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Michael S. Exton · Tillmann H. C. Krüger · Norbert Bursch · Philip Haake · Wolfram Knapp · Manfred Schedlowski · Uwe Hartmann

# Endocrine response to masturbation-induced orgasm in healthy men following a 3-week sexual abstinence

**Abstract** This current study examined the effect of a 3-week period of sexual abstinence on the neuroendocrine response to masturbation-induced orgasm. Hormonal and cardiovascular parameters were examined in ten healthy adult men during sexual arousal and masturbation-induced orgasm. Blood was drawn continuously and cardiovascular parameters were constantly monitored. This procedure was conducted for each participant twice, both before and after a 3-week period of sexual abstinence. Plasma was subsequently analysed for concentrations of adrenaline, noradrenaline, cortisol, prolactin, luteinizing hormone and testosterone concentrations. Orgasm increased blood pressure, heart rate, plasma catecholamines and prolactin. These effects were observed both before and after sexual abstinence. In contrast, although plasma testosterone was unaltered by orgasm, higher testosterone concentrations were observed following the period of abstinence. These data demonstrate that acute abstinence does not change the neuroendocrine response to orgasm but does produce elevated levels of testosterone in males.

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M. S. Exton (✉) · T. H. C. Krüger · P. Haake
M. Schedlowski
Institut für Medizinische Psychologie,
Universitätsklinikum Essen, Hufelandstr. 55,
45122 Essen, Germany
e-mail: michael.exton@uni-essen.de
Tel.: +49-201-723-4282; Fax: +49-201-723-5948

N. Bursch · U. Hartmann
Division of Clinical Psychiatry, Hannover Medical School,
30623 Hannover, Germany

W. Knapp
Division of Nuclear Medicine, Hannover Medical School,
30623 Hannover, Germany

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Although sexual abstinence is a common behavioral pattern in humans, there has been little examination of the psychological and physiological consequences of this behavior. Nevertheless, limited data have demonstrated that sexual abstinence may impact on the physiological regulation of sexual function. Specifically, retrospective studies have shown that features of semen quality are reduced during long periods of sexual abstinence [5, 22]. In contrast, periods of abstinence between 12 h and 10 days have generally revealed enhanced sperm quality parameters [9, 26, 28, 33], although this is not consistent across all measures of sperm quality [28, 33]. Nevertheless, acute sexual abstinence is commonly employed prior to clinical sperm donation to enhance sperm quality.

Despite knowing that acute abstinence affects reproductive function, no data exist that examines the effect of abstinence on the physiological response to sexual arousal and orgasm. We have established a method for examining the neuroendocrine response to masturbation-induced orgasm in men and women [15–17, 24], based upon a continuous blood sampling technique. These investigations demonstrated that masturbation-induced orgasm produced a pronounced increase in cardiovascular responses and plasma catecholamine concentrations. Furthermore, sexual arousal was characterised by a large, persistent increase in concentrations of plasma prolactin. Since hyperprolactinemia is known to inhibit sexual arousal and function [13, 35], these data suggest that prolactin may act as a peripheral and/or central feedback signal in controlling sexual arousal following orgasm.

Therefore, the purpose of this current study was to investigate the effect of acute abstinence on the physiological response to sexual arousal. Specifically, we examined the neuroendocrine responses to sexual arousal and orgasm following separate periods of regular sexual activity and sexual abstinence in healthy men by using an established paradigm.

## Materials and methods

### Participants

Ten healthy male volunteers (mean age of 25.8 ± 0.8 years, range of 22–29 years) participated in this investigation. Participants were screened by completing a general medical/health questionnaire and gave their written consent before being admitted into the study. The protocol for this study was approved by the Ethics Committee for Investigations involving human subjects of the Hannover Medical School, Germany. Individuals taking medication, abusing drugs/alcohol, or exhibiting endocrinological, psychological or sexual dysfunction/disorders were excluded from the study. All participants reported that they had an exclusively heterosexual orientation and a relaxed attitude toward pornography. Further, all subjects were currently in a stable relationship and reported having sexual intercourse approximately 2–3 times per week.

### Design and procedure

A repeated measures design was used so each participant viewed a videotape and masturbated to orgasm on two separate days. Two different videos were shown in a crossover design for the two sessions. The first session took place at 1630 hours on day 0. Immediately after that first session (day 0–day 20), the participant refrained from any type of sexual activity. At 16:30 on day 21 each subject once again participated in the sexual arousal paradigm. The procedure of sexual arousal and orgasm both before (day 0) and after (day 21) were identical, with each session lasting 2 h.

Experiments were conducted in a separate sound-attenuated room equipped with a clinical bed, a color television and a video cassette player. All leads, including the blood line, passed through the wall into the adjacent room where the cardiovascular data and blood samples were collected, allowing the subject to be completely isolated throughout the experiment. At the beginning of each session participants were placed on the bed in front of the video screen. The cardiovascular monitor was then engaged 30 min prior to the film and a steady baseline reading was obtained before the cannula was inserted (20 min before the beginning of the film). The session was composed of three sequences, each lasting 20 min. The first and last sections of the video tape were composed of sections of an emotionally neutral documentary film. However, the middle section consisted of a 20 min pornographic film that showed different couples engaged in foreplay and sexual intercourse. Blood sampling was initiated at the beginning of the film. After 10 min of the pornographic video had been watched (anticipatory phase), subjects in the experimental session were required to masturbate until orgasm. Blood was drawn continuously with the samples divided into six 10 min intervals [15–17, 24]. Specifically, the first two samples represented basal values (10, 20 min), the third sample represented the response to film-induced sexual arousal (30 min), the fourth demonstrated the response to orgasm (40 min) and the final two samples showed the recovery phase (50, 60 min).

### Apparatus and materials

#### *Subjective sexual arousal*

To provide a measurement for sexual arousal, participants completed a visual analogue scale (VAS) by rating their subjective level of sexual arousal from 'not at all sexually aroused' to 'extremely sexually aroused' [15–17, 24]. Subjective sexual arousal was measured at three time points–before, during and after the session–for both controlled and experimental conditions.

Additionally, subjective assessment of the quality of the orgasm was completed using 5-point Likert scales. These scales examined the duration, intensity and speed of orgasm in absolute value and as compared to a typical orgasm. These questions were administered following both the experimental and controlled situations.

#### *Cardiovascular measures*

The cardiovascular parameters heart rate (HR) and systolic and diastolic blood pressure (BP) were monitored continuously via a finger cuff connected to a blood pressure monitor (Critikon Cuff & Dinamap Vital Data Monitor; Critikon Ltd, USA) that was located in the adjoining room. Cardiovascular activity was recorded by computer every 30 s, and the HR and BP values were averaged over 10 min intervals and analysed simultaneously with the blood samples taken in 10 min interval.

#### *Endocrine measures*

For blood sampling, an IV cannula (Vasofix Braunüle, 18G) was connected to a 1.25 m heparinized silicon tube (inner ∅ 2.0 mm, Reichelt Chemie, Heidelberg, Germany) by a plastic three-way stop-cock (Cook, Mönchengladbach, Germany). The silicon tubing passed through the wall into the adjacent room and was positioned through a peristaltic pump (Fresenius, Homburg, Germany). Blood flow was adjusted to 2 ml/min, so that approximately 10 mls of blood per 5 min were collected (i.e. more than 150 mls per session). Blood was collected in EDTA tubes (Sarstedt, Nümbrecht, Germany), and the collection of each sample was delayed by the time it took for blood to pass through the dead space in the tube. Blood was stored on ice until the samples were centrifuged. Plasma was stored in glass aliquots at −20 °C until it was time for the hormone assays.

All samples from the one participant were analysed in duplicate within the same assay for a particular hormone. Plasma prolactin was evaluated by immunoradiometric assay (IBL, Hamburg, Germany) and testosterone, LH, cortisol (Diagnostic System Laboratories, Texas, USA) and catecholamines (IBL, Hamburg, Germany) were assessed by radioimmunoassay. Inter and intra-assay variability were 8.0% and 6.2%, respectively, for noradrenaline; 5.1% and 4.0%, respectively, for adrenaline; 7.1% and 5.0%, respectively, for prolactin; 7.9% and 5.2%, respectively, for testosterone; 5.2% and 3.8%, respectively, for LH; and 4.3% and 2.8%, respectively, for cortisol.

### Statistical analyses

Data from all subjects were analysed by 2-factor repeated measures (condition x time) analyses of variance (ANOVA). If not stated otherwise, only the condition x time interaction effect is reported. An $\alpha$ level of 0.05 was used for all ANOVAs. Post hoc simple effects were evaluated by using paired samples *t*-tests with Bonferroni $\alpha$ corrections made for multiple comparisons. Additionally, the Wilcoxon test was completed for questionnaire data.

## Results

### Subjective sexual arousal

Participants rated themselves as being significantly sexually aroused during the erotic film ($F(2, 16) = 189.51$, $P < 0.001$; time effect) and greater subjective arousal was observed following abstinence ($F(1,8) = 9.21$, $P = 0.016$; condition effect, Fig. 1a). Moreover, participants reported a longer duration (before, 2.1 ± 0.3; after, 3.5 ± 0.3; $Z = -2.34$, $P = 0.019$) and greater intensity (before, 2.5 ± 0.2; after, 3.7 ± 0.3; $Z = -2.28$, $P = 0.023$) of orgasm following abstinence as compared to the controlled session, whilst abstinence did not alter the subjective speed to orgasm (before, 3.1 ± 0.2; after, 3.5 ± 0.2; $Z = -0.71$, $P > 0.05$). Similar results were revealed when subjects compared the orgasmic characteristics to those of a typical orgasm (duration: before, 1.8 ± 0.2; after, 3.1 ± 0.4; $Z = -2.26$, $P = 0.023$; intensity: before, 2.1 ± 0.2; after, 3.0 ± 0.3; $Z = -1.73$, $P = 0.080$; speed: before, 3.1 ± 0.2, after, 3.8 ± 0.3; $Z = -1.35$, $P > 0.05$).

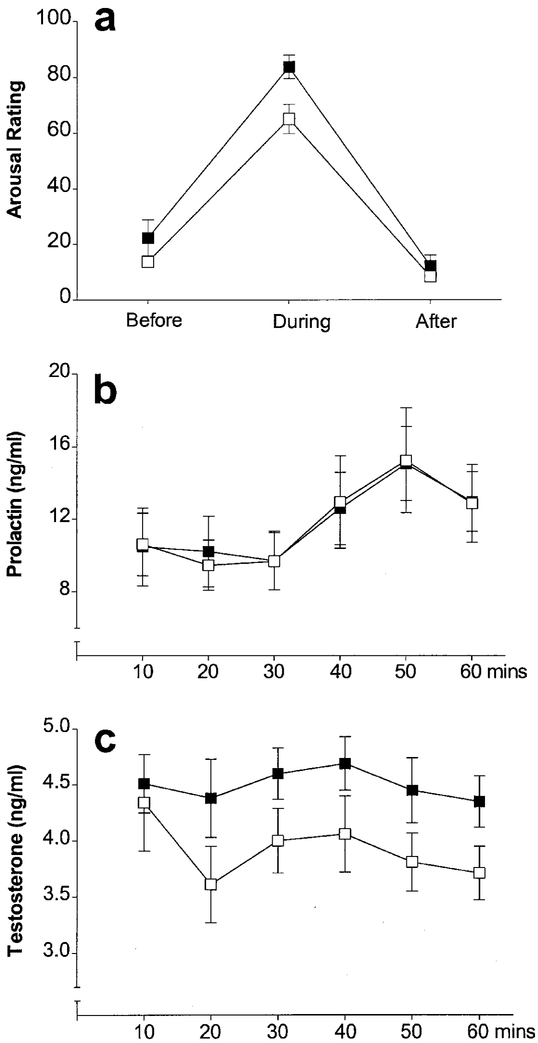


**Fig. 1a–c** Subjective sexual arousal before, during and after sexual arousal; plasma prolactin (ng/ml) and testosterone (ng/ml) concentrations during baseline (10, 20 min), in response to film-induced sexual arousal (30 min), following orgasm (40 min), and recovery after orgasm (50, 60 min). Each subject participated before sexual abstinence (□) and following 3 weeks of abstinence (■) (Data displayed as mean ± SE). Subjective sexual arousal was increased by masturbation-induced orgasm (a). Furthermore, this response was enhanced following sexual abstinence. Plasma prolactin concentrations (b) were significantly elevated by sexual arousal and orgasm both before and after sexual abstinence. In contrast, plasma testosterone concentrations (c) were unaffected by sexual arousal and orgasm both before and after sexual abstinence. However, plasma testosterone was significantly elevated after abstinence when compared to basal values (Data displayed as mean ± SE)

## Prolactin, LH and testosterone response to sexual arousal

Sexual arousal (timepoint: 30 min) and orgasm (timepoint: 40 min) increased plasma prolactin levels markedly in participants ($F(5, 45) = 5.80$, $P < 0.001$; time effect, Fig. 1b). However, no significant difference was observed in this response before and after acute abstinence ($P > 0.05$).

Sexual arousal (timepoint: 30 min) and masturbation-induced orgasm (timepoint: 40 min) did not affect testosterone concentrations either before or after abstinence ($P > 0.05$). However, a significant increase in basal testosterone levels were observed following the period of abstinence ($F(5, 45) = 6.72$, $P = 0.029$; condition effect, Fig. 1c). A significant difference between the conditions was observed at the 20 min baseline period, with this difference remaining throughout the session. Increased testosterone was further accompanied by a small increase in plasma LH following abstinence; however this change did not reach statistical significance ($P > 0.05$; data not shown).

## Cardiovascular response to sexual arousal

Sexual arousal (timepoint: 30 min) and orgasm (timepoint: 40 min) increased heart rate ($F(5, 45) = 14.18$, $P < 0.001$; time effect), systolic blood pressure ($F(5, 45) = 16.35$, $P < 0.001$; time effect) and diastolic blood pressure in participants ($F(5, 45) = 18.11$, $P < 0.001$; time effect, Fig. 2). However, no significant difference was observed in any of these responses before and after acute abstinence (all $P > 0.05$).

## Sympathoadrenal response to sexual arousal

Sexual arousal (timepoint: 30 min) and orgasm (timepoint: 40 min) increased both adrenaline ($F(5,45) = 7.95$, $P < 0.001$; time effect) and noradrenaline ($F(5,45) = 10.90$, $P < 0.001$; time effect) in participants (Fig. 3). However, no significant difference was observed in either of these responses before and after acute abstinence ($P > 0.05$). Sexual arousal and masturbation-induced orgasm did not affect cortisol concentrations either before or after abstinence. However, reflecting circadian rhythm, cortisol levels decreased consistently over time during both sessions ($F(5, 45) = 4.66$, $P = 0.02$; time effect).

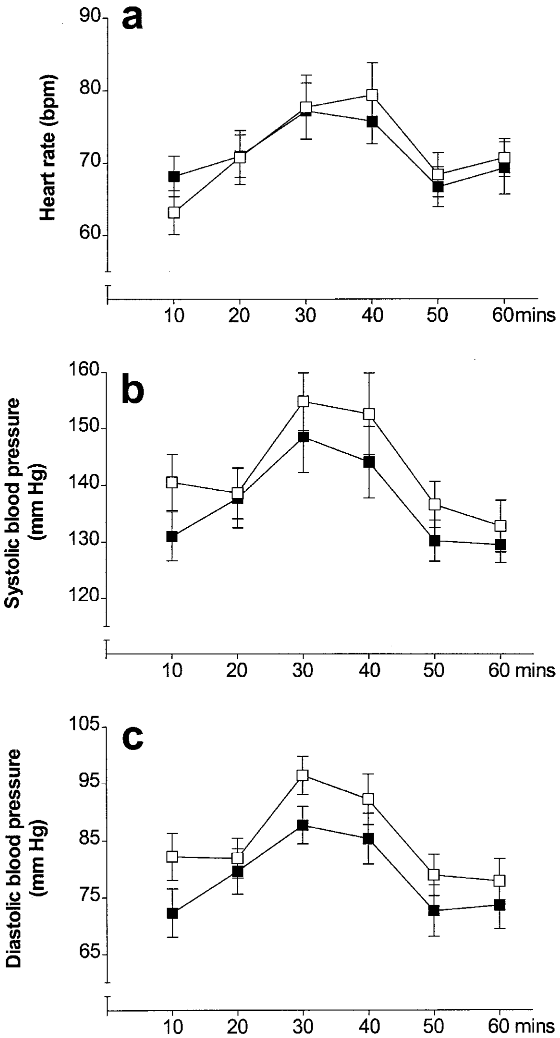


**Fig. 2a–c** Cardiovascular activity during baseline (10, 20 min), in response to film-induced sexual arousal (30 min), following orgasm (40 min) and recovery after orgasm (50, 60 min). Each subject participated before sexual abstinence (□) and following 3 weeks of abstinence (■) (Data displayed as mean ± SE). Heart rate (beats/min; a), systolic blood pressure (mm Hg; b) and diastolic blood pressure (mm Hg; c) were significantly elevated by sexual arousal and orgasm before and after sexual abstinence

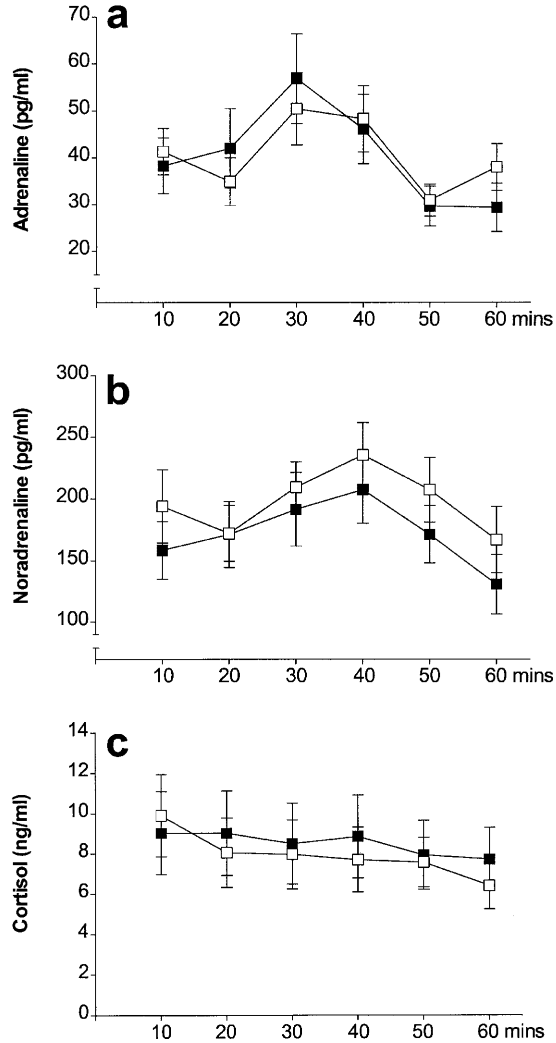


**Fig. 3a–c** Plasma adrenaline (pg/ml), noradrenaline (pg/ml), and cortisol (ng/ml) concentrations during baseline (10, 20 min), in response to film-induced sexual arousal (30 min), following orgasm (40 min), and recovery after orgasm (50, 60 min). Each subject participated before sexual abstinence (□) and following 3 weeks of abstinence (■) (Data displayed as mean ± SE). Adrenaline (a) and noradrenaline (b) concentrations were significantly elevated by sexual arousal and orgasm both before and after sexual abstinence. In contract, plasma cortisol concentrations (c) were unaffected by sexual arousal and orgasm both before and after sexual abstinence

## Discussion

The current study demonstrated that 3 weeks of sexual abstinence elevated basal testosterone concentrations and increased both subjective sexual arousal and subjective interpretation of the quality of the orgasm. However, despite these subjective reports, acute abstinence did not alter the well-characterised cardiovascular and endocrine responses to orgasm.

Sexual arousal and masturbation-induced orgasm produced pronounced increases in sympathetic activity, which were reflected by increased heart rate, blood pressure and plasma catecholamine concentrations. Furthermore, orgasm produced a pronounced increase in plasma prolactin concentrations. These results mirrored previous data obtained by studying masturbation in both men and women [15, 24], which have been shown to be orgasm specific [16].

We have suggested that prolactin secretion following orgasm may act as a peripheral and/or central feedback signal in controlling post-orgasm sexual arousal. This position is supported by a wealth of data from studies of animals and humans that demonstrate the marked inhibitory effect that hyperprolactinemia has on sexual arousal and behavior [11, 13, 30, 32, 35], as well as a decrease in inhibition following the normalization of circulating prolactin [13, 35].

Prolactin may also act as an acute negative controller of sexual function by inhibiting the function of sexual organs. Although not extensively examined, some data clearly demonstrate that acute increases in prolactin inhibit erectile function by hindering the smooth muscle relaxation of the corpus cavernosum [4]. Alternatively, acute increases in prolactin may contribute to a sexual-satiation mechanism following orgasm by means of feedback to the central nervous system (CNS) structures that control sexual arousal [10, 21, 29].

Should prolactin act as a negative regulator of sexual arousal, increased prolactin secretion may be expected to accompany increased sexual arousal produced by abstinence. However, the current data show that acute abstinence does not alter the prolactin response to masturbation-induced orgasm. Nevertheless, although it is clear that the prolactin response is orgasm dependent, the magnitude of the response may be independent of the quality of the orgasm. Alternatively, the subjective scores used presently may not provide an accurate assessment of physiological quality of the orgasm. Thus, future studies should incorporate a more complete assessment of orgasm quality, by integrating a more robust measurement of orgasmic characteristics (e.g. exact timing of orgasm duration, erectile response).

In contrast to previous data using punctual blood sampling [8, 34], sexual arousal in the current paradigm induced elevations in plasma testosterone concentrations that did not reach statistical significance. However, basal testosterone levels were elevated throughout the session following abstinence when compared to values obtained prior to abstinence. Enhanced testosterone may have been a physiological response to re-initiate sexual activity throughout the abstinence period, since increasing testosterone concentrations have been shown to stimulate the initiation of coital and other partnered sexual activity [19, 20]. However, this appears unlikely because baseline testosterone levels at the beginning of the video sequence were similar before and after abstinence.

On the other hand, the increased testosterone concentrations may have resulted from enhanced sensitivity to anticipatory cues, since testosterone secretion increases in anticipation of sexual interaction [18]. Thus, the increase in testosterone following abstinence may have resulted from increased anticipation of the erotic film. Although initial basal testosterone values (10 min timepoint) were not altered by abstinence, the difference between conditions that appeared after the start of the session (20–60 min) support this position.

Such anticipatory or preparatory sex hormone responses are suggested by several other studies [3, 25, 31] and seem to reflect more general appetitive responses in the expectation of the potentially rewarding consequences of sexual activity. As appetitive responses are behaviors that represent a central motivational state for contact with a goal object [2], increased testosterone concentrations may represent an enhancement of the affective properties of the goal object by sexual abstinence. Furthermore, a close interaction exists between steroid hormones and the brain systems involved in reward. Specifically, acute increases in testosterone have possible neural rewarding qualities [2]; this effect is potentially mediated by dopaminergic systems [14]. With regard to our current results, one can speculate that sexual abstinence has a twofold effect on this interaction pattern: It may enhance the anticipated rewarding properties of re-initiated sexual activity and it may lower the threshold at which testosterone produces rewarding effects. Both factors may interact to trigger the elevated testosterone levels observed following abstinence.

The enhanced subjective qualities of sexual arousal and orgasm intensity following sexual abstinence may also be explained by increased testosterone concentrations. Indeed, elevated levels of testosterone in men are known to enhance nocturnal erections [12], probably by acting on peripheral neurons that control copulatory reflexes [23]. Additionally, some evidence suggests that testosterone stimulates the erectile response to experimentally-induced sexual arousal; however, this position remains equivocal [7]. Nevertheless, increased testosterone elevates feelings of sexual arousal and libido [1, 6], and increases both orgasmic frequency [27] and coital initiation [20]. Thus, our data suggest that testosterone may have influenced the subjective assessment of sexual arousal either by directly modifying genital reactions, or alternatively, by manipulating the CNS structures that regulate sexual motivation and arousal.

Summarizing, the current study demonstrated that masturbation-induced orgasm produces pronounced elevated cardiovascular responses and plasma concentrations of catecholamines and prolactin. Although these responses were unaltered by acute abstinence from sexual activity, abstinence increased subjective sexual arousal and the quality of the orgasm, as well as basal testosterone concentrations. Thus, although acute abstinence enhances subjective sexual arousal it has little impact on the acute neuroendocrine response to orgasm.

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