The Roles of Testosterone and Alpha-Amylase in Exercise-Induced Sexual Arousal in Women

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ABSTRACT -

Introduction. Several studies have demonstrated that moderate exercise increases genital response to erotic stimuli in women. The increase in genital arousal could be the result of various changes that can occur in response to exercise including changes in hormone levels, neurotransmitter levels, mood, and autonomic nervous system activity.

Aim. The present study was an attempt to shed light on two such mechanisms through which exercise enhances sexual arousal.

Method. Sixteen participants came into the lab on two separate occasions: during one visit, they filled out questionnaires for 20 minutes, and during the other visit, they exercised on a treadmill for 20 minutes. The questionnaires and exercise were both followed by the presentation of a neutral then erotic film during which the women's physiological sexual arousal was measured. Saliva samples were taken at baseline, prefilm, and postfilm.

Main Outcome Measures. Subjective arousal was measured using a self-report questionnaire, and genital arousal was measured by a vaginal photoplethysmograph. Testosterone and α -amylase (a marker of sympathetic nervous system [SNS] activity) were measured via saliva assays.

Results. Findings replicated previous studies showing a significant increase in physiological sexual arousal with exercise. There was a significant increase in α -amylase across the study in the exercise condition, but not in the no-exercise condition. There were no differences in testosterone levels between the exercise and no-exercise conditions.

Conclusions. SNS activity is one mechanism through which exercise increases genital sexual arousal. Testosterone does not mediate the relationship between exercise and genital sexual arousal. Hamilton LD, Fogle EA, and Meston CM. The roles of testosterone and alpha-amylase in exercise-induced sexual arousal in women. J Sex Med 2008;5:845–853.

Key Words. Arousal; Testosterone; Sympathetic Nervous System; Exercise; Alpha-Amylase

Introduction

D ynamic, cardiovascular exercise has many well-documented health benefits. One of the lesser known benefits is the positive effect of acute exercise on female sexual arousal. In a series of studies, Meston and Gorzalka [1–3] demonstrated that moderate-intensity exercise increased vaginal blood volume (VBV) and vaginal pulse amplitude (VPA) responses to an erotic film. In the first such study, 35 sexually functional, premenopausal women participated in two counterbalanced conditions: exercise and no-exercise. In the exercise condition, they exercised on a stationary bicycle for 20 minutes at 70% of their VO2 max (maximum volume of oxygen consumed over time during intense full-body exercise). After the exercise, the women watched a film sequence that consisted of a 3-minute neutral film clip followed by a 3-minute erotic film clip. In the no-exercise condition, participants filled out questionnaires and watched a similar film. Both VBV and VPA were significantly higher during the erotic portion of the film in the exercise condition as compared with

the no-exercise condition [1]. Using the same experimental paradigm, Meston and Gorzalka replicated the postexercise increase in physiological arousal among sexually functional women and women with low sexual desire, but not among anorgasmic women [2]. A further study examining the effects of exercise at 5-, 15-, and 30-minute postexercise suggested a curvilinear relationship: VPA was significantly increased at 15 minutes, marginally increased at 30 minutes, and decreased at 5-minute postexercise [3].

In these studies, the authors suggested that exercise was enhancing genital arousal via activation of the sympathetic nervous system (SNS). This assumption was based on the findings that indicate exercise at moderate to high levels increases SNS activity, as measured by heart rate [4] and by catecholamine release [5]. These studies were some of the first to suggest that increased SNS activity may have a facilitatory, as opposed to inhibitory, influence on female sexual arousal. A follow-up study showing that clonidine, an antihypertensive medication that blunts normal SNS responses, blocked the effects of exercise on genital arousal supported the authors' speculation [6].

There are a number of limitations to the inferences made in these studies however. First, SNS activation was not directly measured; heart rate was used as an indirect marker of SNS activity. Because heart rate declines after exercise, and sexual arousal was measured at postexercise, it is impossible to know whether the SNS was in fact still active, or whether the parasympathetic nervous system (PNS) had become dominant. Second, in addition to increasing SNS activity, exercise affects several other mechanisms that are thought to be involved in sexual arousal. For example, exercise has been shown to increase both dopamine and serotonin in women [7], and depending on the site of action and the receptor subtype affected, both dopamine and serotonin could have either an inhibitory or facilitatory effect on female sexual arousal (for reviews, see dopamine [8] and serotonin [9]). In general, adequate levels of both serotonin and dopamine are needed for normal sexual functioning, but it is unclear whether an increase due to exercise would be helpful or harmful to sexual arousal.

Exercise has also been shown to affect a variety of hormones such as testosterone [10,11], cortisol [11,12], estrogen [10,13], prolactin [14], and oxytocin [15], which have been linked with sexual arousal in women. For the purposes of the present study, we are particularly interested in the effects of exercise on testosterone. Most studies have found moderate levels of exercise increase plasma testosterone [10,11], although some studies have found no change in testosterone from pre- to postexercise [16]. In women, testosterone is most often linked to increased sexual desire and positive mood, but evidence is mounting that testosterone affects the genital tissues as well. Women administered with exogenous androgens show increased genital arousal [17], although the mechanisms of this change are not well understood [18]. Thus, exercise could exert its positive effects on sexual arousal by increasing testosterone.

Aims

The present study was designed to better elucidate the mechanism(s) by which intense acute exercise enhances genital blood flow in women. The study had two primary goals: (i) to provide a more direct assessment of whether, and to what degree, the SNS is activated with exercise and sexual arousal, and (ii) to examine whether salivary measures of testosterone change with acute exercise and, if so, whether they could account for the exerciseinduced increase in genital engorgement. SNS activation was assessed via salivary measures of α -amylase, a metabolite that is highly correlated with plasma levels of norepinephrine (NE) [19]. Because of its relationship to NE, α -amylase is thought to be a good indicator of adrenergic activity, which is intimately linked to SNS activity. α -Amylase has been shown to increase in response to physical and psychological stress including exercise [20]. Salivary testosterone is highly correlated with plasma free testosterone, the bioavailable form of the hormone [21].

Method

Participants

Participants were 16 women recruited from a community sample (see Table 1 for detailed demographics). All women had engaged in sexual activity with men in the past month. Prior to their first visit, the participants were screened by telephone to determine their eligibility for participation. Study criteria were as follows: women aged 18–45 and not yet menopausal; free of sexual problems; free of any drugs or medical conditions or medications that could affect sexual arousal, SNS activity, or test-osterone (except hormonal contraceptives, N = 4); Download English Version:

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