THE EFFECTS OF SYMPATHETIC ACTIVATION VIA ACUTE EXERCISE ON PHYSIOLOGICAL AND SUBJECTIVE SEXUAL AROUSAL IN WOMEN

by

CINDY MAY MESTON

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Department of Psychology
The University of British Columbia
Vancouver, Canada

Date Sept 8, 1992
ABSTRACT

This investigation was designed to examine the effects of sympathetic activation on physiological and subjective sexual arousal in women. The purpose was to empirically test the widely held assumption that sympathetic activation inhibits sexual arousal. In experiment I, subjects participated in two experimental sessions in which they viewed a neutral preexposure stimulus followed by an erotic stimulus. In one of these sessions, subjects were exposed to 20 minutes of intense exercise, designed to increase sympathetic activity, prior to viewing the films. In experiment II, subjects viewed two consecutive neutral stimuli preceded by 20 minutes of intense exercise. Experiment II was designed primarily to verify the results of experiment I. Sexual arousal was measured physiologically with a vaginal photoplethysmograph and subjectively with a self-report rating scale. Acute exercise increased both vaginal pulse amplitude and vaginal blood volume responses to erotic stimuli but not to neutral stimuli. Subjective perceptions of sexual arousal were unaltered with exposure to exercise. The present findings have implications for the enhancing effects of anxiety upon sexual arousal in women, Wolpe's reciprocal inhibition hypothesis, desynchrony between subjective and physiological sexual arousal in women, and the treatment of sexual dysfunction.
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The Effects of Sympathetic Activation via Acute Exercise on Physiological and Subjective Sexual Arousal in Women

It is commonly assumed that increases in sympathetic nervous system activity, as in the case of anxiety, inhibit sexual arousal in both men and women (Kaplan, 1974). Furthermore, it is believed that female sexual arousal is primarily under the influence of the parasympathetic nervous system (Heiman & Hatch, 1981). Based on these assertions, treatments for sexually dysfunctional men and women, such as sensate focus, are largely anxiety-reduction techniques designed to decrease sympathetic activity. However, these etiological assumptions of over 30 years are based, in large, on clinical reports which indicate performance anxiety causes erectile difficulties in men, and on Wolpe's (1958) theory that anxiety and sexual arousal are mutually inhibitory. Empirical evidence supporting these assertions has not been offered. Hence, the autonomic pathways and processes by which sexual arousal is initiated and heightened remain a matter of conjecture (Schnieden & Rees, 1985).

Despite the widely held assumption that sympathetic activation inhibits sexual arousal, research conducted in both animals and humans indicates that increased sympathetic activity may actually facilitate sexual arousal. For example, recent research conducted in both men and women has shown that, under certain conditions, anxiety can heighten sexual arousal. Unfortunately, it is difficult to ascertain
whether the increases in sexual arousal reported in these studies are attributable to increased sympathetic activity resulting from anxiety exposure, or whether they are the result of cognitive factors associated with the anxiety stimuli.

The present study is the first examination of the effect of sympathetic activation via acute exercise on subjective and physiological measures of sexual arousal in women. If sympathetic activation is shown to enhance sexual arousal, then the findings from this study will provide a possible explanation for the recent reports of a facilitatory effect of anxiety on sexual arousal in women. On the other hand, if sympathetic activation fails to enhance sexual arousal, then the assertion that anxiety enhances sexual arousal via cognitive factors will be strengthened. Furthermore, identification of the effects of sympathetic activation on female sexual arousal has major implications for deriving an etiological theory of sexual arousal, and for developing effective treatments for the alleviation of sexual dysfunction in women.

**Sympathetic Activation and Physiological Sexual Arousal**

Both male and female sex organs receive innervation from the sacral parasympathetic and thoracolumbar sympathetic nerves. In the male, activation of the parasympathetic pathways elicits erection. The parasympathetic signal activates penile nerves which cause dilation of the penile arteries and relaxation of muscles in
the walls of the sinusoids and arterioles that supply erectile tissue. As a result, blood flow in erectile tissue increases which leads to a rise in intracavernous pressure and expansion of the cavernous spaces to produce extension and rigidity of the penis (deGroat & Steers, 1990). Sympathetic fibers may also alter blood flow to the erectile tissue (deGroat & Steers, 1990). Research conducted on male spinal cord injury patients indicates that the sympathetic pathway alone may be sufficient to elicit erection. For example, Comarr (1970) described several patients with lesions at the sacral segment of the spinal cord who were still able to experience erections.

While penile erection is primarily under sacral parasympathetic control, ejaculation is primarily under sympathetic control. Activation of the sympathetic pathways provides an excitatory input to the vas deferens, seminal vesicles, prostate gland, and internal urinary sphincter (deGroat & Steers, 1990). During emission, the sympathetic nerves which innervate each of these organs are activated so that semen is transmitted into the posterior urethra, and the internal urinary sphincter is closed to prevent retrograde ejaculation into the bladder (deGroat & Steers, 1990).

The mechanisms by which the autonomic nervous system influences female sexual behavior are less understood. The majority of research has focused on reproductive functioning and broad assumptions have been made that the control of
arousal and orgasm in women is directly comparable to erection and ejaculation in men. These assumptions are not entirely accurate and may be overstated. Vasocongestion in the female, which is considered analogous to erection in the male, is thought to be regulated by the sympathetic nervous system (Geer et al., 1984). By contrast, erection in the male is controlled primarily by the parasympathetic nervous system. With respect to orgasm, more direct comparisons may be made between men and women. Sympathetic nerves located in the lumbar section of the spinal cord provide input to the bulbocavernosus and ischiocavernosus muscles which are responsible for both ejaculation in males and contribute to the rhythmic perineal contractions during orgasm in women (deGroat & Steers, 1990).

Overall, the direct influence of autonomic pathways on sexual arousal remains unclear. It seems however that in males, erection is under parasympathetic control and ejaculation is under sympathetic control. In females, both vasocongestion and orgasm appear to be under sympathetic control.

**Sympathetic Activation Enhances Sexual Arousal**

Support for a facilitatory effect of sympathetic activity on sexual arousal comes from the fact that several physiological changes associated with increased sympathetic activity accompany the sexual response. For example, sexual arousal has been reported to increase heart rate, perspiration, and muscle tension; alter galvanic skin
conductance; increase systolic and diastolic blood pressure; and cause nipple erection (e.g. Hoon et al., 1976; Masters & Johnson, 1966; Wenger et al., 1968). These responses are known to be under the influence of the sympathetic nervous system. It should be noted, however, that changes in heart rate and skin conductance do not always accompany the sexual response. For example, Hoon and associates (1976) reported that increases in sexual arousal, measured by increases in vaginal blood volume, were accompanied by increases in systolic and diastolic blood pressure but not by changes in heart rate or skin conductance. Similarly, in a study of sexual arousal in men, Wenger and associates (1968) reported significant increases in systolic blood pressure, diastolic blood pressure, and skin conductance with exposure to erotic stimuli, but no change in heart rate. The finding that sexual arousal is sometimes, but not always, accompanied by changes in heart rate and skin conductance may be attributable to the fact that the studies which reported such findings were conducted in a laboratory setting and used erotic films as sexual stimuli. In contrast, studies which reported these measures to accompany sexual arousal, such as that conducted by Masters and Johnson (1966), used more natural sexual interactions and attained higher levels of sexual responding.

More indirect evidence that sympathetic activity may enhance sexual arousal is derived from animal studies. Beach (1947) was one of the first animal researchers to
report that general activation induced by "batting the animals sharply about the cage" could increase sexual excitability in rats. Since then, a number of studies have demonstrated that electric shock or tail pinch can increase the number of prepubertal male rats copulating, increase the rate of copulation in experienced males, decrease the length of the postejaculatory period of sexual inactivity, increase the rate of mounting behavior in aging males, and temporarily restore copulatory activity suppressed by various forms of brain damage (for review, see Antelman & Caggiula, 1980). Calhoun (1962) also reported increases in sexual activity in male and female rats following a succession of aggressive encounters. Generally arousing events, such as fear, have also been reported to facilitate erection and ejaculation in male dogs and chimpanzees (Beach, 1947).

Anxiety and Aversive Stimuli Enhance Sexual Arousal

Further support for a facilitatory effect of sympathetic activity on sexual arousal is provided by clinical and experimental evidence which indicates that anxiety and various forms of aversive stimuli known to increase sympathetic activity enhance sexual arousal in men and women. Early clinical research conducted by Ramsey (1943) revealed that approximately 50% of adolescent boys have experienced erections in response to fearful situations such as being in an accident or being chased by the police. Similar findings were reported by Bancroft (1970). In an
interesting report by Sarrel and Masters (1982), men were able to perform sexual intercourse repeatedly despite direct threats with knives and other weapons if they failed. A similar phenomenon has been reported amongst exhibitionists and voyeurs who are often unable to become aroused without first experiencing the threat of being arrested (Stoller, 1976). Related findings have been reported in women. In the well known Kinsey report, Kinsey and associates (1953) point out that many women experience erotic sensations to being bitten. In a sample of 2200 women, 26% reported "definite and/or frequent erotic responses", 29% reported "some erotic responses", and 45% reported "never" experiencing erotic responses to being bitten. Kinsey and associates also point out that biting is a part of sexual activity in a number of mammalian species including the baboon, various monkeys, mink, marten, ferret, skunk, horse, pig, sheep, lion, shrew, etc.

More recently, the effects of anxiety on sexual arousal have been examined directly in the laboratory. Heiman and Rowland (1983) reported increases in subjective sexual arousal in males who were given demand instructions to maintain an erection. In a study conducted by Dutton and Aron (1974), increases in subjective sexual arousal were reported in men who were asked to cross a fear-arousing suspension bridge. With respect to physiological sexual arousal in the male, Wolchik and associates (1980), Barlow and associates (1983), and Heiman and Rowland (1983)
demonstrated an increase in penile tumescence with exposure to an anxiety arousing videotape, a shock-threat condition, and performance demand instructions. The few studies that have been conducted in the female reveal similar increases in physiological sexual arousal with exposure to anxiety-evoking stimuli. For example, Hoon, Wincze, and Hoon (1977) exposed sexually functional women to either an anxiety-evoking film followed by an erotic film or a neutral film followed by an erotic film. Higher levels of sexual arousal, indicated by increased vaginal blood volume (VBV) levels, were reported amongst women who viewed an anxiety-evoking film prior to the erotic stimuli. Palace and Gorzalka (1990) replicated the findings of Hoon and associates (1977) in both sexually functional and sexually dysfunctional women. In contrast to the reported increase in VBV levels, subjective measures of sexual arousal indicated that preexposure to an anxiety-evoking film caused a slight but significant decrease in ratings of sexual arousal (Palace & Gorzalka, 1990). Based on this finding, the authors hypothesized a possible desynchronous relationship between physiological and subjective components of the sexual response in women. This is the only study known to the author that has measured both subjective and physiological sexual responses to anxiety stimuli.

Cognitive Explanations for the Enhancing Effects of Anxiety on Sexual Arousal
The results of the above studies indicate that exposure to an anxiety-evoking situation may enhance sexual arousal. Researchers who have attempted to explain this finding have focused primarily on cognitive, as opposed to physiological, factors. For example, Wolpe (1978) claimed that the increase in sexual arousal following exposure to an anxiety-evoking stimulus may be attributable to an "anxiety relief" phenomenon as opposed to a facilitatory effect of anxiety. This explanation was based, in part, on a series of experiments whereby patients reported a feeling of relief at the cessation of electric shock. Often their reported sense of relief was greatly out of proportion to the discomfort they had experienced (Wolpe, 1958).

Social psychologists have described the enhancing effects of anxiety on sexual arousal as a form of residual arousal which has moved from one emotional experience to another. For example, Dutton and Aron (1974) and Beggs and associates (1987) explained the increase in sexual arousal within the conceptual framework of Schacter's excitation transfer theory (Schacter, 1964). This theory asserts that when the context for heightened arousal is not apparent, the individual relies on the current context for interpretation. Faced with anxiety when viewing the erotic film, subjects mislabeled the anxiety arousal as sexual arousal. This, in turn, served to heighten sexual responding.

In contrast to the many cognitive explanations for the enhancing effects of anxiety on sexual arousal, Palace and
Gorzalka (1990) were the first to propose that anxiety enhances sexual arousal through the direct instigation and facilitation of sympathetic activation. They asserted that the increase in physiological sexual arousal reported in their study could not be explained by anxiety relief (Wolpe, 1978), given that anxiety was reported throughout the erotic exposure (Palace & Gorzalka, 1990). In order to successfully determine whether the enhancing effects of anxiety are attributable to physiological, versus cognitive, factors, two issues must be addressed. First, in order to rule out several of the cognitive explanations, such as anxiety relief and residual excitation theory, the anxiety-inducing situation must be presented simultaneously with the erotic stimulus. Second, in order to determine whether the physiological components of anxiety alone enhance sexual arousal, the heightened arousal, representative of an anxiety state, must be presented without the accompanying anxiety cognitions.

**Sympathetic Activation Inhibits Sexual Arousal**

*Reciprocal Inhibition Theory.* Joseph Wolpe was the first and most influential researcher to assert that sympathetic activity inhibits sexual arousal. In his theory of reciprocal inhibition, Wolpe (1958) proposed that emotional states such as relaxation, hunger, and sexual arousal are antagonistic to an anxiety state. Furthermore, he proposed that this mutual inhibition was operative at not only a behavioral and cognitive level, but at a neural or
autonomic level as well. He asserted that sympathetic arousal, such as that generated by an anxiety state, interfered or competed with parasympathetic arousal generated by a sexual state.

Wolpe's theory is based on a series of experiments with cats which aimed at determining methods to decrease anxiety and avoidance behavior. Cats were first conditioned via shock to display intense fear behavior in a small cage. They were then fed while exposed to gradually increasing "doses" of anxiety-evoking stimuli. Wolpe noted that when anxiety was intense, eating was inhibited and, when anxiety was lower, allowing eating to take place, the anxiety response weakened.

Since Wolpe's theory was originally proposed, there has been only one study aimed directly at testing the assertion that anxiety and sexual arousal are mutually inhibitory (Hoon et al., 1977). Consistent with Wolpe's theory, this study demonstrated that anxiety exposure following an erotic film decreased physiological sexual arousal in women (measured by VBV levels) more rapidly than did a neutral film. Presumably, the sympathetic influence of anxiety extinguished the parasympathetic influence of the erotic stimuli. However, contrary to the theory, anxiety stimuli prior to an erotic film increased physiological sexual arousal. Wolpe (1978), criticized this study on the grounds that the anxiety stimuli did not increase heart rate measures. As a result, there was no evidence of an anxiety
response during presentation of the erotic stimuli. According to Wolpe, the study did not provide an adequate basis for testing the reciprocal inhibition hypothesis because in order for two events to be mutually inhibitory, they must be presented simultaneously, not sequentially.

Anxiety Inhibits Sexual Arousal

Support for Wolpe's assertion that anxiety inhibits sexual arousal is provided by clinical reports which link generalized anxiety and impaired erectile ability. For example, Johnson (1965), in his retrospective study of males with erectile dysfunction, reported that 48% of the men studied suffered from an anxiety disorder. In a later study, Cooper (1969) reported that only 12% of his sexually dysfunctional subjects were clinically neurotic but 51% had a "marked coital anxiety." Coital anxiety was most frequently reported to be a consequence of fear of failure (73%), being regarded as sexually inferior (43.5%), or being ridiculed (40%). Consistent with Cooper's findings, Ansari (1975) reported that 66% of the patients studied reported that their erectile dysfunction developed as a reaction to discrete sexual experiences. Taken together, these findings support the connection between anxiety and erectile dysfunction. However, they also indicate that erectile dysfunction may be more a consequence of negative cognitions associated with specific sexual activities than of generalized anxiety.
Laboratory studies also indicate that, given certain situations, anxiety may inhibit sexual arousal. Hale and Strassberg (1990) reported decreased physiological sexual arousal in a group of 54 men in response to either shock threat or false feedback indicating atypically low levels of sexual response. The authors suggested that the threatening cognitions associated with these conditions became the subjects' focus of attention. This, in turn, left less attention available for erotic stimuli and resulted in lower levels of arousal. This hypothesis was partially supported by data which indicated lower levels of memory recall for sexual stimuli in the threatening versus control condition. Differences in memory recall between conditions did not, however, reach statistical significance. In a study conducted by Bozman and Beck (1991), decreases in subjective sexual arousal were reported in males who were exposed to an erotic film containing statements designed to evoke anxiety. These anxiety stimuli had no significant effect on physiological sexual arousal. Using the threat of electric shock as the source of anxiety, Beck and Barlow (1986) and Beck and associates (1987) reported that anxiety decreased physiological sexual arousal in men. The above studies contradict those reviewed earlier which indicate that anxiety enhances sexual arousal. Individual differences in sexual responding, different operational definitions of anxiety, and/or a number of methodological differences between studies could explain these contradictory results.
Treatments for Sexual Dysfunction

The majority of techniques used in the treatment of sexual dysfunction focus on reducing anxiety. The foremost of these, systematic desensitization, was proposed by Wolpe in 1958. This technique involves four stages. First, the patient is trained to relax the muscles in his/her body. Second, a hierarchy of anxiety-provoking stimuli or situations is constructed. Third, beginning with the least fearful stimuli, the client imagines the feared situation while remaining relaxed. Finally, the client engages in the sexual activities in real life, while maintaining a state of relaxation. Presumably, the effectiveness of this treatment is attributable to the muscular relaxation component which induces a state of parasympathetic dominance (Wolpe, 1958).

A large number of outcome studies report high success rates in treating sexual dysfunction with systematic desensitization (for review, see Norton & Jehu, 1984). This success has been used as support for the assertion that sympathetic activation inhibits sexual arousal. However, studies conducted in women seem to indicate that systematic desensitization is effective in treating only specific types of sexual dysfunction. For example, in an elaborate study by Sotile and Kilmann (1978), systematic desensitization was shown to produce reductions in several measures of coital anxiety, improved pleasure during intercourse, improved satisfaction with overall sexual function, and increased extracoital stimulation. However, systematic
desensitization was not effective in increasing orgasmic ability. This finding is consistent with other research that indicates systematic desensitization is ineffective in treating anorgasmia (e.g., Andersen, 1983; Cooper, 1981; Crown & D'Ardenne, 1982; Kuriansky & Sharpe, 1981).

Since the introduction of Wolpe's theory in 1958, little has changed in the understanding and treatment of sexual dysfunctions.

The present study is the first examination of the effect of sympathetic activation, via acute exercise, on subjective and physiological measures of sexual arousal in women. In experiment I, subjects were exposed to two experimental sessions in which they were shown a neutral film stimulus followed immediately by an erotic film stimulus. In one of the conditions, the subjects were exposed to 20 minutes of intense exercise prior to viewing the films. This experiment was designed to test the effects of sympathetic activation on subjective and physiological sexual responses to erotic stimuli. In experiment II, subjects were shown two different, consecutive neutral film sequences, preceded by 20 minutes of intense exercise. This experiment was designed to test the effects of sympathetic activation on subjective and physiological sexual responses to neutral stimuli. The results of this experiment were necessary to determine whether the physiological responses reported in experiment I were representative of a sexual as opposed to a cardiovascular or hormonal response to
sympathetic activation. In both experiments, subjective sexual arousal was measured using a questionnaire. Physiological sexual arousal was measured using both vaginal blood volume and vaginal pulse amplitude as indices. Heart rate was used as an indicator of general autonomic arousal.

The purpose of the present investigation is threefold. First, the study is an attempt to test the direct effects of sympathetic activation on female sexual arousal. Exposure of subjects to an intense exercise condition would increase activity of the sympathetic nervous system similar to exposure to an anxiety-evoking stimulus (Obrist et al., 1978). However, unlike anxiety-producing stimuli, exercise would presumably have less of an effect on cognitive set. If exercise and anxiety-evoking stimuli have similar effects on sexual arousal, this would strengthen the argument that anxiety may act via sympathetic activation rather than cognitively to increase physiological sexual arousal.

The second purpose of this study is to provide an indirect test of the assertion that sympathetic activation and sexual arousal are mutually inhibitory. Previous research in the female conducted by Hoon and associates (1977) and Palace and Gorzalka (1990) has been inadequate to test this theory because, according to Wolpe (1978), the two events must occur simultaneously to be mutually inhibitory. In these studies, the anxiety and sexual stimuli were presented sequentially. The design of the present investigation ensured sympathetic influences during
presentation of the erotic stimuli by evoking intense activation of the sympathetic nervous system and by ensuring (via heart rate measures) its persistence throughout the erotic exposure.

The third purpose of this study is to replicate the findings of Palace and Gorzalka (1990) whereby anxiety exposure had an opposite effect on subjective and physiological measures of sexual arousal. That is, the study is designed to determine whether sympathetic activation has dissimilar or similar effects on physiological and subjective sexual arousal. In experiment II, subjects viewed only consecutive neutral stimuli. This provides baseline data and permits comparisons between experimentally manipulated levels of subjective sexual arousal and "nonsexual" baseline measures. It is the first study of its kind to include this type of control group. Previous research has allowed only comparisons between experimental conditions and, hence, has been unable to discern whether subjects are aware and/or willing to report changes in physiological sexual arousal from a "nonsexual" state.

Specifically, the following questions are addressed:
1. Does sympathetic activation enhance sexual arousal?
2. Are sympathetic activation and sexual arousal mutually inhibitory?
3. Does a desynchronous relationship exist between cognitive and physiological components of the sexual response in women?

EXPERIMENT I

Method

Subjects

Twenty-five women participated in the study (Mean age = 24 years, range = 18-34 years). The subjects were recruited through a psychology department undergraduate research participant pool or by personal contact. Nine subjects were graduate students in psychology, 11 subjects were first or second year students in psychology, and five subjects were undergraduate or graduate students in disciplines other than psychology. Subjects were given course credit and/or a fitness assessment profile as an incentive for their participation. Inclusion criteria were: between the ages of 18-35 years, no use of medications known to affect vascular or sexual functioning, no history of treatment for sexual dysfunction, and current involvement in a heterosexual relationship.

Profile descriptions of all subjects were obtained via the Derogatis Sexual Functioning Inventory (DSFI; Derogatis, 1978). The Sexual Functioning Index (SFI) and the Global Sexual Satisfaction Index (GSSI) of the DSFI were used to screen for absence of sexual dysfunction. All subjects employed in the study scored greater than or equal to the
40th percentile on both of these measures. Previous research conducted by Palace and Gorzalka (1992) considered subjects below this percentile to be sexually dysfunctional. In addition, the Brief Symptom Inventory (BSI; Derogatis, 1975) subtest of the DSFI was used to screen for absence of general psychopathology. All subjects employed in the study scored greater than or equal to the 30th percentile on the BSI (see Table I.). The 30th percentile cutoff criterion for general psychopathology was used by Palace and Gorzalka (1992).

Apparatus and Materials

Film Stimuli. Preexposure stimuli consisted of two four-minute color videotapes: a one-minute display of the word relax followed by a three-minute neutral travelogue sequence. The experimental, or erotic stimuli, consisted of two three-minute color videotapes of a nude, heterosexual couple engaging in foreplay and intercourse. The erotic films were accompanied by fast-paced music and included explicit sexual communication by the couple. The two erotic films were matched on the number, order, type, and duration of sexual acts, and included the same actors and settings. The films have been shown to reliably elicit sexual arousal in women (Palace and Gorzalka, 1990; 1992).

Physiological Measurement. Physiological measures were obtained using a vaginal photoplethysmograph (Sintchak & Geer, 1975). Changes in vaginal blood volume (VBV), vaginal pulse amplitude (VPA), and heart rate (HR) were monitored
Table I.

DEMOGRAPHIC AND PSYCHOMETRIC CHARACTERISTICS OF SUBJECTS

<table>
<thead>
<tr>
<th>Measure</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>24.28</td>
<td>4.91</td>
<td>18-34</td>
</tr>
<tr>
<td>VO2 Max</td>
<td>27.40</td>
<td>8.35</td>
<td>16-45</td>
</tr>
<tr>
<td>Duration of sexual experience</td>
<td>8.88</td>
<td>5.13</td>
<td>1-17</td>
</tr>
<tr>
<td>Derogatis Sexual Functioning Inventory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affect</td>
<td>50.60</td>
<td>9.61</td>
<td>33-68</td>
</tr>
<tr>
<td>Experience subtest</td>
<td>50.44</td>
<td>8.12</td>
<td>31-63</td>
</tr>
<tr>
<td>Sexual Functioning Index</td>
<td>51.64</td>
<td>10.94</td>
<td>42-70</td>
</tr>
<tr>
<td>Global Sexual Satisfaction Index</td>
<td>54.96</td>
<td>9.45</td>
<td>41-70</td>
</tr>
<tr>
<td>Brief Symptom Inventory</td>
<td>46.72</td>
<td>8.28</td>
<td>32-61</td>
</tr>
</tbody>
</table>

Note. Duration of Sexual Experience is calculated as the difference between age at first intercourse and current age. Means for the Derogatis Sexual Functioning Inventory are based on raw scores that were converted to established percentile rankings (T scores).
simultaneously during all experimental sessions. VBV, the dc signal, reflects slow changes in the pooling of blood in the vaginal tissue (Hatch, 1979), while VPA, the ac signal, reflects short-term changes in engorgement (Rosen and Beck, 1988).

Contradictions exist in the literature as to whether VBV or VPA is the more appropriate indicator of female sexual arousal. Several investigators have found VPA to be a more sensitive measure of sexual arousal (Geer et al., 1974; Heiman, 1977; Osborn & Pollack, 1977), to show a more rapid return to baseline following stimulus presentation (Henson et al., 1979), to demonstrate greater within-session reliability (Heiman, 1976; 1977), and to be unaffected by temperature and drift confounds (Beck et al., 1983). In contrast, Hoon and associates (1976) and Palace and Gorzalka (1990; 1992) claim that VBV is the more sensitive indicator of genital vasocongestion as changes in VPA account for only a fraction of total blood volume during engorgement.

Light and heating effects were minimized by allowing the photoplethysmograph a 45-minute warm-up period prior to insertion, followed by a 10-minute recorded adaptation period before the experiment began. The signal from the Geer gauge and module (Farral Instruments, Grand Island, NE) was channeled through an optical isolator-power supply. The VBV signal was recorded at a sampling rate of 5 times/second with a Data Translation (Marlborough, MA) analog-digital converter and Labtech Acquire Program (Laboratory
Technologies Corp., 1986) installed on a Samtron SC-386 microcomputer. The VPA and heart rate signals were channeled and recorded on a Beckman model R612 dynagraph (Scheller Park, IL). The software program timed the administration of the stimuli and used an audio trigger signal to mark all stimulus changeovers.

**Subjective Measurement.** A self-report rating scale, adapted from Heiman and Rowland (1983), was used as a subjective measure. The scale has been used in previous research (Palace & Gorzalka 1990; 1992), and has been shown to be a sensitive indicator of emotional reactions to erotic stimuli (Heiman 1980; Heiman and Hatch 1980; Heiman & Rowland 1983; Morokoff & Heiman 1980). Research indicates that there are no significant differences in subjective reports of sexual arousal obtained by methods of discrete versus continuous subjective measurement (Steinman et al., 1981). The scale consists of 32 items: sexual arousal (1 item), perceptions of physical sexual change (4 items), autonomic arousal (5 items), positive affect (11 items) and negative affect (11 items) (see Appendix). Subjects rated each of these items, depending on the degree to which they experienced the sensations, on a 7-point Likert Scale, from (1) not at all to (7) intensely. Subjective sexual arousal was defined by the following 5 items: sexually aroused, warmth in genitals, genital wetness or lubrication, genital pulsing or throbbing, and any genital feelings. Subjective autonomic arousal was defined by the following five items:
faster breathing, faster heart beat, perspiration, feelings of warmth, and any physical reaction at all. Positive affect was defined by the following 11 items: sensuous, a desire to be close to someone, pleasure, interested, attracted, excited, sexy, loving, sexually attractive, easy to arouse, feminine. Negative affect was defined by the following 11 items: worried, angry, disgusted, embarrassed, guilty, dirty, inhibited, incompetent, sexually turned off, offended, bored.

Derogatis Sexual Functioning Inventory. The DSFI is a standardized self-report inventory designed to assess current levels of sexual functioning. Eight subtests (information, experience, drive, attitudes, symptoms, affects, gender role, fantasy) constitute an overall Sexual Functioning Inventory score (SFI) and one item (satisfaction rating of present sexual relationship) constitutes a Global Sexual Satisfaction Index (GSSI). The GSSI is believed to reflect the respondents' perception of their level of sexual functioning. Research has shown the DSFI to be a valid and reliable measure for differentiating sexually functional and dysfunctional women (Derogatis & Melisaratos, 1979; Derogatis & Meyer, 1979). The symptomology subtest of the DSFI, the BSI, is a distinct psychometric instrument designed to assess psychopathology. Nine symptom dimension scores and three global scores constitute the BSI.

Fitness Tests. Fitness levels were assessed for each subject. These assessments provided the necessary
information to set cycle speed and intensity to the same relative work load for all subjects, during the exercise conditions.

A continuous, submaximal, multi-stage bicycle ergometer test was used to estimate each subject's maximum volume of oxygen uptake (VO2 max). VO2 max is the maximal volume of oxygen that one can consume during exhausting work; it is the point at which increased workload is no longer associated with increased oxygen uptake. Because heart rate and oxygen uptake increase linearly in response to increased workload, this test allows the prediction of VO2 max from heart rate response (Pollock et al., 1978). VO2 max is an established indicator of cardiorespiratory endurance and subsequent physical fitness (Pollock et al., 1978). The submaximal multi-stage ergometer testing and scoring procedures used in this investigation were identical to those suggested for women by the YMCA (Golding et al., 1982).

Experimental Design

A within-subject repeated measures design was used in which each subject was exposed to each experimental condition in separate sessions. The two experimental conditions, exercise and no-exercise, were counterbalanced across subjects. During each of these sessions, subjects viewed a one-minute display of the word relax, followed by a three-minute neutral travelogue film, followed by a three-
minute erotic film. The films were counterbalanced across sessions.

Procedure.

The procedure consisted of four sessions: a one-hour orientation screening and questionnaire session, a 20-minute bicycle ergometer fitness test, a 45-minute no-exercise experimental session, and a one-hour exercise experimental session. The sessions were identical for all subjects. All subjects were asked to abstain from psychoactive drugs (including caffeine and alcohol) and to refrain from engaging in any strenuous physical activity for 24 hours prior to each session.

Session 1. Following an initial telephone screening, subjects were scheduled for a first session with the female experimenter. During this session, subjects were shown the laboratory facilities and equipment, were given verbal instructions on the use of the photoplethysmograph, and were encouraged to ask any questions related to the experiment. Subjects were told that they would be participating in an experiment which involved the effects of exercise on female sexual arousal. They were told that they would be viewing brief visual stimuli, some of which could include erotic content. To minimize a possible sense of coercion, subjects were given the option of telephoning within a week, regarding their decision to participate. Two subjects telephoned within the week and informed me of their decision not to participate. Subjects who chose to take part in the
study signed the standard consent form and completed the DSFI in a private room. To minimize potential experimental demand, subjects were instructed as follows:

When you fill out this questionnaire, please be as honest as possible. Every person is unique and there are absolutely no right or wrong answers. I realize that this questionnaire asks very personal information and so I would like to assure you again that all information is kept strictly confidential. All of the forms that you will be filling out are numerically coded; there will be no identifying information on any of the forms. Only myself and one female research assistant will have access to this coded information.

Session 2. During the second session, subjects received a submaximal multi-stage bicycle ergometer fitness test. Prior to taking the test, subjects completed a Physical Readiness Exam For Fitness Test adapted from the Physical Activity Readiness Questionnaire developed by the B.C. Ministry of Health (see Appendix). Subjects who reported medical conditions that may have put them at risk when exercising were excluded from the study. One subject was excluded because of breathing difficulties related to asthma. All fitness tests were conducted in accordance with the guidelines set forth by The American College of Sports Medicine (Pollock et al., 1978). A Monarch 814E ergometer was used to conduct the fitness tests. The bicycle seat height
was adjusted for each individual to ensure a slight bend in
the knee joint when the pedal was in its lowest position.
The test began with a workload of 150 kg/minute. The
subject cycled at a constant 50 rpm while the workload was
increased 150 kg/minute every three minutes. The test
lasted between 9-12 minutes depending on the subjects' heart
rate response. Heart rate was monitored manually from the
carotid artery during the final 30 seconds of each third
minute. The subjects' heart rate over time, in relation to
workload, was used to predict each subject's maximum volume
of oxygen uptake (VO2 max).

Sessions 3 & 4. The third and fourth sessions were the
two experimental sessions: exercise and no-exercise. These
two sessions were counterbalanced across subjects. In order
to minimize any potential within-subject menstrual cycle
influences, the two experimental sessions were scheduled at
approximately three day intervals. Research indicates that
sexual arousability to erotic stimuli, measured both
subjectively and physiologically, is only minimally, if at
all, influenced by menstrual cycle phases (Hoon et al.,
1982; Meuwissen & Over, 1992).

All experimental sessions were conducted inside the
Sexual Psychophysiology Laboratory at the University of
British Columbia. This laboratory has an adjoining,
private, internally-locked subject room. Communication with
subjects is made possible via an intercom system between
subject and experimenter rooms. The room is kept at a
constant 21.7°C. A color television monitor is positioned where subjects can sit comfortably in a recliner with a full view of the screen. A bicycle ergometer is positioned to the rear of the room.

During the no-exercise session, the subject entered the private, internally locked room together with the female experimenter. With the aid of diagrammed instructions, subjects were told to insert the photoplethysmograph so as to allow approximately a 2.5 cm distance between the end of the probe and the vaginal opening. The photoplethysmograph was washed with Hibitane and sterilized for 10 hours in Cidex (long-life activated dialdehyde solution: Surgikose Canada, Peterborough, Ontario) between uses. Subjects were informed of these sterilization procedures, and then instructed as follows:

I will be giving you step by step instructions, via the intercom system as we go along, but I'll also explain to you now what will take place during this session. When I leave the room, I'd like you to sit in this chair and insert the photoplethysmograph. Once you are ready, please say "ready". I will be able to hear you via the intercom system. At that point, I'd like you to just sit back and relax. You can recline the chair a little and cover yourself with this blanket if it makes you feel more comfortable. Once you are ready, I will take a 10 minute
baseline recording. This time period is necessary for the photoplethysmograph to adapt to your body temperature. After the 10 minute baseline period, a series of short films will begin on the television. They will last about 10 minutes. It is very important that you remain as still as possible during the baseline period and especially during the film presentations. The photoplethysmograph is very sensitive and any movement makes it very hard to read the information. Immediately after the films have ended, I'd like you to fill out this short questionnaire. Please be as honest as possible about how you feel about the films. Remember, every person is unique in their response and there are no right or wrong answers. Also remember that all information is kept strictly confidential.

Following the 10 minute adaptation period, subjects viewed one of two videotaped sequences (the sequences were counterbalanced across subjects and experimental conditions). Each sequence consisted of a one-minute display of the word relax, followed by a three-minute neutral travelogue film, followed by a three-minute erotic film. Immediately following the erotic film, subjects were asked to fill out the subjective rating scale.

During the exercise session, the subject entered the private, internally locked room with the female experimenter
and cycled for 20 minutes on a Get Fit 200-II stationary bicycle. The subjects' heart rate was monitored continuously using a Heart Speedometer model 8719 (Computer Instruments Corp., Westbury N.Y.) and the workload was adjusted by the experimenter throughout the 20 minutes to ensure that the subjects cycled at a constant 70% of their estimated VO2 max. The exercise physiology literature has shown that exercising for 20 minutes at 70% of VO2 max is sufficient to elicit significant changes in sympathetic nervous system activity (Grossman & Moretti, 1986). In addition, by ensuring that all subjects exercised at a constant 70% of their estimated VO2 max, confounds relating to fitness levels were eliminated. Research indicates that differences in physiological responses resulting from variation in fitness levels are minimized when subjects work at equivalent levels of their VO2 max (Grossman & Moretti, 1986). During the first few minutes of cycling time, the experimental procedures were repeated. When one minute of cycling time remained, the experimenter left the room. With the exception of 20 minutes of cycling, all experimental procedures were identical to those of the no-exercise session.

Subjects who would not be participating in experiment II 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 subjects were given personal fitness profiles indicating their level
of cardiovascular fitness as per Canadian Fitness Standards. First year psychology students were given three credit points for their participation.

Data Sampling and Reduction

**Vaginal Pulse Amplitude.** VPA was recorded during the entire neutral and erotic film sequences. The data were hand scored from the polygraph recordings. 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 seconds of the neutral or erotic film stimulus and dividing by the number of peaks per interval. Percentage amplitude change was computed for each experimental condition by subtracting the average VPA during the neutral film from the average VPA during the erotic film multiplied by 100. Mean VPA scores were used in the analyses of responses between stimulus presentations within conditions. Percentage amplitude change was used for all other statistical analyses. These statistical procedures are comparable with those of Morokoff and Heiman (1980).

**Vaginal Blood Volume.** VBV was sampled at a rate of 5 times/second during the last 80 seconds of neutral film, and during the entire 180 seconds of erotic stimuli (1,300 data points/subject/condition). The data were measured as 0.00001 mV units of VBV deviation from a baseline reference level defined as the mean of the last 80 seconds of the neutral stimulus. Scores +/- 2 sd. of each 10 second time block
mean were eliminated from the analyses. This procedure was used in order to control for potential movement artifacts. Mean VBV scores were used in the analyses of responses between stimulus presentations within conditions. Deviation scores, averaged across the entire 180 seconds of erotic stimuli (900 data points/subject/condition), were used for all other statistical analyses.

Heart Rate. Heart rate was scored from the VPA polygraph record by counting the number of beats per minute. The entire 180 seconds of neutral and 180 seconds of erotic film were scored to yield 20 measures (bpm) for each subject per experimental condition.

Results

Analyses of the Validity of Erotic Stimuli

In order to verify that the erotic films caused a significant increase in VBV responding, VBV responses averaged across the entire 180 seconds of erotic stimuli (no-exercise, Mean=.068; exercise, Mean=2.986) were compared to baseline VBV levels averaged across the last 80 seconds of neutral stimuli (no-exercise, Mean=-1.39; exercise Mean=.043). Analyses revealed that the erotic films caused a significant increase in VBV in both the no-exercise, t(24)=3.78, p=.001, and exercise conditions, t(24)=4.44, p<.001. Similar analyses were conducted with VPA scores. Mean VPA scores, averaged across the mid 20 seconds of neutral stimuli (no-exercise, Mean=10.609; exercise,
Mean=7.414) were compared with mean VPA scores averaged across the mid 20 seconds of erotic stimuli (no-exercise, Mean=16.133; exercise, Mean=17.536). Analyses revealed that the erotic films caused a significant increase in pulse amplitude in both the no-exercise, $t(24)=5.89, p<.001$, and exercise conditions, $t(24)=6.89, p<.001$. These results indicate that the experimental stimuli were effective in manipulating sexual arousal.

**Analyses of Order Effects**

Analyses were conducted on all physiological and subjective measures recorded during both the exercise and no-exercise sessions in order to determine whether the order of presentation of experimental conditions had influenced the results. The data indicate there were no significant differences attributable to the order of presentation (VPA no-exercise, $t(23)=.38, p=.711$; VPA exercise, $t(23)=.51, p=.613$; VBV no-exercise, $t(23)=.61, p=.546$; VBV exercise, $t(23)=.31, p=.757$; subjective sexual arousal (SSA) no-exercise, $t(23)=.35, p=.728$; SSA exercise, $t(23)=.95, p=.352$; autonomic arousal (AA) no-exercise, $t(23)=.43, p=.674$; AA exercise, $t(23)=.15, p=.882$; positive affect (PA) no-exercise, $t(23)=1.18, p=.249$; PA exercise $t(23)=.87, p=.394$; negative affect (NA) no-exercise $t(23)=2.60, p=.02$; NA exercise $t(23)=.54, p=.597$).

**Analyses of the Control for Fitness Levels**

In order to verify that variations in individual fitness levels did not influence levels of physiological
sexual responding during the exercise conditions, correlational analyses were calculated between fitness levels (as indicated by VO2 max scores) and mean VBV deviation scores, and between fitness levels and mean percentage change in pulse amplitude scores. There were no significant correlations between fitness levels and VPA responses $r(25)=.126, p=.274$, or between fitness levels and VBV responses $r(25)=.025, p=.452$. These results indicate that individual variations in physiological sexual responses cannot be attributed to variations in fitness levels.

**Analyses of Physiological Sexual Arousal**

**Vaginal Pulse Amplitude.** Percentage changes in VPA between erotic stimuli and neutral stimuli were compared between no-exercise and exercise conditions. Mean percentage change scores are presented in Fig. 1. Analyses revealed a significant increase in VPA during the exercise condition, $t(24)=4.56, p<.001$.

**Vaginal Blood Volume.** Deviation scores in VBV between erotic stimuli and neutral stimuli were compared between no-exercise and exercise conditions. Mean deviation scores are presented in Fig. 2. Analyses of these data showed a significant increase in VBV during the exercise condition, $t(24)=1.71, p=.05$ (one-tailed).

**Analyses of Heart Rate**

A 2x2x10 (Condition x Stimulus x Time) repeated measures ANOVA was computed to investigate the effects of exercise and erotic stimuli on heart rate. Results indicate
Figure 1. Mean percentage change in vaginal pulse amplitude (mm of pen deflection) ± S.E.M. between stimulus presentations during the No-Exercise (neutral-erotic) condition in experiment I, the Exercise (neutral-erotic) condition in experiment I, and the Exercise Control (neutral-neutral) condition in experiment II.
Figure 2. Mean vaginal blood volume (mV deviation from baseline averaged across 900 data points/subject) ± S.E.M. during the No-Exercise (neutral-erotic) condition in experiment I, the Exercise (neutral-erotic) condition in experiment I, and the Exercise Control (neutral-neutral) condition in experiment II.
a significant increase in heart rate during the exercise condition, $F(1, 24) = 54.09, p < .001$. No difference in heart rate was found between neutral and erotic films, $F(1, 24) = 3.03, p = .094$, or across time, $F(9, 216) = .85, p = .567$.

These results suggest that exercise, but not the erotic films, caused a significant change in sympathetic activation.

**Analyses of Subjective Measures**

**Subjective Sexual Arousal.** Ratings of sexual arousal, in response to erotic stimuli, were compared between the no-exercise and exercise conditions. Mean subjective ratings are presented in Fig.3. Analyses revealed no significant difference between the no-exercise and exercise conditions, $t(24) = 1.01, p = .320$.

These subjective ratings are not consistent with physiological measures of sexual arousal which indicate significantly higher levels of sexual arousal in response to erotic stimuli during the exercise condition.

**Subjective Autonomic Arousal.** Subjective perceptions of autonomic arousal in response to erotic stimuli were compared between the no-exercise and exercise conditions. Mean subjective ratings are presented in Fig.3. Analyses revealed no significant difference in subjective ratings of autonomic arousal, in response to erotic stimuli, between the no-exercise and exercise conditions, $t(24) = .84, p = .412$.

These subjective ratings parallel physiological
measures of heart rate which also indicate no change in autonomic arousal with the presentation of erotic stimuli.

**Affective Response.** Positive and negative affective responses to erotic stimuli were analyzed separately between the no-exercise and exercise conditions. Mean subjective ratings of positive and negative affect are presented in Fig. 3. There were no significant differences in positive affect, \( t(24) = .33, p = .741 \), or negative affect, \( t(24) = .42, p = .682 \) between the no-exercise and exercise conditions.

These findings imply that subjective ratings of both positive and negative affect in response to erotic stimuli were unaltered with exposure to exercise.

**Analyses of the Relationship Between VBV and VPA Responses**

In order to determine the relationship between VBV and VPA responses, Pearson product-moment correlation coefficients were calculated for both the exercise and no-exercise conditions. Analyses revealed no significant correlation between VBV and VPA during the no-exercise, \( r(25) = .242, p = .122 \), or exercise, \( r(25) = -.063, p = .383 \), conditions.

**Analyses of the Relationship Between Physiological and Subjective Responses**

Pearson product-moment correlation coefficients were calculated separately for each experimental condition in order to investigate the degree of association between physiological and subjective ratings of sexual arousal. There were no significant correlations between VPA and
Figure 3. Mean subjective ratings ± S.E.M. of subjective arousal, autonomic arousal, positive affect, and negative affect during No-Exercise (neutral-erotic) condition in experiment I, the Exercise (neutral-erotic) condition in experiment I, and the Exercise Control (neutral-neutral) condition in experiment II.
subjective ratings during the no-exercise, $r(25) = .018$, $p = .465$, or exercise $r(25) = -.033$, $p = .437$, conditions, or between VBV and subjective ratings during the no-exercise, $r(25) = -.09$, $p = .336$, or exercise, $r(25) = .19$, $p = .182$, conditions.

Discussion

The results reveal increases in both VPA and VBV with the presentation of erotic stimuli. This indicates that the experimental stimuli were successful in altering physiological sexual arousal. Analyses of heart rate showed significantly higher heart rate levels throughout the film presentations during the exercise condition. This indicates that cycling for 20 minutes at 70% of $V_{O2}$ max was sufficient to elicit significant sympathetic activity. The absence of a correlation between fitness levels and physiological measures of sexual arousal indicates that exercising subjects at relative, as opposed to absolute, work loads eliminated any potential confounds relating to variations in fitness levels. Analyses of order effects revealed that the order of presentation of experimental conditions did not influence subjective or physiological measures of sexual arousal. In other words, familiarity with the experimental procedures and/or with viewing an erotic film, did not influence subjects' responses. Clearly, the experimental manipulations and controls were effective.
The finding that both VPA and VBV responses to erotic stimuli were significantly higher during the exercise versus no-exercise condition provides strong support that acute exercise facilitates sexual arousal in women. Interestingly, VPA measures revealed a much stronger effect and less variability than VBV measures. Furthermore, results revealed a lack of correlation between subjects' VPA and VBV responses.

This is the first study to test the effects of acute exercise on VBV and VPA measures of sexual arousal hence, direct comparisons with previous research cannot be made. Comparisons can, however, be made between the results of the no-exercise condition and research which has examined the effects of erotic stimuli on VBV and VPA measures and, indirect comparisons can be made between the results of the exercise condition and research which has looked at the effects of anxiety on these measures. Data from the present study indicate a 62% increase in VPA with the presentation of erotic stimuli. This finding is consistent with previous research conducted in a nonclinical sample (Morokoff & Heiman, 1980) which showed a 63% increase in VPA with exposure to an erotic film. There have been no studies, to my knowledge, which have looked at VPA responses to anxiety stimuli. With respect to VBV measures, data from this study reveal an average 1.5 millivolts deviation from baseline with the presentation of erotic stimuli. This finding is lower than that reported by Palace and Gorzalka (1990), who
found an average 2.1 millivolts deviation from baseline using the same erotic stimuli. Data from the exercise condition revealed an average 2.9 millivolts deviation from baseline with the presentation of erotic stimuli. Again, the magnitude of this effect is somewhat lower than that reported by Palace and Gorzalka (1990) which indicated an average 3.4 millivolts deviation from baseline with the presentation of an anxiety followed by an erotic film. Based on the above comparisons, subjects used in the present study appear to be comparable to those used in previous research in their VPA responses to erotic stimuli. With respect to their VBV responses, it appears that they may have demonstrated atypically low levels for a nonclinical sample. If so, this may help to explain why VPA responses during the exercise versus no-exercise condition were significant at $p<.001$, while VBV responses to acute exercise were significant at $p=.05$ (one-tailed). However, it is also possible that the subjects used in the Palace and Gorzalka study (1990) demonstrated atypically high levels of VBV responses in which case comparisons with this study cannot be made.

A second possible reason why VPA measures showed a much stronger effect than VBV measures relates to methodological issues concerning the photoplethysmograph. Research indicates that the dc signal, used in the measurement of VBV, is more likely affected by temperature confounds than the ac signal used in the measurement of VPA
(Beck et al, 1983). It is possible that exercise caused fluctuations in body temperature which influenced the VBV but not the VPA response. Changes in body temperature may also help to explain the increase in VBV variability during the exercise condition.

The discrepancy between VPA and VBV responses is consistent with previous research that has shown a lack of correlation between these two measures (Zingheim & Sandman, 1978; Rosen & Beck, 1988). The reason for this discrepancy remains open to speculation. One possibility is that VPA is a more sensitive indicator of sexual arousal in women. This suggestion is consistent with assertions made by Geer et al. (1974), Heiman (1977), and Osborn and Pollack (1977). One must keep in mind, however, that it is questionable whether VPA and VBV responses can be reliably compared, given that they reflect different aspects of the vasocongestive response and are sampled and analysed differently.

Data indicate higher levels of physiological sexual arousal in response to erotic stimuli during the exercise versus no-exercise condition. Despite this finding, there were no differences in self-reported levels of arousal between these conditions. Furthermore, there was a lack of correlation between subjects' physiological and subjective ratings of sexual arousal. In other words, subjects were either unable to detect and/or unwilling to report higher levels of sexual arousal in response to erotic stimuli.
during the exercise versus no-exercise condition. This is consistent with other research that has demonstrated desynchrony between subjective and physiological components of the female sexual response (Heiman et al., 1991; Morokoff & Heiman, 1980; Palace & Gorzalka, 1990; 1992; Steinman et al., 1981; Wincze et al., 1976).

EXPERIMENT II
Method

Subjects

Prior to the debriefing in experiment I, subjects were asked if they were willing to take part in another experimental session. Ten of the 25 women who participated in experiment I agreed to take part (Mean age = 25 years, range = 19-34 years). Five of the subjects were graduate students in psychology, two were first or second year psychology students, and three were graduate or undergraduate students in disciplines other than psychology. Participation was strictly voluntary; no incentives were offered.

Apparatus and Materials

The film stimuli consisted of a one-minute display of the word relax; followed by a three-minute novel, neutral travelogue film; followed by a second, novel, three-minute neutral travelogue film.

All subjective and physiological measurements were identical to those used in experiment I.
Experimental Design

A within subject repeated measures design was used in which each subject viewed two neutral travelogue sequences.

Procedure

The procedure was identical to that used in the exercise condition in experiment I with the exception of the film stimulus which consisted of a one-minute display of the word relax, followed by two consecutive three-minute neutral travelogue sequences.

Data Sampling and Reduction

Data sampling and reduction procedures were identical to those used in experiment I.

Results

Analyses of Physiological Sexual Arousal

Vaginal Pulse Amplitude. VPA scores were averaged across the mid 20 seconds of each of the two neutral stimuli. No significant difference in VPA responses between the two neutral stimuli was revealed, t(9)=.22, p=.829.

Vaginal Blood Volume. VBV responses were averaged across the last 80 seconds of the first neutral travelogue sequence and compared to the mean VBV response averaged across the entire 180 seconds of the second travelogue sequence. Analyses revealed no significant difference in VBV responses between neutral stimuli, t(9)=.28, p=.788.
Analyses of Heart Rate

A one-way repeated measures ANOVA was computed to investigate the effects of film change and time on heart rate. Results indicated no difference in heart rate with the presentation of a novel stimulus $F(1, 9) = .02, p = .882$, or across time, $F(9, 81) = .82, p = .598$.

Analyses of the Validity of VPA Scores in Experiment I

Analyses were conducted in order to verify that the higher levels of VPA during the exercise condition (reported in experiment I) were not attributable to differences in baseline levels between the no-exercise and exercise conditions. A one-way repeated measures ANOVA was computed between average VPA scores during the neutral segments of the exercise and no-exercise conditions in experiment I and the first neutral sequence in experiment II (mean VPA scores: 7.28, 9.36, 8.90, respectively). Analyses revealed no significant difference in VPA between neutral films, $F(2, 18) = 1.3, p = .296$. This suggests that exercise per se had no effect on VPA during presentation of neutral films.

Analyses of the Validity of Physiological Measurements

Analyses of heart rate reveal that exercise caused a significant increase in autonomic arousal in both the exercise condition in experiment I, $F(1, 24) = 54.09, p < .001$, and the exercise condition in experiment II, $F(1, 9) = 12.76, p = .006$. Analyses of physiological sexual arousal indicate that both VPA, $t(24) = 6.89, p < .001$, and VBV, $t(24) = 4.44, p < .001$, were increased with the presentation of erotic
stimuli during the exercise condition in experiment I. Neither VPA, \( t(9) = .22, p = .829 \), nor VBV, \( t(9) = .28, p = .788 \) were increased with the presentation of the second neutral stimulus during the exercise condition in experiment II. In other words, exercise alone, without the presence of erotic stimuli, was sufficient to increase autonomic arousal but not sexual arousal. These data indicate that the photoplethysmograph differentiated physiological sexual arousal from generalized autonomic arousal.

**Analyses of Subjective Measures**

**Subjective Sexual Arousal.** Ratings of sexual arousal in response to erotic stimuli during the exercise condition in experiment I were compared to ratings of sexual arousal in response to neutral stimuli during the exercise condition in experiment II. Mean subjective ratings are presented in Fig.3. Analyses revealed that reports of sexual arousal in response to erotic stimuli were significantly higher than in response to neutral stimuli, \( t(9) = 4.20, p = .002 \).

These subjective reports are consistent with physiological measures which indicate higher levels of sexual response during the presentation of erotic versus neutral stimuli.

**Subjective Autonomic Arousal.** Subjective perceptions of autonomic arousal were compared between the two exercise conditions. Mean subjective ratings are presented in Fig.3. Subjects reported significantly higher levels of autonomic arousal in response to erotic stimuli in experiment I than
in response to neutral stimuli in experiment II, $t(9)=3.48$, $p=.007$.

These subjective ratings do not parallel physiological measures of heart rate which revealed no change in autonomic arousal with either the presentation of erotic stimuli in experiment I or the presentation of neutral stimuli in experiment II.

Affective Response. Positive and negative affective responses to film stimuli were analysed separately between the two exercise conditions. Mean subjective ratings of positive and negative affect are presented in Fig. 3. Subjects reported significantly higher levels of positive affect in response to the erotic stimuli in experiment I than in response to the neutral stimuli in experiment II, $t(9)=3.24$, $p=.01$. There were no significant differences in ratings of negative affect with the presentation of erotic versus neutral stimuli, $t(9)=.77$, $p=.46$.

These findings imply that subjective ratings of positive affect were increased with the presentation of erotic stimuli while subjective ratings of negative affect were unaltered.

Discussion

VPA and VBV responses remained unchanged with the presentation of the second neutral film. This finding tends to confirm that the increases in VPA and VBV responses to erotic stimuli (reported in experiment I) can be attributed
to the sexual content of the film. If, instead, the increases in VBV and VPA were simply a result of the passage of time or an orienting reflex resulting from the presentation of a novel stimulus, one would expect a change in VBV and VPA during presentation of the second neutral stimulus.

Exercise did not influence VPA responses to the neutral film segments in either experiment I or experiment II. This finding has several implications.

First, the fact that there were no significant differences in VPA responses to neutral stimuli across conditions rules out the possibility that the higher levels of VPA responses to erotic stimuli during the exercise condition in experiment I were attributable to different baseline measurements between exercise and no-exercise conditions. In other words, this finding helps to verify the VPA results reported in experiment I.

Second, the fact that exercise did not increase VPA responses to neutral stimuli indicates that the higher levels of VPA in response to erotic stimuli during the exercise condition represent a purely sexual response. If the higher levels of VPA were attributable to other "non sexual" cardiovascular or hormonal responses to exercise, one would also expect higher levels of VPA in response to neutral stimuli during the exercise versus no-exercise conditions.
Lastly, and most importantly, the fact that VPA was not increased during the presentation of neutral stimuli indicates that in the absence of erotic stimuli, exercise did not increase VPA. In other words, sympathetic activation alone does not enhance physiological sexual arousal. This finding illustrates the complex interplay between cognitive and physiological components of the sexual response.

The finding that subjects reported higher levels of sexual arousal with exposure to exercise in experiment I than in experiment II indicates that subjects were able to detect and/or willing to report increases in sexual arousal with exposure to erotic stimuli. In contrast to experiment I, the subjects' reports of sexual arousal were in synchrony with their physiological responses.

GENERAL DISCUSSION

The results of this study indicate that activation of the sympathetic nervous system via physical activity enhances physiological sexual arousal in women. These effects include an increase in both vaginal pulse amplitude and vaginal blood volume. Despite increased physiological sexual responses, exercise did not alter subjective reports of sexual arousal. These findings hold both theoretical and practical implications.
Anxiety Enhances Sexual Arousal via Sympathetic Activation

The findings from this study provide a possible explanation for the effects of anxiety upon female sexual arousal. Several studies in both men and women have demonstrated that anxiety increases physiological sexual arousal (e.g. Hoon et al., 1977; Palace & Gorzalka, 1990). In these studies, anxiety was induced by exposing subjects to stimuli such as shock threats or films depicting events such as threatened amputation. In addition to enhancing sympathetic activity, these stimuli undoubtedly altered cognitions. Thus, it is difficult to determine to what extent the findings were attributable to physiological, versus cognitive, factors. In the present study, the physiological component of anxiety was simulated, using acute exercise, without altering cognitions. The assertion that cognitions were not altered with exposure to exercise is supported by the fact that no differences were reported between subjective ratings of positive affect, negative affect, autonomic arousal, or sexual arousal between the exercise and no-exercise conditions. Results indicated that the physiological component of anxiety alone (i.e. increased sympathetic activity) is sufficient to enhance sexual arousal. This finding supports the notion that reported increases in physiological sexual arousal in women following exposure to anxiety-evoking stimuli may be attributable to physiological as opposed to cognitive factors. Anxiety
stimuli serve to activate the sympathetic nervous system which, in turn, enhances sexual arousal.

**Sympathetic Activation and Sexual Arousal are not Mutually Inhibitory**

The finding that sympathetic activation enhanced physiological sexual arousal challenges the theory that sympathetic activation and sexual arousal are mutually inhibitory. The present data indicate that heart rate was significantly increased in the exercise condition and that this increase remained unchanged throughout the entire 180 seconds of erotic exposure. These data verify that the effects of sympathetic activation and erotic stimuli were presented simultaneously, as opposed to sequentially as in previous studies. Hence, this study provided an adequate test of the reciprocal inhibition hypothesis set forth by Wolpe (1958).

If sympathetic activity and sexual arousal are mutually inhibitory, one would expect either a decrease in sexual arousal during the exercise condition or, alternatively, a decrease in heart rate during the erotic exposure. Neither of these outcomes occurred. Increased sympathetic activity facilitated physiological sexual arousal, and increased physiological sexual arousal did not alter sympathetic activity. Perhaps, then, anxiety and sexual arousal are mutually inhibitory at a cognitive level only or, at a physiological level but only when accompanied by anxiety cognitions. Perhaps sympathetic activation can be
considered an "engine" that drives the cognitions to a behavioral response. If anxiety cognitions are in play, then sympathetic activation provides the force to decrease sexual arousal. In this regard, sympathetic activation and sexual arousal are mutually inhibitory. On the other hand, if sexual cognitions are in play, sympathetic activation is the force behind increasing sexual arousal. In this sense, sympathetic activation and sexual arousal are mutually facilitatory. In addition, if Wolpe is correct in his assertion that sympathetic and parasympathetic nervous system functions are mutually inhibitory, then perhaps female sexual arousal, at least in the initial stage, is largely under the influence of the sympathetic rather than the parasympathetic nervous system as has long been assumed.

Subjective and Physiological Components of the Female Sexual Response are in Synchrony

The finding that increased physiological sexual arousal during the exercise condition was not accompanied by subjective reports of increased sexual arousal is consistent with previous research which shows a lack of concordance between these two measures in women (Palace & Gorzalka, 1990; 1992; Morokoff & Heiman, 1980; Steinman et al, 1981; Wincze et al., 1976). The majority of research done in the male indicates a high correlation between physiological and subjective components of the sexual response (Heiman & Rowland, 1983; Rosen & Beck, 1988; Steinman et al., 1981).
However, recent research indicates that this correlation is by no means perfect (Bozman & Beck, 1991).

Authors have explained the lack of concordance between physiological and cognitive components of the female sexual response in terms of a potential desynchronous relationship (Palace & Gorzalka, 1990). Heiman (1977) suggested that this desynchrony may be attributable to a less direct feedback system (erection vs. vasocongestion) in the female, thus allowing bodily cues to be more easily ignored. Data from this study do not entirely support this explanation. Subjects reported significantly higher levels of sexual arousal in response to erotic versus neutral stimuli. Physiological measures also indicated higher levels of sexual arousal in response to erotic versus neutral stimuli. In this sense, subjective measures of sexual arousal were in synchrony with physiological responses.

With respect to the finding in experiment I, whereby higher levels of physiological arousal in the exercise condition were not accompanied by higher ratings of subjective sexual arousal, three potential explanations are offered. First, it is possible that the subjective tests employed were able to detect changes from a "non-sexual state" to a sexually aroused state but were not sufficiently sensitive to detect more subtle differences between a sexually aroused state and a "more" sexually aroused state. This explanation is consistent with research which indicates a lower concordance between subjective and physiological
components of the sexual response during low versus high levels of sexual arousal (Heiman, 1976). One must also keep in mind that this is an analogue laboratory setting. Perhaps one would need natural sexual interactions to achieve much higher, "detectable" levels of sexual arousal.

Second, it is possible that subjects were willing to report a certain level of sexual arousal but were not willing to report intense levels of sexual arousal. In other words, social dictates allowed subjects to report being sexually aroused but not "too aroused". The findings from this study provide partial support for this explanation. Subjects reported significantly higher levels of autonomic arousal in response to erotic stimuli than in response to neutral stimuli. This is surprising given that physiological heart rate measures indicated there were no changes in autonomic arousal with the presentation of either neutral or erotic stimuli. It is tempting to speculate that the subjects reported increases in sexual arousal as increases in autonomic arousal. Perhaps they felt more comfortable describing their sexually aroused state using descriptives such as "any physical reaction at all" and "faster heart beat" versus descriptives such as "genital wetness and lubrication" and "genital pulsing and throbbing". This explanation cannot, however, fully account for the findings from this study. Consistent with this assertion, subjects reported higher levels of autonomic arousal during the exercise versus no-exercise condition;
however, these results did not reach statistical significance.

A third potential explanation for the lack of concordance between physiological and subjective measures of sexual arousal relates to the possibility of a ceiling effect on subjective ratings of sexual arousal. Mean ratings of subjective sexual arousal during the no-exercise and exercise conditions were 4.2 and 3.8, respectively. These results are comparable to a study conducted by Palace and Gorzalka (1990) which used the same subjective rating scale. Palace and Gorzalka (1990) reported mean subjective ratings of 4.5 and 3.8 with exposure to a neutral-erotic and an anxiety-erotic film, respectively. Together, these findings indicate that, on a scale of 1 to 7, 4 may be the highest level of arousal that subjects can attain viewing an erotic film in a laboratory setting.

To test the possibility of a ceiling effect on subjective sexual arousal, a post-hoc questionnaire was distributed to subjects (see appendix). The questionnaire asked (on a scale of 1 to 7) "What is the highest level of sexual arousal you think that you could experience viewing an erotic film alone?". Ten of 25 questionnaires were returned. The mean rating of expected sexual arousal was 4.2. The reported level of sexual arousal for these ten subjects during the exercise condition was 4.0. These findings support the notion that reported levels of sexual
arousal were possibly as high as could be expected in a laboratory setting. 

With respect to studies that have shown discordance between physiological and subjective reports of sexual arousal in women, I propose that this is attributable to methodological and social factors as opposed to a desynchrony between cognitive and physiological components of the sexual response. Experimental manipulation of sexual arousal using an erotic film is sufficient to increase sexual arousal both physiologically and cognitively. However, the degree to which either of these components can be raised in a laboratory setting differs. An erotic film increases physiological sexual arousal and further increases are induced with exposure to stimuli that increase sympathetic activity such as an anxiety-evoking film or acute exercise. In other words, stimuli have been added to further "boost" the physiological component. Furthermore, given the precision of the physiological measurement devices, detection of even minute changes in arousal is possible. With respect to cognitions however, an erotic film in a laboratory setting can raise subjective sexual arousal but a second erotic film in the same laboratory setting does not further increase arousal. In other words, nothing has been added to "boost" the cognitive component; subjects' "cognitive" level of sexual arousal has already been raised as high as an erotic film will allow. Furthermore, given the comparatively unsophisticated
measures used and social demands which may limit the
subjects' willingness to report being sexually aroused, it
is likely that only large changes in subjective sexual
arousal would be detected.

**Implications**

The findings from this study have broad implications
for deriving an etiological theory of sexual arousal. For
over 30 years, it has been assumed that female sexual
arousal is largely under the influence of the
parasympathetic nervous system and that sympathetic
influences play an inhibitory role. This study demonstrated
that sympathetic activation may enhance physiological sexual
arousal in women.

The mechanism by which sympathetic activation enhances
sexual arousal remains undetermined. Basic physiology
indicates that signals from the central nervous system are
sent along vascular pathways to the pituitary gland and
along sympathetic pathways to the adrenal medulla. In turn,
the pituitary gland secretes a variety of hormones, many of
which act upon other target glands, and the adrenal medulla
signals the secretion of catecholamines (Bunt, 1986).

Physical exercise at the intensity induced in the
present study has been documented to increase cortisol
secretion secondary to ACTH release (Carr et al., 1981); to
increase serum levels of prolactin and growth hormone
(Sutton & Lazurus, 1976); to increase (Guglielmini et al.,
1984), decrease (Kuusi et al., 1984), or not change (Bunt,
1985) testosterone levels; to increase (Bonen et al., 1979), or not change (Loucks & Horvath, 1984) estrogen and progesterone responses; to markedly increase circulating levels of norepinephrine and epinephrine (Hartley et al., 1972); to increase circulating levels of dopamine (Grossman & Moretti, 1986), and to trigger a rise in plasma levels of beta-endorphin (Gambert et al., 1981; Farrel et al., 1982).

Conceivably, any number of the above alterations could be responsible for influencing the sexual response. For example, numerous drugs known to affect monoaminergic systems have been reported to influence the sexual response (for review see Meston & Gorzalka, 1992). Both acute and chronic administration of opioids have been reported to inhibit sexual behavior in humans and animals (for review see Pfaus & Gorzalka, 1987). With respect to hormonal responses to sympathetic activation, research indicates that changes in hormones such as luteinizing hormone, testosterone, and cortisol sometimes facilitate the sexual response in males (Stearns et al., 1973; Pirke et al., 1974; LaFerla et al., 1978; Rowland et al., 1987). In women, the possibility that hormonal responses facilitate sexual arousal has been only minimally explored. Hoon and associates (1982) and Meuwissen and Over (1992) reported that sexual arousability to erotic stimuli is unaffected by menstrual cycle influences. This is consistent with evidence indicating that estrogen and progesterone may not be important in altering sexual arousal (Rosen & Beck,
Heiman and associates (1991) reported that exposure to an erotic film did not significantly alter levels of cortisol, prolactin, luteinizing hormone, or testosterone. The above studies argue against the hormonal control of sexual arousal in women, at least in the short term. It is possible, however, that hormonal influences on sexual arousal in women occur via the release of epinephrine and/or norepinephrine. Indirect support for this hypothesis is provided by a classic study by Schacter and Singer (1962). Subjects were injected with epinephrine and either informed or misinformed of the side effects they would experience. Subjects who were misinformed of the side effects and, hence, did not know that the increase in arousal (i.e., increased heart rate, rapid breathing) was attributable to the drug, looked to their environment for cues about their emotions. For example, subjects exposed to a confederate who acted in an angry manner, supposedly in response to the drug, also acted in an angry manner. In contrast, subjects who knew that the drug was responsible for their arousal did not acquire the mood of the confederate. This study highlights the possibility that, given the appropriate cues for interpretation, epinephrine may alter both cognitive and physiological states of arousal. Lange and associates (1981) directly tested the possibility that epinephrine alters sexual arousal in men. Epinephrine injections, designed to artificially increase sympathetic activity, facilitated the loss of erection, but only after, and not
during, the sexual stimulus. The effects of epinephrine on female sexual arousal have not been explored.

In sum, the manner in which sympathetic activation enhances sexual arousal remains open to speculation. Based on the above studies, it seems unlikely that the increases in sexual arousal reported in the present study are attributable to hormonal changes. A more likely possibility is that the sympathetic nervous system activates sexual arousal through a neural pathway or a nonhormonal system. For example, an increase in vasocongestion may be the direct result of sympathetic activation of the circulatory system.

The finding that sympathetic activation enhances sexual arousal by no means implies that parasympathetic influences are not important in sexual arousal. Furthermore, it does not mean that sexual arousal is dominated by sympathetic activity. Rather, the findings from this study point to the possibility that both sympathetic and parasympathetic influences are integral to the sexual response, and neither should be considered dominant. It is possible that during the initial stages of sexual arousal, an increase in sympathetic activity prepares the individual for sexual arousal or provides a "boost" to already increasing levels of arousal.

Future research is necessary to determine the precise role of sympathetic and parasympathetic influences on female sexual arousal. It would be interesting to compare the effects of deep relaxation exercises versus acute physical
exercise on sexual arousal. This would provide a more
direct indication of the relative effects of parasympathetic
versus sympathetic dominance on sexual arousal.

The finding that sympathetic activation without the
presence of erotic stimuli did not increase sexual arousal,
emphasizes that sexual arousal cannot be viewed as arising
from a state of generalized arousal. In other words,
physiological changes alone do not sufficiently define the
sexual response. The results of this study highlight the
critical role of cognitive labeling and subjective
experience in interpreting an aroused state as sexual.

The findings from this study also have implications for
the treatment of sexual dysfunction. Since the early 1960s,
little has changed in the treatment of sexual disorders.
The majority of sex therapy techniques, such as systematic
desensitization, continue to focus on anxiety reduction
techniques which decrease sympathetic activity. The
effectiveness of these techniques is mixed. Several studies
report the effectiveness of systematic desensitization in
reducing coital anxiety and improving pleasure during
intercourse (see Norton & Jehu, 1984 for review), while
others indicate that systematic desensitization is
ineffective in treating anorgasmia (see Norton & Jehu, 1984
for review). Perhaps systematic desensitization is
effective in treating sexual desire disorders or disorders
in which cognitive factors play a primary role. Perhaps it
is ineffective in treating sexual disorders in which
physiological processes constitute the primary component. Such may be the case with anorgasmia.

This study focused only on sexually functional women. Future research is necessary to determine whether sympathetic activation enhances sexual arousal in sexually dysfunctional women. If this is the case, then treatments for sexual dysfunction need to be re-evaluated. Techniques need to focus not only on altering a negative cognitive set, but must also give further consideration to the role of sympathetic activity in enhancing the physiological response.
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APPENDIX

Film Scale 73
Physical Readiness Exam for Fitness Test 75
Arousal Scale 76
FILM SCALE

Instructions: Please use the following scale to evaluate how you felt during the last film. Please answer honestly and carefully. On the scale, circle any of the numbers from 1 (not at all) to 7 (intensely).

During the film, I felt:

1. Faster breathing__________ 1 2 3 4 5 6 7
2. Faster heart beat__________ 1 2 3 4 5 6 7
3. Perspiration_______________ 1 2 3 4 5 6 7
4. Feelings of warmth__________ 1 2 3 4 5 6 7
5. Any physical reaction at all______ 1 2 3 4 5 6 7

Continue on to the next page.
Instructions: Please use the following scale to evaluate how you felt during the last film. Please answer honestly and carefully. On the scale, circle any of the numbers from 1 (not at all) to 7 (intensely).

During the film, I felt:

6. Breast sensations
7. Warmth in genitals
8. Genital wetness or lubrication
9. Genital pulsing or throbbing
10. Any genital feelings
11. Sexually aroused
12. Worried
13. Anxious
14. Angry
15. Disgusted
16. Embarrassed
17. Guilty
18. Sensuous
19. A desire to be close to someone
20. Pleasure
21. Interested
22. Attracted
23. Excited
24. Sexy
25. Dirty
26. Loving
27. Sexually attractive
28. Inhibited
29. Easy to arouse
30. Incompetent
31. Sexually turned off
32. Offended
33. Bored
34. Feminine

Stop and wait for further instructions.
For most of us, physical activity poses no problem or hazard. This test has been designed to identify that small number of individuals who should seek medical advice prior to participating in a fitness test.

YES NO

Has your doctor ever said you have heart trouble?

Do you often have pains in your heart or chest?

Do you often feel faint or have spells of severe dizziness?

Has a doctor ever said your blood pressure was too high or too low?

Has your doctor ever told you that you have a bone or joint problem that has been aggravated by exercise, or might be made worse with exercise?

Do you have any menstrual period problems?

Do you have problems with recurrent itching or discharge?

Are you currently using any prescription medications?

If yes, what are they?

Are you using any recreational drugs?

Have you ever smoked?

If you currently smoke, how many cigarettes per day?

If you have quit smoking, when was it?

Do you drink coffee?

If yes, how many cups per day?

Are you currently involved in a regular exercise program?

Do you regularly walk or run one or more miles continuously?

If yes, average no. of miles per day

What is your average time per mile?

Do you practice weight lifting or home calisthenics?

Are you involved in an aerobics program?

If yes, how frequently?

Do you frequently participate in competitive sports?

If yes, how frequently?

Have you suffered any serious medical problems that you consider important for us to know?

Is there a good physical reason not mentioned here why you should not receive a fitness test?

Signature
Instructions: Please answer the following questions using the scale provided. Circle any of the numbers from 1 (low) to 7 (intense). Please answer honestly and carefully.

1. Given the appropriate situation, what is the highest level of sexual arousal you think that you could experience?
   1 2 3 4 5 6 7

2. What is the highest level of sexual arousal you have ever experienced?
   1 2 3 4 5 6 7

3. Prior to the films used in this research, had you ever viewed an erotic film?
   Yes    No

4. What is the highest level of sexual arousal you think that you could experience viewing an erotic film alone?
   1 2 3 4 5 6 7