivers (2009) and by Chivers, Seto, Lalumière, Laan, and Grimbos (2010).

However, one also needs to consider how cognitive processes potentially interfere with the physiological dynamics of sexual responding in subjects with an anxiety disorder. A fundamental contribution from this perspective was offered by van den Hout and Barlow (2000): They suggested that anxious subjects react selectively to stimuli perceived as threatening. In these subjects sexual responding activates an anxiety response, which is perceived as a threat and causes fear. Such a threat results in a diminishing of sexual arousal.

Laboratory studies regarding the relations among anxiety, distraction, general activation of the sympathetic nervous system, and sexual responding seem to demonstrate that "anxiety-evoking stimuli do not necessarily disrupt sexual functioning [. . .] This suggests the possibility that anxiety, by itself, may be insufficient to produce sexual dysfunctions" (Norton & Jehu, 1984, p. 176). However, studies that refer to anxiety in an absolute and general sense often have contradictory findings: Women give different answers about the effect that anxiety has on their sexual life:

> Negative mood states, such as anxiety, were reported by some women as reducing their ability to become aroused and by others as increasing it [. . .] In addition to individual variability, women also noted that context and timing were important in this regard. (Graham, Sanders, Milhausen, & McBride, 2004, p. 531)

Examining anxiety in relation to sexual functioning requires not only studying state anxiety (acute emotional response, characterized by a subjective feeling of apprehension and accompanied by increased activation of the autonomic nervous system; Spielberger, 1966), as is often done in experimental laboratory studies; it is also important to understand the effect of trait anxiety (a personal tendency to perceive a wide range of living conditions as threatening and react to them with a high intensity of anxiety; Spielberger, 1966). There is scant empirical literature regarding the interaction between sexual functioning and chronic levels of anxiety: van Minnen and Kampman (2000) maintained that little is known about the sexual functioning of subjects with an anxiety disorder.

An epidemiological study by Dunn, Croft, and Hackett (1999) found that women with medium to high scores on self-report measures of anxiety were particularly at risk of developing sexual problems, especially related to the arousal phase, orgasm, and the inhibition of pleasure. Van Minnen and Kampman (2000) investigated sexual functioning of women with diagnoses of an anxiety disorder: These subjects mostly reported lack of sexual desire, whereas in other phases of the sexual response—such as excitation and

orgasm—their responses were no different from those of women in the nonclinical group. Figueira et al. (2001), extending the studies of Kaplan, Fyer, and Novick (1982) and Sbrocco, Weisberg, Barlow, and Carter (1997), evaluated sexual functioning of people diagnosed with social phobia and panic disorder. Subjects with panic disorder reported a significantly higher percentage of sexual disturbances compared with patients with social phobia. However, none of the women seemed to experience disturbances related to the arousal phase.

Bradford and Meston (2006) conducted a more extensive study that compared physiological and subjective measurements of sexual arousal in a nonclinical group of women. They differentiated for the first time among three types of anxiety: state anxiety (momentary, reactive), trait anxiety (stable, dispositional), and anxiety sensitivity (fear of anxiety-induced sensations). They found a curvilinear relation between state anxiety and physiological sexual arousal (vaginal pulse amplitude) and observed a negative correlation between trait anxiety and arousal scores on the Female Sexual Function Index: dispositional anxiety was associated with lower levels of subjective sexual arousal. However, there was no correlation between trait anxiety and physiological levels of sexual arousal (lubrication scores on the Female Sexual Function Index). The authors suggested that the effect of dispositional anxiety on sexual functioning was more cognitive than physiological: Trait anxiety was negatively correlated with all domains of sexual functioning with the exception of lubrication, which was a more physiological than cognitive phenomenon.

In addressing the issue of sexuality, it is important to consider individual variability in sexual responsiveness: Such responsiveness is regulated by a host of inhibitory and excitatory neurophysiological processes (Janssen & Bancroft, 2007). The dual control model (Bancroft, 2009; Bancroft & Janssen, 2000) proposed a conceptual theory that takes into account the presence of a neurophysiological system related to sexual response activation and another one related to its suppression. Furthermore, this theory refers to the balance between sexual excitation and inhibition and its influence on personal sexual arousal and sexual behaviors. Sexual arousal should not be considered as one-dimensional, such as something that is activated or not; rather, sexual inhibition should be considered as a potentially adaptive pattern, because it blocks the action in situations in which sexual behavior would be potentially dangerous. Because individuals vary in their propensity for sexual excitation and inhibition, those high in a propensity to sexual inhibition and/or low in a propensity to sexual excitation would exhibit more sexual problems in contrast with individuals who show the opposite propensities. Most research regarding the role of cognitive processes in sexual dysfunction have emphasized the excitatory mechanisms (excitation or lack of excitation) and not the inhibitory ones, yet both mechanisms should be taken into consideration. Moreover, it is important to consider the difference between state and trait levels of these mechanisms: Janssen and Bancroft (2007) suggested that such

mechanisms are independent at a trait level, whereas they could be connected at a state level. Thus, consistent with the dual control model, in the present study on sexual arousal it was relevant to investigate not only the excitatory process but also the inhibitory one.

On the basis of these conceptual and empirical underpinnings, the present study aimed to replicate that part of Bradford and Meston's (2006) findings concerning the effect of anxiety on sexual functioning at a subjective level. Given previous evidence regarding the presence of sexual dysfunction in women with an anxiety disorder, and following the recommendation of Bradford and Meston (2006) to involve groups of women with anxiety disorders in future research, our central objective was to compare the sexual responses of women with low levels of anxiety with women with a diagnosis of an anxiety disorder. It could then be ascertained whether elevated levels of anxiety predict the development of sexual dysfunction generally and, in particular, predict difficulties associated with the arousal phase in the female heterosexual population. Moreover, we decided to evaluate the capacity for excitation and inhibition (in reference to the dual control model) from the subjective perspective of each participant. Hence, the aim of this study was also to assess the relation between anxiety levels and the propensity toward sexual inhibition and excitation in the female heterosexual population. We were also interested in exploring hypotheses regarding a direct effect of anxiety levels and excitation/inhibition dimensions on sexual functioning. In consequence, the present study evaluated the following hypotheses:

1. The sexual responses of women with low levels of anxiety will reveal better levels of functioning than those of women with diagnoses of an anxiety disorder.
2. The propensity toward sexual inhibition and excitation of women with low levels of anxiety will be significantly different from that of women with a diagnosis of an anxiety disorder, presenting a lower tendency toward inhibition and a greater tendency toward excitation.
3. Levels of anxiety, particularly of trait anxiety and anxiety sensitivity, will negatively influence sexual functioning, increasing the propensity toward sexual inhibition and decreasing the propensity toward sexual excitation, with these effects being more pronounced in women with an anxiety disorder.

METHOD

Participants

Participants were 130 women (30 in the clinical group and 100 in the control group), between 20 and 40 years of age. (The clinical and control groups did not differ significantly in mean age.) All participants resided in Florence, Italy, and were heterosexual and sexually active at the time of the study. Excluded

from the study were women who were pregnant or who had given birth within a few months before the study; women with hypertension, diabetes or cholesterol problems requiring medication; women with partners who had prostate problems; women with serious pathologies, and any women using medications that could interfere with sexual functioning. Exclusion criteria were monitored using a dedicated questionnaire.

As part of the informed consent process each participant was provided the name, scientific and professional status of the researchers, and the general purpose of the research before completing the questionnaires. Each woman was also assured of the privacy and anonymity of her responses. None of the women received any kind of compensation for their participation.

Clinical Group

The clinical group comprised 30 White/Caucasian women (M age = 30.17 years, SD = 5.96 years). Women were referred by psychologists, psychiatrists, and psychotherapists working in the Sanitary District 11 of Empoli (in Florence, Italy) or in private practise from the surrounding area. The patients had sought these services as a result of anxiety problems and were found by the various specialists to be suffering from anxiety disorders (panic disorder or generalized anxiety disorder, in particular) according to findings of clinical interviews and specific tests; the diagnosis was always made on the basis of criteria described in the Diagnostic and Statistical Manual of Mental Disorders (4th edition, text revision; American Psychiatric Association, 2000). In particular, the clinical group included 15 women diagnosed with panic disorder and 15 women with generalized anxiety disorder. No additional assessment was done to confirm the specialists' diagnosis. On the basis of our selection criteria, specialists provided us with only pure cases: There were no cases of comorbidity with other disorders. In this group the response rate among women approached to participate was 100%. Table 1 presents data regarding similarity in anxiety levels and sexual functioning measures across panic disorder and generalized anxiety disorder subjects; therefore, these subjects were all considered as part of a single group.

Nonclinical Group

The nonclinical group comprised 100 White/Caucasian women (M age = 28.8 years, SD = 5.7 years).

We used a convenience sample of volunteer women recruited from the general population in various public gathering places in Florence, Italy, selected by asking them to take part in research aimed at gathering information on some psychophysical sensations and on certain aspects of their sex life. During recruitment, we tried to avoid using the word *anxiety* in order to

TABLE 1. Anxiety and Sexual Functioning Differences Between Panic Disorder and Generalized Anxiety Disorder Patients in the Clinical Group

	Panic disorder		Generalized anxiety disorder			
Variable	*M*	*SD*	*M*	*SD*	$t_{(14)}$	*p*
State anxiety	50.20	9.45	51.27	10.69	−0.290	.774
Trait anxiety	55.80	5.98	55.60	6.02	0.091	.928
Anxiety sensitivity	34.80	7.39	31.20	7.37	1.336	.192
Desire	3.56	1.03	3.40	0.90	0.455	.653
Arousal	4.62	0.87	4.52	0.88	0.313	.757
Lubrication	5.56	0.49	5.30	0.71	1.171	.252
Orgasm	4.21	0.85	3.79	0.84	1.380	.179
Satisfaction	4.69	0.99	4.16	1.08	1.415	.168
Pain	4.99	0.95	4.27	1.93	1.293	.207
Global sexual function	27.63	4.21	25.43	4.99	1.304	.203

avoid influencing the responses of participants. In this group the response rate was 90%. As shown in Table 2, our nonclinical group did not differ from the general population norms for state and trait anxiety and had lower levels of anxiety sensitivity in relation to general population norms.

Measures

Four self-report measures were used in this study: the State and Trait Anxiety Inventory—Version Y; the Anxiety Sensitivity Index; the Female Sexual Function Index; and the Sexual Inhibition-Sexual Excitation Scale.

State and Trait Anxiety Inventory—Version Y

State anxiety and trait anxiety were measured using the State and Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970), which is a self-report questionnaire consisting of 20 items measuring state anxiety (momentary, reactive), and 20 items measuring trait anxiety (stable, dispositional). Responses on each item on the inventory are reported on a 4-point Likert-type scale ranging from 1 (*not at all*) to 4 (*very much so*). This psychometric instrument is reliable (Cronbach's alpha = .93 for state anxiety, .90 for trait anxiety; the stability to the test–retest is high for trait anxiety) and valid. The discriminant validity was established through the low correlation between State and Trait Anxiety Inventory (especially trait anxiety scale) and the California Psychological Inventory (Gough, 1957), which measures different aspects of personality. The Italian adaptation of the instrument, published by Pedrabissi and Santinello (1996), yielded high test–retest stability for trait anxiety (.82), and Cronbach's alpha ranged from .91 to .95 for state anxiety and from .85 to .90 for state anxiety.

TABLE 2. Single-Sample *t* Test for Anxiety in the Clinical and Nonclinical Groups

	Clinical group				Nonclinical group				Standardization of nonclinical group		Clinical group vs. nonclinical group	
	M	*SD*	$t_{(29)}$	*p*	*M*	*SD*	$t_{(99)}$	*p*	*M*	*SD*	*t*	*p*
State anxiety	50.73	9.92	**6.13**	**.00**	40.69	10.92	0.98	.33	39.62	10.64	4.51	**.00**
Trait anxiety	55.70	5.89	**12.67**	**.00**	42.25	9.76	0.19	.85	42.06	9.67	13.36	**.00**
Anxiety sensitivity	33.00	7.48	**10.18**	**.00**	11.99	7.57	**−9.39**	**.00**	19.10	9.11	7.15	**.00**

Results shown in bold are significant at $p < .05$.

Anxiety Sensitivity Index

We measured anxiety sensitivity, defined as the fear of anxiety-induced sensations, with the Anxiety Sensitivity Index (Peterson & Reiss, 1993; Reiss, Peterson, Gursky, & McNally, 1986). The index is a self-report questionnaire consisting of 16 items: For each item, the subject is asked to indicate the level at which the anxiety sensation or physiological arousal (e.g., accelerated heartbeat) generates fear or worry. The fear of possible deleterious effects, determined by anxiety symptoms, is evaluated on each item using a 5-point Likert-type scale ranging from 0 (*very little*) to 5 (*very much*). Scores are calculated by summing ratings across the 16 items, with high scores on the Anxiety Sensitivity Index indicating high levels of anxiety sensitivity. For nonclinical samples (based on findings from about 12 studies conducted with more than 4,500 participants), the average Anxiety Sensitivity Index score is 19.1 ($SD = 9.11$; Peterson & Reiss, 1993). The index is reliable (Cronbach's alpha from .82 to .91; and test–retest stability from .71 to .75) and valid, especially for criterion and construct validity (Peterson & Heilbronner, 1987; Peterson & Reiss, 1993; Reiss et al., 1986; Taylor, Koch, McNally, & Crockett, 1992), and translated into several languages (this study uses the Italian version by Fava and Grandi [1991] with reliability and validity for this version reported by Bernini, Pennato, Berriocal Montiel, & Guazzelli, 2008).

Female Sexual Function Index

We measured female sexual functioning with the Female Sexual Function Index (Rosen et al., 2000). This is a brief, self-report questionnaire consisting of 19 items designed to evaluate female sexual response by deriving scores in six specific areas: desire (two items); subjective arousal (four items); lubrication (four items); orgasm (three items); satisfaction (three items); and pain related to sexual activity (three items). Each item asks about experiences in the past 4 weeks and gives a choice of responses with varying scores (from 0 to 5 or from 1 to 5). The scoring method provides for a specific score for each of the six areas and for an overall score. High scores correspond to high levels of sexual functioning; the lowest possible overall score is 2, and the highest possible overall score is 36. The Female Sexual Function Index can be administered to women of different ages, including those in menopause, and is used as a clinical instrument for evaluating the multidimensional nature of female sexual functioning (Basson et al., 2003; Rosen et al., 2000). It is a reliable (Cronbach's alpha more than .82; test–retest stability from .79 to .82) and valid (especially for construct and discriminant validity) instrument (Meston, 2003; Wiegel, Meston, & Rosen, 2005). The Italian translation was developed by Jannini, Lenzi, and Maggi (2007), who did not report separate psychometric characteristics for this adaptation.

Sexual Inhibition Scale/Sexual Excitation Scale

We evaluated the propensity toward sexual inhibition or sexual excitation in these women using the Sexual Inhibition Scale/Sexual Excitation Scale (Janssen, Vorst, Finn, & Bancroft, 2002a, 2002b). The theoretical model underlying the Sexual Inhibition Scale/Sexual Excitation Scale holds that, in humans, sexual response and related sexual behaviors depend on a twofold control mechanism present at the cerebral level and based on the balance created between the excitatory system and the inhibitory system (Bancroft, 1999; Bancroft & Janssen, 2000). Each item is scored using self-reports on a 4-point scale ranging from 1 (*strongly agree*) to 4 (*strongly disagree*). Some questions on this measure regard specific situations to which subjects respond on the basis of their own, real experiences; other questions require that subjects imagine being in particular situations and hypothesize about their reactions. A factorial analysis revealed three factors:

1. Sexual excitation (SES) consisting of 20 items and four subfactors: 9 items relate to social interactions with sexually attractive persons (F1SES), 4 items relate to excitation resulting from visual stimulations (F2SES), 4 items relate to proclivity to sexual excitation resulting from sexual thoughts or daydreaming (F3SES), and 3 items relate to excitation resulting from nonspecific sexual stimulations (F4SES).
2. Sexual inhibition caused by the threat of failure in sexual performance (SIS1) consisting of 14 items and three subfactors: 8 items relate to the loss of excitation (F1SIS1), 3 items relate to worries emerging during sexual activity with a partner (F2SIS1), and 3 items relate to fears and worries associated to performance and other external sources of distraction (F3SIS1).
3. Sexual inhibition caused by the threat of the consequences of sexual performance (SIS2) consisting of 11 items and three subfactors: 4 items relate to the risk of being caught during sexual activities (F1SIS2), 3 items relate to negative consequences of sex (F2SIS2), and 4 items relate to physical pain or norms and values (F3SIS2).

The Sexual Inhibition Scale/Sexual Excitation Scale has been shown to possess good psychometric properties for men (Janssen et al., 2002a, 2002b) and for women (Carpenter, Janssen, Graham, Vorst, & Wicherts, 2008): In women, the three factors showed significant levels of internal consistency and test–retest reliability (SES, $r = 0.70$; SIS1, $r = 0.68$; SIS2, $r = 0.41$). Moreover, this instrument showed high convergent and discriminant validity. A preliminary validation of this instrument was conducted in the Italian population (Panzeri et al., 2008) and also supported a hierarchical factorial structure consisting of one excitation and two inhibition factors. The Sexual Inhibition Scale/Sexual Excitation Scale has an inverted score: The higher the score is, the less present is the measured characteristic and vice versa.

Statistical Analysis

All data were transferred into an electronic database and processed using the Statistical Package for the Social Science (SPSS) 17.0 for Windows (SPSS, Inc., Chicago, IL). After having observed the data distribution (Kolmogorov & Smirnoff one-sample test), we tested the differences in anxiety levels comparing the clinical and nonclinical group as well as the clinical and nonclinical group versus standardization sample (two independent samples analysis). By this test, we also evaluated the discrepancy between the sexual functioning of the two groups. In the clinical and nonclinical groups, a Pearson's correlation matrix was calculated for anxiety measures (State and Trait Anxiety Inventory—Version Y and Anxiety Sensitivity Index), Sexual Inhibition Scale/Sexual Excitation Scale and Female Sexual Function Index scores. On the basis of the results of these correlations, we were able to assess the associations between the three types of anxiety and sexual response as well as between the three types of anxiety and tendency toward inhibition and excitation. We performed a simple linear regression to investigate the influence and predictive power of aspects of anxiety on sexual response and on inhibition and excitation propensity, in the clinical and nonclinical groups. Models were derived using the stepwise method controlling for tolerance, variance inflation factor, collinearity, and part and partial correlations.

We chose an alpha level of .05 with Holm's stepwise multiple comparisons correction (Sankoh, Huque, & Dubey, 1997) as the level of significance in all analyses (Tables 4 and 5 show the corrected p values). The Holm's method begins with the consideration that only the nonrejected hypothesis must be controlled and corrected for. The Holm's method is less conservative than the Bonferroni approach, and is well suited to our experimental design, because it does not use a clinical population or a repeated measures analysis about treatment efficacy, where a more restricted control would be more appropriate.

RESULTS

Differences Between Anxiety and Demographic Data of the Two Groups

As reported in Table 1, women with panic disorder and generalized anxiety disorder, who made up our clinical group, did not differ on anxiety nor on sexual functioning measures. Using the State and Trait Anxiety Inventory—Version Y (Spielberger, Gorsuch, & Lushene, 1970), the average level of state anxiety in women belonging to the clinical group was 50.73 ($SD = 9.92$) and the average level of trait anxiety was 55.7 ($SD = 5.89$). A series of t tests (Table 2) showed that state anxiety and trait anxiety levels

were significantly higher than those reported in the standardization nonclinical sample. Also, the average scores obtained on anxiety sensitivity in the clinical group were compared with those obtained in Peterson and Reiss's (1993) study. Women in the clinical group had anxiety levels significantly higher than those of women in the nonclinical population. The same statistical analyses were performed on the nonclinical group. These results (Table 2) indicated no statistically significant differences between the nonclinical group and the general population norms with regard to state anxiety and trait anxiety as measured by the State and Trait Anxiety Inventory—Version Y. For anxiety sensitivity a statistically significant difference was found between the nonclinical group and scores for the healthy population obtained in the study by Peterson and Reiss (1993); however, the difference was negative, indicating that the women in the nonclinical group had lower levels of anxiety sensitivity compared with the healthy population examined by Peterson and Reiss (1993).

Table 3 shows demographic information for women in the clinical and nonclinical groups. The table shows that both groups have similar demographic characteristics and few exceptions. In the month preceding the study, 86.6% (26/30) of the women belonging to the clinical group reported having had stable sexual partners and 13.4% (4/30) reported having had casual sexual partners. The average duration of sexual relationships among those women who reported stable partners was 5.5 years. The duration of the shortest relationship was 1 month and that of the longest was 18 years. In the nonclinical group, 94% (94/100) reported having had a stable partner during the month preceding the study, and 6% (6/100) reported having had a casual

TABLE 3. Demographic Data of the Clinical and Nonclinical Groups

	Clinical group	Nonclinical group
Marital status		
Married/cohabiting	40.00%	39.00%
Separated/divorced	13.33%	5.00%
Widowed	0.00%	0.00%
Single	46.67%	66.00%
Education		
Primary school	0.00%	1.00%
Secondary school	6.66%	7.00%
Professional certificate (3 years)	6.66%	3.00%
High school diploma (5 years)	53.28%	52.00%
Undergraduate degree	33.00%	34.00%
Master's or doctoral degree	0.00%	3.00%
Number of children		
0	69.93%	74.00%
1	16.65%	12.00%
2	9.99%	14.00%
3 or more	3.33%	0.00%

partner. The average duration of relationships reported as stable was 8 years, with the shortest of these being of 1 month, and the longest of 24 years.

Differences in Sexual Functioning Between the Two Groups

Results reported in Table 4 indicate significant differences between the two groups in the phase of arousal ($t = -2.56$, $p < .05$), orgasm ($t = -3.99$, $p < .05$), satisfaction ($t = -4.80$, $p < .05$) and in global sexual function ($t = -2.84$, $p < .05$), but not in desire, lubrication, and pain. Results reported in Table 5 indicate significant differences between the clinical group and the nonclinical group with respect to subfactors F1SIS1 ($t = -6.89$, $p < .05$), F3SIS1 ($t = -4.40$, $p < .05$), F2SIS2 ($t = -3.62$, $p < .05$) and factors SIS1 ($t = -6.40$, $p < .05$) and SIS2 ($t = -4.06$, $p < .05$). Women in the clinical group had a greater tendency to lose excitation and report that they were more worried and more likely to attend to external distractions. These women were also more worried about possible negative consequences from sexual activity itself. Furthermore, women in the clinical group had higher levels of sexual inhibition, either as a result of the threat of possible performance failure or as a result of the possible consequences from the activity itself. To interpret these tables, it is important to note that the Sexual Inhibition Scale/Sexual Excitation Scale has an inverted score: The higher the score is, the less present is the measured characteristic, and vice versa.

Relation Between Anxiety Level and Sexual Functioning

In the clinical group (see Table 6), there was a significant negative relation between state anxiety and arousal as well as between state anxiety and global sexual function. Furthermore, there was a significant relation, in different ratios, between trait anxiety and all phases of female sexual response except for the phase of desire as well as between trait anxiety and global sexual function: Higher levels of trait anxiety were associated with lower levels of

TABLE 4. Results of *t* Test for Independent Samples (for Female Sexual Function Index)

	Clinical group		Nonclinical group			
Variable	*M*	*SD*	*M*	*SD*	$t_{(128)}$	*p*
Desire	3.48	0.95	3.92	0.92	−2.28	.06
Arousal	4.57	0.86	5.02	0.84	**−2.56**	**.01**
Lubrication	5.43	0.61	5.24	0.97	1.26	.21
Orgasm	4.00	0.86	4.96	1.22	**−3.99**	**.00**
Satisfaction	4.43	1.05	5.16	1.14	**−4.80**	**.00**
Pain	4.63	1.54	5.07	1.44	−1.46	.15
Global sexual function	26.53	4.68	29.37	4.82	**−2.84**	**.00**

Results shown in bold are significant at $p < .05$ (Holm stepwise correction).

TABLE 5. Results of *t* Test for Independent Samples on the Subfactors and Factors of the Sexual Inhibition Scale/Sexual Excitation Scale

	Clinical group		Nonclinical group			
Variable	*M*	*SD*	*M*	*SD*	$t_{(128)}$	*p*
F1SES: Sexual excitation related to social interaction with sexually attractive person	2.90	0.39	3.01	0.57	−0.96	.34
F2SES: Sexual excitation resulting from visual stimulations	2.85	0.78	2.99	0.74	−0.86	.39
F3SES: Sexual excitation resulting from sexual thoughts or daydreaming	2.04	0.22	2.01	0.53	0.44	.66
F4SES: Sexual excitation resulting from not specifically sexual stimulations	3.17	0.54	3.29	0.60	−0.98	.33
F1SIS1: Sexual inhibition resulting from the loss of excitation	1.96	0.41	2.71	0.55	**−6.89**	**.00**
F2SIS1: Sexual inhibition resulting from the worries emerging during sexual activity with a partner	1.69	0.41	1.82	0.55	−1.38	.17
F3SIS1: Sexual inhibition resulting from the worries associated with performance or other external source of distraction	1.93	0.56	2.52	0.66	**−4.40**	**.00**
F1SIS2: Sexual inhibition related to the risk of being caught during sexual activities	1.68	0.57	2.00	0.73	−2.19	.06
F2SIS2: Sexual inhibition related to negative consequences of sex	1.49	0.59	2.01	0.71	**−3.62**	**.00**
F3SIS2: Sexual inhibition related to physical pain or norms and values	1.90	0.45	2.07	0.61	−1.63	.11
SES: Sexual excitation	2.74	0.33	2.82	0.49	−0.87	.38
SIS1: Sexual inhibition caused by the threat of failure in sexual performance	1.94	0.44	2.61	0.52	**−6.40**	**.00**
SIS2: Sexual inhibition caused by the threat of the consequences of sexual performance	1.69	0.34	2.03	0.54	**−4.06**	**.00**

Results shown in bold are significant at $p < .05$ (Holm stepwise correction).

arousal, lubrication, orgasm, satisfaction, and total sexual function and to higher levels of pain. (This area has a reversed score in contrast with the other ones.)

In the nonclinical group (see Table 7), there were statistically significant, albeit weak, negative relations between state anxiety and orgasm, state

TABLE 6. Pearson Correlations Between Anxiety Measures and Scores in Areas of the Female Sexual Function Index in the Clinical Group

Domain	Desire	Arousal	Lubrication	Orgasm	Satisfaction	Pain	Total
State anxiety	−.27	**−.37***	−.32	−.23	−.30	−.27	**−.37***
Trait anxiety	−.13	**−.38***	**−.52****	**−.41***	**−.44***	**−.38***	**−.48****
Anxiety sensitivity	−.01	−.25	−.36	−.11	−.15	−.30	−.27

*$p < .05$, two-tailed. **$p < .01$, two-tailed. Correlations shown in bold are significant.

TABLE 7. Pearson Correlations Between Anxiety Measures and Scores in Areas of Female Sexual Function Index in the Nonclinical Group

Domain	Desire	Arousal	Lubrication	Orgasm	Satisfaction	Pain	Total
State anxiety	−.04	−.18	−.16	**−.24***	**−.28****	−.19	**−.26****
Trait anxiety	−.02	−.14	−.03	**−.32****	**−.26***	−.10	**−.20***
Anxiety sensitivity	−.04	−.06	−.00	−.17	−.04	−.16	−.11

*$p < .05$, two-tailed. **$p < .01$, two-tailed. Correlations shown in bold are significant.

anxiety and satisfaction, state anxiety and global sexual function; and between trait anxiety and orgasm, trait anxiety and satisfaction; and trait anxiety and global sexual function.

Relation Between Anxiety Level and the Propensity Toward Inhibition and Excitation

In the clinical group (Table 8), trait anxiety correlated negatively, at a moderate level, with the subfactors F1SIS1, F3SIS1, and F1SIS2. It therefore appears that an increase in trait anxiety corresponds to a decrease in the scores on those domains, indicating (in consideration of Sexual Inhibition Scale/Sexual Excitation Scale inverted scores) a greater tendency to lose excitation, a greater propensity toward worry, a greater tendency toward nonsexual cognitive distractions, and a greater tendency to worry about being discovered while engaging in sexual activity. Anxiety sensitivity was negatively correlated with the subfactor F3SIS1. Furthermore, there was a statistically significant negative correlation between trait anxiety and SIS1 and between trait anxiety and SIS2, as reported in Table 9.

In the nonclinical group (Table 8) there was a significant correlation between state anxiety and F1SIS1, indicating a greater tendency to lose excitation with increasing levels of state anxiety as well as between trait anxiety and the subfactors F1SIS1 and F3SIS1. Consequently, an increase in trait anxiety was also correlated with a greater tendency to lose excitation along with greater levels of worry and attention turned toward nonsexual cognitive distractions. In the nonclinical group there were also negative correlations, at moderate levels, between anxiety sensitivity and the subfactors F1SIS1, F2SIS1, and F3SIS1. From these analyses, as reported in Table 9, results indicated that state anxiety, trait anxiety and anxiety sensitivity correlated negatively and significantly only with SIS1.

Influence of Anxiety on Sexual Functioning and on the Propensity Toward Inhibition and Excitation

We derived six multiple linear regression models for the clinical and nonclinical group, considering the full-scale Female Sexual Function Index and the

TABLE 8. Pearson Correlations Between Anxiety Measures and Scores on Subfactors of the Sexual Inhibition Scale/Sexual Excitation Scale in the Clinical and Nonclinical Groups

	State anxiety		Trait anxiety		Anxiety sensitivity	
Domain	Clinical group	Nonclinical group	Clinical group	Nonclinical group	Clinical group	Nonclinical group
F1SES: Sexual excitation related to social interaction with sexually attractive person	−.34	−.09	.23	−.13	−.07	−.12
F2SES: Sexual excitation resulting from visual stimulations	.03	.05	−.09	−.01	−.07	.06
F3SES: Sexual excitation resulting from sexual thoughts or daydreaming	−.09	.00	−.10	−.03	.04	−.07
F4SES: Sexual excitation resulting from not specifically sexual stimulations	−.21	.08	.09	.04	.18	.08
F1SIS1: Sexual inhibition resulting from the loss of excitation	−.12	**−.28****	**−.37***	**−.34****	−.11	**−.46****
F2SIS1: Sexual inhibition resulting from the worries emerging during sexual activity with a partner	.04	.04	−.08	−.16	.08	**−.28****
F3SIS1: Sexual inhibition resulting from the worries associated with performance or other external source of distraction	−.11	−.15	**−.42***	**−.30****	**−.48****	**−.34****
F1SIS2: Sexual inhibition related to the risk of being caught during sexual activities	.20	−.15	**−.51****	−.07	−.12	−.05
F2SIS2: Sexual inhibition related to negative consequences of sex	−.05	−.07	−.09	−.08	−.23	.06
F3SIS2: Sexual inhibition related to physical pain or norms and values	.08	−.06	−.18	−.08	−.29	−.01

*$p < .05$, two-tailed. **$p < .01$, two-tailed. Correlations shown in bold are significant.

TABLE 9. Pearson Correlations Between Anxiety Measures and Scores on Three Factors of the Sexual Inhibition Scale/Sexual Excitation Scale in the Clinical and Nonclinical Groups

	State anxiety		Trait anxiety		Anxiety sensitivity	
Domain	Clinical group	Nonclinical group	Clinical group	Nonclinical group	Clinical group	Nonclinical group
SES: Sexual excitement	−.18	.02	.04	−.04	.02	−.01
SIS1: Sexual inhibition caused by the threat of failure in sexual performance	−.13	**−.25***	**−.45***	**−.37****	−.36	**−.46****
SIS2: Sexual inhibition caused by the threat of the consequences of sexual performance	.12	−.12	**−.42***	−.10	−.33	.00

*$p < .05$, two-tailed. **$p < .01$, two-tailed. Correlations shown in bold are significant.

orgasm, lubrication, arousal, pain, satisfaction, and desire scores as dependent variables. Trait anxiety, the Anxiety Sensitivity Index, F1SIS1, F2SIS1, F3SIS1, F1SIS2, F2SIS2, and F3SIS2 were inserted as predictors. The rationale for these choices was based on the correlation matrix among all these factors (dependent and predictor variables) that highlighted several positive and negative correlations, in the clinical and nonclinical groups. Using a stepwise regression model, for the first step, we inserted all the scales that had shown a statistically significant coefficient with at least one other variable. The results of each single model are shown in Table 10 (clinical group) and Table 11 (nonclinical group).

TABLE 10. Linear Regression Models Examining the Influence of Trait Anxiety and the Sexual Inhibition Scale/Sexual Excitation Scale in the Clinical Group

Dependent variable	Independent variables	Standardized coefficient beta	R^2	t	p
Female Sexual Function Index	STAI-T	−.501	.422	−3.410	.002
	F2SIS1	−.457		−3.115	.004
Orgasm	STAI-T	−.411	.169	−2.384	.024
Satisfaction	STAI-T	−.466	.313	−2.914	.007
	F2SIS1	−.350		−2.185	.038
Lubrication	STAI-T	−.556	.490	−4.030	.000
	F2SIS1	−.475		−3.444	.002
Pain	F3SIS1	.431	.186	2.525	.017
Arousal	F2SIS1	−.508	.401	−3.398	.002
	STAI-T	−.422		−2.827	.009
Desire	F2SIS1	−.524	.274	−3.254	.003

Note. F2SIS1 = sexual inhibition resulting from the worries emerging during sexual activity with a partner; F3SIS1 = sexual inhibition resulting from the worries associated with performance or other external source of distraction; STAI-T = trait anxiety.

TABLE 11. Linear Regression Models Examining the Influence of Trait Anxiety and the Sexual Inhibition Scale/Sexual Excitation Scale in the Nonclinical Group

Dependent variable	Independent variables	Standardized coefficient beta	R^2	t	p
Female Sexual Function Index	F3SIS1	.340	.116	3.578	.001
Orgasm	STAI-T	−.261	.138	−2.647	.009
	F3SIS1	.197		1.993	.049
Satisfaction	F3SIS1	.295	.087	3.059	.003
Pain	F1SIS1	.321	.103	3.357	.001
Arousal	F3SIS2	.246	.061	2.513	.014
Lubrication	STAI-T	−.556	.490	−4.030	.000
	F2SIS1	−.475		−3.444	.002
Desire	F3SIS2	.274	.075	2.822	.006

Note. F1SIS1 = sexual inhibition resulting from the loss of excitation; F2SIS1 = sexual inhibition resulting from the worries emerging during sexual activity with a partner; F3SIS1 = sexual inhibition resulting from the worries associated with performance or other external source of distraction; F3SIS2 = sexual inhibition related to physical pain or norms and values; STAI-T = trait anxiety.

With the exception of pain (which was more related to "worries associated to performance itself or other sources of distraction" [F3SIS1] in the clinical group), trait anxiety and "worries emerging during sexual activity with a partner" (F2SIS1) proved to be the most important predictors of global and specific sexual functions. Considering the R^2 values, the models based on trait anxiety and "worries emerging during sexual activity with a partner" (F2SIS1) provided the most reliable explanation for the anxiety and sexual functioning relation (Female Sexual Function Index total score $R^2 = .422$; arousal $R^2 = .401$; lubrication $R^2 = .490$). With respect to the effect of trait anxiety and F2SIS1 on the Female Sexual Function Index and arousal, there was a stronger link between these two predictors and lubrication, with an amount of explained variance that reached nearly 50%.

Table 11 indicates a reduction of trait anxiety influence on sexual functioning and the appearance of the sexual inhibition subfactor related to physical pain or norms and values (F3SIS2).

DISCUSSION

Results confirm the first hypothesis: women with anxiety disorders, specifically with panic disorder or generalized anxiety disorder (who were considered as a single group because they did not differ in anxiety nor sexual functioning measures) appeared to have worse sexual functioning than nonclinical women, at a global level. These differences were particularly evident with respect to arousal, orgasm, and sexual satisfaction; however, there were no significant differences in desire, lubrication, and pain. It appeared, therefore, that women with high levels of anxiety were capable of experiencing

physiological sexual excitation similar to that experienced by women with normal levels of anxiety, because the major differences were in subjective sexual excitation. This result confirms the necessity of distinguishing between two types of sexual excitation (physiological and subjective), as suggested by previous literature reviewed in the introduction. The fact that high levels of anxiety resulted in difficulties pertaining to subjective sexual arousal, but not in lubrication, might be explained by the importance of the cognitive dimension and its interaction with the physiological mechanisms of sexual response in women with anxiety disorders. The results of the present study (a) are congruent with Bradford and Meston's (2006) research, in which trait anxiety had a differential effect on physiological and subjective sexual arousal; and (b) confirm findings by van Minnen and Kampman (2000) and Figueira et al. (2001) with regard to worse sexual functioning in women with anxiety disorders.

In addition, results of the present study indicate that women with a diagnosis of an anxiety disorder showed greater sexual inhibition compared to women with normal levels of anxiety, confirming the second study hypothesis. Inhibition was caused by the threat of performance failure (SIS1) or the threat of the consequences of sexual performance itself (SIS2). Such women had, in particular, a greater tendency to lose excitation (F1SIS1), and were more worried by and likely to turn their attention toward nonsexual cognitive distractions (F3SIS1). Furthermore, they were particularly worried about possible negative consequences of sexual activity itself (F2SIS2). Van den Hout and Barlow (2000) previously noted that greater levels of anxiety result in cognitive interference followed by less attention to sexually arousing stimuli and an increase in erroneous interpretations of such stimuli. Our own findings indicate that in women with high scores on anxiety measures, an increase in state anxiety reduced sexual functioning and arousal in particular; whereas an increase in trait anxiety diminished sexual functioning—especially arousal, lubrication, orgasm, satisfaction, and raised pain. In women without an anxiety disorder, an increase in state or trait anxiety only diminished sexual functioning with respect to orgasm and satisfaction. State anxiety and trait anxiety correlated negatively with global sexual functioning in both groups of women, although such relations were more pronounced in the clinical group. These results demonstrated the importance of considering the level and the nature of anxiety and also the personal history of each individual, as suggested by van Minnen and Kampman's study (2000). Only in women with panic disorder or generalized anxiety disorder was there a negative correlation between state and trait anxiety and subjective arousal. A possible explanation is offered by van Minnen and Kampman (2000), who suggested that anxious subjects perceive physiological sexual excitation as anxious arousal and tend to focus their attention on threatening information, thus producing greater levels of anxiety and less subjective reactions to sexual excitation; healthy subjects might confuse their anxiety

with sexual excitation itself, thus increasing the level of excitation so that their anxiety does not negatively influence their sexual arousal.

Our results can be compared with Bradford and Meston's study (2006), in which higher levels of state and trait anxiety were associated with diminished global sexual functioning and capacity for experiencing sexual satisfaction. Our results confirmed Bradford and Meston's (2006) findings with respect to women with nonpathological levels of anxiety. Furthermore, this study showed that in women with normal levels of anxiety an increase in trait anxiety is associated with diminished capacity to reach orgasm. The finding that anxiety sensitivity was not correlated with any phase of sexual response nor with global sexual functioning corroborates the findings of Bradford and Meston (2006), although in the latter study, there was a slight correlation between anxiety sensitivity and difficulties in subjective sexual arousal. Bradford and Meston (2006) did not offer an explanation as to the lack of association between anxiety sensitivity and sexual functioning; in our opinion, this might be because the Anxiety Sensitivity Index only measures, in a general and undifferentiated sense, the extent to which physiological responses to anxiety are interpreted as threatening. It could be possible that this instrument is not sufficiently sensitive. More useful information was obtained from the correlations between the Anxiety Sensitivity Index and the Sexual Inhibition-Sexual Excitation Scale, especially with respect to the sexual inhibition scale, with clearer identification of the concerns that resulted in anxiety. Our results indicate that in the clinical group greater trait anxiety also increased "sexual inhibition due to the loss of excitation" (F1SIS1), "sexual inhibition due to the worries associated to performance or other external source of distraction" (F3SIS1), and "sexual inhibition related to the risk of being caught during sexual activities" (F1SIS2): there was a consequent increase in "sexual inhibition caused by the threat of failure in sexual performance" (SIS1) and in "sexual inhibition caused by the threat of the consequences of sexual performance" (SIS2). Furthermore, in the clinical group, an increase in anxiety sensitivity also corresponded to an increase in the tendency to develop "sexual inhibition due to the worries associated to performance or other external source of distraction" (F3SIS1).

These findings confirm previous theories and demonstrate the importance of cognitive mechanisms related to filtering of information, the role of attention, negative expectations, and excessive control in regulating the propensity toward sexual excitation. Our results confirmed only a part of the third experimental hypothesis. Through a multivariate analysis, findings indicated that in women with pathological levels of anxiety (women with panic disorder or generalized anxiety disorder in particular), dispositional anxiety (trait anxiety) is a good predictor of various types of sexual dysfunctions, alone and in combination with other sexual inhibition factors such as "worries emerging during sexual activity with a partner" (F2SIS1) and "worries associated to performance or other external source of distraction" (F3SIS1). In the nonclinical group, these relationship results differed,

showing a reduction of trait anxiety effect on sexual functioning and a prevalence of inhibition factors including "physical pain or norms and values" (F3SIS2) and "worries associated to performance or other external sources of distraction" (F3SIS1). Results did not entirely confirm the hypothesis in that anxiety sensitivity does not appear to influence sexual functioning in either of the two groups. However, in the clinical group anxiety sensitivity was nevertheless a good predictor of the tendency to develop concerns and to turn attention toward nonsexual cognitive distractions (F3SIS1), but this cannot be considered to be the only predictor. In the nonclinical group, trait anxiety was linked to sexual dysfunction only in the orgasm phase. Trait anxiety and anxiety sensitivity are linked to the tendency toward sexual inhibition as a result of "the loss of excitation" (F1SIS1) and of "worries associated to performance or other external source of distraction" (F3SIS1) and also of the propensity toward "sexual inhibition caused by the threat of failure in sexual performance" (SIS1), but in none of these cases were these factors the only predictors. Greater levels of anxiety increased the likelihood of developing sexual dysfunction. Results therefore confirmed what has been noted previously in the literature: the complexity of the female sexual response, and in particular its wide variability, is influenced by a wide range of physiological, psychological, contextual, and social factors (Boncinelli et al., 2008; Graziottin et al., 2009; Wylie & Mimoun, 2009).

At a physiological level, it has frequently been observed that anxiety facilitates rather than inhibits sexual functioning: this has been explained as resulting from a process that involves the activation of the sympathetic nervous system as described by Meston and Gorzalka (1995), Palace and Gorzalka (1990), and Palace (1995). However, this study demonstrated a difference between the effect of anxiety on women with pathological levels of anxiety compared with women with normal levels of anxiety: Whereas in the former group, anxiety appears to have a negative influence on the excitation and orgasm phases, in the latter group it appeared to only alter the orgasm phase. This might be explained, as indicated earlier, by the greater importance of cognitive factors in subjective reactions to sexual excitation, whereas physiological factors might have greater importance for orgasm.

Some comments are warranted concerning our findings that the two clinical groups did not differ regarding sexual desire, nor did our data show any correlation between anxiety and desire. The Female Sexual Function Index measures desire using only two items that evaluate female desire without noting that the lack of initial/spontaneous desire is fundamentally normal in women; hence, forcing the respondent to use a male-centered and not a female-centered model of sexual desire (Basson et al., 2003) probably alters the evaluation itself. Therefore, in the Female Sexual Function Index, the area pertaining to desire is the weakest one. We note, however, that the p value of the difference in desire between the clinical and nonclinical groups was close to significance ($p = .06$), with the clinical group scoring lower for sexual desire than the nonclinical group.

Results of the present study affirm the importance of considering the relation between anxiety and sexual functioning and between anxiety and sexual arousal, particularly as it relates to selecting or developing optimal therapeutic interventions for women with anxiety problems and such interventions' ability to prevent or treat problems in the sexual sphere. This study contributes to the area of debate regarding the possible effect of anxiety on female sexual responses, but it does not establish with absolute certainty the presence of a totally inhibitory effect, as already expressed by Hoon et al. (1977).

Last, we acknowledge limitations of the present study. A small number of subjects made up the two groups, and the size between the nonclinical and clinical groups differed (the sample size of the nonclinical group was more than three times that of the clinical one). Furthermore, the participants of the nonclinical group were not drawn randomly from the population but were volunteers selected on the basis of their willingness to take part in the study, whereas participation in the clinical group was through the specialists we contacted. Consequently, the extent to which the results can be generalized is limited. Also, to more fully understand the relation between anxiety and female sexual excitation, it would be useful to have objective investigations into physiological sexual excitation (vaginal pulse amplitude); however, such procedures were not possible in this study given that the women with an anxiety disorder were drawn from community mental health clinics where such physiological measures cannot be obtained. It is also unlikely that participants in the nonclinical group, having volunteered to take the questionnaires, would have accepted such invasive procedures. It would have been interesting to measure state anxiety immediately before sexual activity, as this would be the optimal way to ascertain the direct relation between state anxiety and sexual functioning. In those cases, the relation between anxiety and female sexual excitation would certainly have been more clearly demonstrated, and it might have been possible to evaluate more closely the relation between state anxiety and vaginal pulse amplitude and to establish whether this relation is curvilinear, as suggested by Bradford and Meston (2006).

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