SHORTER COMMUNICATIONS

The effects of immediate, delayed, and residual sympathetic activation on sexual arousal in women

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Summary—In a recent experiment, Meston and Gorzalka (1995) [*Behaviour, Research and Therapy, 33,* 651–664] demonstrated a facilitatory effect of sympathetic activation, via acute exercise, on female sexual arousal. The present investigation was designed to examine the time course of this effect. Thirty-six sexually functional women participated in two experimental sessions in which they viewed a neutral film followed by an erotic film. In one of these sessions, *S*s were exposed to 20 min of intense exercise (stationary cycling) prior to viewing the films. Subjective (self-report) and physiological (photoplethysmograph) sexual arousal were measured at either 5 min, 15 min, or 30 min post-exercise. Acute exercise marginally decreased vaginal pulse amplitude (VPA) and had no effect on vaginal blood volume (VBV) responses to an erotic film when measured 5 min post-exercise. At 15 min post-exercise, exercise significantly increased VPA and marginally increased VBV responses. At 30 min post-exercise, both VPA and VBV responses to an erotic film were marginally increased. Acute exercise had no significant effect on subjective perceptions of sexual arousal in any of the experimental conditions.

INTRODUCTION

It is well known that the sympathetic and parasympathetic branches of the autonomic nervous system do not function independently, and that nerve fibers from both systems innervate many bodily systems involved in the sexual response. It is generally assumed, however, that one or the other of these systems becomes dominant during increased sexual arousal and serves to perpetuate further arousal and/or orgasm. Which of these two systems better facilitates sexual arousal has been open to conjecture.

For over 30 yr, clinicians, researchers, and theorists in the field of human sexuality have worked largely under the assumption that the sympathetic nervous system (SNS) plays an inhibitory role, and the parasympathetic nervous system (PNS) plays a facilitatory role in initiating and maintaining the early stages of sexual arousal. In men, this assumption is based on neurophysiological studies which reveal PNS mediation of the erectile response, and on an abundance of clinical reports which link generalized anxiety (i.e. SNS activation) and erectile failure. In women, however, this assumption is based primarily on analogies that have been drawn between the erectile response in men and the vasocongestive response in women. Direct, empirical evidence for a PNS-mediated sexual response in women has not yet been offered. Determination of the precise role of the SNS in sexual arousal not only has important implications for deriving an etiological theory of sexual dysfunction, but for developing effective treatments for the alleviation of sexual difficulties.

The present study follows from a recent investigation (Meston & Gorzalka, 1995) which found that intense, acute exercise significantly increased physiological sexual arousal in women. Because exercise at the intensity and duration used in that investigation has been shown to elicit significant SNS activity (e.g. Mazzeo & Marshall, 1989; Robinson, Epstein, Beiser & Braunwald, 1966), the results of Meston and Gorzalka (1995) challenged the long-held notion that activation of the SNS inhibits sexual arousal in women. Moreover, the findings suggested that SNS activation may play a facilitatory role in enhancing the initial stages of sexual arousal in women. The present study aims at further elucidating the role of the SNS in female sexual arousal by examining the temporal boundaries by which exercise facilitates physiological sexual arousal in women. In the Meston and Gorzalka (1995) study, the effects of exercise on sexual arousal were measured at only one time point (i.e. 15 min) post-exercise. Evidence indicates that, at 15 min post-exercise, SNS influences remain significantly elevated, but have declined considerably from intensities during and immediately following exercise (Savin, Davidson & Haskell, 1982). This raises the question of whether exercise would have an even greater facilitatory effect on sexual arousal if measured when SNS activation was at a peak, and whether the sexually facilitatory effects of exercise are in some way directly related to the *level* of SNS activation. To investigate this possibility, subjective and physiological sexual arousal were measured in response to erotic films at either 5 min, 15 min, or 30 min following 20 min of intense (i.e. *70% HR$_{max}$) exercise. These time intervals allowed for examination of the effects of high, moderate, and low levels of SNS activation on sexual arousal in women. The exercise intensity and time period were chosen based on findings which indicate that SNS activity becomes prominent during moderate to heavy exercise (e.g. Mazzeo & Marshall, 1989; Robinson *et al.*, 1966), and remains significantly elevated at 30 min post-exercise (e.g. Kraemer, Patton, Knuttgen, Hannan, Kettler, Gordon, Dziados, Fry, Frykman & Harman, 1991; Strobel, Hack, Kinscherf & Weicker, 1993).

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*HR$_{max}$ = 220 − age in years (Golding, Meyers & Sinning, 1982).

METHOD

Subjects

Thirty-six sexually functional women (*M* age = 25.6 yr, range = 18–45) participated in one of three experimental conditions: Immediate (*n* = 12), Delayed (*n* = 12), and Residual (*n* = 12) sympathetic activation. Mean ages of *S*s in the Immediate, Delayed, and Residual conditions were 23.8, 29.4, and 22.6, respectively. Because of reported ethnic differences in sexual activity (e.g. Meston, Trapnell & Gorzalka, 1996), subject background information was recorded. Racial background of the *S*s was: Caucasian (35), and Southeast Asian (1). All *S*s were currently involved in sexual relationships; 2 of the *S*s were married. Initial telephone screening criteria were: between the ages of 18–45 yr, no use of medications known to affect vascular or sexual functioning, no history of treatment for sexual dysfunction, no medical condition that might put the *S* at risk when exercising, and current involvement in a heterosexual relationship.

To screen for absence of sexual dysfunction, profile descriptions of all *S*s were obtained via the Derogatis Sexual Functioning Inventory (DSFI; Derogatis, 1978), and the Orgasmic Functioning Questionnaire (OFQ; Meston, Jung, Hanson & Gorzalka, 1993). All *S*s employed in the study scored greater than or equal to the 40th percentile on the Sexual Functioning Inventory (*M* = 46.6, range = 40–64), the Global Sexual Satisfaction Index (*M* = 54.4, range = 40–70), and the Drive (*M* = 59.6, range = 40–71) subscale of the DSFI. In addition, the Brief Symptom Inventory (BSI; Derogatis, 1975) subtest of the DSFI was used to screen for absence of general psychopathology. All *S*s employed in the study scored greater than or equal to the 30th percentile (i.e. within two SDs of the normative mean) on the BSI (*M* = 42.0, range = 30–61). Data from the Experience subtest of the DSFI was used to ensure that all *S*s were within the normative range of sexual experience. All *S*s scored above the 30th percentile on the Experience subtest (*M* = 51.4, range = 35–64). Data from the OFQ was used to screen for absence of orgasmic dysfunction. All *S*s employed in the study were able to achieve orgasm by some means (e.g. intercourse, oral sex, masturbation) on at least 50% of the attempted trials (*M* = 95%).

Design and procedure

The procedure consisted of three sessions: a 1-h orientation screening and questionnaire session; a 45-min No-exercise experimental session; and a 1-h Exercise experimental session. Order of the two experimental sessions was counterbalanced across *S*s within each experimental condition. During each experimental session *S*s viewed one of two 7-min videotaped sequences which consisted of a 1-min display of the word "relax" followed by a 3-min neutral travelogue film and then a 3-min erotic film. The films were identical to those used by Meston and Gorzalka (1995). The two experimental sessions were scheduled at approximately 3 day intervals and excluded times during which the *S*s were menstruating. Phase of the menstrual cycle was not controlled given that sexual arousability to erotic stimuli is only minimally, if at all, influenced by the menstrual cycle (e.g. Hoon, Bruce & Kinchloe, 1982). All *S*s were asked to abstain from psychoactive drugs (including caffeine and alcohol) and to refrain from engaging in any strenuous physical activity for 24 h prior to each experimental session.

During the No-exercise session, *S*s entered a private, internally locked room. They were told that, when the experimenter notified them, via an intercom system, they were to sit in the chair and insert the photoplethysmograph so as to allow approximately a 2.5 cm distance between the end of the probe and the vaginal opening. They were also asked to remain as still as possible throughout the session in order to minimize potential movement artifacts. Subjects in the Immediate condition were notified to insert the plethysmograph after 1 min had passed, *S*s in the Delayed condition were notified after 11 min had passed, and *S*s in the Residual condition were notified to insert the plethysmograph after 26 min had passed. This procedure was used to ensure that the plethysmograph adaptation period was equivalent for all experimental conditions. As soon as *S*s had inserted the plethysmograph, the film began. Immediately following the erotic film, *S*s were asked to fill out the subjective rating scale.

During the Exercise session, prior to viewing the erotic films, *S*s were asked to cycle for 20 min on a Get Fit 200-II stationary bicycle. The *S*s were given continual heart rate feedback, and asked to cycle faster or slower in order to maintain an exertion level of approximately 70% HR_{max}. By ensuring that all *S*s worked at equivalent levels of their HR_{max}, differences in physiological responses resulting from variations in fitness levels are minimized (Grossman & Moretti, 1986). Fitness levels were not assessed, given that Meston and Gorzalka (1995) reported no correlation between fitness levels and physiological measures of sexual arousal when *S*s exercised at equivalent levels of their maximum heart rate. The total time from the cessation of exercise to the onset of the erotic stimulus was approximately 5 min for the Immediate condition (1-min rest period, 1-min display of the word "relax", 3-min neutral film), 15 min for the Delayed condition (11-min rest period, 1-min display of the word "relax", 3-min neutral film), and 30 min for the Residual condition (26-min rest period, 1-min display of the word "relax", 3-min neutral film). Immediately following the erotic film, *S*s were asked to fill out the subjective rating scale. With the exception of 20 min of cycling, all experimental procedures were identical to those of the No-exercise session. Upon completion of the experimental sessions, *S*s were thoroughly debriefed, informed about the additional purposes and goals of the study, and given an opportunity to view the records of their vaginal responses. All *S*s were paid $15.00 for their participation.

Data sampling and reduction

Physiological measurements. Physiological measures were obtained using a vaginal photoplethysmograph (Sintchak & Geer, 1975). Changes in VBV, VPA, and heart rate were monitored simultaneously during all experimental sessions. Vaginal pulse amplitude, the ac signal, reflects short-term changes in engorgement (Rosen & Beck, 1988). Vaginal blood volume, the dc signal, reflects slow changes in the pooling of blood in the vaginal tissue (Hatch, 1979). Vaginal pulse amplitude was recorded throughout the entire 180 s of neutral film and 180 s of erotic film. The data were hand scored from the polygraph recordings by a research assistant who was kept blind to the experimental manipulations. For each experimental condition, an average peak to peak amplitude was computed for both the neutral and erotic films by summing the amplitudes of each peak during the middle 20 s of the neutral or erotic film stimulus and dividing by the number of peaks per interval. Difference scores were computed for each experimental condition by subtracting the average VPA score during the neutral film from the average VPA score during the erotic film. Vaginal blood volume was sampled during the last 80 s of neutral film, and during the entire 180 s of erotic stimuli. Because there is no absolute method of calibrating VBV and, hence, no zero point, the data were scored as 0.0001 mV units of blood volume deviation from a baseline reference level defined as the mean of the last 80 s of the neutral stimulus. Heart rate was scored from the VPA polygraph records by counting the number of beats across the entire 180 s of neutral and 180 s of erotic film. The scores were averaged across

time to yield 2 measures (bpm) for each S per experimental session (one measure during each of the neutral and erotic films).

Subjective measurements. A self-report rating scale, adapted from Heiman and Rowland (1983), was used to assess subjective measures of sexual arousal (5 items), positive affect (11 items) and negative affect (11 items). Subjects rated each of these items, depending on the degree to which they experienced the sensations, on a 7-point Likert Scale, from *not at all* (1) to *intensely* (7). Subjective sexual arousal was defined by the following 5 items on the scale: Sexually aroused, warmth in genitals, genital wetness or lubrication, genital pulsing or throbbing, and any genital feelings.

For a detailed summary of the data reduction/sampling procedures, see Meston and Gorzalka (1995).

RESULTS

Analyses of physiological sexual arousal

Vaginal pulse amplitude. A Session (Exercise vs No-exercise) × Condition (Immediate vs Delayed vs Residual) analysis of variance was conducted on VPA difference scores. Mean VPA difference scores for the No-exercise and Exercise sessions are presented in Fig. 1. Results revealed a significant main effect of exercise on VPA difference scores, $F(1, 33) = 5.85$, $P = 0.021$, and a marginally significant main effect of condition on VPA difference scores, $F(2, 33) = 3.10$, $P = 0.058$. There was a significant interaction between session and condition, $F(2, 33) = 6.63$, $P = 0.004$. A follow-up one-way analysis of variance between VPA difference scores during the No-exercise sessions revealed no significant difference in VPA difference scores between the Immediate, Delayed, or Residual conditions $(F < 1)$. A follow-up one-way analysis of variance between VPA difference scores during the Exercise sessions revealed significant differences in VPA difference scores across conditions, $F(2, 33) = 7.93$, $P = 0.002$. Newman Keuls tests with a significance level set at $P < 0.05$ indicated a significant difference in VPA difference scores between the Immediate and Delayed conditions, and between the Immediate and Residual conditions during the Exercise session. Planned follow-up t-tests were conducted between the No-exercise and Exercise conditions in each of the Immediate, Delayed, and Residual conditions. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $P < 0.02$ $(P < 0.05/3)$ should be considered statistically reliable. Results revealed a marginally significant decrease in VPA scores with exposure to exercise in the Immediate condition, $t(11) = -2.57$, $P = 0.026$, a significant increase with exposure to exercise in the Delayed condition, $t(11) = 3.65$, $P = 0.004$, and a marginally significant increase with exposure to exercise in the Residual condition, $t(11) = 2.31$, $P = 0.041$.

In order to verify that the erotic films facilitated VPA responses, one-tailed, paired samples t-tests were conducted on VPA raw scores between neutral and erotic films within each experimental condition and session. Results revealed a significant increase in pulse amplitude responses to an erotic film in the No-exercise Immediate condition, $t(11) = 6.82$, $P < 0.001$; Exercise Immediate condition, $t(11) = 5.83$, $P < 0.001$; No-exercise Delayed condition, $t(11) = 9.99$, $P < 0.001$; Exercise Delayed condition, $t(11) = 8.33$, $P < 0.001$; No-exercise Residual condition, $t(11) = 4.46$, $P < 0.001$; and Exercise Residual condition, $t(11) = 7.08$, $P < 0.001$.

Vaginal blood volume. Deviation scores in blood volume were compared using a Session (Exercise vs No exercise) × Condition (Immediate vs Delayed vs Residual) analysis of variance. Mean VBV deviation scores during the Exercise
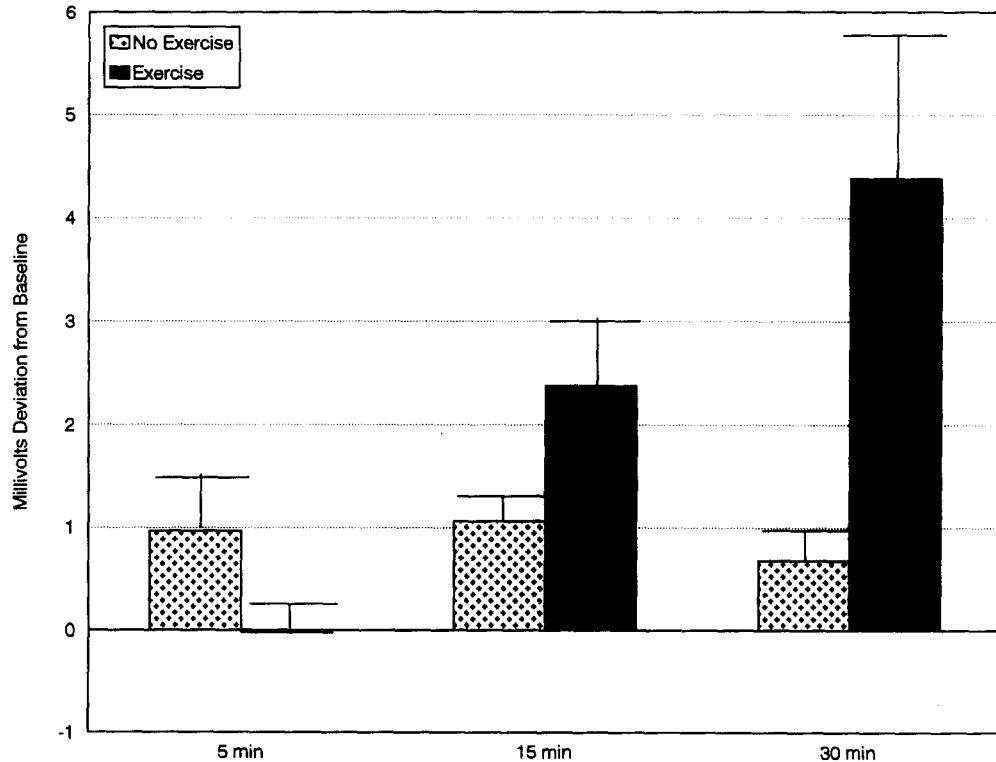

Fig. 1. Mean vaginal pulse amplitude (mm of pen deflection) ±SEM between neutral and erotic stimulus presentations during the No-exercise and Exercise conditions measured at 5 min, 15 min, and 30 min post-exercise.

and No-exercise sessions are presented in Fig. 2. Results revealed a significant main effect of exercise on VBV deviation scores, $F(1, 33) = 5.50$, $P = 0.025$, a significant main effect of condition on VBV deviation scores, $F(2, 33) = 4.45$, $P = 0.020$, and a significant interaction between session and condition, $F(2, 33) = 5.57$, $P = 0.008$. A follow-up one-way analysis of variance between VBV deviation scores during the No-exercise sessions revealed no significant difference in VBV deviation scores between the Immediate, Delayed, or Residual conditions ($F < 1$). A follow-up one-way analysis of variance between VBV deviation scores during the Exercise sessions revealed significant differences in VBV deviation scores across conditions, $F(2, 33) = 5.60$, $P = 0.008$. Newman Keuls tests with a significance level set at $P < 0.05$ indicated a significant difference in VBV deviation scores between the Immediate and Residual conditions during the Exercise session. Planned follow-up t-tests were conducted between the No-exercise and Exercise conditions in each of the Immediate, Delayed, and Residual conditions. Due to accumulating Type I error on mean comparisons across the variables, only mean differences of $P < 0.02$ ($P < 0.05/3$) should be considered statistically reliable. Exercise had no significant effect on VBV deviation scores in the Immediate, $t(11) = 1.47$, $P = 0.169$, or Delayed condition, $t(11) = -2.07$, $P = 0.063$, and produced a marginally significant increase in the Residual condition, $t(11) = -2.55$, $P = 0.027$.

In order to verify that the erotic films facilitated VBV responses, one-tailed, paired samples t-tests were conducted on VBV raw scores between neutral and erotic films within each experimental condition and session. Results revealed a significant increase in blood volume responses with exposure to an erotic film in the No-exercise Immediate condition, $t(11) = -2.18$, $P < 0.05$; No-exercise Delayed condition $t(11) = -1.95$, $P < 0.05$; Exercise Delayed condition, $t(11) = -2.66$, $P < 0.01$; No-exercise Residual condition, $t(11) = -2.63$, $P < 0.01$; and Exercise Residual condition, $t(11) = -3.02$, $P < 0.01$. The erotic films failed to increase VBV responses during the Exercise Immediate condition.

Heart rate. A repeated-measures Session (Exercise vs No-exercise) × Film (neutral vs erotic) analysis of variance of heart rate was conducted within each experimental condition to examine whether heart rate was altered with exposure to exercise and/or an erotic film. Results revealed a significant increase in heart rate with exposure to exercise during each of the Immediate, $F(1, 11) = 40.13$, $P < 0.001$; Delayed, $F(1, 11) = 51.91$, $P < 0.011$; and Residual conditions, $F(1, 11) = 21.05$, $P < 0.01$. No difference in heart rate was found between neutral and erotic films in either the Immediate, Delayed, or Residual conditions (all $Fs < 1$), and there was no significant interaction between session and film for any of the experimental conditions.

Results from a one-way analysis of variance indicated that there were no significant differences in mean heart rate across films, between conditions during the No-exercise sessions, ($F < 1$), but significant differences in heart rate between conditions during the Exercise sessions, $F(2, 33) = 7.08$, $P = 0.003$. *Post hoc* analyses using Newman Keuls tests with a significance level of $P < 0.05$ revealed differences in heart rate between the Exercise Immediate and Exercise Delayed conditions, and between the Exercise Immediate and Exercise Residual conditions. There was no significant difference in heart rate between the Exercise Delayed and Exercise Residual conditions. Mean heart rates following the Immediate, Delayed, and Residual
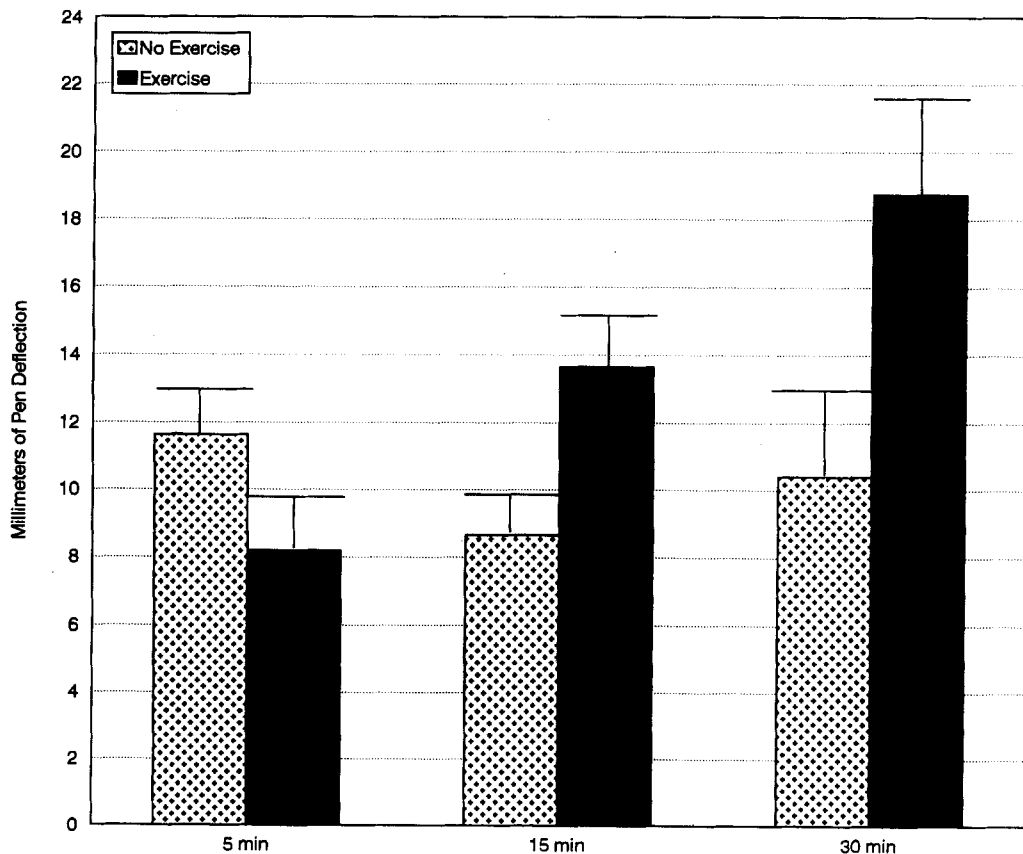

Fig. 2. Mean vaginal blood volume (mv deviation from baseline) ±SEM between neutral and erotic stimulus presentations during the No-exercise and Exercise conditions measured at 5 min, 15 min, and 30 min post-exercise.

conditions respectively, were 70.1, 67.9, 70.1 during the No-exercise neutral film; 70.2, 68.5, 70.2 during the No-exercise erotic film; 96.0, 87.4, 80.9 during the Exercise neutral film; and 97.2, 86.9, 80.5 during the Exercise erotic film.

Analyses of subjective measures

Subjective ratings of sexual arousal, positive affect, and negative affect, in response to erotic stimuli, were analyzed using 2 × 3 (Session × Condition) analyses of variance. There were no significant effects of session or condition, nor were there significant interactions between session and condition for any of the subjective measures. Mean subjective ratings of sexual arousal, positive affect, and negative affect by experimental condition were: No-exercise Immediate, 4.1, 3.2, 1.4; Exercise Immediate 3.8, 3.1, 1.4; No-exercise Delayed, 4.2, 3.5, 1.4; Exercise Delayed, 4.0, 3.3, 1.3; No-exercise Residual, 3.8, 2.5, 1.6; Exercise Residual, 3.7, 2.9, 1.4, respectively.

DISCUSSION

The present investigation examined the effects of low, moderate, and high levels of SNS activation, via acute exercise, on subjective and physiological sexual arousal in women. Exercise was used to elicit SNS activity based on evidence which suggests that exercise, at the intensity, duration, and time frame used in the present study, produces significant SNS arousal (e.g. Mazzeo & Marshall, 1989; Robinson *et al.*, 1966). Indirect support for the effectiveness of the experimental procedures and controls in eliciting SNS activity was provided by the finding that heart rate remained significantly elevated during the Exercise vs No-exercise sessions in each of the Immediate, Delayed, and Residual conditions. The finding that heart rate was significantly higher during the Exercise Immediate than the Exercise Residual condition suggests that, as intended, levels of SNS activity had declined to markedly lower levels post-exercise. The finding that the erotic films elicited significant increases in VPA during each of the experimental conditions indicates that the experimental stimuli were successful in altering physiological sexual arousal. These results suggest that the experimental manipulations and control procedures used in the present investigation were effective.

In the presence of an erotic stimulus, acute exercise inhibited physiological sexual arousal when measured immediately following exercise, and facilitated physiological sexual arousal when measured 15 or 30 min post-exercise. These effects included a marginally significant decrease in VPA at 5 min post-exercise, a significant increase in VPA and a marginal increase in VBV at 15 min post-exercise, and a marginally significant increase in both VPA and VBV at 30 min post-exercise. These findings suggest that an optimal level of SNS activation exists beyond and below which physiological sexual arousal is suppressed or unaffected. The notion of a curvilinear relationship between SNS activation and sexual arousal was suggested by Jupp and McCabe (1989) who found that moderate vs low or high, levels of self-reported, general, physiological arousability were optimal in facilitating sexual function.

In contrast to the reported changes in physiological sexual arousal with exposure to exercise, exercise had no significant effect on subjective ratings of sexual arousal in either of the Immediate, Delayed, or Residual conditions. These findings replicate those of Meston and Gorzalka (1995) who found significant increases in physiological but not subjective ratings of sexual arousal at 15 min post-exercise. Exercise also had no significant effect on subjective ratings of positive or negative affect in any of the experimental conditions. This suggests that the exercise-induced changes in physiological sexual arousal cannot be explained exclusively in terms of cognitive factors. That is, the reported increases or decreases in physiological sexual arousal cannot be attributed to differences in *S*s' mood with exposure to exercise, or to a positive feedback system between cognitive and physiological components of the sexual response.

The fact that exercise marginally decreased VPA responses at 5 min post-exercise leads one to question whether too much SNS activity may inhibit sexual arousal in women. However, other physiological changes, secondary to intense, acute exercise make this hypothesis rather speculative. Research indicates that during and immediately following exercise, a decrease in vascular resistance of working muscles causes a significant increase in blood flow to the exercising muscles, and arterial vasoconstriction decreases blood flow to the nonexercising muscles, skin, kidney, spleen, liver, and intestines (Christensen & Galbo, 1983). Consequently, immediately following exercise, blood flow may have been shifted away from the genital region to help restore working muscles. The lack of available blood in the genital region may have impaired sexual responding. This assertion is supported by the finding that, at 5 min post-exercise, VBV responses to erotic stimuli were not only suppressed but extinguished. Further research is needed to examine the effects of intense levels of SNS activation elicited by means that do not cause significant increases in peripheral muscle activity (e.g. epinephrine injections) on sexual responding. Research of this nature would provide insight into whether the inhibitory effects noted at 5 min post-exercise are attributable to intense SNS activity or, alternatively, to residual, *nonsexual* effects of acute exercise.

The notion that low to moderate levels of SNS activation facilitate physiological sexual responding contrasts with previous assumptions regarding the nervous system regulation of sexual arousal in women. Early researchers (e.g. Kinsey, Pomeroy, Martin & Gebhard, 1953; Wolpe, 1958) concluded that female sexual arousal was largely dependent upon PNS rather than SNS activation. More recently, Kaplan (1974) proposed a widely accepted biphasic model in which the initial stage of sexual arousal, characterized by engorgement of tissue, is mediated by the PNS, and the later stage of arousal, characterized by orgasm, is mediated by the SNS. The present results, together with those of Meston and Gorzalka (1995), suggest that SNS influences may be important in facilitating sexual responding not only during the later stages of arousal/orgasm, but during the initial stages of arousal as well. The possibility for a facilitatory role of SNS activation in female sexual function may help to explain the high reported incidence of inhibited sexual arousal/orgasm secondary to psychotherapeutic drug use. Numerous antipsychotic and anxiolytic medications have been reported to inhibit sexual arousal and orgasm in women (Meston & Gorzalka, 1992; Shen & Sata, 1990). Many of these same drugs have also been shown to inhibit peripheral nervous system activity (Physicians' Desk Ref., 1993).

The finding that moderate levels of SNS activation facilitate sexual arousal has potential treatment implications for sexual dysfunction in women. Since Wolpe's introduction of systematic desensitization (1958), anxiety-reduction techniques have been widely adopted in the treatment of sexual dysfunction. These techniques are thought to facilitate sexual responding by decreasing negative cognitions which disrupt the processing of erotic cues, and by inducing a state of relaxation which increases PNS and decreases the presumably inhibitory SNS influences. With respect to the cognitive aspect of these treatments, numerous outcome studies have shown anxiety-reduction techniques to be highly successful in altering negative performance cognitions (for review, see Andersen, 1983). With regard to the physiological component of these treatments, however, the effects of decreasing SNS activity on sexual arousal have not yet been examined. Together with the findings of Meston and Gorzalka (1995), the present results suggest that treatments for sexual dysfunction which decrease SNS activity by inducing a state of relaxation may, in fact, be counterproductive to the sexual response at a physiological level.

Because research supports the use of relaxation techniques in facilitating cognitive change, it may be the case that techniques such as systematic desensitization and sensate focus are desynchronous in their effectiveness for treating components (i.e. cognitive vs physiological) of the female sexual response. This would not be surprising given that, although linked, cognitive, behavioral, and physiological response systems do not necessarily change at the same time, in the same manner, or even in the same direction (Rachman & Hodgson, 1974).

Although the present study was limited to the effects of SNS activation in sexually functional women, it is conceivable that women with sexual difficulties would benefit from treatments which focus on altering cognitive factors while maintaining or even increasing (vs decreasing) SNS activity. A possible method for treating sexual dysfunction at both a cognitive and physiological level, would be to teach women, via imagery techniques, to associate increases in SNS activity (e.g. increased heart rate, muscle tension, sweating) with sexually pleasurable stimuli, and to dissociate those physiological changes and negative performance-anxiety cognitions. Of course before new treatment models can be considered, future research is needed to examine the effects of both SNS activation and SNS inhibition on sexual arousal in women with sexual difficulties.

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