

# Cardiovascular and Endocrine Alterations After Masturbation-Induced Orgasm in Women

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**Objective:** The present study investigated the cardiovascular, genital, and endocrine changes in women after masturbation-induced orgasm because the neuroendocrine response to sexual arousal in humans is equivocal. **Methods:** Healthy women ( $N = 10$ ) completed an experimental session, in which a documentary film was observed for 20 minutes, followed by a pornographic film for 20 minutes, and another documentary for an additional 20 minutes. Subjects also participated in a control session, in which participants watched a documentary film for 60 minutes. After subjects had watched the pornographic film for 10 minutes in the experimental session, they were asked to masturbate until orgasm. Cardiovascular (heart rate and blood pressure) and genital (vaginal pulse amplitude) parameters were monitored continuously throughout testing. Furthermore, blood was drawn continuously for analysis of plasma concentrations of adrenaline, noradrenaline, cortisol, prolactin, luteinizing hormone (LH),  $\beta$ -endorphin, follicle-stimulating hormone (FSH), testosterone, progesterone, and estradiol. **Results:** Orgasm induced elevations in cardiovascular parameters and levels of plasma adrenaline and noradrenaline. Plasma prolactin substantially increased after orgasm, remained elevated over the remainder of the session, and was still raised 60 minutes after sexual arousal. In addition, sexual arousal also produced small increases in plasma LH and testosterone concentrations. In contrast, plasma concentrations of cortisol, FSH,  $\beta$ -endorphin, progesterone, and estradiol were unaffected by orgasm. **Conclusions:** Sexual arousal and orgasm produce a distinct pattern of neuroendocrine alterations in women, primarily inducing a long-lasting elevation in plasma prolactin concentrations. These results concur with those observed in men, suggesting that prolactin is an endocrine marker of sexual arousal and orgasm. **Key words:** sexual arousal, masturbation, orgasm, cardiovascular measures, catecholamines, prolactin.

LH = luteinizing hormone; FSH = follicle-stimulating hormone; VPA = vaginal pulse amplitude.

## INTRODUCTION

A definitive model of the endocrine response to sexual arousal has yet to be established. Nevertheless, evidence from animal studies clearly demonstrates a modulatory role of hormones in sexual behavior. Sexual stimulation or behavior increases secretion of such hormones as testosterone (1–3), oxytocin (4), adrenal steroids (5, 6), and prolactin (1, 6, 7). Furthermore, neuroendocrine regulation is responsible for complex sexual behavior, such as monogamy (8), mate selection (9), and sexual preference (10). Indeed, it is apparent from animal models that hormone/neuropeptide/neurotransmitter combinations provide stimulatory and inhibitory control of sexual behavior (11, 12).

However, the findings to date of experiments conducted in humans are equivocal; thus, knowledge in

this area is far less advanced than our knowledge about animals (13). Although sexual arousal reliably stimulates cardiovascular responses (14–17), the effect on sympathetic adrenal hormones is inconsistent. For example, plasma adrenaline and urinary adrenaline and noradrenaline levels have been found to increase as a result of viewing a sexually arousing film (18, 19); however, this effect was not replicable in a subsequent investigation (20). Concentrations of plasma cortisol have been demonstrated to increase after masturbation (21) and were positively correlated with film-induced sexual arousal (22). Nevertheless, other studies have shown that cortisol remains unchanged after sexual stimulation (20, 23).

Discrepant results have also been found between studies examining pituitary hormones, such as  $\beta$ -endorphin, prolactin, LH, and FSH. Although increased plasma concentrations of LH and decreased concentrations of prolactin have been reported to follow sexual arousal (23–25), the concentrations of LH, FSH, growth hormone, prolactin, and  $\beta$ -endorphin have been found to remain unchanged after film-induced sexual arousal, masturbation, or coitus (20–22, 25–27).

Similar unclear patterns emerge when gonadal hormones are tested. In particular, testosterone has been shown to increase (21, 24, 25, 27, 28) or remain unchanged (20, 23) in response to visual sexual stimulation. Furthermore, plasma testosterone concentrations have been shown to increase after coitus in a single subject (29), but a subsequent study failed to replicate this finding (26).

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It is likely that a number of methodological differences contribute to the contrasting results, making general interpretation difficult. A number of methods have been used to induce sexual arousal, such as viewing of stimulating films, imagery of fantasies, masturbation, and coitus. The differences between such studies are further compounded because some studies requested that participants achieve orgasm whereas others did not. In addition, the method of collecting blood, which is often taken at single discrete time points, may not efficiently detect the short-term alterations of certain neuroendocrine variables.

Therefore, we designed a method to examine the neuroendocrine response to masturbation-induced orgasm in men (30) based on a continuous blood sampling technique that we previously established (31–33). This investigation demonstrated that masturbation-induced orgasm produced a pronounced increase in cardiovascular responses and plasma catecholamine concentrations. Furthermore, sexual arousal was characterized by a large, persisting increase in plasma prolactin concentrations (30). Therefore, the present study replicated our methodology in women to elucidate whether changes in neuroendocrine activity may be used as a universal marker of human sexual arousal in healthy individuals independent of gender.

### Methods

#### Subjects

Ten healthy female volunteers were recruited through an advertisement at the Hannover Medical School. All volunteers underwent an intensive nonstructured clinical interview to exclude those with confounding physical or mental health problems. Subjects with drug or alcohol abuse, medication intake, or sexual dysfunction were also excluded. Table 1 describes the demographic characteristics of participants. Subjects had an average body mass index and a regular menstrual cycle. All participants were exclusively heterosexual and reported a relaxed attitude toward masturbation and pornography. Volunteers were paid 100 Deutsche marks for participation in the study. All experimental procedures were approved by the ethics committee for investigation using human subjects of the Hannover Medical School.

**TABLE 1. Demographic Characteristics (mean  $\pm$  SD) of Participants (N = 10)**

Age (yr)	24.8 $\pm$ 2.3
Height (cm)	168 $\pm$ 5.1
Weight (kg)	60.2 $\pm$ 8.2
Body mass index (kg/m <sup>2</sup> )	21.2 $\pm$ 2.9
Menstrual cycle duration (days)	28.2 $\pm$ 2.4
Menstruation duration (days)	4.7 $\pm$ 1.0

### Experimental Paradigm

Subjects were initially informed about the procedure of the study. Subsequently, they were scheduled to be studied during the midfollicular phase of their menstrual cycle because of the evidence suggesting that sexual interest is highest at this time (34–36) and that a stable profile of gonadal hormones occurs during this period. Volunteers were then asked to refrain from any kind of sexual activity and to avoid alcoholic beverages or other drugs 24 hours before the laboratory investigation. The study paradigm is shown in Figure 1. A crossover design was used, involving two sessions on consecutive days. Each session began at 1500 hours. Subjects laid on a comfortable bed in front of a video screen, with the head propped by pillows to allow viewing of the video. In the control session, volunteers viewed a neutral documentary video about the culture of Nepal for 60 minutes. The video for the experimental session was composed of three sequences, each lasting 20 minutes. The first and last sequences consisted of the same documentary film. The middle sequence was a pornographic film showing different heterosexual couples having sexual intercourse. The order of presentation was balanced, with five subjects watching the experimental film on the first day and five on the second. Continuous blood withdrawal and cardiovascular monitoring allowed the subject to remain in a separate room as the investigators without being interrupted during the session. Blood sampling was initiated at the start of each film. Blood was drawn continuously, with the samples divided into six 10-minute intervals. After viewing the pornographic video for 10 minutes (anticipatory phase), subjects in the experimental session were asked to masturbate until orgasm.

### Continuous Blood Sampling and Endocrine Assessment

A small portable pump (Fresenius, Hamburg, Germany) allowed accurate, noninvasive monitoring of plasma hormone concentrations (30–32). An intravenous cannula (Vasofix Branüle 18G, Mel-sungen, Germany) was inserted into a brachial vein 30 minutes before the start of each session. The catheter was connected to a heparinized silicon tubing (1 mm  $\phi$ , Reichelt Chemie, Heidelberg, Germany) that passed through the test room wall into the adjoining room. Blood was collected into ethylenediamine tetraacetic acid tubes (Sarstedt, Nümbrecht, Germany). Blood flow was adjusted to 1 ml/min. Tubes were changed every 10 minutes to allow a time kinetic of endocrine variables (33). Samples were centrifuged at 4°C and stored at  $-70^{\circ}\text{C}$  until assay.

All samples of each subject for each particular hormone were examined in the same assay. Plasma catecholamine concentrations were measured by high-performance liquid chromatography as previously described (37). Plasma testosterone levels were analyzed by radioimmunoassay (ICN Biomedicals, Eschwege, Germany). All other hormones were determined using commercially available radioimmunometric, immunoradiometric, or chemoluminescence immunometric assays: FSH (MAIAclone, Biodata Diagnostics, Rome, Italy); LH (LH-CTK, Sorin Biomedica, Saluggia, Italy); prolactin (MAIAclone);  $\beta$ -endorphin (radioisotopic assay, Nichols Institute Diagnostics, San Juan Capistrano, CA); progesterone (CoatRIA, BioMérieux, Lyon, France); estradiol (CoatRIA 2); cortisol (Coat-A-Count RIA, DPC Diagnostics, Los Angeles, CA). The FSH, LH, and cortisol assays had inter- and intraassay variation of  $<5\%$ , and the variation of the testosterone, prolactin, and  $\beta$ -endorphin assays was  $<10\%$  (30).

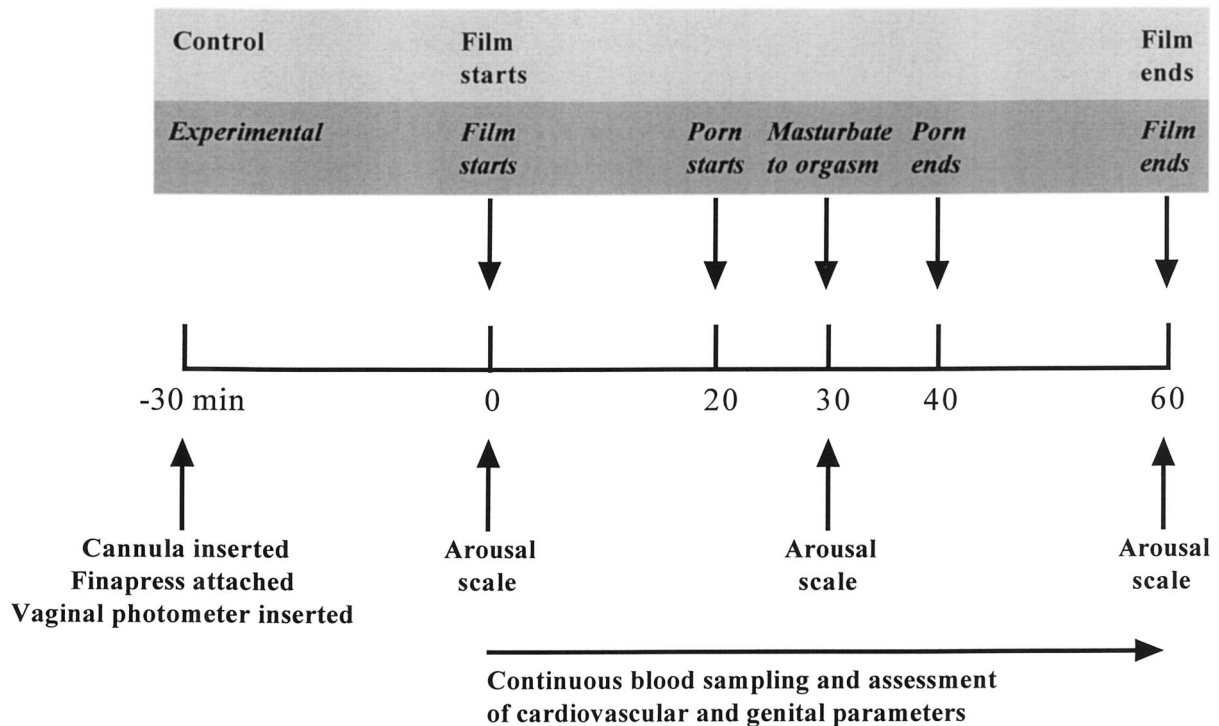


Fig. 1. The experimental paradigm consisted of an experimental and a control session, each lasting 60 minutes. Each subject completed both sessions, with endocrine, genital, and cardiovascular parameters monitored continuously throughout each session.

### Cardiovascular Monitoring

Heart rate and systolic and diastolic blood pressure were measured continuously in parallel with the continuous blood sampling, by means of a finger cuff connected to a blood pressure monitor (Finapres, Ohmeda, Louisville, KY). The cables of the finger cuff also passed through the wall, with the monitor connected to a personal computer in the adjoining room. Sampling of cardiovascular parameters occurred every 30 seconds, with the data averaged over the 10-minute intervals for statistical analysis and direct comparison to the 10-minute blood samples (30, 33).

### Physiological Assessment of Sexual Arousal

A vaginal photoplethysmograph (38) was used to monitor physiological sexual arousal. When placed in the vagina, the device shines a light onto the vaginal lumen, and a photosensitive receptor monitors the amount of reflected light. The photometer thus measures changes in vaginal vasocongestion, because changes in the vascular bed of the vaginal wall alter the degree of light that is reflected. The AC signal, measuring VPA, monitors the pulse wave of vaginal blood with each heartbeat. This parameter has been shown to be a reliable physiological indicator of female sexual arousal (39–41). The tampon-shaped device (1.5 × 6 cm) was inserted vaginally by the subject before each session. VPA was recorded continuously throughout each session, via an amplifier and pen-and-paper polygraph (SP300, Farrall Instruments, San Diego, CA). Data were scored using standard methodology (42–44). The continuous VPA paper polygraph was divided into six 10-minute intervals, corresponding to the analysis of endocrine and cardiovascular parameters. Within each 10-minute interval, the VPA was determined at every 20-second time point. Using a ruler, the distance of pen deflection (mm) resulting from a single pulse wave was

measured on the polygraph. For each 20-second time point, the average amplitude (mm) of three consecutive pulse waves was determined (to eliminate error caused by random variation of the VPA). The scores from each 20-second probe were then averaged for each of the six 10-minute periods, allowing direct comparison to the endocrine and cardiovascular data. The device was disinfected according to the standardized procedure of the manufacturer.

### Subjective Assessment of Sexual Arousal

Subjects rated the grade of arousal and excitement before, during (after 30 minutes of film), and immediately after each session using visual analogue rating scales (30, 45). Additionally, volunteers were asked to rate three qualities of the orgasm itself (duration, speed, and intensity), as well as to compare it with previous orgasms, using a five-point rating scale. The perceived arousability of the pornographic video was also assessed using a six-point rating scale.

### Statistical Analysis

Data were analyzed using two-factor (condition × time) repeated-measures analysis of variance. Post hoc differences between groups were examined using Tukey's test for multiple comparisons. All results of analysis of variance refer to the condition × time interaction unless otherwise stated.

### Results

#### Quality of Erotic Film and Masturbation

The pornographic film was judged to be sexually arousing (mean = 4.4 ± 0.47). The duration, speed,

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and intensity of orgasm were considered to be average ( $2.8 \pm 0.20$ ,  $3.0 \pm 0.29$ , and  $3.1 \pm 0.27$ , respectively) as well as average compared with past orgasms ( $2.8 \pm 0.29$ ,  $2.9 \pm 0.34$ , and  $3.0 \pm 0.29$ , respectively).

### Subjective and Physiological Sexual Arousal

The film and masturbation also produced a significant increase in subjective arousal ( $F(2,18) = 29.19$ ,  $p < .001$ ), which returned to control levels immediately after the session (Figure 2, *a*). Furthermore, orgasm produced a marked increase in physiological sexual arousal. VPA was raised significantly by the film and masturbation ( $F(5,45) = 57.79$ ,  $p < .0001$ ) and, in contrast to subjective arousal, remained significantly elevated for the remainder of the session (Figure 2, *b*).

### Cardiovascular Parameters

Cardiovascular activity was increased by sexual arousal and orgasm. The arousal procedure produced significant increases in heart rate ( $F(5,45) = 8.06$ ,  $p <$

$.001$ ; Figure 3, *a*), systolic blood pressure ( $F(5,45) = 5.59$ ,  $p < .01$ ; Figure 3, *b*), and diastolic blood pressure ( $F(5,45) = 3.43$ ,  $p < .05$ ; Figure 3, *c*).

### Sympathetic Adrenal Hormones

The basal level of adrenaline was nonsignificantly higher during the experimental session than during the control session (Figure 4, *a*). However, arousal increased plasma adrenaline to levels significantly

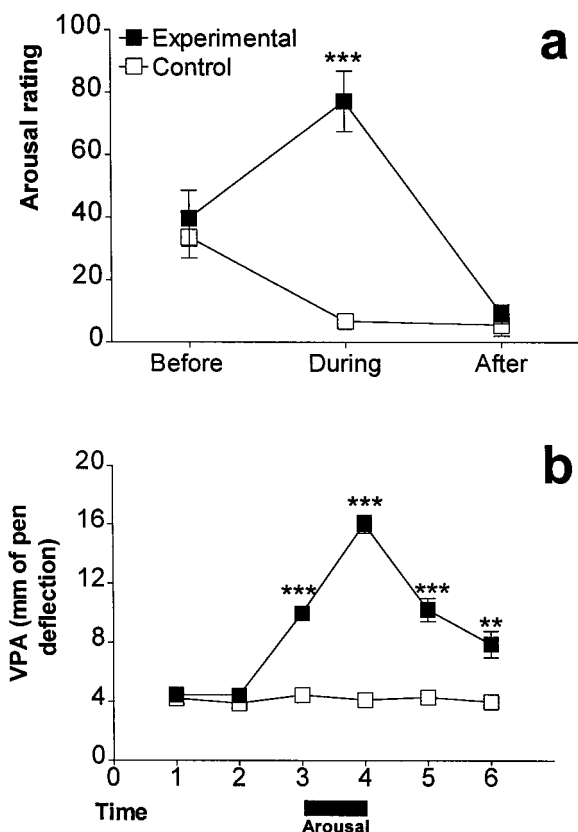


Fig. 2. Subjective arousal rating (before, during, and after experiment) (*a*) and VPA (mm of pen deflection) (*b*), analyzed in 10-minute intervals for the experimental and control conditions. Data are mean  $\pm$  SE. \*\*\* $p < .001$ ; \*\* $p < .01$ .

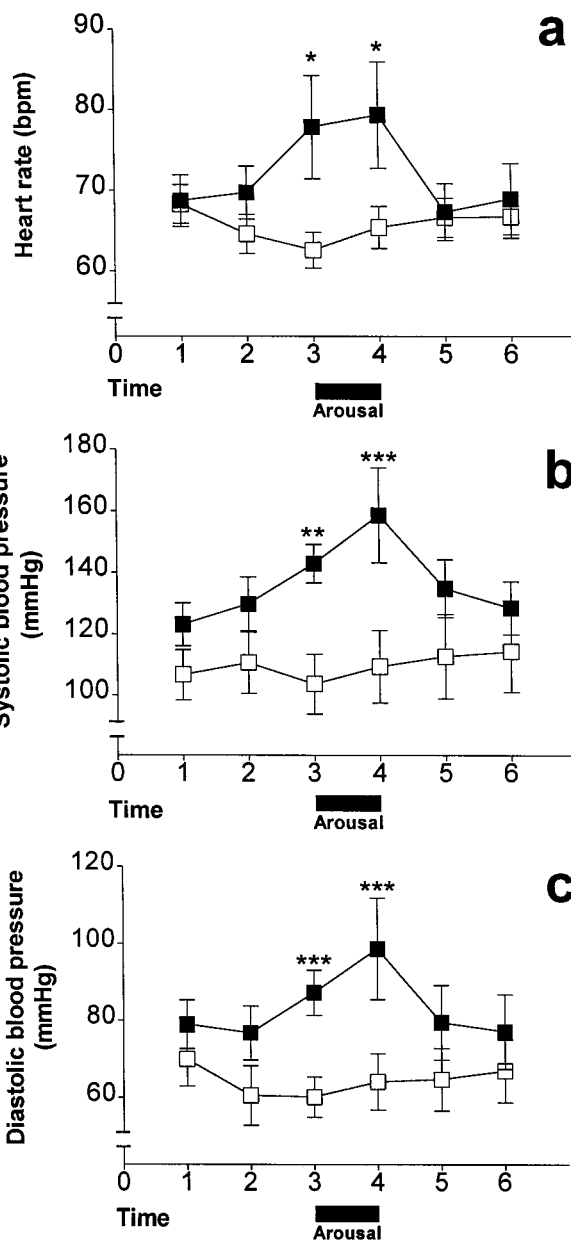


Fig. 3. Heart rate (beats per minute) (*a*) and systolic (*b*) and diastolic (*c*) blood pressure (mm Hg), analyzed in 10-minute intervals for the experimental and control conditions. Data are mean  $\pm$  SE. \*\*\* $p < .001$ ; \*\* $p < .01$ ; \* $p < .05$ .

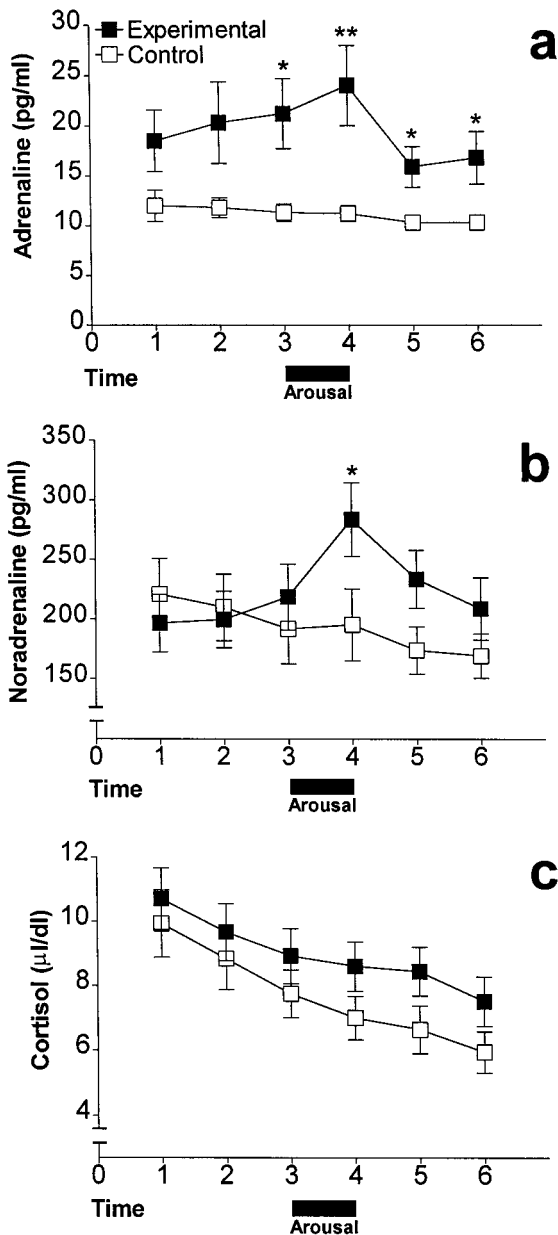


Fig. 4. Plasma concentrations of adrenaline (a), noradrenaline (b), and cortisol (c), analyzed in 10-minute intervals for the experimental and control conditions. Data are mean  $\pm$  SE. \*\* $p < .01$ ; \* $p < .05$ .

greater than control levels, with plasma adrenaline levels remaining significantly elevated thereafter ( $F(1,9) = 10.67$ ,  $p < .01$ ; condition effect). Plasma noradrenaline showed no difference between control and experimental sessions at baseline; however, noradrenaline was increased significantly during the masturbation stage of arousal ( $F(5,45) = 4.94$ ,  $p < .001$ ; Figure 4, b). Sexual arousal had no significant impact on plasma cortisol (Figure 4, c), with participants showing a continuous decline in plasma cortisol levels

during both sessions ( $F(5,45) = 48.88$ ,  $p < .001$ ; time effect).

#### Pituitary Hormones

The most significant impact of sexual arousal on plasma hormone concentrations was observed for prolactin. Small but significant increases in plasma prolactin were observed during the basal phase during the experimental session compared with control. However, prolactin increased during the masturbation phase of sexual arousal and remained highly elevated for the remainder of the session ( $F(5,45) = 8.45$ ,  $p < .001$ ; Figure 5, a). Because we had previously shown that prolactin plasma concentrations remain elevated during the experimental session in men (30), we took an additional blood sample from all participants 40 minutes after the end of both sessions (60 minutes after sexual arousal). This sample was drawn at the same rate as other samples (1 ml/min for 10 minutes). Interestingly, this sample revealed that prolactin plasma concentrations remained elevated significantly during the experimental session, 60 minutes after the end of arousal (control =  $8.99 \pm 1.09$  ng/ml, experimental =  $14.1 \pm 1.83$  ng/ml,  $p < .05$ ).

Sexual arousal produced a small albeit significant increase in plasma LH concentrations at the time of masturbation ( $F(5,45) = 3.01$ ,  $p < .05$ ; Figure 5, b) that returned to control values at the end of the session. However, plasma concentrations of  $\beta$ -endorphin ( $F(5,45) = 0.53$ ,  $p > .05$ ; Figure 5, c) and FSH ( $F(5,45) = 1.79$ ,  $p > .05$ ; Figure 5, d) were unaffected by sexual arousal and orgasm.

#### Gonadal Hormones

Sexual arousal produced an increase in plasma testosterone that approached statistical significance ( $F(5,45) = 2.34$ ,  $p = .05$ ; Figure 6, a). Progesterone levels were elevated significantly during the experimental session ( $F(1,9) = 12.05$ ,  $p < .01$ ; group effect; Figure 6, b), with both sessions producing a decrease in plasma concentrations across the session ( $F(5,45) = 13.91$ ,  $p < .001$ ; time effect). No significant alterations were revealed for estradiol concentrations ( $F(5,45) = 2.81$ ,  $p > .05$ ; Figure 6, c).

#### Discussion

The results of the present study demonstrated that masturbation-induced orgasm increased both subjective and physiological sexual arousal. Heightened sexual arousal, in turn, increased heart rate and blood pressure as well as raised plasma adrenaline and nor-

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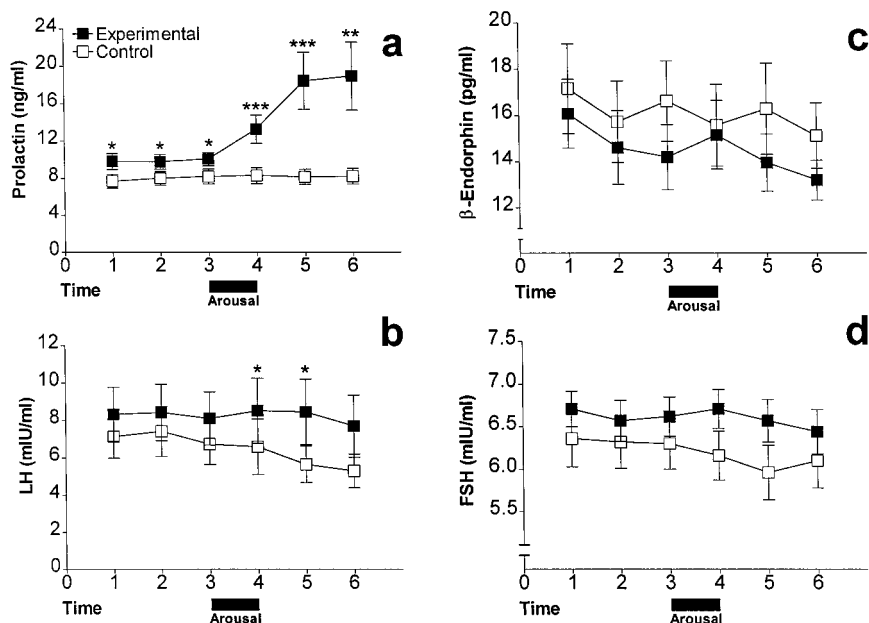


Fig. 5. Plasma concentrations of prolactin (a), LH (b),  $\beta$ -endorphin (c), and FSH (d), analyzed in 10-minute intervals for the experimental and control conditions. Data are mean  $\pm$  SE. \*\*\* $p < .001$ ; \*\* $p < .01$ ; \* $p < .05$ .

adrenaline concentrations. Furthermore, orgasm induced a large increase in plasma concentrations of prolactin, which remained elevated 60 minutes after completion of arousal. Additionally, small increases in LH and testosterone concentrations were observed. In contrast, sexual arousal did not alter plasma concentrations of  $\beta$ -endorphin, FSH, progesterone, or estradiol. These results mirror those achieved using the identical experimental paradigm with male participants (30). That is, in both men and women, sexual arousal produced a rapid increase in cardiovascular parameters and plasma catecholamine concentrations with a concomitant, sustained increase in prolactin plasma concentrations. The small increases in LH and testosterone observed in women are also mirrored by small increases of these hormones in men using the current paradigm (30), although these effects were not statistically significant. Thus, to our knowledge, this is the first study in which, men and women have been shown, using a identical paradigm, to display a similar neuroendocrine response to sexual arousal.

The increases in heart rate and blood pressure are robust responses to sexual arousal (14–17). However, the effect of sexual stimulation on sympathetic adrenal hormones remains equivocal. The present data confirm previous findings demonstrating that sexual arousal produces immediate increases in plasma noradrenaline and urinary adrenaline and noradrenaline, which rapidly return to control levels after termination of stimuli (18, 19). Importantly, results from the present study, together with our previous results (30),

reveal that this effect occurs in both men and women. In men, noradrenaline acts peripherally to inhibit erectile function via  $\alpha$ -adrenoceptors in the penile corpus cavernosum (46, 47). The noradrenergic response to sexual arousal has been shown to be functionally relevant, because  $\alpha_2$ -adrenoceptor antagonists prolong the duration of the erectile response to erotic stimuli in humans (48–50). Nevertheless, the relevance of this response to female sexual functioning is unknown.

Indeed, noradrenaline secretion in response to sexual arousal may play either an inhibitory or facilitatory role in modulating sexual arousal. Noradrenaline secretion may act to arrest sexual arousal via inhibition of vaginal blood flow (51, 52). Nevertheless, although it is suggested that sympathetic hormones and peptides may oppose the known facilitation of vaginal blood flow by vasoactive intestinal peptide (VIP) (53–57), this position remains equivocal. In contrast, the noradrenergic response to masturbation and orgasm may facilitate sexual arousal. Stress and exercise-induced sympathetic activation before sexual stimulation have been shown to increase physiological sexual arousal in women (44, 58). However, to confirm that these effects are due to catecholamines, the potentiating effect of sympathetic activation on sexual arousal needs to be blocked via application of an adrenoceptor antagonist. Thus, the precise relevance of peripheral noradrenaline secretion after during sexual arousal remains unclear.

We previously showed that sexual arousal produces a large, sustained increase in plasma prolactin concen-

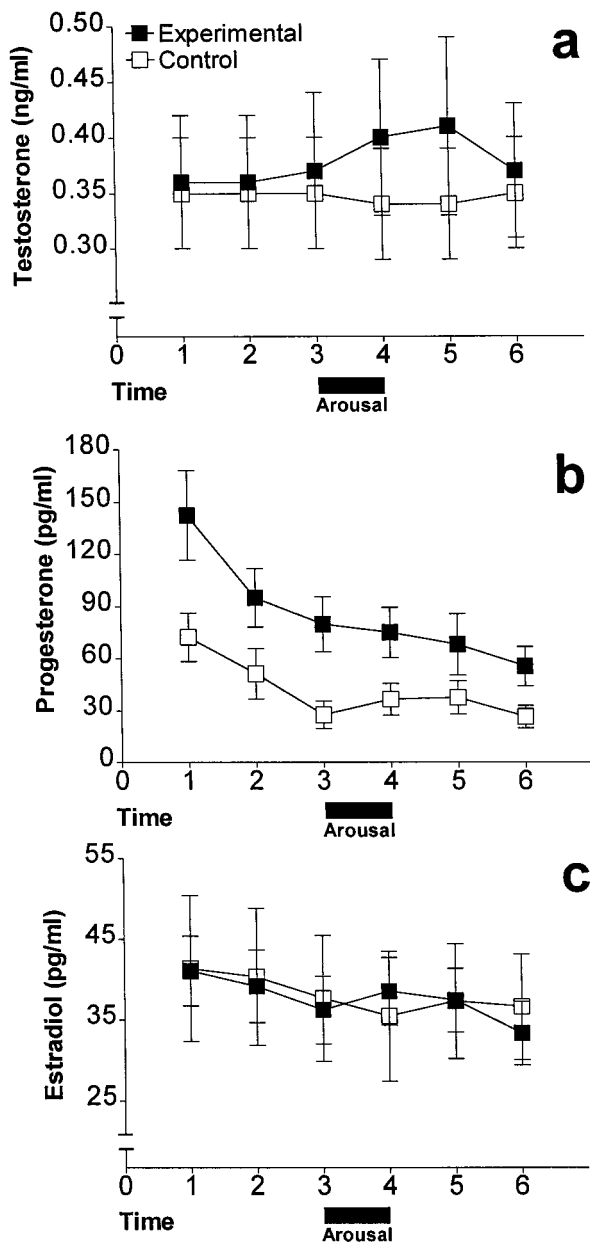


Fig. 6. Plasma concentrations of testosterone (a), progesterone (b), and estradiol (c), analyzed in 10-minute intervals for the experimental and control conditions. Data are mean  $\pm$  SE.

trations in healthy men (30). The results of the current study extend these findings, showing that arousal doubled the concentration of prolactin in women and remained highly elevated 60 minutes after completion of the arousal component. Thus, a similar time kinetic in sexual arousal-induced release of prolactin into blood was observed in both men and women. This contrasts with results of other studies showing either unchanged or decreased prolactin levels after sexual arousal (20, 23). Nevertheless, it is possible that the increase in

prolactin observed presently is related specifically to orgasm associated with sexual arousal.

Therefore, our current data, combined with the effect demonstrated in men (30), show that a large and sustained increase in plasma prolactin concentrations is a specific marker of sexual arousal and orgasm in males and females. The clinical significance of this response is presently unclear. Data from animal experiments indicate that prolactin has an overall inhibitory response on male sexual behavior and function (12, 59–63). The inhibitory effect of prolactin may occur peripherally, arresting erection via direct action on the penile corpus cavernosum (62). In contrast, peripheral prolactin may alter sexual behavior via central mechanisms, because it is able to pass into the cerebrospinal fluid (64). Peripheral prolactin administration has been shown to modulate dopaminergic function at particular brain sites (65–67). Prolactin may thus alter sexual behavior and arousal via dopaminergic regulation, which is posited to play a major role in controlling sexual functions (68–70). Although less well documented, it is also apparent that prolactin inhibits female sexual behavior in animal models (71). However, despite chronic hyperprolactinemia producing strong reductions in libido in both men and women (67, 72), the role of prolactin in modulating human sexual behavior after arousal remains equivocal. Nevertheless, an attractive possibility is that prolonged prolactin secretion after sexual arousal may act as a neuroendocrine negative feedback loop, such as that which occurs to limit other behaviors, such as food consumption (73, 74).

The present study revealed small increases in testosterone and LH after masturbation-induced orgasm. This neuroendocrine pattern is also similar to the response found in men using the current methodology, although the effects did not reach statistical significance (30). Sexual arousal-induced increases in testosterone and LH have been documented (23–25, 28), although other studies have revealed no alterations of these hormones (20, 21, 26, 27, 29). Data from the present study suggest that a possible cause of the discrepant findings is that plasma concentrations of these hormones are only slightly affected by sexual arousal. Thus, small differences in methodology and data interpretation may easily produce contrasting results. However, the discrepant effects with regard to LH may be due to the pulsatile secretion pattern of this hormone after sexual arousal (25).

This study shows that when an identical methodology is used, females produce a neuroendocrine response to sexual arousal similar to that demonstrated for males (30). It is likely that the inconsistencies in previous studies largely reflect an abundance of differ-

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ent methodologies. A number of studies did not use a continuous blood sampling technique, prohibiting blood withdrawal at the time of highest sexual arousal. Thus, changes in hormones with a short half-life, such as catecholamines, would not be detected. A major factor that contributes to the conflicting data is the type of arousal stimulus or paradigm used. Film, slides, imagery, fantasy, masturbation, and intercourse have been used to produce sexual arousal. Because different techniques produce different levels of sexual arousal (75, 76), it is not surprising that studies have produced nonconfirming or even opposite results. Nevertheless, we report here a method that is relatively simple yet effective for monitoring the neuroendocrine pattern of sexual arousal. However, it must be recognized that by sampling blood over 10-minute periods, the current method increases the risk of not identifying very short peaks in hormone concentrations. Furthermore, because of the 10-minute sampling of blood, the current methodology does not allow differentiation of the immediate pre- and postorgasmic endocrine status. Distinction of pre- and postorgasmic endocrine responses would allow a theoretical separation of hormone secretion that may act to either enhance preorgasmic arousability or inhibit sexual arousal after orgasm. Future studies should consider using a system in which the participant can notify the investigators of orgasm (77–79) and create a new blood sample for the postorgasmic phase. Thus, the current technique is sufficient to detect large changes in catecholamines and prolactin in plasma; the method also may be adjusted to sample blood over much shorter time intervals to allow discrimination between the pre- and postorgasmic periods as well as to measure hormones with rapid pulsatile characteristics.

In summary, the present study established that women and men have a similar neuroendocrine response to sexual arousal. These data aid the understanding of the effect of sexual arousal on hormonal status. An explicit knowledge of the neuroendocrine response to sexual arousal in healthy humans may facilitate further research into the role of endocrine factors in sexual dysfunction.

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